

Pediatric Hip

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Developmental Dislocation of the Hip

- Can occur at birth or be diagnosed later in infancy. All series report significant incidence of late diagnosis. Not known if these developed dislocation later or if missed at birth. Teratologic dislocations occur well before birth, are not reducible at birth and are resistant to routine treatment.

I. Etiology

A. Ligamentous laxity

- Constitutional (15% people have) or maternal hormonal

B. Pre- and postnatal positioning

- Oligohydramnios, breech, mechanical factors

C. Genetic factors

- Multifactorial inheritance pattern

D. Incidence

- 0.12 to 1.5 per 1,000 established DDH
- 6 to 9 per 1,000 unstable in newborns but most resolve spontaneously within week

E. Risk factors

- Female
- First born
- Breech position – tenfold increased risk
- Torticollis 20%
- Metatarsus adductus – 1.5-10% of Met Add have DDH (probably lower end)
- Ligamentous laxity
- Family history
- Lower limb deformity
- Other significant musculoskeletal abnormalities

F. Definitions

- Dislocation – femoral head not in contact with acetabulum
- Subluxation – lack of congruous, concentric reduction
- Subluxatable – femoral head can be moved from reduced position to out of the socket.
- Instability – femoral head does not remain concentrically reduced.
- Dysplasia – upper part of acetabulum does not develop normally, usually due to instability of the hip.
- Teratologic – severe form of dislocation occurring prenatally with very poor development of the acetabulum

G. Natural history of DDH

1. Newborn – possible outcomes
 - a. Normal
 - b. Subluxation

- c. Dislocation
- d. Dysplasia
2. Late diagnosed
 - a. Limited abduction
 - b. Limb shortening (apparent)
 - c. Waddling gait, hyperlordosis (in bilateral cases) due to flexion adduction contractures
3. Adult
 - a. Bilateral
 - 1) False acetabulum
 - Well developed – degenerative joint disease, clinical disability
 - Absent – no degenerative joint disease, function better
 - 2) Low back pain secondary to hyperlordosis
 - b. Unilateral
 - 1) False acetabulum (same as above)
 - 2) Limb length inequality
 - 3) Ipsilateral knee deformity and pain
 - 4) Scoliosis
 - 5) Gait disturbance

H. Natural history of subluxation

1. Clinical disability
2. Radiographic degenerative changes
3. Time course
 - Onset of DJD related to severity of anatomic dysplasia
 - Clinical symptoms predate radiographic changes by approximately 10 years.

I. Natural history of dysplasia

- Incidence unknown
- DJD – associated with degenerative arthritis especially in females
- Conventional radiographic parameters not predictive
- In absence of subluxation – natural history unpredictable

J. Diagnosis of DDH

1. History – looking for risk factors, family Hx, signs of prenatal distress
2. Physical exam
 - Cornerstone of Dx early
 - **Barlow sign** (subluxation with adduction), **ortolani sign** (reduction with abduction), **limited or asymmetric abduction** are most important findings.
 - **Galeazzi sign** helpful to suggest further evaluation
 - **Asymmetric skin folds** sometimes helpful but not reliable
3. In walking child, increased lordosis, limp, slight delay in walking, short leg, painless Trendelenburg gait, toe walking on affected side are typical signs.

K. Ultrasound

- Excellent modality but is labor intensive and dependent on skills of the operator.
- Can demonstrate reduction vs subluxation vs dislocation. Lower Graf angle suggests hip dysplasia. Not in common use as screening tool in United States.
- Preferred imaging tool before three months of age.
- Radiograph? – helpful after three months. Confusing before then.

II. Treatment

A. Double diapers not acceptable treatment, Hbut sometimes used as reminder to recheck.

B. Pavlik harness (or Von Rosen splint)

- “The main aim of the harness is to achieve concentric reduction and to prevent avascular necrosis, which cripples the child for the whole of his life.” Pavlik
- Kicking harness with hips in flexion and mild abduction
- Use up to 6 months of age
- Must demonstrate reduction in the device within three to four weeks. Ultrasound is good for this.
- Failure to achieve reduction in approximately 8%
- Full time in brace for twelve weeks minimum, then to age at diagnosis
- AVN rate 2.5%

C. Closed reduction and casting

- 6 months until ?
- Precede with skin traction
- Adductor release can be done to increase “safe zone” of abduction. (Range of abduction enough to keep hip located but not enough to cause AVN)
- Position in hip flexion and slight internal rotation with only mild abduction (“human position”)
- Confirm position with arthrogram (rule out obstructions and confirm reduction)
- CT after casting to rule out posterior subluxation
- Ultrasound gaining use to confirm reduction in cast

D. Open reduction (+/- pelvic osteotomy)

1. Indications – failure to achieve reduction or need to leave hip in so extreme a position to keep it reduced that AVN becomes major concern
 - Obstructions to reduction include inverted labrum, iliopsoas tendon and elongated ligamentum teres.
 - By age three the femur will need to be shortened to achieve reduction, maybe earlier.
2. Approaches – anterior Smith-Peterson allows full access to obstructions, hip joint and pelvis and is necessary in older children.

- a. Ludloff approach (medial or adductor) preferred by some in younger infants < 1
 - Direct approach to the major obstructions
 - Cannot do capsulorrhaphyStability depends on immobilization.
- b. Acetabulum – continues to develop until age 4, with some change up to age 8. Therefore varus femoral procedures may be sufficient until age 4, but pelvic osteotomies are probably necessary after that if dysplasia or subluxation still evident. Newer evidence is supporting likelihood of osteoarthritis as sequellae of hip dysplasia.
 - Preferred osteotomies are Salter, Pemberton, Dega, double (Sutherland) or triple or Ganz.Avoid salvage type osteotomies such as Chiari.

III. Complications

A. Avascular necrosis – result of treatment

- Wide range of effects
- Wide abduction may cause AVN.
- Traction or shortening femoral osteotomy seems to decrease incidence.
- Medial circumflex artery may be damaged with medial approach.
- Treatments: femoral varus osteotomy, trochanteric arrest or lateral transfer

B. Recurrent dislocation/dysplasia

- Progressive subluxation must be treated: femoral osteotomy, pelvic osteotomy or both.
- Dysplasia probably also should be treated to avoid osteoarthritis but this is controversial.

C. Arthritis(long term)

D. Teratologic dislocation

- Commonly found in many syndromes such as arthrogyrosis
- Closed treatment has poor success and high AVN.
- Although in past the recommendation was not to relocate hips, modern techniques have yielded better results.
- Recommend open reduction, probably through anterior approach for unilateral
- Bilateral may be better left out for symmetry, better than one in, one out.

E. Secondary operative treatments

1. Indications are controversial in young child.
 - Yes if hip not stable at time of open reduction
 - Yes if acetabulum doesn't remodel with growth
 - Yes in child over 8 (maybe younger)
2. Prerequisite for most osteotomies is concentric reduction of hip, otherwise use salvage procedure.

3. Femoral osteotomies –shortening or correction of anteversion with or without varus, excellent in patients < 4 years old
4. Pelvic osteotomies
 - Salter – reorients acetabulum, increases anterolateral coverage at expense of posterior coverage. Pivot point at opposite triradiate cartilage
 - Double (Sutherland) and triple (Steele) – pivot closer to joint so can shift acetabulum much further than Salter.
 - Spherical osteotomy (Ganz) – very technically demanding. Indicated if triple still won't provide enough coverage. Typically only being done by small number of people.
 - Pemberton, Dega – bend top of acetabulum through open triradiate cartilage. Makes joint smaller. Doesn't sacrifice posterior coverage
 - Chiari-Salvage procedures for noncongruous joints, reduction not possible. Complete osteotomy through ilium with substitution of joint capsule for articular cartilage. Medializes the hip. Indications are painful hip, subluxed and irreducible due to scarring or incongruity of the femoral head and acetabulum.
 - Shelf – same indications as above; often resorbs but can work as salvage.

IV. Legg-Calvé-Perthes' Disease

A. Avascular necrosis of hip joint, idiopathic

B. Symptoms

- Limp
- Pain, gradual onset, intermittent, activity related, in groin, thigh or knee
- History of trauma 17%

C. Physical findings

- Pain with motion of hip early is due to synovitis.
- Limitation of abduction and medial rotation
- Adduction contracture

D. Epidemiology

- Age – 4 to 8 years usually (2 to mid teens possible)
- Sex M:F 4 or 5:1
- Bilateral – 12%, usually sequential. If both sides equal, consider skeletal dysplasia.
- Family history – 1.6-20%
- Abnormal birth presentation
- Older parental age
- Later births
- Lower socioeconomic groups
- Delayed skeletal maturation
- Delayed skeletal growth distal > proximal

- Increased frequency of associated anomalies

E. Natural history

- Process runs its course over 18 months. Circulation almost always regenerates. If head doesn't collapse while necrotic then sphericity maintained. Goals of treatment are to protect head from collapse.
- 20 to 40 years after the onset of LCP most patients maintain a good ROM despite few normal radiographs. Pain and limitations of motion are mostly limited to those with flattening at the time of primary healing or growth arrests with short necks and trochanteric overgrowth. After 45 years, only half remain relatively symptom free, 40% have arthroplasties and 10% have lower Iowa Hip Scores (< 80 of 100).

F. Etiology

- Inflammatory, synovitis: transient synovitis has been noted to precede LCP
- Infectious
- Traumatic
- Congenital
- Generalized disorder: abnormalities found in opposite "normal" hip histologically and radiographically
- Primary cartilage disorder
- Cytotoxic agents
- Vascular, venous? Double arterial infarction model, venous hypertension has been reported several times.

G. Pathoanatomy

1. "Initial stage"
 - a. Epiphyseal cartilage
 - Superficial layers normal but thickened
 - Middle layer hyper cellular or has fibrocartilaginous areas.
 - Deepest layer (growth cells) enchondral ossification ceased in areas affected and matrix fibrillated and degenerated.
 - b. Physeal growth plate
 - Cleft formation and areas of extravasated blood
 - Some areas of normal cells, other areas that have proliferating cells that are separated by a layer that does not degenerate or calcify, leaving tongues of bone growing into the metaphysis as the normal areas grow.
 - c. Ossific nucleus
 - Necrosis of marrow and trabeculae
 - Marrow filled with necrotic debris
2. "Fragmentation stage"
 - a. Ossific nucleus
 - Bone necrosis
 - Invasion of vascularized connective tissue

- Osteoclastic activity
- Resorption of necrotic bone
- New osteoid formation on old lamellar bone
- b. Epiphyseal and physeal bone same as initial phase
- 3. “Reparative stage”
 - a. Ossific nucleus
 - Increased new bone
 - Little necrotic bone
 - Mature bone formation
 - Normal marrow
 - b. Epiphyseal cartilage: resumption of enchondral bone formation
 - c. Physeal growth plate: resumption of enchondral bone formation

H. Radiographic findings

1. “Initial stage”
 - a. Failure of ossific nucleus to grow, ie, smaller than other side
 - b. Increased medial joint space
 - Probably due to cartilage hypertrophy
 - Possibly due to synovitis or hypertrophy of ligamentum teres
 - c. Subchondral radio lucent zone or crescent sign
 - Due to fracture of subchondral bone
 - d. Increased density of femoral head
 - e. Physeal growth plate irregular
 - f. Metaphyseal radiolucency
2. “Fragmentation stage”
 - Fragmentation of ossific nucleus
 - Areas of increased radiodensity and radiolucency
 - Physeal plate irregular
 - Metaphyseal lesions
3. “Reparative stage”
 - Return to normal densities of femoral head
 - Head deformity w/wo plate closure
 - Neck widening

I. Residual head deformities

1. Coxa magna – almost always present
2. Premature physeal plate closure
 - Complete – short neck, head round
 - Partial (lateral) – head externally tilted, oval and acetabulum adapts
3. Irregular head
 - Partial growth arrest
 - Iatrogenic from forcing hinge abduction
4. Osteochondritis dissecans
 - 3% incidence
 - Late onset of disease or incomplete healing

J. Prognostic signs

1. Age
 - a. Age at onset
 - < 8-year-old good prognosis, little treatment needed
 - > 8-year-old poorer prognosis (less time to remodel?)
 - b. Age at healing
 - Can remodel until end of growth, same for acetabulum – growth maturation is usually delayed.
 - Head at risk signs rare under age five
2. Sphericity and congruency at skeletal maturity: probably best indicator but not available to make treatment decisions
3. Extent of involvement
 - a. Partial vs total
 - b. Catterall classification
 - 1) Anterior head involvement
 - No sequestrum
 - No subchondral fracture
 - No metaphyseal abnormalities
 - 2) Sequestrum
 - Clear junction between involved and uninvolved
 - Subchondral fracture anterior 1/2
 - Anterolateral metaphyseal lesions
 - 3) Large sequestrum – 3/4 of head
 - Junction between involved and uninvolved areas sclerotic
 - Metaphyseal changes diffuse
 - Subchondral fracture extends to posterior 1/2 of epiphysis
 - 4) Whole head, diffuse metaphyseal lesion
 - c. Salter-Thompson classification
 - 1) Group A
 - Less than half-head involvement
 - Similar to Catterall I and II
 - 2) Group B
 - More than 50% involvement
 - Catterall III and IV
 - 3) Main determinant is presence of lateral pillar of viable bone which shields remaining bone from collapse.
4. “At risk” signs
 - a. Radiographic
 - 1) Gage’s sign – lytic area in lateral epiphysis and adjacent metaphysis
 - 2) Calcification lateral to the epiphysis
 - 3) Diffuse metaphyseal lesion
 - 4) Lateral subluxation
 - 5) Horizontal growth plate – indicates adduction.

- b. Clinical
 - 1) Adduction contracture
 - 2) Decreases range of motion. **Only universally accepted principle is that excellent motion correlates with good outcome.** Treatment if motion lost is controversial
 - 3) Obesity

K. Treatment

- 1. Goals
 - Prevent deformity
 - Favorably alter growth disturbance
 - Prevent degenerative joint disease
- 2. Basic principle – better motion = better outcome. No treatment needed if Catteral I with good motion and no “at risk” signs.

L. Agreement ends here

- 1. Basic schools of thought
 - a. Range of motion exercises, mainly abduction
 - Traction as needed for synovitis, maybe ASA, then ROM with PT
 - If can get motion will get good outcome.
 - If cannot get motion, then what?
 - b. Containment treatment
 - 1) Requires ROM adequate
 - 2) Can fit head into socket.
 - 3) Head will stay contained with one of the procedures/orthotics.
 - 4) Confirm containment in new position with x-ray.
 - 5) Orthotics
 - a) Scottish Rite type – abduction external rotation
 - Easy to use, well-tolerated
 - May put weight on weak area of bone (check x-ray)
 - b) Petrie
 - Abduction internal rotation
 - Good position for hip
 - Nearly impossible to walk, tolerate for long time
 - c) Advantages: no surgery
 - d) Disadvantages
 - Wear brace for up to 18 months
 - When to stop
 - May not work or be detrimental
 - May still end up with surgery
 - Psychological effects
 - 6) Osteotomy
 - Femoral – varus
 - Pelvic – Salter, shelf for advanced disease
 - Combined femoral and Salter pelvic

2. No clear superiority of any of the above methods.
3. Uncontainable femoral head: maintain ROM.
4. Consider abduction femoral osteotomy.
5. Salvage or reconstructive procedures
 - a. Cheilectomy – usually doesn't work.
 - b. Trochanteric advancement – may use if good hip but + trochanteric overgrowth and + Trendelenburg
 - c. Chiari osteotomy
 - d. Shelf arthroplasty
 - e. Fusion
 - f. THA

V. Slipped Capital Femoral Epiphysis

A. Definition

1. Displacement of the proximal femoral epiphysis sideways off the metaphysis

B. Epidemiology

1. Incidence .71 to 3.4 per 100,000
2. M > F 2.5:1
3. Left more common than right
4. Age range 10-14 in girls, 11-15 in boys
5. Bilateral in 25% to 40%. Since many asymptomatic, probably much higher.
6. Obesity – 90% heavy
7. Skeletal maturation delay
8. Blacks more common

C. Etiology

1. All factors affect strength of “zone of hypertrophy” of physis or shear stresses acting on physis
 - a. Hormonal/endocrine
 - 1) If have endocrine abnormality (eg, hypothyroid, hypogonadism, renal osteodystrophy, etc), then higher incidence of slips.
 - 2) If test patients with SCFE, rare to find endocrine problem
 - 3) 80% occur during adolescent growth spurt.
 - b. Generalized systemic disorder
 - c. Genetic?
 - d. Trauma: may contribute, particularly in acute slips

D. Pathology

1. Physeal changes
 - a. Widened physis up to 12 mm (normal 2.6 to 6 mm)
 - b. Hypertrophic zone up to 80% width (normal 15-30%)
 - c. Slips occur through zone of hypertrophy.
2. Synovitis with increased hypervascularity and round cell infiltration

E. Clinical presentation

1. Symptoms
 - a. Pain in groin, thigh or knee

- b. Limp with leg turned out
- c. Onset is gradual or sudden.
- 2. Physical findings
 - a. Limited internal rotation
 - b. Pain with motion (internal rotation)
 - c. With flexion may have obligatory need to externally rotate.
 - d. May have apparent LLD due to adduction contracture
- 3. Radiographs
 - a. AP and lateral needed. Get true lateral rather than frog, if acute slip expected, to avoid further displacement.
 - b. AP view
 - 1) Widened irregular plate
 - 2) Kleins line won't intersect epiphysis
 - 3) Blush sign
 - 4) Decreased height of epiphysis
 - c. Lateral view
 - 1) Best view to detect slip
 - 2) Head posterior to neck
 - d. Classification
 - 1) Preslip – widened physis
 - 2) Mild – max displacement 1/3 diameter of neck
 - 3) Moderate – > 1 cm displacement but less than 1/2 diameter of neck
 - 4) Severe > 1/2 diameter of neck

F. Classification (traditional)

- 1. Preslip
 - a. Early symptoms
 - b. Radiographs normal except possible widening. No displacement of epiphysis
 - c. Exam may be positive for pain with internal rotation.
- 2. Acute
 - a. Abrupt displacement through physis with pre-existing epiphyseolysis
 - b. 10% of all slips
 - c. Symptoms less than 2 or 3 weeks
 - d. 75% describe prodromal symptoms for 1 to 3 months – acute on chronic
 - e. Onset often associated with an event
 - f. Physical exam
 - 1) Pain usually prevents exam.
 - 2) External rotation of leg
 - 3) Unable to bear weight
 - g. Radiographs: acute disruption of epiphysis from metaphysis
 - h. No evidence of healing or remodeling
- 3. Chronic slip
 - a. Symptoms – 3 weeks to 9 months
 - b. Usually gradual onset

- c. Pain with prolonged standing or walking
 - d. Lack of internal rotation
 - e. Shortening
 - f. Radiographs – displacement of epiphysis with remodeling changes
4. Acute on chronic – symptoms for a while with sudden change for worse

G. Newer classification (Loder)

- 1. Stable vs unstable (acute and acute on chronic)
- 2. Better correlation with treatment
- 3. Stable
 - a. Able to bear weight on hip
 - b. Single pin fixation usually adequate
- 4. Unstable
 - a. Unable to bear weight
 - b. Fracture table to avoid movement
 - c. May need more than one pin

H. Natural history

- 1. Controversial
 - Few natural Hx studies
 - Few long-term F/U studies
- 2. Deformity related to long-term prognosis
 - Mild – good long-term prognosis
 - Moderate – good long-term prognosis
 - Severe deformity – early degenerative joint disease
- 3. Complications of treatment may lead to poor prognosis even in mild slips.

I. Treatment

- 1. Goals
 - Prevent further displacement – orthopaedic emergency or urgency
 - Avoid AVN, chondrolysis
 - Promote closure of physis? May still close at normal time
- 2. Chronic slip
 - a. Stabilization
 - Pin in situ
 - Open epiphyseodesis-rarely done
 - b. Long-term results good or excellent for mild and moderate displacement
 - c. Severe slips get DJD so debate as to whether to realign
 - Neck osteotomy
 - Intertrochanteric osteotomy
 - Subtroch osteotomy
 - d. Due to higher rates of complications, should reserve above for people with restricted motion after physal plate closure.
- 3. Acute, acute on chronic
 - a. Reduce acute component **only**

- Skin traction
- Manipulation to get onto the table may reduce–
Warning: overreduction may lead to AVN.
- b. Stabilize epiphysis
 - 1) Multiple pins, cannulated screws
 - Need good radiographic control
 - Stay out of joint.
 - Stay out of superolateral quadrant due to AVN (lat epiphyseal vessels).
 - Never remove pins before plate closure.
 - 2) Postop
 - Touch weight bearing until pain free
 - Follow until plate closure.
 - Watch for opposite side slip (most occur within one year, in less mature patients at time of presentation).
- c. Epiphyseodesis
 - 1) Advantages
 - Rapid fusion of physeal plate (3 months)
 - Avoid hardware removal.
 - 2) Disadvantages
 - Increased surgery time
 - Blood loss
 - Postop cast 3 months
 - Continued slippage if graft fails to close plate

J. Complications

1. Avascular necrosis – most devastating complication
 - a. Risk factors
 - Acute slips
 - Overreduction of slip
 - Pins in superolateral quadrant
 - Neck osteotomies
 - b. Treatment
 - Avoid risk factors.
 - Sugioka procedure
 - Intertrochanteric osteotomy
 - Vascularized graft
 - Hip fusion
2. Chondrolysis – cartilage necrosis
 - a. Clinical
 - 1) Pain
 - 2) Loss of motion
 - 3) Contracture
 - 4) Limp
 - b. Radiographically
 - 1) Loss of joint space
 - 2) Irregularity of subchondral bone both sides of joint
 - 3) Disuse osteopenia
 - c. Etiology unknown

- 1) Autoimmune?
- 2) Interruption of cartilage nutrition
- d. Risk factors
 - 1) Cast immobilization
 - 2) Pin in joint
 - 3) Severe slips
 - 4) Long duration of symptoms prior to Rx
- e. Treatment
 - 1) Anti-inflammatories
 - 2) Bedrest, traction
 - 3) CPM with surgical capsulectomy???

K. Controversies

- Should pins be removed at all? High rate of pin breakage during removal.
- Should opposite hip be prophylactically pinned?
- Probably in very immature patients because incidence of contralateral slip is so high
- * Role of osteotomies

VI. Septic Hips

- Most common in children under age 2
- Big problem is damage done to hip articular and growth cartilage before infection is under control.

A. Signs

1. Fever
2. Limited motion with pain
3. Increased ESR or C-reactive protein
4. + fluid in hip by ultrasound or tap
5. Look for evidence of nearby osteomyelitis in femoral neck or pelvis.

B. Bacteriology

1. Most common *Staph aureus*
2. Hemophilus influenza not so common now due to immunization programs

C. Treatment

1. Early surgical drainage
2. Antibiotics IV or oral for three weeks
3. Watch for instability, may need spica to prevent dislocation.
4. Must watch for evidence of growth problems or hip dysplasia over following years.

VI. Toxic Synovitis

- Must rule out infection
- ESR or CRP normal
- Patient not sick
- Look for future Perthes' type condition, but may not ever develop

VII. PFFD

A. Hypoplastic femur

B. Aitken classes A-D

- A – head neck trochanter
- B – head, shaft, no connection
- C – no head
- D – no head, no acetabulum

C. PFFD treatment

1. If discrepancy < 17 cm
 - Hypoplastic femur
 - Aitken A
 - Fix pseudoarthrosis or coxa vara
 - Lengthen femur.
2. If discrepancy > 17 cm
 - Knee fusion Syme's amputation
 - Treat like AK amputation.
 - Or consider rotation plasty.

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