Conceptual Orthopedics
The Learning Never Ends

Pediatric Orthopedics

Dedicated To

The Man Who Taught Us
Examination, Evaluation and Management of
Orthopedic Problems in Children

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Authored By The Faculty of Conceptual Orthopedics

Table of Content

SECTION 1
General Pediatric Orthopedics

CHAPTER | 1.1

Epiphyseal Injuries

Epiphysis

- Region of long bone between ends of the bone with the growth plate.
- At birth the end of the bone is completely cartilaginous [Except for distal femur / occasionally proximal tibia].
- It is known as chondroepiphysis.
- It gradually enlarges until cartilaginous arc has been completely replaced by bone.
- The appearance of ossification centers differs between different bones & this needs to be taken into account when diagnosing the fracture.
- Ossification causes more rigidity which causes change in fracture pattern with age.

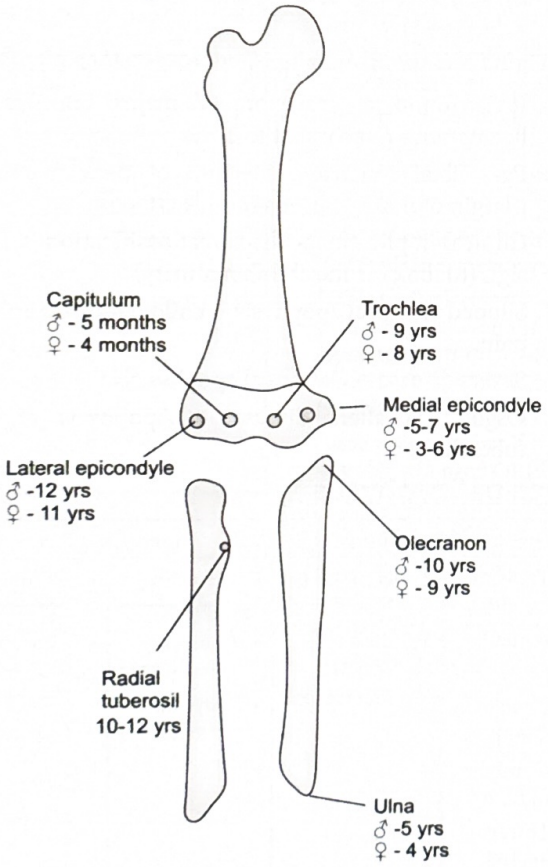


Fig.1.1.1: Ossification centres of upper limb

- The external surface of epiphysis is composed of articular cartilage/ perichondrium.
- Muscle fibres, tendons, ligaments attach to the perichondrium also responsible for centrifugal enlargement of epiphysis.
- The perichondrium blends in periosteum.
- The perichondrial/ periosteal tissue continuity contributes to biomechanical strength of the epiphyseal / metaphyseal junction at region called zone of Ranvier.
- The hyaline cartilage below articular cartilage contributes to the growth of epiphysis.

Four Zone's: of physis

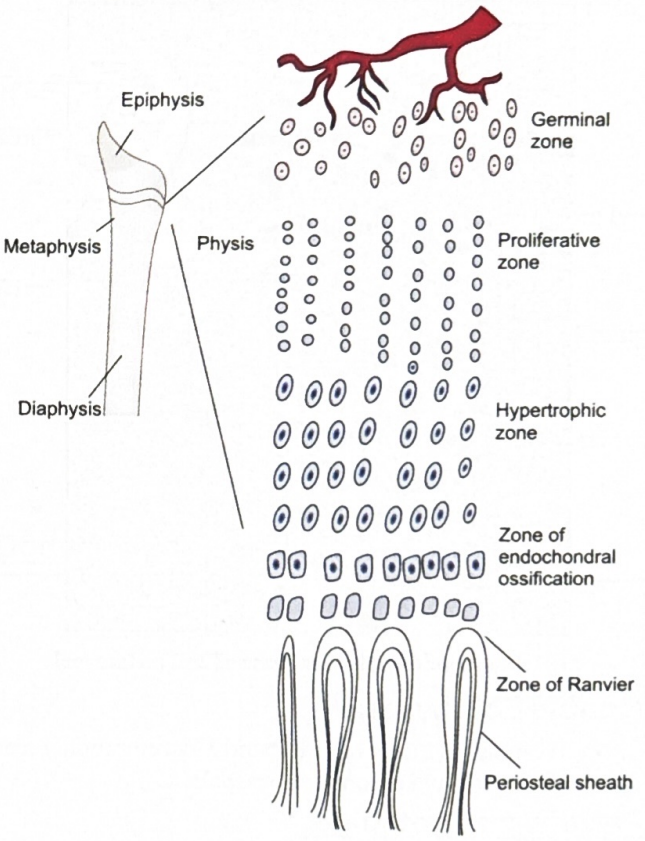


Fig.1.1.2: Zone's of physis

Physis

- Highly organized and also a dynamic structure consisting chondrocytes
- Collagen Type 10 is seen in limited quantity in the hypertrophic zone.

Blood Supply of physis

Type A: Easily compromised by epiphyseal separation.

- Proximal humerus, Proximal femur
- Covered with articular cartilage
- Blood supply enters through perichondrium.
- Distal to proximal
- Complete disruption of epiphyseal vasculature may not produce an extensive ischemia.

Type B: Less susceptible to devascularization.

- Proximal/ Distal tibia, distal radius.
- Partially covered with articular cartilage.

Pseudoepiphyses-

- Persistent expression of distal epiphysis [thumb – Most common]
- Appears earlier than proximal epiphysis.
- Fuses rapidly.
- Radiological differentiation from acute fracture

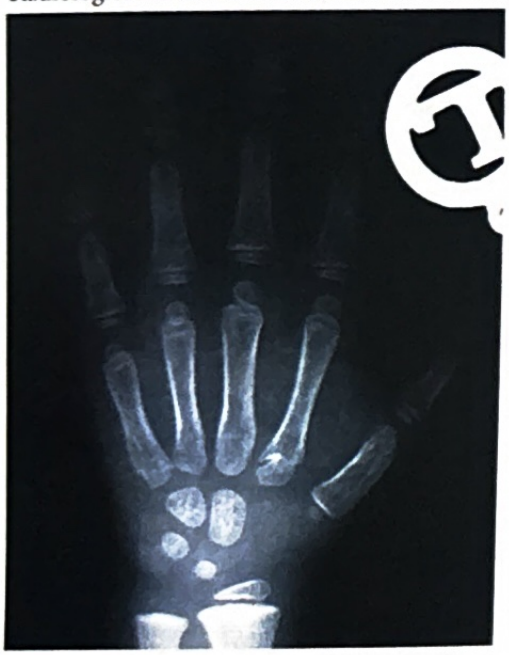


Fig 1.1.3: Pseudoepiphysis at base of 2nd metacarpal

Double Epiphyses:

- It can occur in any bone of hand Most commonly seen in the first and second metacarpals.

Periphyseal Notching

- It is usually confused with fracture or pseudoepiphysis or a double epiphysis
- In such cases clinical examination of the involved digit and comparing it radiologically with other digits of the same hand and the other hand will be helpful.

Hypertrophic cell zone [Maturation zone]

- It is either enlarged/ swollen and they eventually die.
- Fractures can occur in around 5% of children through this region.

Apophysis:

- It is a growing center which grows upon the mother bone.
- There is no direct articulation of the bone with the joint.
- It is a normal developmental outgrowth of bone which arise from separate ossification center and faces to the mother bone later in development [limited growth potential]

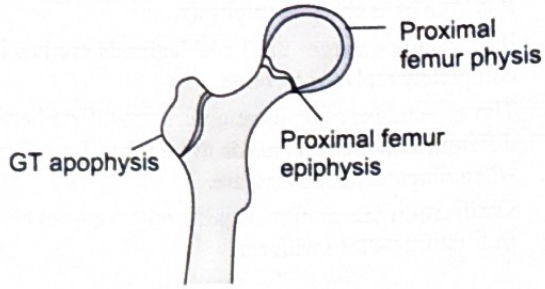


Fig 1.1.4: Showing Apophysis, Intracapsular epiphysis

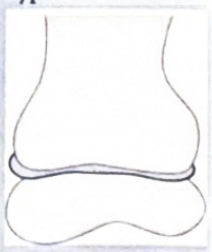
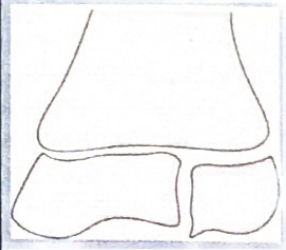
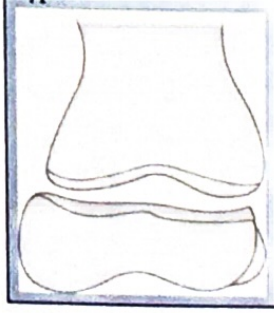
- It is found at insertion of major tendons and ligamentous attachment to bone
- Eg- Tibial tubercle- insertion of patellar tendon/ Margin of ilium- various muscle attachment.
- Clinical implications- Iliac crest ossification → Risser sign (to look for the child's maturity)
- Slipped vertebral apophysis → child complains of back pain.
- Severs disease → Calcaneal apophysitis.
- Osgood Schalters disease → Apophysis at tibial tubercle.

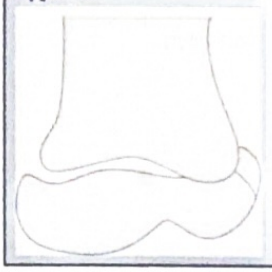
Types of Epiphyses

Pressure	Aberrant	Traction	Atavastic
• Transmit weight • Articular E.g.: 1. Head of femur 2. Condyle of tibia 3. Lower and of radius	• Head of 1st Metacarpal • Base 2nd 3rd Metacarpal	• Greater trochanter • Lesser trochanter • Styloid process	• Coracoid process • Posterior tubercle of talus.

Pediatric fractures

Salter Harris classification

Type-I 	• Transverse fracture through hypertrophic or calcified zone • Even if fracture is displaced growing zone of physis is usually not injured & growth disturbance is uncommon. • Prognosis is good.
Type-II 	• Most common type • Towards edge, fracture deviates away from physis & splits off triangular metaphyseal fragment of bone. [Thurston Holland] • Prognosis – excellent
Type-III 	• Fracture that splits epiphysis and exists off transversely to one side through hypertrophic layer. • Damages reproductive layers of physis and may result in growth disturbances. • Good prognosis but for any intra articular displacement one might have to consider an ORIF.
Type-IV 	• Fracture splits epiphysis but it extends into metaphysis • Liable to displacement with misfit between the separated parts of physis, resulting in asymmetric growth • Good prognosis but if unstable will need ORIF.
Type-V 	• A longitudinal compression injury of physis • No visible fracture but growth plate is crushed and may result in growth arrest • Poor prognosis as can result in growth arrest.

RANG CLASSIFICATION	
Type-VI 	• Injury to perichondrial ring (Peripheral zone of Ranvier) • There is significant growth disturbance. • May cause angular deformity.

Mechanism of injury

- Fall or traction
- Most common in RTA and during sporting activities

Clinical features

- Boys: girls- 3:2
- Infancy or age group of 10-12 years
- Deformity usually minimal
- Injury in child followed by pain and tenderness near joint.

X-Ray:

- Physis itself is radiolucent & epiphysis incompletely ossified.
- Widening of physeal gap, incongruity of joint or tilting of epiphyseal axis.

Treatment

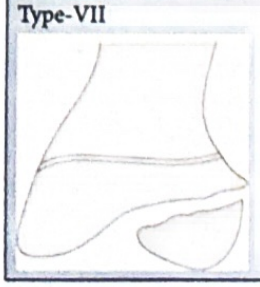
Undisplaced

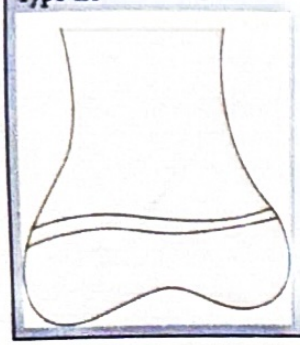
- Splinting 2-4 weeks
- Type 3,4: Repeat x-ray after 4-10 days to rule out late displacement.

Displaced

- Type 1 & 2 → Closed reduction followed by splinting for 3-6 weeks
- 3 & 4 → Anatomical reduction of fractures
- Gentle manipulation followed by cast for 4-5 weeks & longer for type 4
- If not – ORIF with screws.

Ogden type VII, VIII, IX

Type-VII 	• Fracture involving epiphysis only • Includes osteochondral fracture epiphyseal avulsion.

Type-VIII 	• Fracture of metaphysis • Circulation to remodeling region disputed
Type-IX 	• Diaphyseal fracture • Avulsion injury to periosteum.

CHAPTER | 1.2

Rickets

Before understanding these diseases, one has to know how the levels of serum calcium is maintained in our body. The ingested calcium is absorbed from the gut, regulated by vitamin D. However, if there is a reduced absorption of calcium from the gut due to deficiency of vitamin D or deficient activation of the vitamin D, the level of serum calcium falls. This gives feedback to the parathyroid glands which responds by increasing the levels of parathyroid hormone. This increases the release of calcium from the bone, which is the main source of calcium within our body. For this release however the problem that occurs is that there is lower mineralization of the bones and at a later stage due to the excessive osteoclast activity there can be a more resorption than bone formation. 8 to 10 grams of calcium reach the glomeruli also, most of which are reabsorbed (65%- Proximal convoluted tubule, 20%- ascending loop of Henle, 10%- distal convoluted tubule). This is majorly controlled by Parathormone either directly or indirectly via vitamin D. Parathormone doesn't have a direct action on gut absorption of calcium.

Phosphate has a similar journey of that of calcium from the time of its absorption from the gut, with minor differences that its reabsorption from the kidney is inhibited by parathormone and its lowering in the serum doesn't stimulate parathormone secretion.

With this kept in mind let us understand the types of rickets. Rickets affects children and there are more of physeal changes that are predominant whereas osteomalacia is the adult form leading to more of pseudo-fractures or loozer's zone appearance.

Causes of Rickets

1. Vitamin D disorders

a. Nutritional
b. Secondary, malabsorption, decrease in liver (25) hydroxylase activity, CRF
c. VDDR Type I Deficiency of 1 alpha hydroxylase
d. VDDR Type II End organ resistance to 1.25 (OH)2 Vitamin D3 (High prevalence of alopecia, ectodermal defects)

2. Calcium deficiency

3. Phosphorus deficiency

4. Renal losses

a. Fanconi syndrome
b. Distal RTA
c. X-linked dominant (commonest), it is due to PHEX gene mutation, autosomal dominant and autosomal recessive are 3 varieties of hypophosphatemic Rickets (vitamin D resistant rickets)—increase incidence of skeletal deformities, no hypocalcemia, hypophosphatemia, phosphaturia, PTH is normal, vitamin D is normal and ALP is high.

5. Tumor:
   a) Soft tissues tumours like hemangiopericytoma
   b) Bone tumors like non-ossifying fibroma, giant cell tumors, osteoblastoma, fibrous dysplasia, and neurofibromatosis.

Vitamin D deficient rickets- where there is deficiency of vitamin D leading to reduced calcium absorption from the gut. The initial reduction of the serum calcium causes lower calcium available for bone mineralization and the same gives negative feedback to the parathyroid gland which causes an increased release of calcium from the bone. Here there is normal serum calcium with mild reduction of phosphate with high levels of parathormone with definite demineralization of the bone.

Vitamin D resistant rickets- where there is an abnormality in either the proximal or distal end of the glomerulus leading to absent activation of Vitamin D, deranged phosphate and calcium reabsorption and also inability to maintain hydrogen ions. Here there can be a hypocalcemic state as well hypophosphate state. The various types include hypophosphatemic rickets, proximal Fanconi syndrome and Lowe syndrome. The classical hypophosphatemic rickets is an X linked dominant transmitted disease, meaning the affected males if they have daughters all of the are affected and affected females affect half of their sons and daughters.

Clinical manifestation of rickets

In infantile period vitamin D deficiency is usually associated with malnourishment. So, in such cases the child can be irritable and apathetic. They can also have lower body weight as compared to other children of the same age group.

In children of walking age, can have difficulty walking in severe cases. Most of the children also have a stunted height.

Signs are very much evident from head to toe. In the head there is bossing of the skull, craniotabes, flattening of the parietal bone, hot cross bun skull and slow eruption of teeth. On the chest there can be rachitic rosary which are adjacent costochondral enlargements that are firm nodular and seen on either side of the sternum. Pectus carinum and softened lower ribs leading to Harrison's groove. In the abdomen there can be a pot belly due to hypotonia, rachitic catback appearance, and loss of lumbar lordosis at a later stage. In the extremities there are deformities that can form. This is due to the weak physis which has absence of mineralization of the zone of calcification. The loads are transferred on the metaphysis leading to broadening of the metaphysis. The weak physis leads to valgus or varus deformities of the joints, rachitic saber shins, sausage enlargements of the digits, string of pearl appearance of the digits when there is constriction at level of the joints and in florid rickets pathological fractures are also common.

Epiphyseal Plate Abnormalities

The normal epiphyseal plate has five zones

1. The resting zone - adjacent to the epiphyseal nucleus, is composed of cartilage in which the cells are sparse, small, rounded, and randomly dispersed.
2. The proliferative zone - On the metaphyseal side of the above zone; here the cells are regular, flattened, and arranged in columns. This zone is the site of mitotic activity and length growth of the epiphyseal plate.
3. The maturation zone - Below the above level; here the cells, still in columns, become increasingly large and more rounded. These cells stain less densely and contain large amounts of glycogen.
4. At the lowest part of this zone, often called the zone of hypertrophy, the cell lacunae become very large, but the cell nuclei are shrunken and appear faded when stained with hematoxylin and eosin. In this region, vascular buds grow in from the metaphysis, appearing to enter the empty lacunae at the base of the columns, while the bars of cartilage matrix between the columns become heavily calcified. This entire region is called the zone of provisional calcification.
5. The zone of primary spongiosa - Lower in the metaphysis, the calcified cartilage bars become surrounded by osteoblasts, which produce a seam of calcified osteoid around the still present calcified cartilage bars.

The maturation zone is usually affected in rickets. Other zones are quite normal histologically. The chondrocyte arrangement is distorted, the column arrangement is lost and this cartilage can penetrate into the underlying weak bone. The chondrocytes elongate and hence there is an appearance of increased width of the physis and irregularity at the metaphyseal end due to the penetration of cartilage into the bone. There is increase in transverse growth of the bone instead of the vertical growth of the physis. This increased width of the plate produces the palpable enlargement of the bone or rib ends characteristic of rickets and referred to as "cupping" or 'flaring" of the epiphyseal-metaphyseal area.

The second obscure characteristic of the epiphyseal area in rickets is cupping. This due to the piston-cylinder effect on the abnormal bone. On a normal growing bone, the piston and the cylinder are rigid, and hence the force of the piston on the cylinder tends to push it away from the cylinder letting it grow. But in a weak bone these loading ends in cupping.

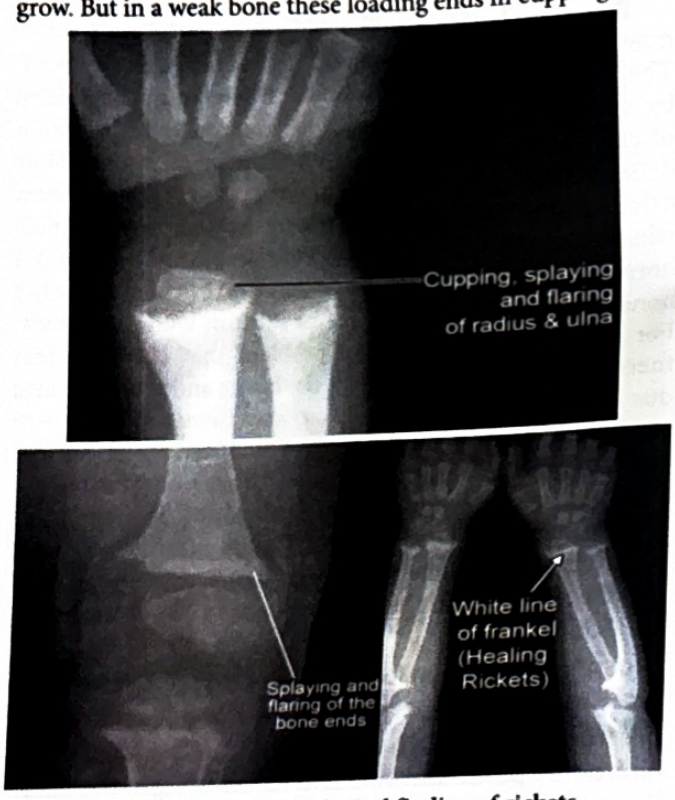


Fig 1.2.1: Classical radiological finding of rickets

Other Systems affected in Rickets and Osteomalacia

- Rickets and osteomalacia are systemic diseases with muscular hypotonia and abdominal and renal disturbances with late onset cardiac and respiratory issues as well.
- Paradox of rickets- As the rickets becomes more severe and the child becomes more sick, the changes in the epiphyseal plate become milder and may actually disappear if the patient lives long enough. This paradoxical behavior is due to the fact that rickets, by definition, is a disease of growth. If the patient becomes chronically ill with respiratory, cardiac, or renal disease, growth is inhibited on a hypoproteinemic or nutritional basis, and the epiphyseal manifestations of rickets (which are directly related to the rapidity of growth) fade or even disappear

Management

Blood tests

Table 1.2.1: Biochemical markers in various rickets

Lab findings in Rickets	Calcium (Usually N↓)	Phosphorus (Usually N↓)	PTH (Usually ↑)	ALP (Usually ↑)	25 (OH) D	1, 25 $(OH)_2$ D
Vit D deficiency	N↓	↓	↑	↑	↓	↓N↑
VDDR Type I	N↓	↓	↑	↑	N	↓
VDDR Type II	N↓	N↓	↑	↑	N	↑↑
CRF	N↓	↑	↑	↑	N	↓
Dietary P deficiency	N	↓	N	↑	N	↑
XLH-Hypophosphatemic rickets	N	↓	N	↑	N	↓
ADHR-Hypophosphatemic rickets	N	↓	N	↑	N	↓
Fanconi syndrome (proximal RTA)	N	↓	N	↑	N	↓
Dietary Ca deficiency	N↓	↓	↑	↑	N	↑

A renal function test has to be performed to rule out renal cause for rickets.

Radiographs

Xrays of the deformed part can give an idea about the angular deformities, status of the physis, metaphysis and prognosis. The findings are the following-

- The characteristic feature of rickets is thickening and widening of growth plate (physis)
- Indistinct and hazy metaphysis that is abnormally wide (splaying) with cupping or flaring (Brush like appearance)
- Bowing of diaphysis, with thinning of cortices
- Looser's zone in 20%.
- Persistent hypocalcemia may cause secondary hyperparathyroidism leading to findings of brown tumour as well.

Therapy

1. Nutritional rickets: Two strategies for administration of vitamin D. Stoss therapy, 300,000–600,000 I.U. of vitamin D are administered orally or intramuscularly. The alternative is daily high dose vitamin D, 2000–5000 I.U./day over 4–6 week. Followed by 400 I.U. Vit D/Day and supplements of calcium for 2–4 months
2. Hypophosphatemic Rickets: Oral phosphate and Vit D supplements. Joule's solution—Dibasic sodium phosphate, phosphoric acid is given in hypophosphatemic Rickets.
3. VDDR I: Calcitriol, calcium, phosphate supplements
4. VDDR II: Treatment not satisfactory large doses of calcitriol and calcium, phosphate supplements

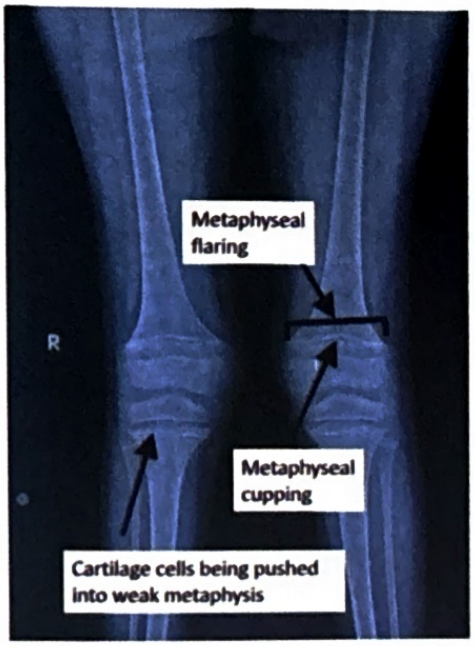


Fig 1.2.2: Look at the changes at metaphyseal region where as epiphyseal end looks normal - Mankin's theory

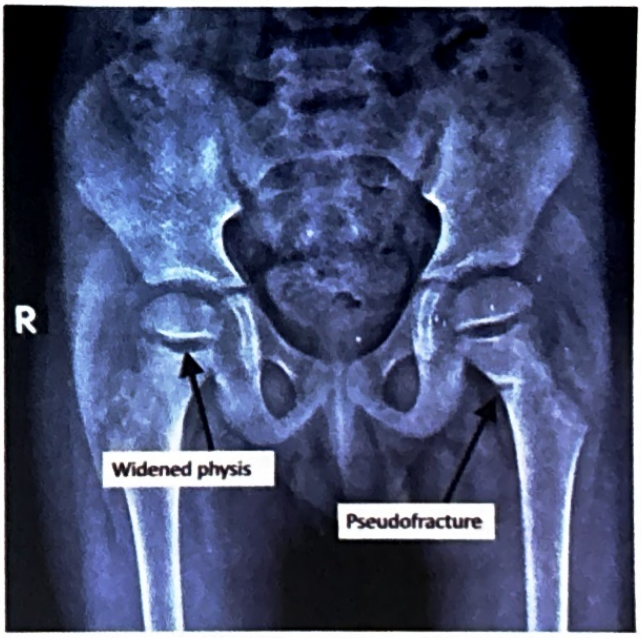


Fig 1.2.3: Looser's zone (Pseudofracture)

CHAPTER 1.3

Causes of Limp in Children

Table 1.3.1. Causes of limp in various age groups.

Toddler (1-3 yrs)	Child (4-10 yrs)	Adolescent (11-15 yrs)
Transient synovitis	Transient synovitis	SCFE
Septic arthritis	Septic arthritis	Hip dysplasia
Diskitis	Perthes disease	Chondrolysis
Toddlers fracture	Discoid meniscus	Overuse syndromes
Cerebral palsy	Limb length discrepancy	Osteochondritis dessicans
Muscular dystrophy		Scoliosis
DDH		
Coxa vara		
Pauciarticular juvenile arthritis		
Leukemia		
Osteoid osteoma		
Rarities		

CHAPTER | 1.4

Cerebral Palsy

What is cerebral palsy?

- Mercer Rang
  - Cerebral palsy is a condition which develops as a result of a lesion in the developing brain which results in a disorder of movement and posture, which is permanent, but not unchanging.
    - Permanent lesion → non-progressive brain lesion ("Static encephalopathy")
    - Movement disorder → keeps changing (progressive musculoskeletal pathology)
- The national workshop on definition and classification of CP (2007)
  - Cerebral palsy describes the group of
    - Permanent disorders of the development of movement and posture causing activity limitation that are attributed to
    - Non-progressive disturbances that occurred in the developing foetal or infant brain.
  - Motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication and behaviour; by epilepsy and by secondary musculoskeletal problems.
  - It is the result of a brain lesion, which is fixed and non-progressive.
- Prevalence:2.4-2.7 per 1000 live births
  - Rates are increasing secondary to an increase in the survival of very low birth weight infants.

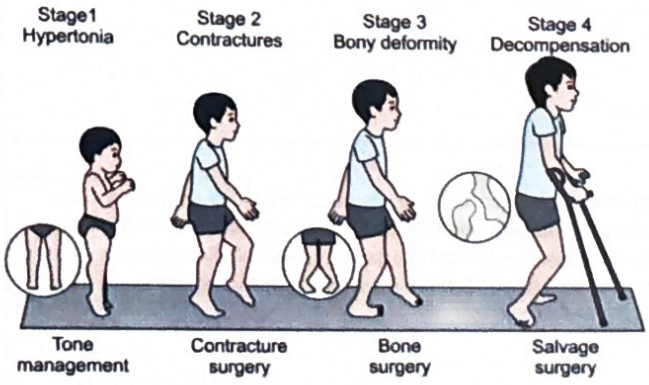


Fig 1.4.1: Musculoskeletal pathology in cerebral palsy

Etiology

- Prenatal
  - Brain Malformations
  - Neurological damages due to
  - Intrauterine infections [TORCH]
  - Intrauterine hypoxia
  - Vascular factors
  - Environmental toxins
- Perinatal causes
  - Birth asphyxia
  - Neonatal jaundice
  - Neonatal hypoglycemia
  - Meningitis/encephalitis
  - Intracranial bleeding
- Postnatal causes
  - Infections
  - Asphyxia due to aspiration or drowning
  - Traumatic brain injury
  - Vascular complications
- Prematurity
  - Risk of CP
    - 1 in 2000 (0.05%) full term births
    - 12.3% for infants born at 24-33 weeks
- Birth weight
  - Risk of CP
    - >2500g → 1.1 per 1000
    - <1000g → 78.1 per 1000

Classification

Physiological classification

- Spastic Cerebral Palsy (80%) is characterised by at least two of:
  - Abnormal pattern of posture and/or movement
  - Increased tone (not necessarily constantly)
  - Pathological reflexes (hyper-reflexia or pyramidal signs e.g. Babinski response)
  - It may be unilateral (hemiplegia) or bilateral
- Dyskinesic Cerebral Palsy (10%) is characterised by both of

- Abnormal pattern of posture and/or movement
- Involuntary, uncontrolled, recurring, occasionally stereotyped movements of affected body parts
- Dyskinesic Cerebral Palsy may be either:
  - Dystonic Cerebral Palsy: Hypokinesia and hypertonia.
  - Choreo-athetotic Cerebral Palsy: Hyperkinesia and hypotonia

- Ataxic Cerebral Palsy (5%) is characterised by both of
  - Abnormal pattern of posture and/or movement
  - Loss of orderly muscular co-ordination, so that movements are performed with abnormal force, rhythm and accuracy

When it is a Mixed CP (5%) form, i.e. spasticity with ataxia and/or dyskinesia, the child should be classified according to the dominant clinical feature.

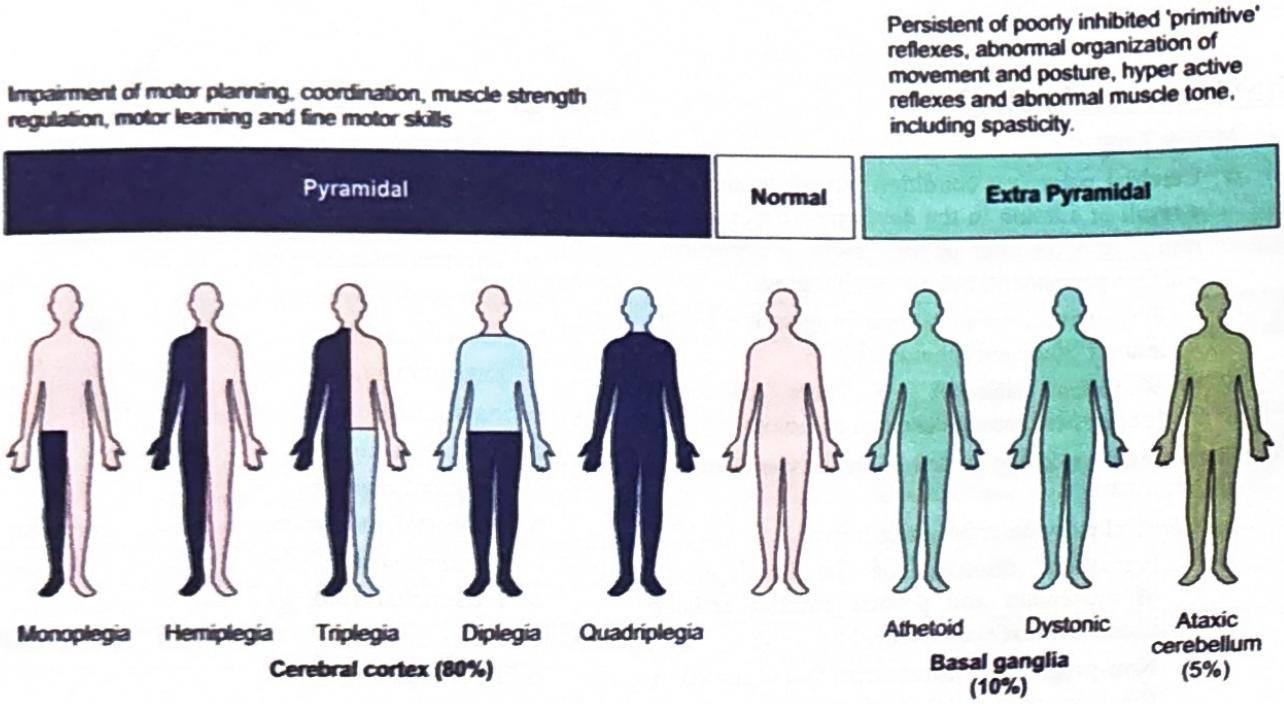


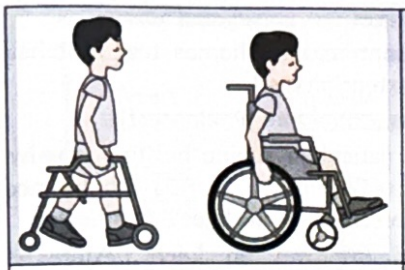
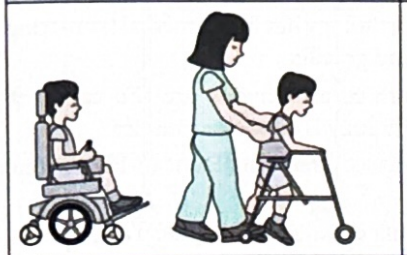
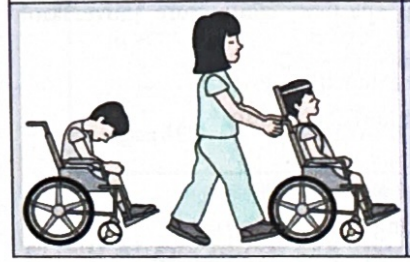
Fig 1.4.2: Physiological classification

Functional classification

- Gross motor functional classification system
  - Between 6th and 12th birthday (figure below)
  - Between 12th and 18th birthday (not discussed here)

Table 1.4.1. Functional classfication

	GMFCS level I Children walk at home, school, outdoors and in the community. They can climb stairs without the use of a railing. Children perform gross motor skills such as running and jumping, but speed, balance and coordination are limited.
	GMFCS level II Children walk in most settings and climb stairs holding onto a railing. They may experience difficulty walking long distances and balancing on uneven terrain, inclines, in crowded areas or confined spaces. children may walk with physical assistance, a hand hold mobility device or used wheeled mobility over long distances. Children have only minimal ability to perform gross motor skills such as running and jumping.

	**GMFCS level III** **Children walk using a hand-held mobility device in most indoor settings. They may climb stairs holding onto a railing with supervision or assistance. Children use wheeled mobility when traveling long distances and may self propel for shorter distances.**
	**GMFCS level IV** **Children use methods of mobility that requires physical assistance or powered mobility in most settings. They may walk for short distances at home with physical assistance or use powered mobility or a body support walker when positioned. At school, outdoors and in the community children are transported in a manual wheelchair or use powered mobility.**
	**GMFCS level V** **Children are transported in a manual wheelchair in all settings. Children are limited in their ability to maintain antigravity head and trunk postures and control leg and arm movements.**

General evaluation [Multi-disciplinary evaluation]

Only focusing on Orthopaedics relevant evaluation here

History

- Birth history – prenatal, natal & post-natal
  - Prematurity
  - Birth weight
  - Neonatal ICU stay
- Developmental milestones
- Preferential use of hand or leg / early handedness (<2 years old)
- Dragging one leg while crawling
- Strabismus, difficulty swallowing, frequent choking, delayed speech development, poor eyesight
- Seizures

Clinical examination

- Muscle tone
- Infantile reflexes persist after 3-6 months
  - Moro reflex / Parachute reflex / Tonic neck reflex
- Exaggerated deep tendon reflexes

Prognosis

- Ability to sit independently by age of 2 years → highly prognostic of ability to walk

Red flag signs

- History
  - No risk factors
  - Developmental milestone regression
  - Fluctuation in motor skills
  - Positive family history
  - Progression of symptoms
  - Onset of disability coincides with a period of metabolic stress → convulsion
- Examination
  - Dysmorphic facies
  - Optic atrophy/retinopathy
  - Pes cavus
  - Evolving sensory signs

Orthopedic evaluation

- Level of ambulatory ability
  - GMFCS
  - FMS (Functional mobility score)
- Muscle tone
  - Modified Ashworth scale
    - 0: No increase in muscle tone
    - 1: Slight increase in muscle tone, with a catch and release or minimal resistance at the end of the range of motion when an affected part(s) is moved in flexion or extension
    - 1+: Slight increase in muscle tone, manifested as a catch, followed by minimal resistance through the remainder (less than half) of the range of motion
    - 2: A marked increase in muscle tone throughout

most of the range of motion, but affected part(s) are still easily moved
- 3: Considerable increase in muscle tone, passive movement difficult
- 4: Affected part(s) rigid in flexion or extension

- Tardieu scale
  - R1 → the angle of catch following a fast velocity stretch
  - R2 → passive range of motion following a slow velocity stretch
    - R2-R1
      - Large difference = spastic component is more
      - Small/no difference = Rigidity/ contracture

- Selective motor control
  - Grade 0 – No ability/only patterned movement observed (poor)
  - Grade 1 – Partial ability/partially isolated movements (fair)
  - Grade 2 – Complete ability/completely isolated movements (good)
- Muscle strength – MRC grading
- Tests for contractures
  - Hip flexion contracture [Thomas test / Staheli prone hyperextension test]
  - Hip adduction contracture [Phelps test]
    - With the patient in supine position, passive abduction of the hip is performed with the knee in extension and with the knee in 90° flexion
    - Abduction improves on knee flexion → primary pathology lies in the medial hamstring muscles and gracilis
    - If both the measurements are the same → prime pathology is adductor muscles
  - Rectus femoris contracture [Duncan-Ely's/Prone Rectus test]
  - Knee hamstring contracture [Popliteal angle test]
  - Gastro-soleus spasticity/contracture [Silverskiold test]
- Torsional profile [Staheli]
- Standing balance and equilibrium
- Gait analysis
  - Observational gait analysis
  - Video recording
  - Computerized gait analysis (Gait lab)

Gait analysis

- Spastic diplegia (Sagittal gait patterns)

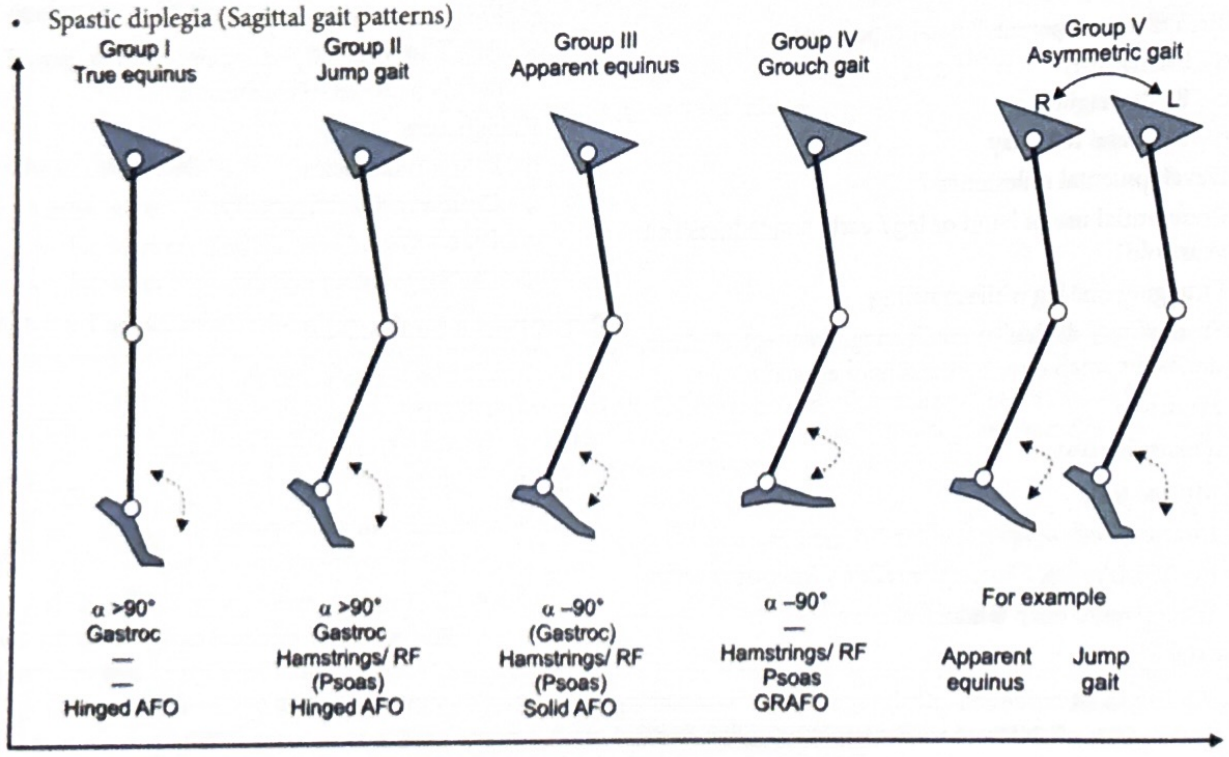


Fig 1.4.3: Gait in spastic diplegia (cerebral palsy)

- Spastic hemiplegia

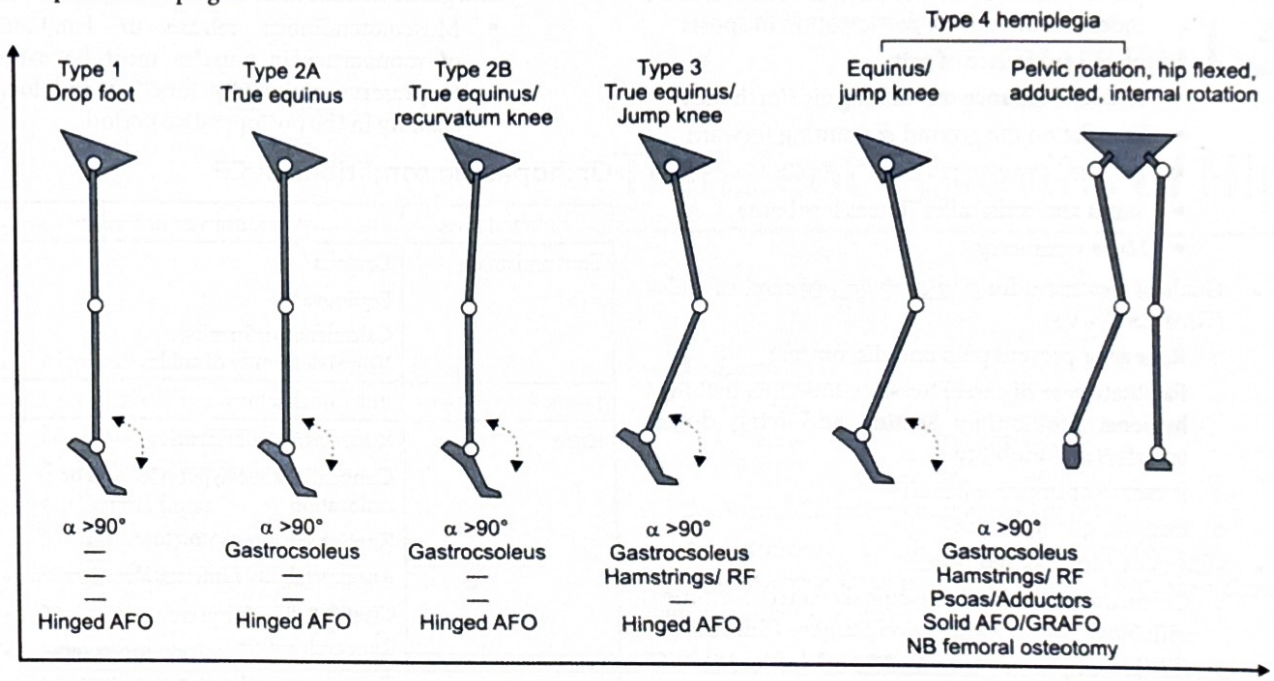


Fig 1.4.4: Gait in spastic hemiplegia

CLINICAL DECISION MAKING : THE DIAGNOSTIC MATRIX

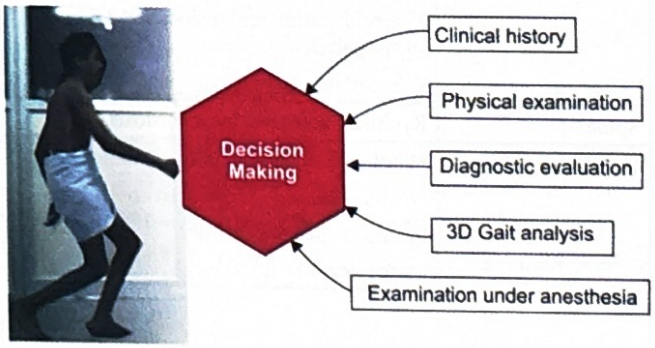


Fig 1.4.5: Plan for treatment

Management

Management of spasticity

- Pharmacologic
  - Baclofen
    - Routes: oral and intrathecal pump
    - Inhibiting signals through the GABA pathway
    - Side-effects: increased somnolence and decreased alertness during the day
  - Phenol intramuscular injection
  - Botulinum toxin A intramuscular injection
    - Presynaptic blockade of neuromuscular junction
    - Effective for 3 to 6 months; not a permanent cure of spasticity
  - Valium and clonazepam (oral)
  - Tizanidine and clonidine (oral)
  - Dantrolene (oral)
- Non-pharmacologic
  - Physiotherapy
  - Occupational therapy
  - Use of adaptive equipment and orthoses
  - Orthopaedic surgical procedures
  - Selective dorsal rhizotomy
    - Performed in ambulatory children with spastic diplegia to complement orthopaedic management
    - Can lead to spinal instability and deformity
    - Contraindicated in athetoid CP, non-ambulatory patients and spastic quadriplegia

Gait disorders [treatment explained under gait analysis graph]

Surgical management

- Goals of treatment for *ambulatory* cerebral palsy (GMFCS I–III)
  - Optimize gait efficiency (correct biomechanics) to optimize energy conservation
    - Preserve or improve physical function, e.g., walk longer distance, walk faster, decrease fatigue, better stability-reduced tripping and fewer falls, keep up with friends
    - Pain relief or pain prevention and increased endurance
    - Preserve or increase activities and

participation, e.g., more physically active, more independence, more participation in sports
    - Improve appearance of gait
        - Reduced reliance on walking aids/orthotics
        - Feet flat on the ground & pointing forward
        - Reduced dragging of feet
        - Stand and walk taller (knees less bent)
        - More symmetry
- Goals of treatment for *non-ambulatory* cerebral palsy (GMFCS IV–V)
    - Relieve or prevent pain and discomfort
    - Facilitate ease of care: Dressing, toileting, bathing/ hygiene; positioning: Seating and lying down; transfers and mobility
    - Preserve or improve health
    - Improve quality of life
- SEMLS vs. birthday syndrome
    - Correction of all fixed musculoskeletal deformities with Single Event Multi-Level Surgery (SEMLS)
    - SEMLS improves the likelihood of achieving sagittal plane balance
    - Reduces the need for repeated anesthetics
    - Reduces episodes of hospitalization
    - Only one major period of rehabilitation
- Principles of surgery
    - Types of surgery include release and lengthening of musculotendinous unit, tendon transfers, osteotomies, and arthrodesis
    - The concept of **"surgical dose"**: The surgical intervention should match the severity of dysfunction
    - **Multiarticular muscles** are more commonly contracted (e.g., psoas, rectus femoris, hamstrings, and gastrocnemii) than the monoarticular muscles.
        - Musculotendinous release or lengthening of monoarticular muscles must be avoided to preserve antigravity function and loss of stability in the postoperative period

Orthopaedic conditions in CP

Affected area	Condition or deformity
Foot and ankle	Equinus Equinovarus Calcaneous deformity, Valgus deformity of ankle
Lower extremities	Rotational deformities
Knee	Knee flexion contractures Congenital knee hyperextension or dislocation Knee extension contracture Knee instability / internal derangements Crouch gait Knee dislocation Genu varum/valgum/recurvatum Patellar subluxation and dislocation
Hips and pelvis	Abduction external rotation contracture Hip flexion deformity Hip subluxation and dislocation / wind-swept pelvis Pelvic obliquity
Spine	Kyphosis, scoliosis, hyperlordosis
Upper extremities	Shoulder contracture and instability Flexion contractures of wrist and fingers thumb-in-palm deformity Elbow flexion contracture

CHAPTER 1.5

Transient Synovitis of Hip

- Also called as
  1. Irritable hip
  2. Toxic synovitis of hip
  3. Irritable hip syndrome
  4. Coxitis fugax
  5. Acute transient epiphysitis.
- Was first described by Lorvett and Morse
- Most common age 16 1-10 years.
- Seen more common in boys than girls
- Most common cause of hip pain in children
- It is a self limiting condition.

Etiology:

1. Unknown
2. Sometimes preceded by respiratory infection.

Clinical features

1. Low grade temperature.
2. Child refuses to walk or bear weight
3. Preceded by Respiratory tract infection
4. Limb is in flexion and extension and internal rotation is painful.

Diagnosis:

1. It is disease of exclusion
2. Blood tests
   a. WBC, ESR, ERP } Increased
3. X-ray: Can show joint space widening
4. Ultrasound: This is the investigation of choice.

If joint space does not show fluid in joint, then should consider other diagnosis.

Differential Diagnosis

1. Septic arthritis:
   - Most common to be excluded
   - Has higher grade of fever and high ESR and CRD and WBC counts.
   - Restriction of movements is much severe compared to transient synovitis.
2. Juvenile arthritis
3. Osteomyelitis of femur
4. Psoas abscess (Pseudo flexion deformity)

Treatment

1. Rest
2. Medications: NSAIDs
3. Aspiration of hip joint.

CHAPTER | 1.6

Limb Length Discrepancy

Introduction

- Causes of Decreased Leg Length
  - Congenital limb deficiency
    - Congenital femoral deficiency
    - Tibial hemimelia / Fibular hemimelia
  - Asymmetric neurologic disorders
    - Hemimyelomeningocele
    - Poliomyelitis
    - Asymmetric static encephalopathy (e.g., hemiparesis)
    - Asymmetric peripheral neuropathy
  - Trauma
    - Malunion
    - Physeal growth disturbance or arrest after fracture
  - Other acquired causes of physeal growth disturbance
    - Infection
    - Tumor
      - Enchondroma/ Osteochondroma / Unicameral bone cyst
    - Irradiation
    - Blounts disease
    - Legg-Calvé-Perthes disease
  - Hemiatrophy
    - Idiopathic, nonsyndromic hemiatrophy
    - Russell-Silver syndrome
    - Unilateral clubfoot deformity
    - Congenital pseudarthrosis of the tibia
- Causes of Increased Leg Length
  - Posttraumatic overgrowth
    - Femoral shaft fracture / Tibial shaft fracture
  - Soft tissue overgrowth syndromes
    - Gigantism with neurofibromatosis
    - Klippel-Trénaunay syndrome
    - Beckwith-Wiedemann syndrome
    - Proteus syndrome
    - Idiopathic hemihypertrophy
  - Inflammatory arthritis

Problems with LLD

- Inefficient gait – increases energy expenditure
- Equinus contracture at ankle
- Scoliosis (postural)
- Low back pain
- Pelvic obliquity
  - Hip osteoarthritis/Hip uncovering of longer side

Assessment of LLD

- History
  - Congenital or developmental
  - To determine cause
  - To determine deformity
  - Onset and mode of progression (static or increasing)
- Examination
  - Spine examination
  - ASIS & GT levels
    - Bryants triangle / Nelatons line / Shoemakers line
  - Trendelenberg test
  - Galeazzi sign
  - True length vs. apparent length
    - Structural LLD – measurable difference in a lower extremity segment
    - Functional (Postural) LLD – asymmetry in positioning one lower extremity relative to the other
  - Wood block test (screening method)

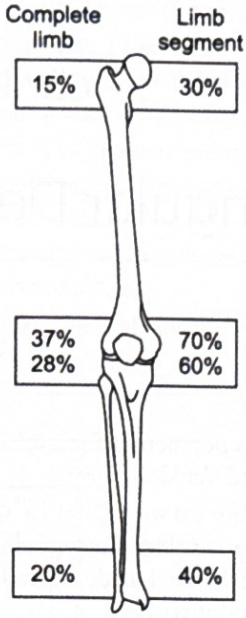


Fig 1.6.1: Contribution to lower limb growth and segmental growth

- Radiographs
  - Standing orthoradiographs
  - Scanogram
  - Orthoroentgenogram
  - CT Scanogram
- Prediction of remaining growth (to predict LLD at maturity)
  - Green-Anderson data
  - Moseley graph
  - Paley multiplier method
- A gross estimate of LLD can be made under the following assumption of the growth per year (up to age 16 years in boys; 14 years in girls)
  - Distal femur: 9 mm growth per year
  - Proximal tibia: 6 mm growth per year
  - Proximal femur: 3 mm growth per year

Treatment

- LLD < 2cm at maturity
  - Observation
  - Shoe lift
  - If symptoms persist, lengthening or shortening
- LLD 2-5 cm at maturity
  - Lengthening
    - Intramedullary
      - Albizzia nail
      - ISKD – intramedullary skeletal kinetic distractor
    - External
      - Unilateral fixator
      - Ring fixator
        - Ilizarov
        - Taylor spatial frame
    - Combined
      - Lengthening over nail
  - Shortening
    - Skeletally immature
      - Yes → Epiphysiodesis
        - Physeal drilling
        - Phemister technique
        - Growth modulation
          - Epiphyseal stapling (Blounts technique)
          - Percutaneous Epiphysiodesis using Transphyseal Screws (Métaizeau)
          - Tension plate epiphysiodesis
      - No → Acute shortening osteotomy
- LLD >5cm at maturity
  - Multiple lengthening procedure
  - Combination of lengthening and contralateral shortening