



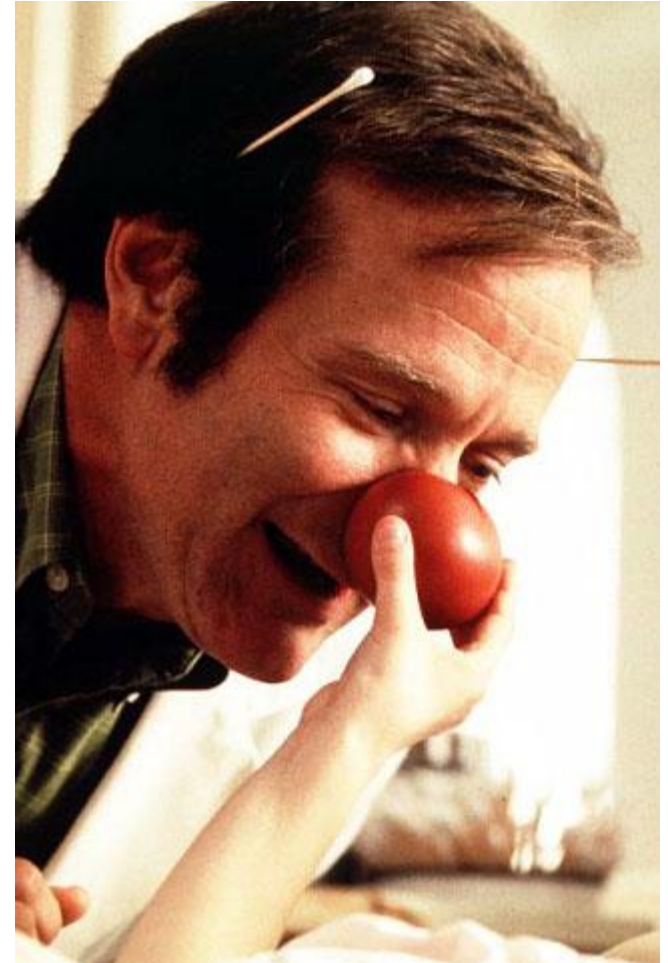
Paediatric Orthopaedics

Dr Ruan Goller



Children are not small adults!

- Physically and emotionally unique
- Specific pathologies
- Listen to parents
- Importance of “treating” the parents
- Children take time!





The Normal Child

- What is normal
- Physiological vs Pathological
- Causes of variations

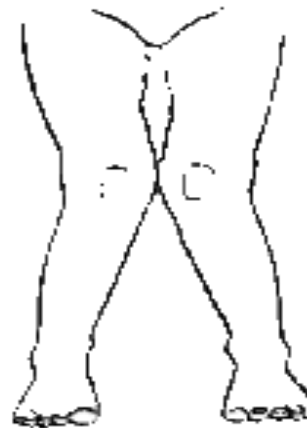




The Normal Child

Normal angular and rotational appearance:

- At Birth: Bowed legs
- Second Year: Straight legs
- Fourth year: Maximal genu valgum (up to 15°)
- Sixth year: Normal genu valgum (7°)





What is Normal??

It is important to differentiate between physiological and pathological:

Physiological	Pathological
<ul style="list-style-type: none">■ Symmetrical■ Mild-Moderate■ Regressive (self-limiting)■ Generalised (eg bow legs)■ Age appropriate	<ul style="list-style-type: none">■ Asymmetrical■ Severe■ Progressive (requires treatment)■ Localised (eg Blounts)■ Not age appropriate



What is Normal??

Symmetrical



Asymmetrical



Generalised



Localised





What is Normal??

Causes of variations:

Physiological	Pathological
<ul style="list-style-type: none">■ Normal■ Worsened by:<ul style="list-style-type: none">- overweight- early weight-bearing- baby-walker	<ul style="list-style-type: none">■ Rickets■ Endocrine disorders■ Metabolic disease■ Injury to epiphysis end plate (infection/trauma)■ Idiopathic



Evaluation

As with any orthopedic complication associated with child growth and development, the key to optimal growth potential lies in the early diagnosis and treatment of conditions. While many variations are not of significant concern, any abnormalities should be closely monitored by parents and where necessary referred to a healthcare professional that specializes in childhood orthopedic disorders.



Evaluation

- History:
 - Onset
 - Severity
 - Progression
 - Developmental history
 - Family history
 - Treatment history
 - Disability
- Blood workup
- Radiological:
 - Measure angles
 - Look for pathology





Evaluation

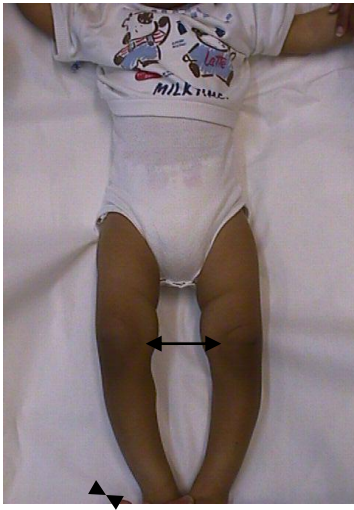
- Angular Profile:
 - Intercondylar distance (bow legs/genu varum)
 - Intermalleolar distance (knock knees/genu valgum)
- Rotational Profile:
 - Foot Progression Angle
 - Femoral version (Ryder's angle)
 - Tibial torsion (Transmalleolar angle)
 - Foot Assessment
 - Thigh foot angle

Many angular deformities are
Associated with rotational deformities
eg bow legs with internal tibial torsion





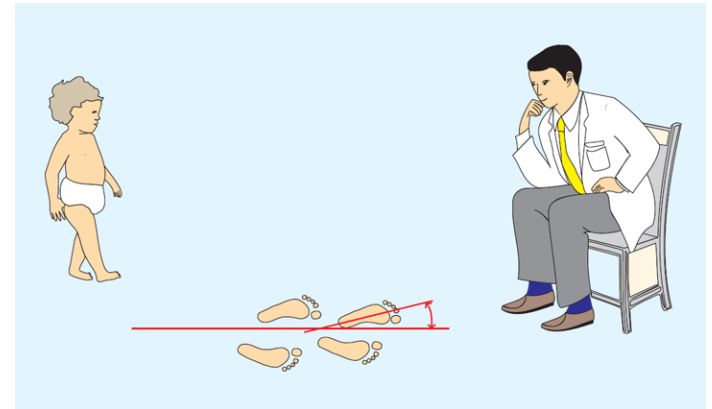
Evaluation



Inter-condylar distance



Inter-malleolar distance



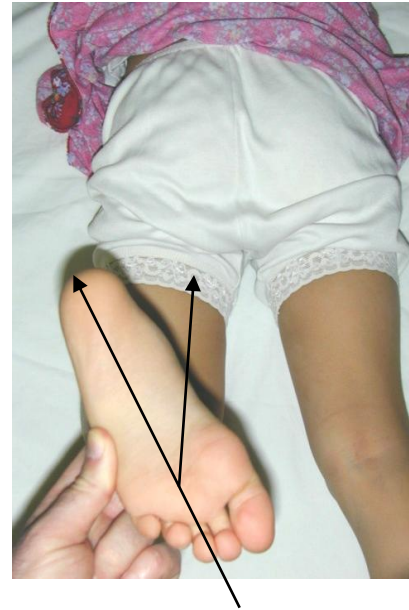
Foot Progression Angle



Evaluation



Trans-Malleolar Axis

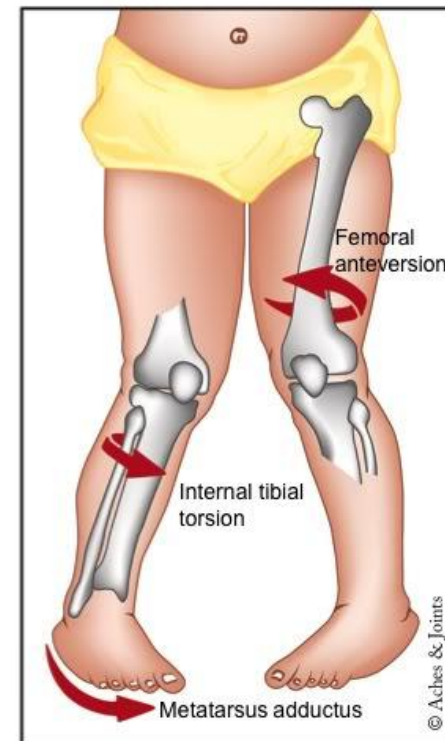


Thigh-Foot Angle



Intoeing

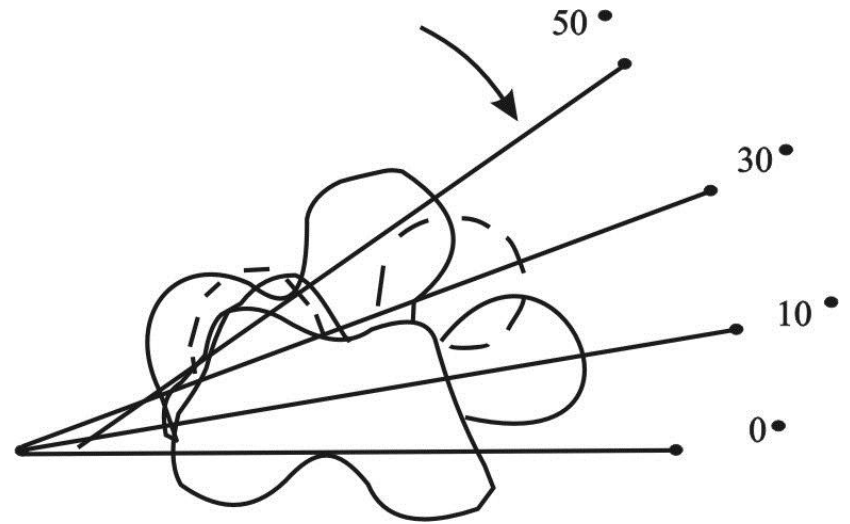
- Rotational profiles change during childhood, but vary widely among healthy children of the same age.
- Causes:
 - Femoral Anteversion
 - Tibial Torsion
 - Metatarsus Adductus





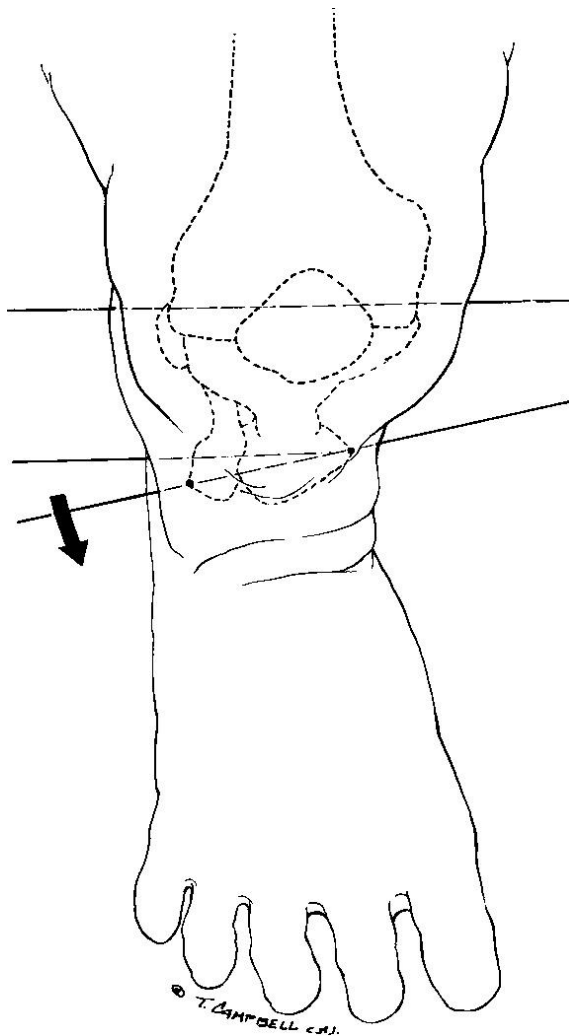
Femoral Anteversion

- Intoeing with medially rotated patella's= femoral anteversion
- Prevalent cause of intoeing 3-6 years
- At birth: normal 30°-40°
- At skeletal maturity: 15°
- Girls:boys 2:1, and often hereditary
- Not directly correlated to degenerative changes in hip and knee
- M/W sitting
- Can cause P/F pain





Tibial Torsion

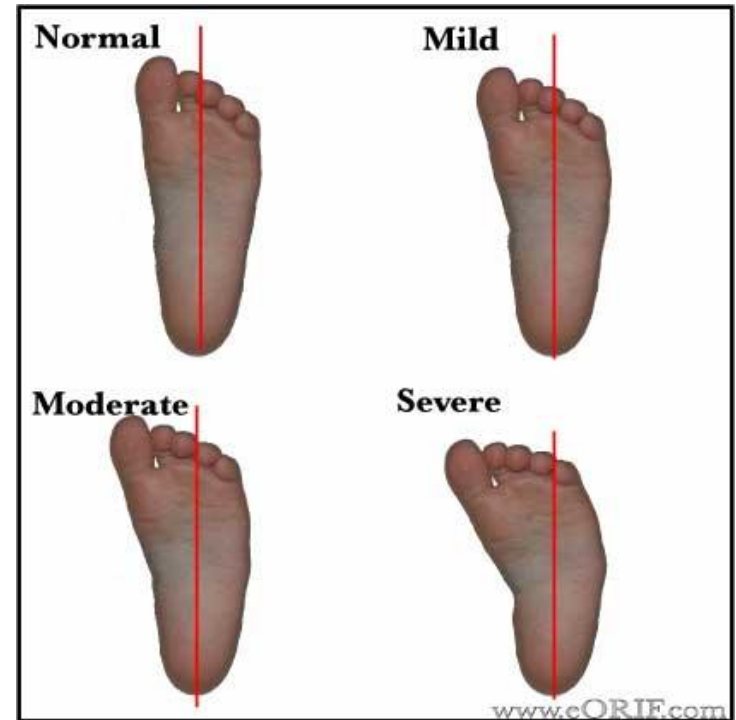


- Most evident 1-2 years
- Usually resolves by 6 years
- Measured as angle between:
 - Bimalleolar axis (ankle)
 - Bicondylar axis (knee)
- Normal: 20° ext rotation



Metatarsus Adductus

- “kidney” shaped foot
- Medial deviation of forefoot
- Normal alignment of hindfoot
- 12% of newborns
- ? Due to intrauterine position
- Assess active correction by tickling foot





Management of Deformities

- Femoral Anteversion:
 - Generally: Monitor progression
 - *Only* were indicated: Derotational osteotomy
- Tibial Torsion:
 - Generally: Monitor progression
 - *Only* were indicated: Derotational osteotomy
- Metatarsus Adductus:
 - Generally: Monitor progression (95% correct by age 5)
 - Mild deformity: stretches
 - Moderate deformity :serial casting
 - *Only* were indicated: incl. open wedge medial osteotomy and closed wedge lateral osteotomy



Management of Deformities

Most angular and rotational deformities
DO NOT and SHOULD NOT require treatment





Specific Joint Pathologies

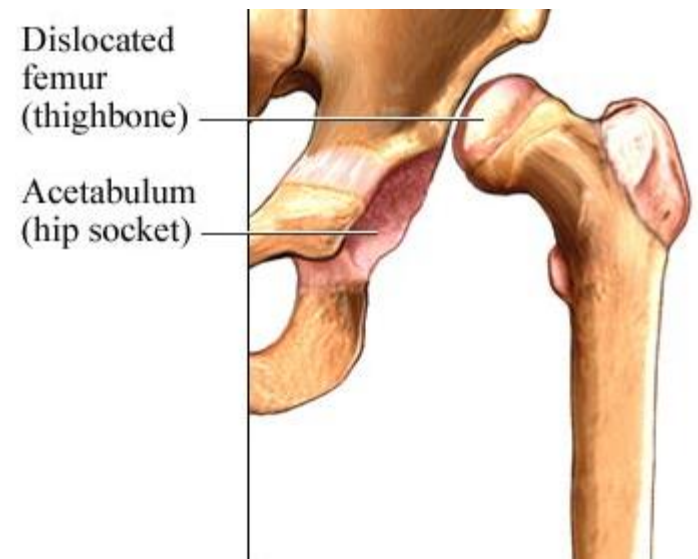
- Hip
 - DDH
 - SUFE
 - Perthes
- Knee
 - Blounts
- Feet
 - Clubfoot
 - Flat foot





Developmental Dysplasia of the hip (DDH)

- 1-2/1000 births
- Hip at risk
 - Family history (10x)
 - Breech presentation (10x)
 - Female
 - First born
 - Other packaging problems
 - Torticollis
 - Genu recurvatum
 - Other congenital abnormalities
 - Syndromic child, spina bifida



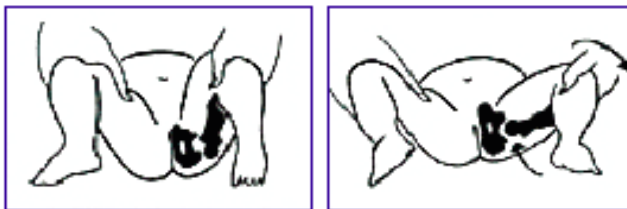


Presentation

- Early/Neonatal period
 - Decreased abduction
 - Asymmetrical skin folds
 - Ortolani test + (Barlow test +)



Barlow Test



Ortolani Test

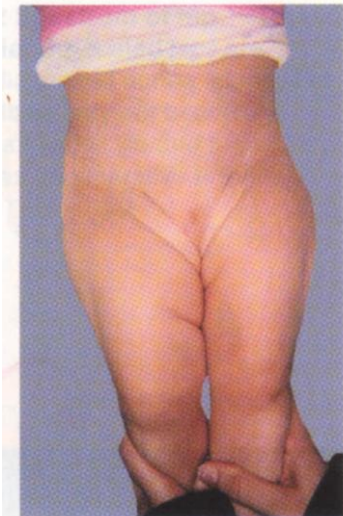
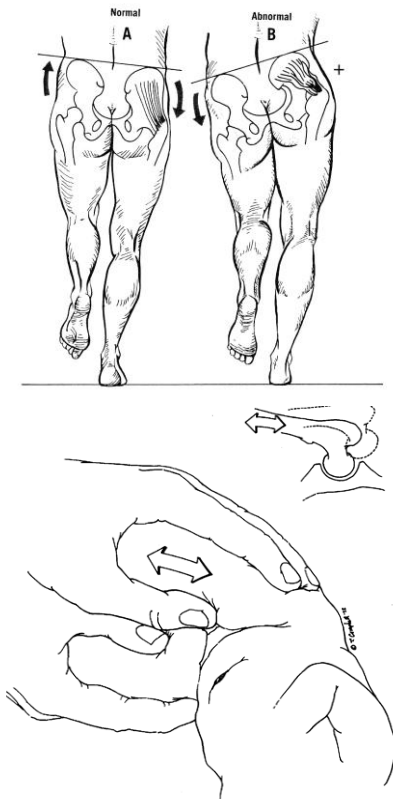


Fig. 7.46 Asymmetrical thigh folds. These occur in up to 20% of normal infants.



Presentation

- Late: after 6 months
 - Limited abduction but excessive internal and external rotation
 - Late walker
 - Leg length discrepancy
 - Telescoping of leg
 - Trendellenburg gait and sign
 - Limping child





Investigations

- **Ultrasound**

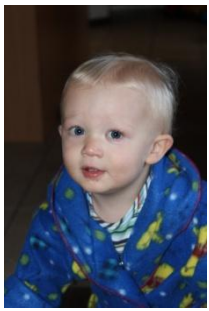
Best modality for confirming DDH in a child of < 3months. Should perhaps be used for selective screening of high risk groups.

Otherwise overdiagnosis.

Wait till 6 weeks if abnormal u/s but normal clinically at birth. Most will improve.

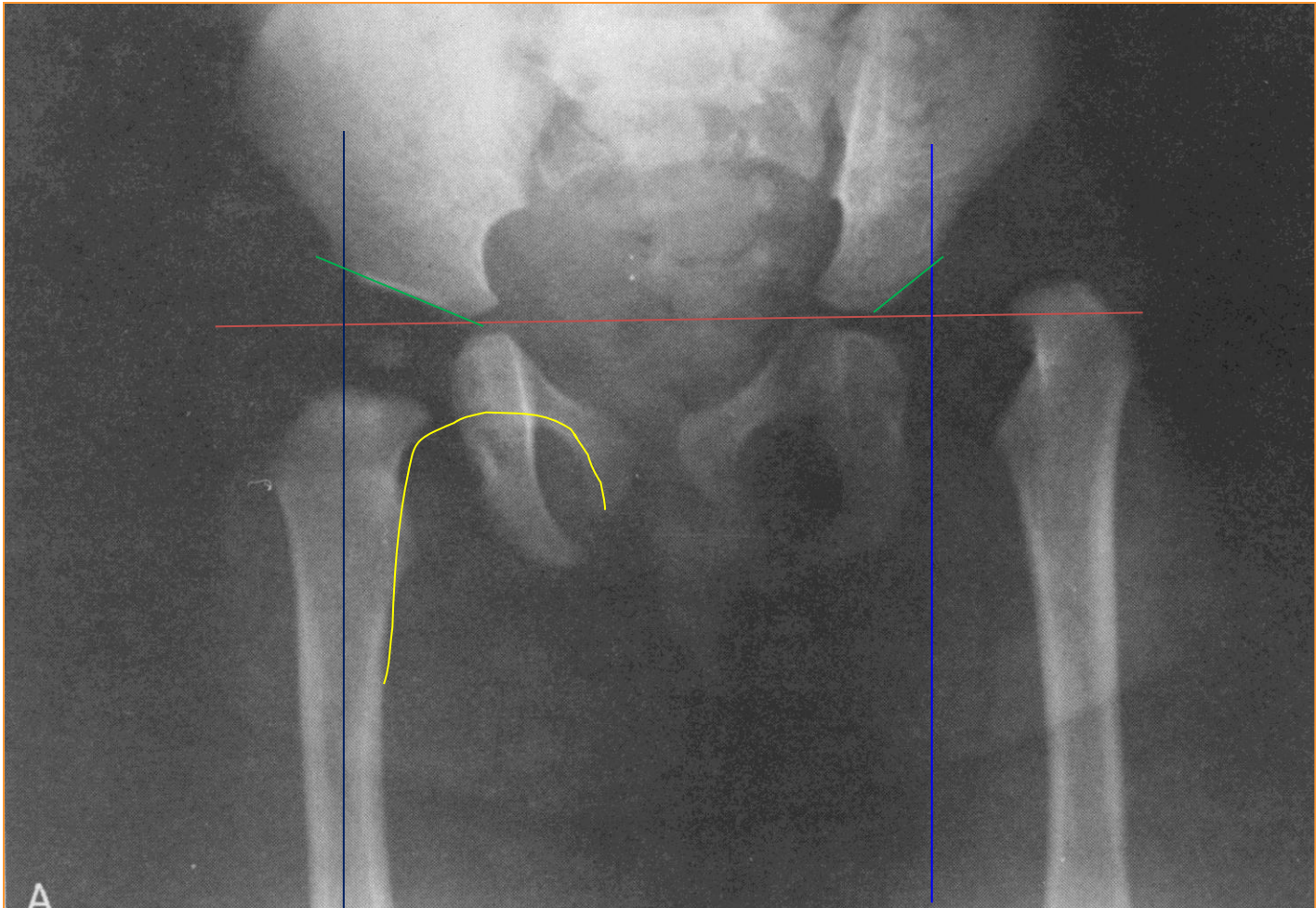
Important in follow up of patients undergoing Rx.





Investigations

- X rays





Management

- Birth to 6 months
 - Pavlik harness



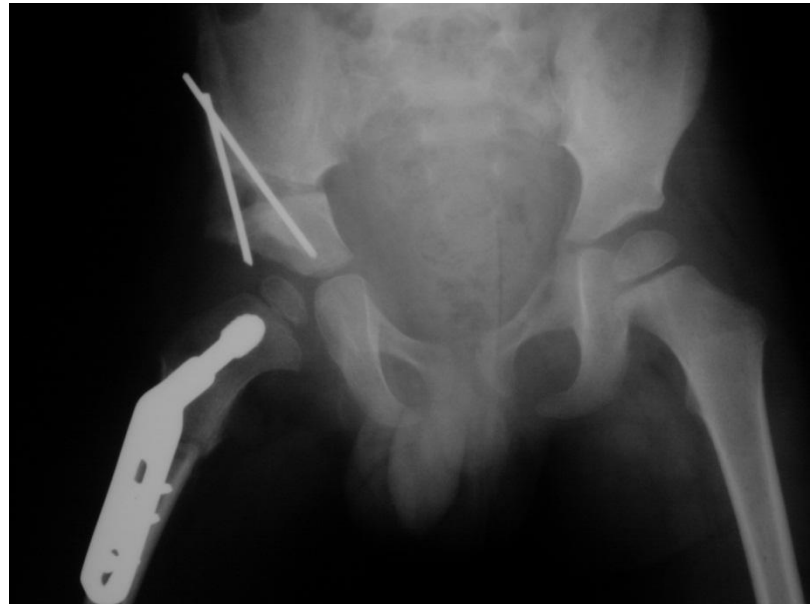
Management

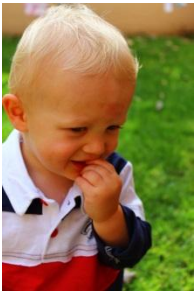
- 6-18 months
 - Closed reduction and spica



Management

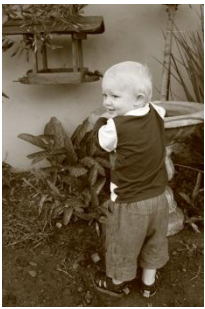
- Older
 - Open reduction +- femoral +- pelvic osteotomy





Slipped Upper Femoral Epiphysis (SUFE)

- Misnomer because epiphysis is held in acetabulum and metaphysis moves upward and outward
- Etiology
 - Mechanical factors
 - Obesity
 - Increased femoral retroversion
 - Increased physeal inclination
 - Deeper acetabulum
 - Greater coverage leads to more shear stress across physis



Slipped Upper Femoral Epiphysis (SUFE)

- Etiology

- Biochemical factors

- Disease of puberty

- Estrogen reduces physeal width and increases strength
 - Testosterone reduces strength

- Hypothyroidism

- Hypogonadism

- Growth hormone supplementation

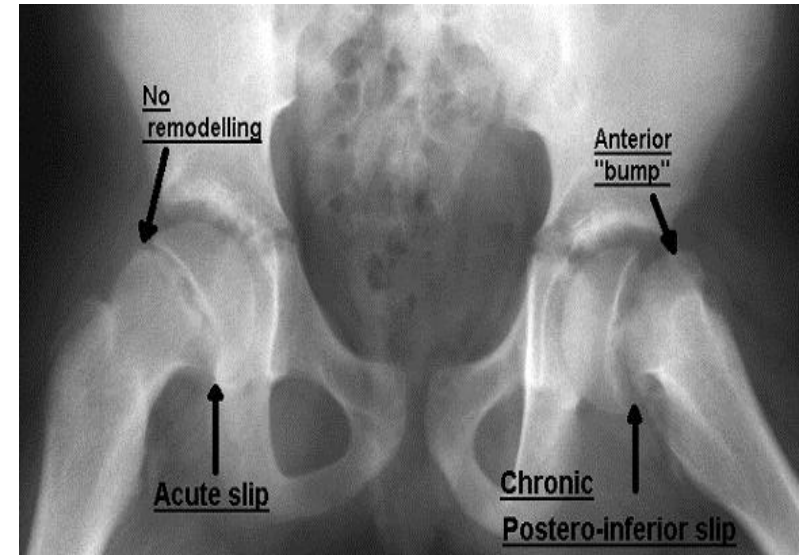
- SUFE in children <10 years and >16 years is almost always associated with endocrinopathy





Epidemiology and Clinical Presentation

- Male predominance
- Typical age 9-16 years
 - Boys average 13.5 years
 - Girls average 12.0 years
- Bilaterality up to 63%
- Acute slip may mimic child with hip fracture
- Chronic slip
 - LLD and antalgic gait
 - Decreased internal rotation
 - Leg held in external rotation
 - With flexion hip goes into abduction and external rotation
 - 46% presents with pain in the knee or distal part of thigh





Classification

- Loder's classification
 - Stable slip
 - Able to walk with or without crutches
 - AVN up to 50%
 - Unstable slip
 - Cannot walk with or without crutches
 - AVN 0%



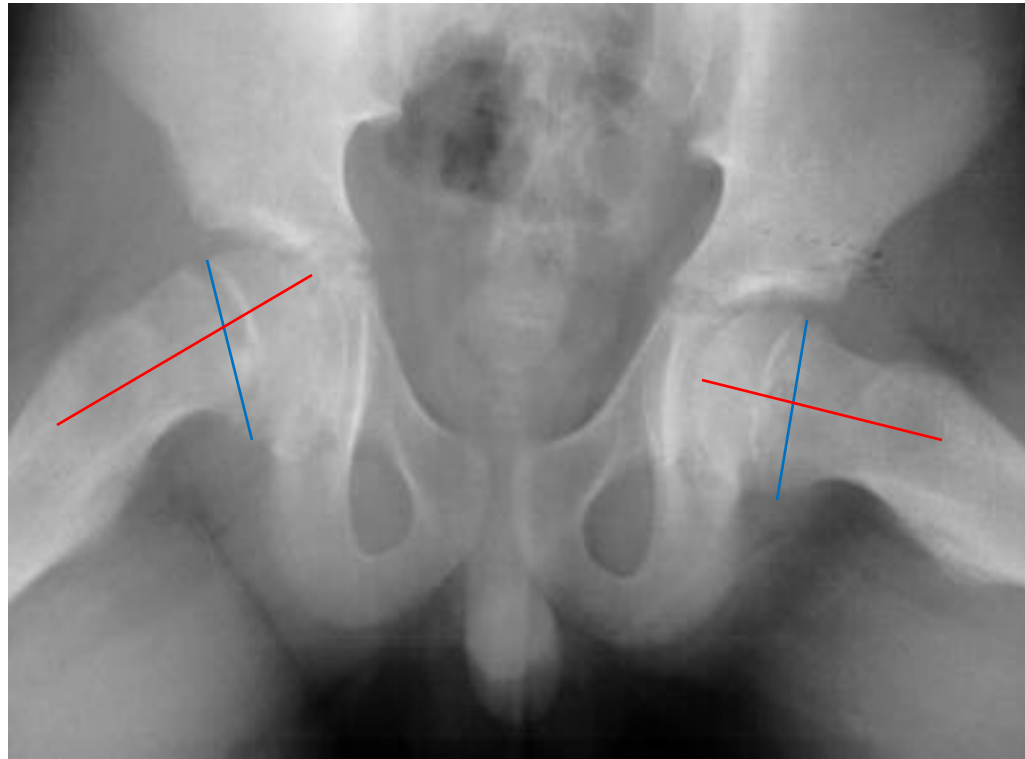
Radiography

- AP Pelvis



Radiography

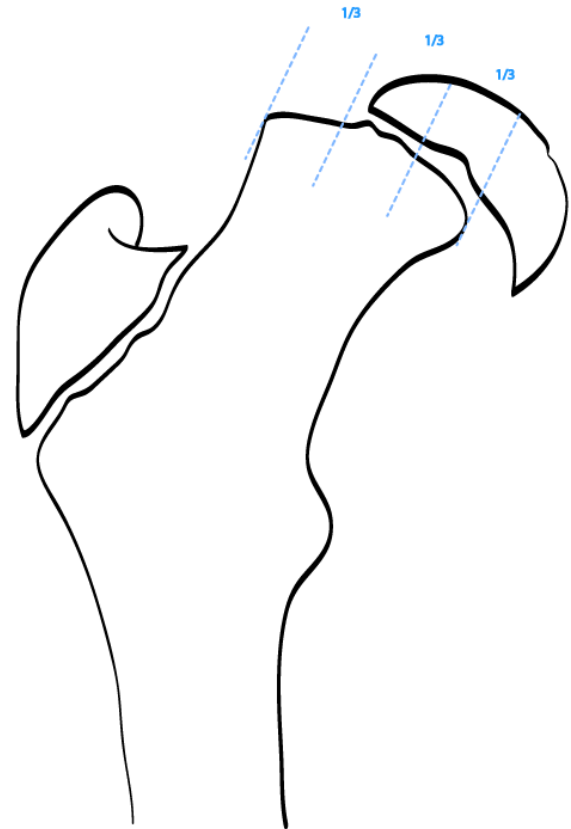
- Lateral X ray





Grading

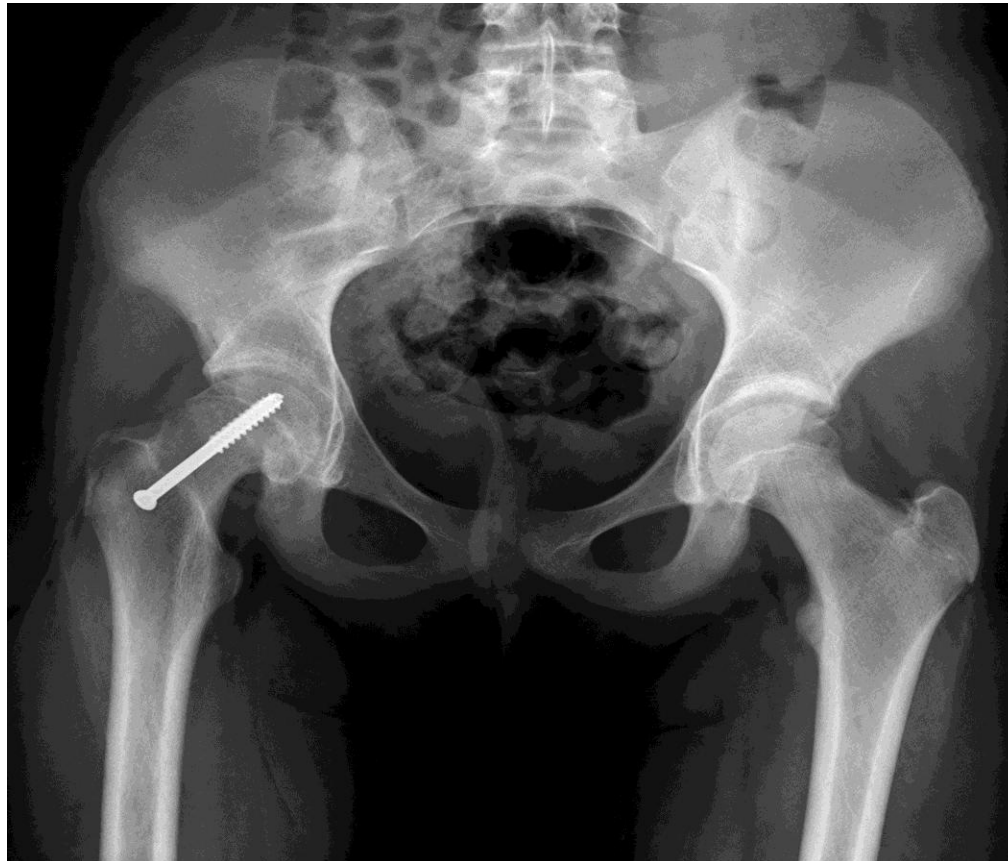
- Amount of displacement of epiphysis on metaphysis
 - Mild $< 30\%$
 - Moderate 30-50%
 - Severe $>50\%$





Treatment

- In situ stabilization with cannulated screw





Treatment

- Very rarely for the severe slip we do a open reduction and femoral neck osteotomy





Treatment

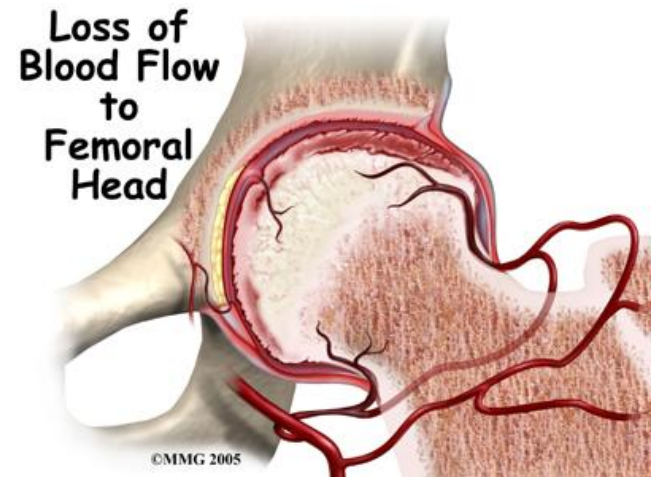
- Residual deformity can be corrected with flexion-internal rotation subtrochanteric osteotomy





Legg-Calve-Perthe's Disease

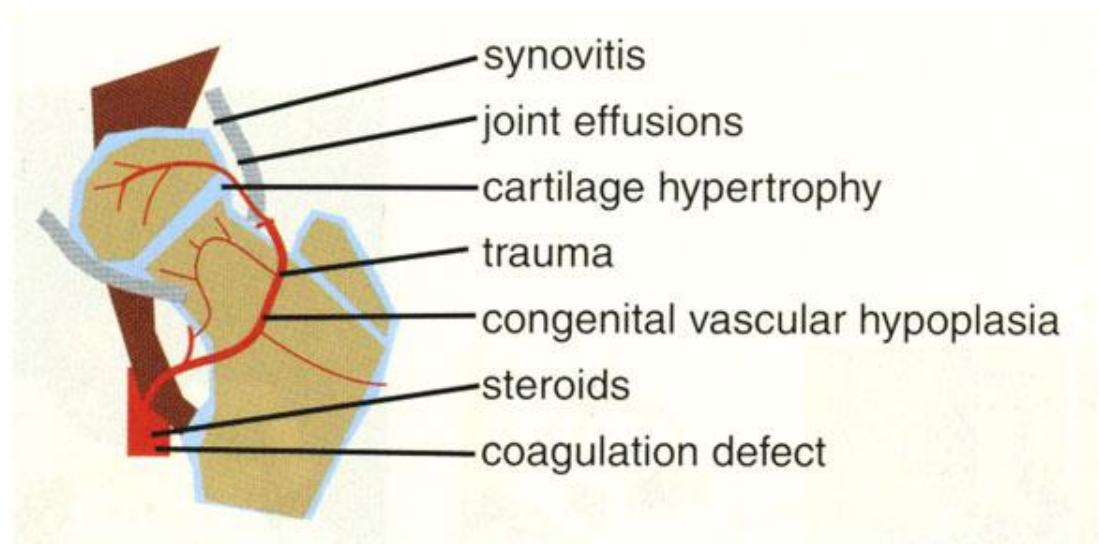
- 100 years old
- Osteonecrosis of femoral epiphysis
- 1.5-4 per 100 000
- Commonly between 5-8 years
- Male predominance 5:1
- Bilaterality 10-15%





Etiology

- Unknown cause
 - Coagulopathy
 - Highly active children (ADHD)
 - Small for age
 - Trauma (Micro and Macro)
 - Synovitis



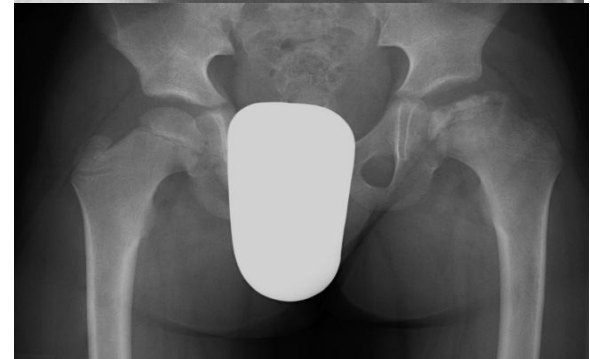


Clinical presentation

- Limp
- Pain
 - May refer to knee, medial thigh
- Decreased abduction and internal rotation
- Quad atrophy
- LLD
- Out toeing
- Trendellenburg gait and sign

Radiology

- 1st phase
 - Smaller ossification femoral head
 - Increased joint space
- 2nd Phase
 - Subchondral fracture (best seen on lateral)
- 3rd Phase
 - Increased density
 - Collapse
 - Calsification of necrotic areas
 - Reossification
- 4th Phase
 - Areas of rarefaction
 - Widening of neck



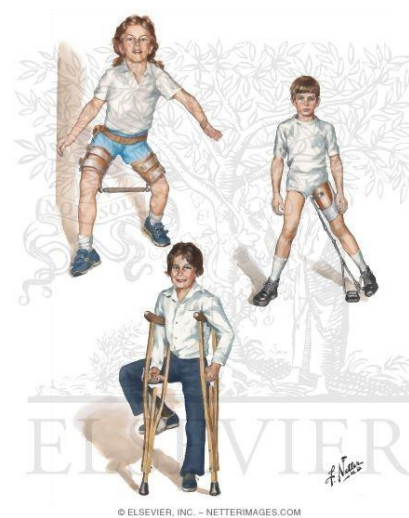
Treatment

- Hip at risk signs
 - Lateral extrusion of femoral head.
 - Calcification lateral to femoral head..
 - Diffuse metaphyseal rarefaction.
 - Horizontal growth plate.

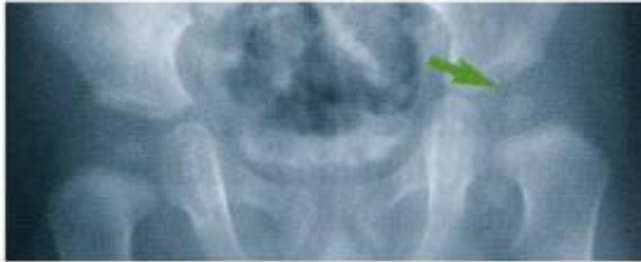


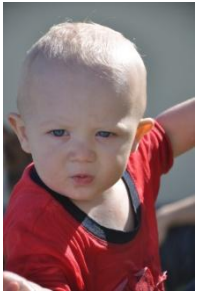
Treatment

- 60% will do well without aggressive treatment
- Nonsurgical treatment
 - Decrease hip irritation with bedrest and traction
 - Several orthotic devices described but not really used



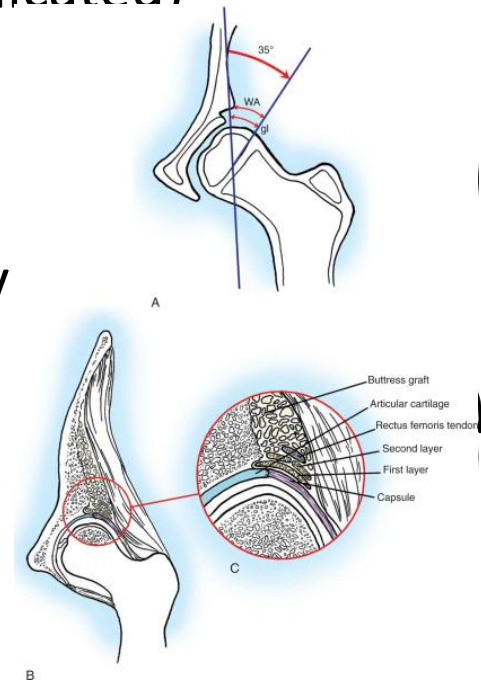
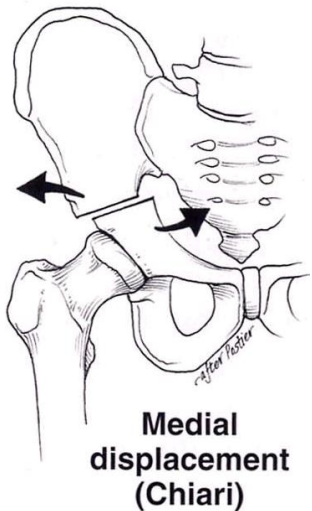
Treatment





Treatment

- Surgical options
 - Femoral osteotomies
 - Varus osteotomy
 - Valgus osteotomy (rarely indicated)
 - Pelvic osteotomies
 - Salter osteotomy
 - Staheli shelf acetabuloplasty
 - Chiari osteotomy





Treatment Protocol

- Patients <6 years
 - Conservative or symptomatic treatment
- Patients 6-9 years
 - Conservative or symptomatic treatment
 - More severe -> femoral osteotomy
- Patients >9 years
 - Conservative or symptomatic treatment
 - More severe -> pelvic osteotomy

Transient Synovitis

- Sterile inflammation of the hip
- Diagnosis made by exclusion of septic arthritis
- Presents with groin pain, refusal to walk
- Little or no fever
- Infective markers normal
- Restricted internal rotation of hip
- Hip normally improves within a few days



Summary: Hip Conditions

	DDH	SUFE	PERTHES
Age	From Birth	Puberty	5-8 years
Male Female	Female > male	Male > Female	Male > Female
Risk Factors/ patient Charac- teristics	Family History Breech Presentation First Born Other congenital abnormalities	Obesity ↑femoral retroversion ↑physeal inclination Deeper acetabulum Endocrinology (age 1-16)	Active children Small for age Trauma



Summary: Hip Conditions

	DDH	SUFE	PERTHES
Presentation	<p>Early:</p> <ul style="list-style-type: none"> -↓ Abduction -Asymmetrical skin fold -Ortolani test + -Barlow test + <p>Late:</p> <ul style="list-style-type: none"> -↓ Abduction -↑ Int and Ext rot -Late walker -LLD -Trendellenburg 	<p>Acute:</p> <ul style="list-style-type: none"> -Mimic hip # <p>Chronic:</p> <ul style="list-style-type: none"> -Antalgic gait -LLD -With hip flex → abd ext rotation -46% knee and thigh pain 	<ul style="list-style-type: none"> -↓ Abduction internal rotation -LLD -Quads atrophy -Trendellenburg -Pain referred to knee -Out-toeing



Blount's disease

- Growth disturbance of medial part of proximal tibial physis
- Acute genu varum with internal rotation
- Divided into
 - Infantile
 - <3 years
 - Usually bilateral
 - Late onset
 - Juvenile 4-10 years
 - Adolescent >11 years





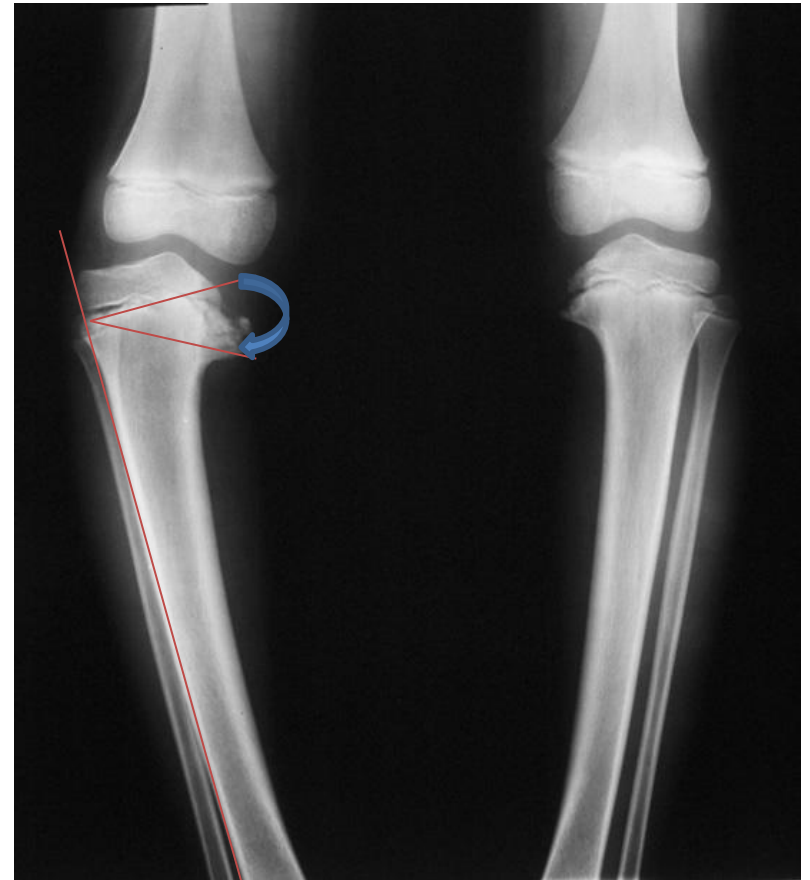
Evaluation

- Suggestive of pathological bowing
 - Proximal tibial location of bowing
 - Sharply angular deformity
 - Asymmetric bowing of legs
 - Progressive deformity
 - Lateral thrust during gait
 - Severe deformity



Radiography

- Standing AP X rays with patellae facing forward
- Metaphyseal-diaphyseal angle
 - $<10^{\circ}$ -> 95% will resolve
 - $>16^{\circ}$ -> 95% will progress





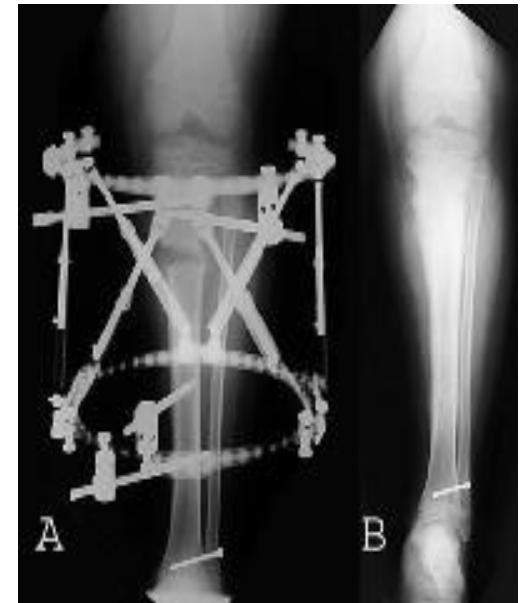
Treatment

- Nonsurgical
 - Bracing efficacy is controversial
 - Only indicated for pts 2-3 years with mild disease
 - Improvement should occur within 1 year



Treatment

- Surgical
 - Proximal tibial osteotomy





Clubfoot

Complex, congenital, contractural malalignment of the bones and joints of the foot and ankle

Classification:

1. Congenital
2. Neurogenic
3. Syndrome complex
4. Positional





Clubfoot

- White population 1.2 per 1000
- Black population 305 per 1000
- Male: female 2:1
- 50% bilateral
- Hereditary
- Many etiological factors

Clubfoot

- Deformity of foot is fixed. Cannot be corrected by manipulation into dorsiflexion in eversion.
- The calf muscles may have the appearance of underdevelopment.
- In unilateral cases the affected foot is shorter.
- The foot is turned inwards so that the sole is directed medially.
- The hindfoot is in plantarflexion (equines) and varus and the heel may be small and situated high against the leg.
- A positive calcaneocuboid angle is present (as viewed from the plantar aspect of the foot)





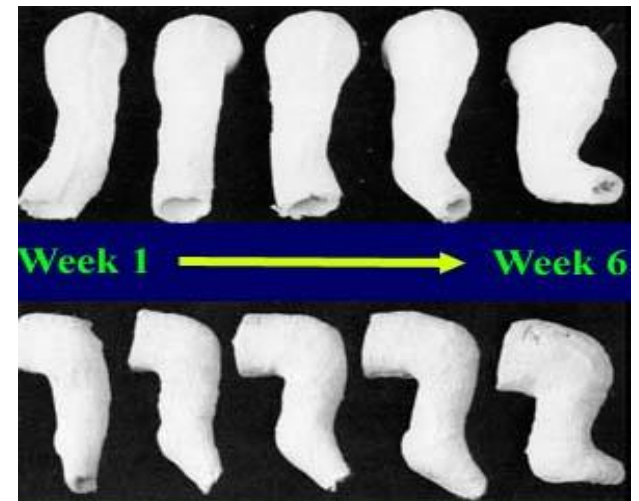
Clubfoot

- Foot position: “CAVE”
 - Cavus (high arch)
 - Adduction (Midfoot)
 - Varus (hindfoot rotated inwards)
 - Equinus (Foot plantar flexed)



Management

- Goals of treatment
 - Plantigrade, painfree, functional foot
 - Cosmetically acceptable, good mobility, no specialised footwear or orthosis
- Main treatment method
 - Ponseti serial casting
 - Casts changed at weekly intervals



Management

- Total number of casts varies from 4 to 10
- After successful serial casting the child is placed in “boots and bar” splint for 10 weeks followed by night and nap times until 4 years of age



Management

- If more than 10 casts or foot not improving -> conservative treatment has failed
- Surgery then indicated followed by special shoes (tarso-pronator type shoes)
- Surgical management:
 - Posterior release
 - Medial release
 - Lateral release



Management

- Surgical management:
 - Posterior release
 - Medial release
 - Lateral release





Flat feet

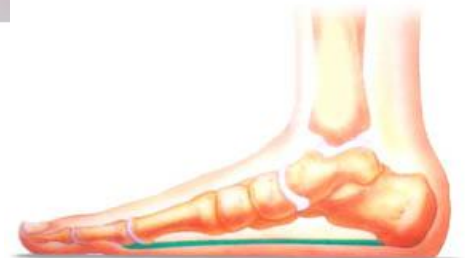
- Physiological flat feet:
 - Flexible flatfoot
 - Normal development (3-5 years old)
 - Generalised laxity
 - Large fat pad medially



In this child with a flexible flatfoot, the longitudinal arch that is normally seen is absent.



Normal Arch



Flat Arch

- Evaluation:
 - Evident arch in toe-standing and heel moves into varus, d/f the big toe (Jack's test) and NWB
 - Full painless subtalar movement





Flat feet

- Treatment:
 - Asymptomatic: no treatment
 - Non-surgical:
 - Shoes and orthoses no benefit to arch development, but can relieve pain
 - Stretches if tight TA
 - Surgical:
 - Failed conservative treatment
 - Calcaneal neck lengthening with soft tissue balancing (preserves motion and growth)

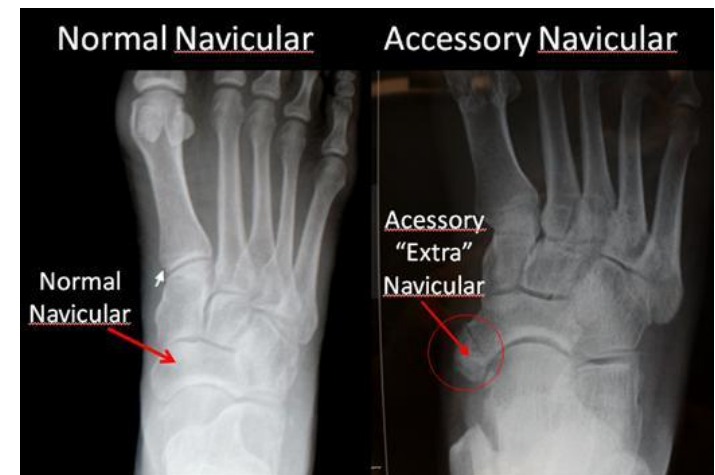


Flat feet

- Pathological flat feet:
 - Talipes calcaneovalgus
 - Congenital vertical talus
 - Tarsal coalitions
 - Hypermobile flatfoot with tight TA
 - Neurogenic
 - Accessory navicular bone



Talipes calcaneovalgus



Cavus feet

- CONGENITAL
- FAMILIAL
- IDIOPATHIC
- NEUROMUSCULAR DISEASE
 - MUSCLE
 - Muscular dystrophy e.g., Duchenne
 - NEUROLOGICAL
 - Peripheral nerve
 - Polyneuritis
 - Charcot-Marie-Tooth
 - Ant. Horn cells
 - Polio
 - Myelomeningocele
 - Diastematomyelia
 - Cord tracts
 - Friedereichs' ataxia
 - Brain
 - Cerebral palsy
- TRAUMA
- BURNS
- IRRADIATION.



Cavus feet

- High medial border (1st metatarsal in plantarflexion)
- Heel in varus

Cavus feet

- Always investigate further, especially in unilateral cavus foot.
- ? + Family history
- Full neurological examination
- Spinal X-rays
 - Spinal dysraphism
 - Diastematomyelia
- Myelography
 - To rule out space occupying lesion

Cavus feet

- **Management**

- Refer to orthopaedic surgeon for investigation and treatment.

- **Surgery**

- Indication varies with the cause, age and severity of the problem.

- Soft tissue procedures

- Osteotomies

- Arthrodesis

IMPORTANT NEUROLOGICAL DISEASES

- BRACHIAL PLEXUS INJURIES
- CEREBRAL PALSY
- MYELOMENINGOCELE
- POLIOMYELITIS
- MUSCLE DYSTROPHY

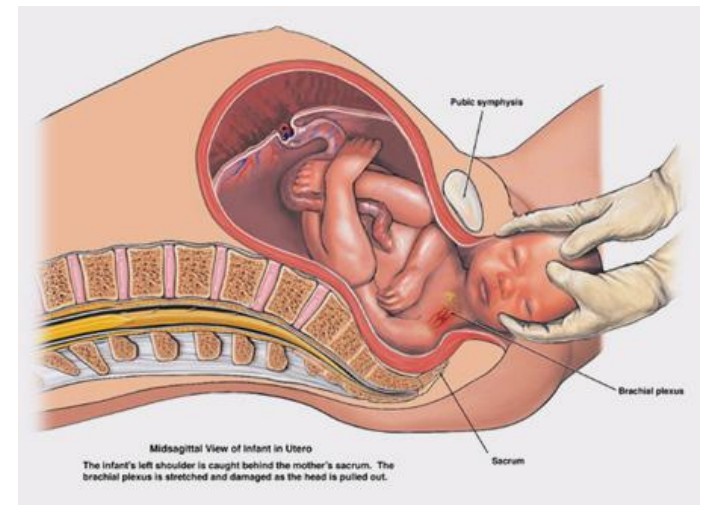
Brachial Plexus Injury

- Upper arm involvement = Erb-Duchenne type.
- Whole arm involvement type.
- Lower arm involvement type = Klumpke type.



Brachial Plexus Injury

- UPPER ARM INVOLVEMENT
 - Cephalopelvic disproportion
 - Impaction of the head with traction to the shoulders causes stretching of the plexus. Often upper part of plexus at ERB'S point (C5 and C6 roots) (ERB'S paralysis).
 - Shoulder and elbow affected. Adduction / medial rotation position. Waiters tip arm.
 - Absence of Moro reflex.



Brachial Plexus Injury

- Diff DX of septic arthritis with pseudo paralysis.
- Early diagnosis essential.
- Document neurological deficit and repeat exam regularly. Ensure mobility of both shoulder and elbow joints: Abduction / external rotation of shoulders 10 X with each nappy change.
- Most pts have fair prognosis.
- Bad prognosis if deltoid and biceps function not visible at 3 months.
- Surgery considered if at 3 months no response: neurolysis or nerve grafts.

Brachial Plexus Injury

- **WHOLE ARM INVOLVEMENT**
 - All 3 cords involved.

Brachial Plexus Injury

- LOWER ARM INVOLVEMENT:
 - Lower 2 cords involved with paralysis of intrinsic muscles of hand and Horner's syndrome. No grip reflex. Intact wrist and finger flexors. Involvement of Cervical sympathetic nerves in 1st dorsal (thoracic) roots → causes ipsilateral Horner syndrome with enophthalmos, myosis and ptosis
 - Difficulties with delivery can also cause hypoxia and cerebral damage presenting as CP.
 - Prognosis: Complete recovery in 80-95% of cases within 3 months.
 - Prevention of contractures crucial in management.

Cerebral Palsy

- **Definition**

- The term refers to a handicap in children, caused by a number of clinical disorders, with one feature in common namely that of a non-progressive primary lesion in the brain.
- Expression of the disease is progressive due to spasticity and contractures
- Except for motor disorders they may also have disorders of vision, hearing, speech and intellectual function.
- Incidence of 1-3 per 1000 live births

Cerebral Palsy

Prenatal factors

- Genetic
- Familial forms e.g. spastic paraplegia and tremor.
- Complications of pregnancy
 - Haemorrhage from placenta causing anoxia of the foetus.
 - Maternal toxemia
 - Maternal infection and trauma
 - Kernicterus
 - Irradiation (?)

Cerebral Palsy

Perinatal factors

- ANOXIA IS THE MOST COMMON CAUSE OF CP
- Increased risk with physical pressure e.g. abnormal position, disproportion, instruments during delivery. Often associated intracranial bleeding.
- Prematurity-Anoxia probably causative factor.
- Jaundice -Bilirubin damages the basal ganglia.
- Purulent meningitis and hydrocephalus

Postnatal factors

- Any brain injury after birth e.g. trauma, meningitis, encephalitis etc. may be followed by cerebral palsy. Remember that brain tumours and cysts may also cause cerebral palsy

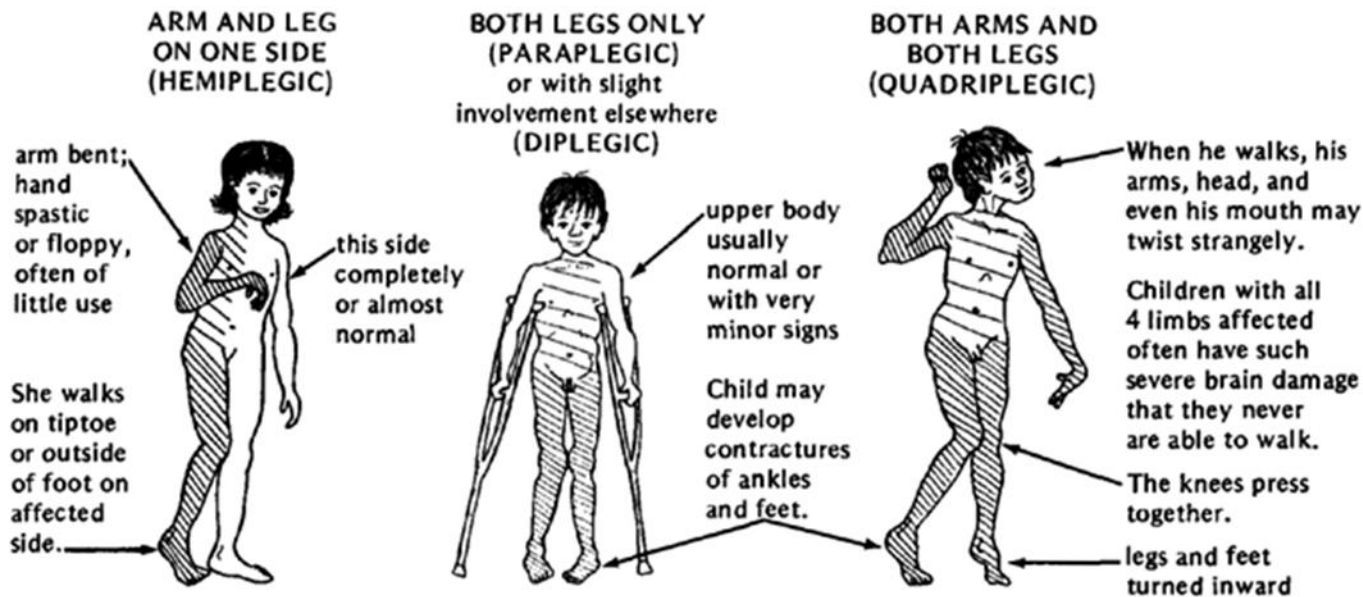
Types of Cerebral Palsy

- **Spastic**
 - Lesion in pyramidal system of cerebral motor cortex.
 - Signs: Hypertonus (mostly in flexors), Increased deep tendon reflexes, Clonus at knee or ankle, Extensor plantar response.
- **Athetoid**
 - Damage to basal ganglion. Exchange blood transfusions prevents this complication (Rh incompatibility)
 - Involuntary uncontrolled movements of muscles in limbs and face.
 - Difficulty with speech.
 - Hypertonus varying degrees.
 - Normal deep tendon reflexes.
 - Intelligence well preserved.

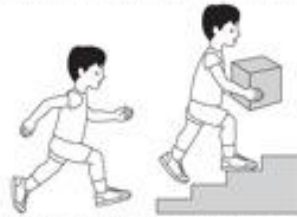
Types of Cerebral Palsy

- **Ataxic**
 - Lesion in cerebellum and brain stem.
 - Disturbed balance
 - Nystagmus
 - Hypotonia
 - Tremor
- **Rigid**
 - Lesion in cortex and basal ganglia
 - Lead pipe rigidity or cogwheel (interrupted) rigidity.
 - Mental retardation often severe.
 - Alone or combined with other types.
- **Floppy infant**
 - A few hypotonic or floppy infants turn out to be cerebral palsied children

Types of Cerebral Palsy



GMFCS for children aged 6-12 years: Descriptors and illustrations



GMFCS Level I

Children walk indoors and outdoors and climb stairs without limitation. Children perform gross motor skills including running and jumping, but speed, balance and coordination are impaired.



GMFCS Level II

Children walk indoors and outdoors and climb stairs holding onto a railing but experience limitations walking on uneven surfaces and inclines and walking in crowds or confined spaces and with long distances.



GMFCS Level III

Children walk indoors or outdoors on a level surface with an assistive mobility device and may climb stairs holding onto a railing. Children may use wheelchair mobility when traveling for long distances or outdoors on uneven terrain.



GMFCS Level IV

Children use methods of mobility that usually require adult assistance. They may continue to walk for short distances with physical assistance at home but rely more on wheeled mobility (pushed by an adult or operate a powered chair) outdoors, at school and in the community.



GMFCS Level V

Physical impairment restricts voluntary control of movement and the ability to maintain antigravity head and trunk postures. All areas of motor function are limited. Children have no means of independent mobility and are transported by an adult.

Cerebral Palsy

- **Diagnosis:**
- The more severe the earlier the diagnosis is made. In mild cases the diagnosis is often made after the child has been walking for several years.
- History
 - Perinatal (convulsions, jaundice etc.).
 - Positive predictors for walking
 - Sitting by 2 years
 - Pulling to stand by 2 years
 - Poor prognostic indicators for walking
 - Persistence of 2 or more primitive reflexes at 1 yo (Moro)
 - Not sitting by 5 yo
 - Not walking by 8 yo

Cerebral Palsy

- **Foot:**

- Equines deformity.
- With or without varus deformity
- Surgery, namely lengthening of tendon Achilles sometimes indicated.



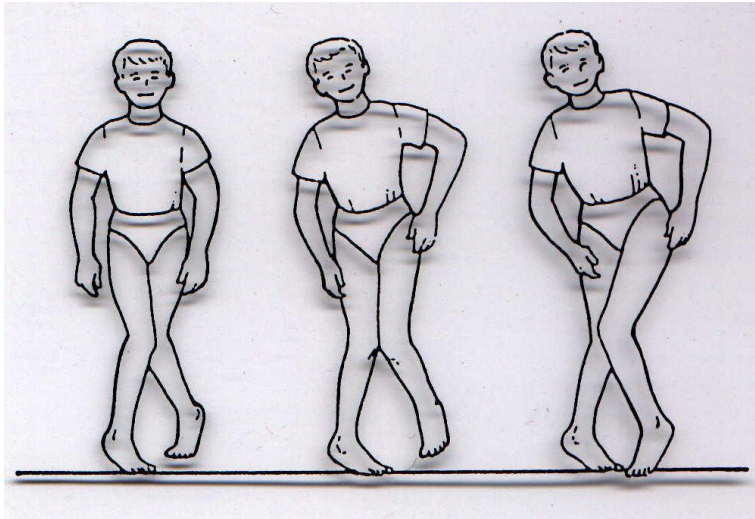
- **Knee:**

- Flexion deformity common.
- May need hamstring lengthening and sometimes extension osteotomy of distal femur



Cerebral Palsy

- **Hip**
 - Flexion/adduction deformity causing:
 - scissoring when walking
 - later hip dislocation.
 - Need regular X-rays and tenotomies of iliopsoas and adductors to prevent hip dislocation.
 - In the older child proximal femoral osteotomies may be necessary



Cerebral Palsy

- **Spine:**
 - 50% with severe CP have scoliosis.
 - Early referral is imperative.



Cerebral Palsy

- **Upper limb:**

- Often severely disabled in CP.
- Surgery for a severely deformed upper limb, especially wrist flexion and a clenched fist, should be performed during the first few years of life.
- The use of a hand, even if only as a paperweight, should be optimized.

Cerebral Palsy

- Treatment
 - Physiotherapy
 - Occupational therapy
 - Speech therapy
 - Splinting and bracing
 - Antispasticity medicine
 - Botox injections
 - Surgical interventions

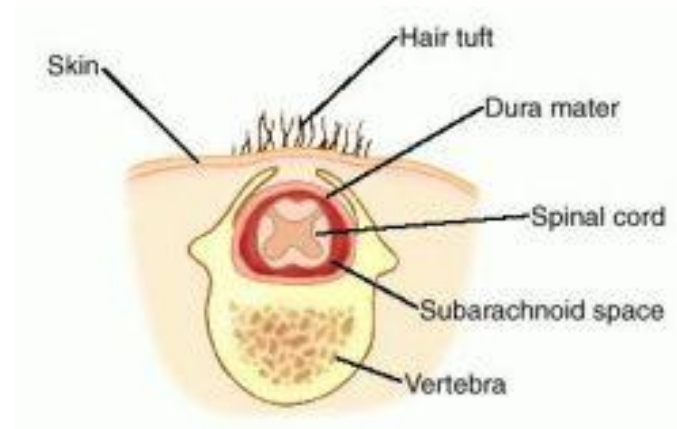
Spina Bifida

- Malformation of spinal column and spinal cord due to failure of closure of neural crests 3-4 weeks post fertilisation
- Prenatal diagnosis
 - α -fetoprotein 60-95% accurate
 - Ultrasound
 - Amniocentesis
- Risk factors
- History of previously affected pregnancy
- Low folic acid intake
- Pregestational maternal diabetes
- In utero exposure to valproic acid carbamazepine

Spina Bifida

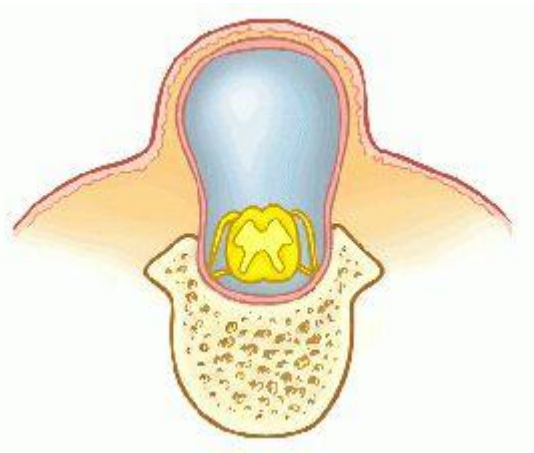
- **Spina bifida occulta**

- There is an unfused segment of the vertebral arches especially at the level of L5 and S1.
- It is found in 10% of adult spines. A small number of patients may develop progressive neurological signs.
- May have associated tuft of hair or skin discolouration



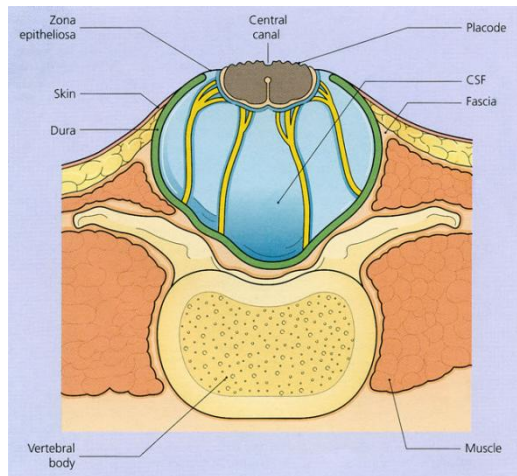
Spina Bifida

- **Meningocele**
 - Relatively uncommon variety of spina bifida. Defect in vertebral arches with a cystic swelling including dura and arachnoid without spinal cord inclusion.



Spina Bifida

- **Myelomeningocele (MM)**
 - In this form there is a fluid-filled cystic swelling formed by dura and arachnoid. The spinal cord and nerve roots are carried into the fundus of the sac.
 - These patients have varying degrees of motor, sensory and visceral paralysis.
 - These lesions may be closed (covered by skin) or open (skin deficient and nerve tissue exposed on the surface of the body).



Spina Bifida

- **THE NEUROLOGICAL LESION:**
 - **Motor lesions:**
 - Muscle imbalance -primarily due to LMN lesion. This is valid for the area of cord involved due to failure of development of that part of the cord = myelodysplasia.
 - Secondary LMN paralysis-develop due to traction on the cord, direct pressure during delivery and post-natal drying and infection of the neural plate.
 - Two thirds of children also have an UMN lesion due to interruption of the long spinal tracts.
 - Several combinations of imbalance can exist between flaccid, normal and spastic muscles.



Spina Bifida

- **Sensory lesion:**
 - Sensory impairment causes trophic ulceration especially of the feet.
- **Visceral paralysis:**
 - Incontinence of bladder and bowel is present in a high proportion of these children.
 - Urinary tract infections are common and often the cause of death in these children.



Spina Bifida

- **MANAGEMENT:**
 - **Genetic counselling and antenatal diagnosis**
 - Alpha fetoprotein estimation at 15 – 17 weeks gestation are diagnostic and abortion can be considered.
 - **Total management**
 - 50% of cases of SB survive the first year of life. Most important is the team approach, which can only be offered by special clinics or hospitals. This includes amongst them the neurosurgeon, urologist and orthopaedic surgeon.
 - The ultimate aim is to get the child walking even if only for part of his or her early childhood.
 - Walking limits pressure sores, obesity, urinary stasis and osteoporoses.
 - The urologist probably plays an even greater role in the management of these children and evaluation by a urologist soon after birth is imperative.

Spina Bifida

- **Orthopaedic management:**
 - Because the level of involvement varies, the management of each case will vary.
 - Obtain plantigrade feet in sitters and walkers in order to facilitate the fitting of protective footwear and to prevent pressure sores.
 - Rebalancing of muscles and release of contractures of the lower limbs.
 - Surgery is sometimes indicated in lower lumbar lesions where the hips are dislocated since this group has a better prognosis for walking.
 - Scoliosis in these children needs early attention.

Poliomyelitis

- An acute viral infection with a wide range of manifestations, including non-specific minor illness, aseptic meningitis (nonparalytic poliomyelitis), and flaccid weakness of various muscle groups (paralytic poliomyelitis).

Poliomyelitis

- **Pathophysiology**

- Virus enters mouth-primary multiplication in lymphoid tissue. Via the bloodstream to the RES. Secondary viremia followed by invasion of CNS. The agent is present in blood, throat, and faeces during incubation period.
- Persists in throat washings for 1 to 2 weeks and in faeces for 3 to 6 weeks or longer. Viremia lasts several days but disappears by the time of onset, when antibodies have already developed.
- Factors predisposing the serious neurologic damage include increasing age, recent tonsillectomy, inoculations (most often DTP) and pregnancy.

Poliomyelitis

- **PATHOPHYSIOLOGY:**

- The spinal cord and brain -only sites of significant virus-induced pathology.
- Motor neurons of the anterior horn of the spinal cord, the medulla, and to a lesser degree certain other parts of the brain, including the cerebellum and the motor cortex, are involved.
- Damage to neurons=intense inflammatory reaction and eventually neuronophagia.
- Site and severity of paralysis determined by the distribution of the neuronal lesions.

Poliomyelitis

- **2 basic clinical patterns:**
 - minor illness (abortive type)
 - major illness (which may be paralytic or nonparalytic).

Poliomyelitis

- **Minor illness**

- 80 to 90% of clinical infections, in young children, is mild, and does not involve the CNS.
- Symptoms are light fever, malaise, headache, sore throat, and vomiting which develop 3 to 5 days after exposure.
- Recovery occurs within 24 to 72 hours.

Poliomyelitis

- **Major illness**

- Appear without a previous minor illness, in older children and adults.
- Incubation 7 to 14 days.
- Fever, headache, stiff neck and back, deep muscle pain, and hyperesthesia's and parasthesias.
- There may be no further progression, or disease may go on, with loss of selective tendon reflexes and asymmetric weakness or paralysis of muscle groups, depending on the location of lesion in the spinal cord or medulla.

Poliomyelitis

- **Major illness**

- Dysphagia, nasal regurgitation, and nasal voice are early signs of bulbar involvement.
- The CSF glucose is normal, the protein is lightly elevated, and the cell count commonly ranges from 10 to 300 cells/ μ L (predominantly lymphocytes). The peripheral WBC counts may be normal or moderately increased.

Poliomyelitis

- **Diagnosis and Differential Diagnosis**
 - Asymmetric flaccid limb paralyses or bulbar palsies without sensory loss during an acute febrile illness in a child or young adult
 - Coxsackieviruses and echoviruses' (Guillain-Barré syndrome) = usually no fever, muscle weakness is symmetric, sensory findings coexist in 70% of cases, and CSF protein is usually elevated in the presence of a normal cell count.

Poliomyelitis

- **Diagnosis and Differential Diagnosis**
 - Mumps or herpes viruses, tuberculous meningitis, or brain abscess or meningoencephalitis due to arboviruses.
 - Virus isolation from throat and/or faeces or demonstration of a rise in specific antibody is required to confirm the diagnosis

Poliomyelitis

- **Prognosis**

- Abortive and nonparalytic forms, recovery is complete.
- Paralytic poliomyelitis,
 - <25% of patients severe permanent disability
 - 25% have mild disabilities
 - >50% recover with no residual paralyses.
- Greatest return of muscle function in the first 6 months
- Improvement may continue for 2 years.
- Mortality is 1 to 4% but may increase to 10% in adults or those with bulbar disease.

Poliomyelitis

- **Post-poliomyelitis syndrome:**
 - Muscle fatigue and decreased endurance, weakness, fasciculation's, and atrophy in selective muscles.
 - Occurs many years after an attack of paralytic poliomyelitis,
 - Affecting older and initially more severely involved patients.
 - Cause is thought to be associated with aging changes and further loss of anterior horn cells in a population of neurons already depleted by earlier poliovirus infection.

Poliomyelitis

- **Therapy is symptomatic.**
 - Bed rest for several days.
 - Analgesics and antipyretics may be useful.
 - Muscle spasm and pain may be relieved by several 20 min applications per day of hot, moist packs.
 - Urinary retention, may respond to a parasympathomimetic. An intermittent catheterisation program is often preferable.
 - Physical therapy is the most important part of management of paralytic poliomyelitis during convalescence.

Poliomyelitis

- The paralysed legs are supported by plaster splints or pillows and sandbags to keep the hip joints in 51 of flexion and in neutral rotation.
- The knee joint is held at 51 of flexion and the foot is supported in a 901 position.
- Splinting relieves pain and spasm and prevents the development of deformities.

Poliomyelitis

- **Respiratory failure**

- Result from spinal cord involvement causing paralysis of the muscles of respiration or from damage by the virus to the respiratory centres in the medulla and paralysis of muscles innervated by the cranial nerves. Ventilation is the treatment for both types.
- Pharyngeal muscle weakness, difficulty in swallowing, inability to cough, and pooling of bronchotracheal secretions. Postural drainage and suction. Intubation or tracheostomy frequently required to keep the airway clear.
- Pulmonary atelectasis should be managed.

MUSCLE DYSTROPHY

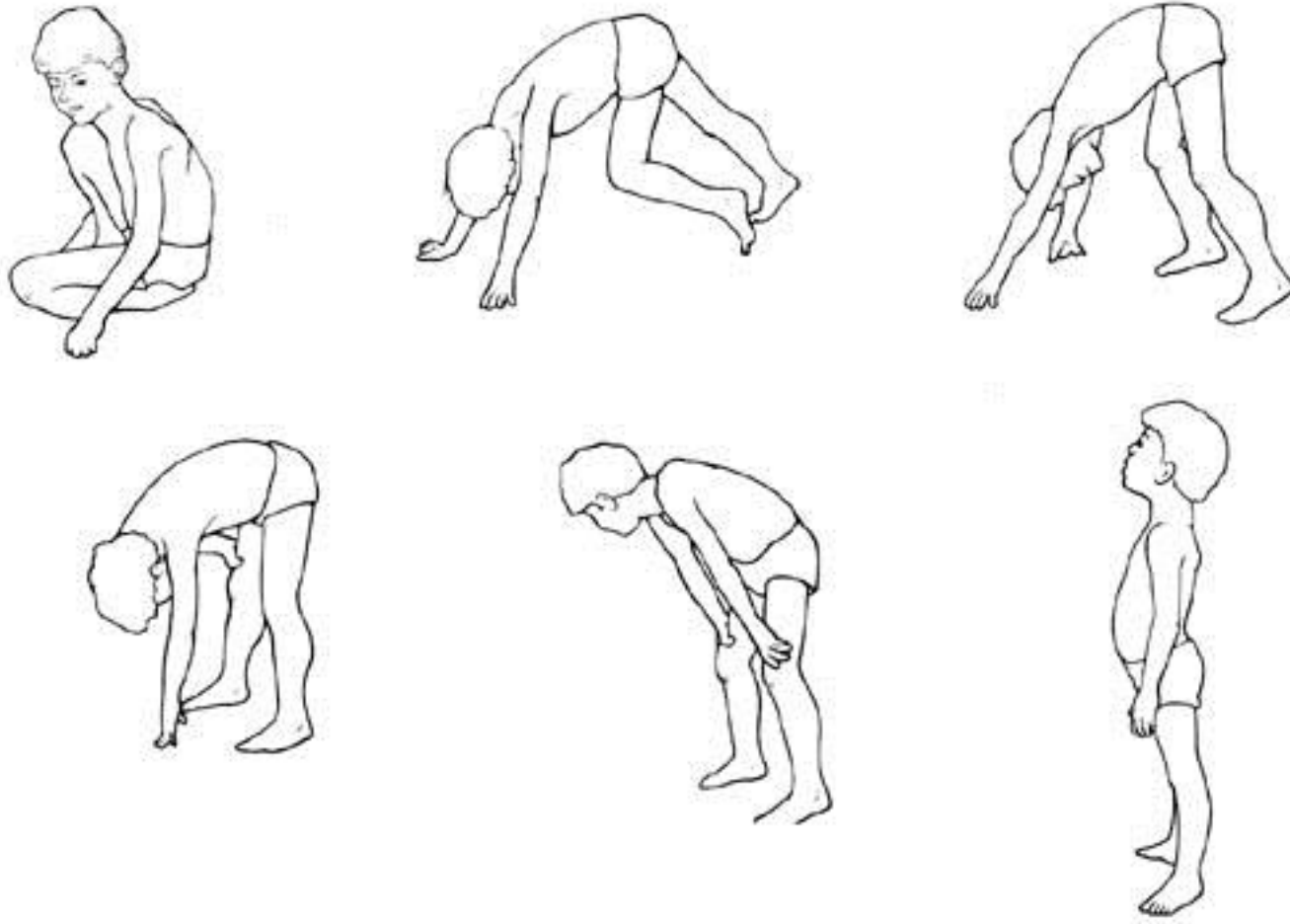
- **Duchenne muscular dystrophy**
 - X-linked recessive inherited trait
 - Absent dystrophin protein
 - Reported to occur in 1 in 3500 live births. There is a family history in 70% of patients, and the condition occurs as a spontaneous mutation in about 30% of patients (spermatogenesis on mother's paternal side)

Duchenne Muscular Dystrophy

- Children usually reach early motor milestones at appropriate times, but independent ambulation may be delayed
- Many are initially toe-walkers.
- Clinical features= large, firm calf muscles, tendency to toe-walking, widely based, lordotic stance, waddling Trendelenburg gait, and positive Gower test indicative of proximal muscle weakness.

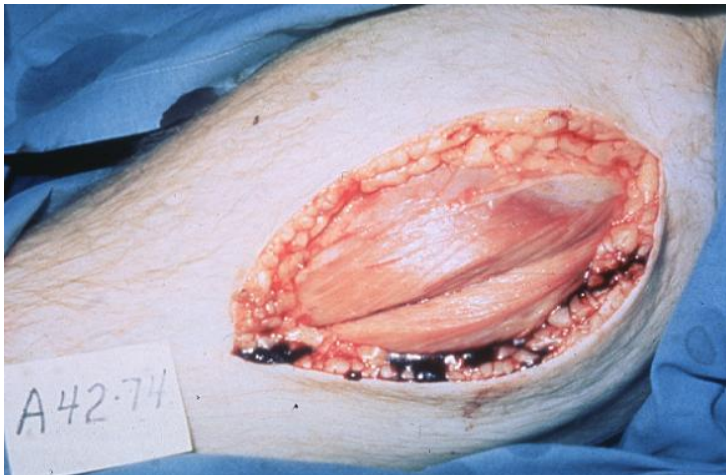


Duchenne Muscular Dystrophy



Duchenne Muscular Dystrophy

- The diagnosis usually obvious by 5 or 6 years old
- Diagnosis confirmed by a dramatically elevated level of creatinine phosphokinase (50 to 100 times normal)
- Muscle biopsy characterized by variations in fiber size in internal nuclei, split fibers, degenerating or regenerating fibers, and fibro fatty tissue deposition.



Duchenne Muscular Dystrophy

- **Maintain functional ambulation as long as possible.**
 - Contractures of the lower extremity require early treatment to prolong the child's ability to ambulate, if even for 1 to 2 years
 - It is easier to keep patients walking than to induce them to resume walking once they have stopped.
 - When children with Duchenne muscular dystrophy stop walking, they also become more susceptible to the development of scoliosis and severe contractures of the lower extremities.
 - Cessation of ambulation also results in rapid pulmonary deterioration.
 - Equines contractures of the feet should not be corrected initially =helps force the knee into extension and prevent the knee buckling caused by severe weakness of the quadriceps.

Duchenne Muscular Dystrophy

- Stretching exercises and nightly bracing can be used to prevent contractures.
- Flexion and abduction contractures of the hip, should be prevented.
- Prevent the child's sleeping in a frog position.
- When surgery is indicated, the foot and hip contractures should be released simultaneously.
- Ambulation should be resumed immediately after surgery if possible.
- Polypropylene braces are preferred to long-term casting.
- Prolonged immobilization must be avoided to prevent or limit the progressive muscle weakness caused by disuse.

Duchenne Muscular Dystrophy

- Loose independent ambulation by age 10
- Usually wheelchair bound by age 15
- Usually die before 20 due to cardiopulmonary causes
- New literature shows that steroid use may prolong disease process

MUSCLE DYSTROPHY

- **OTHER VARIANTS OF MUSCULAR DYSTROPHY**
 - Becker muscular dystrophy.
 - Emery-Dreifuss muscular dystrophy.
 - Limb-girdle dystrophy.
 - Facioscapulohumeral muscular dystrophy.
 - Myotonic dystrophy.

Septic arthritis

‘Every hour that an acute suppurative process continues within a joint is of urgent significance to prognosis’

Paterson 1970

Septic arthritis

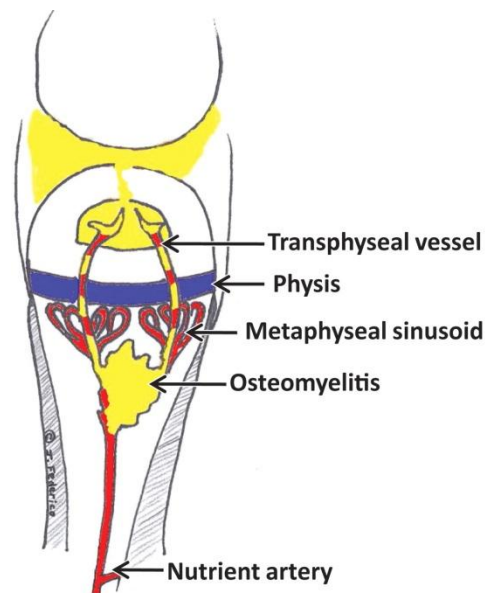
- In USA 1/5000 kids <13yrs
- 2/5000 develop SA
- Outcome extremely poor in Septic arthritis.
- As bad as 40% poor outcome in hip Septic arthritis.

Septic arthritis

- Inflammation involving a joint, for which an infecting micro-organism is either detected or presumed to be the cause
- Different clinical patterns found depend on the 'toxic' characteristics of the causative germ; and the way in which the synovium responds to the organism
- AIM of management:
 - **restoration and maintenance of normal function.**
- Up to 10% of patients, more than one joint is involved

Septic arthritis

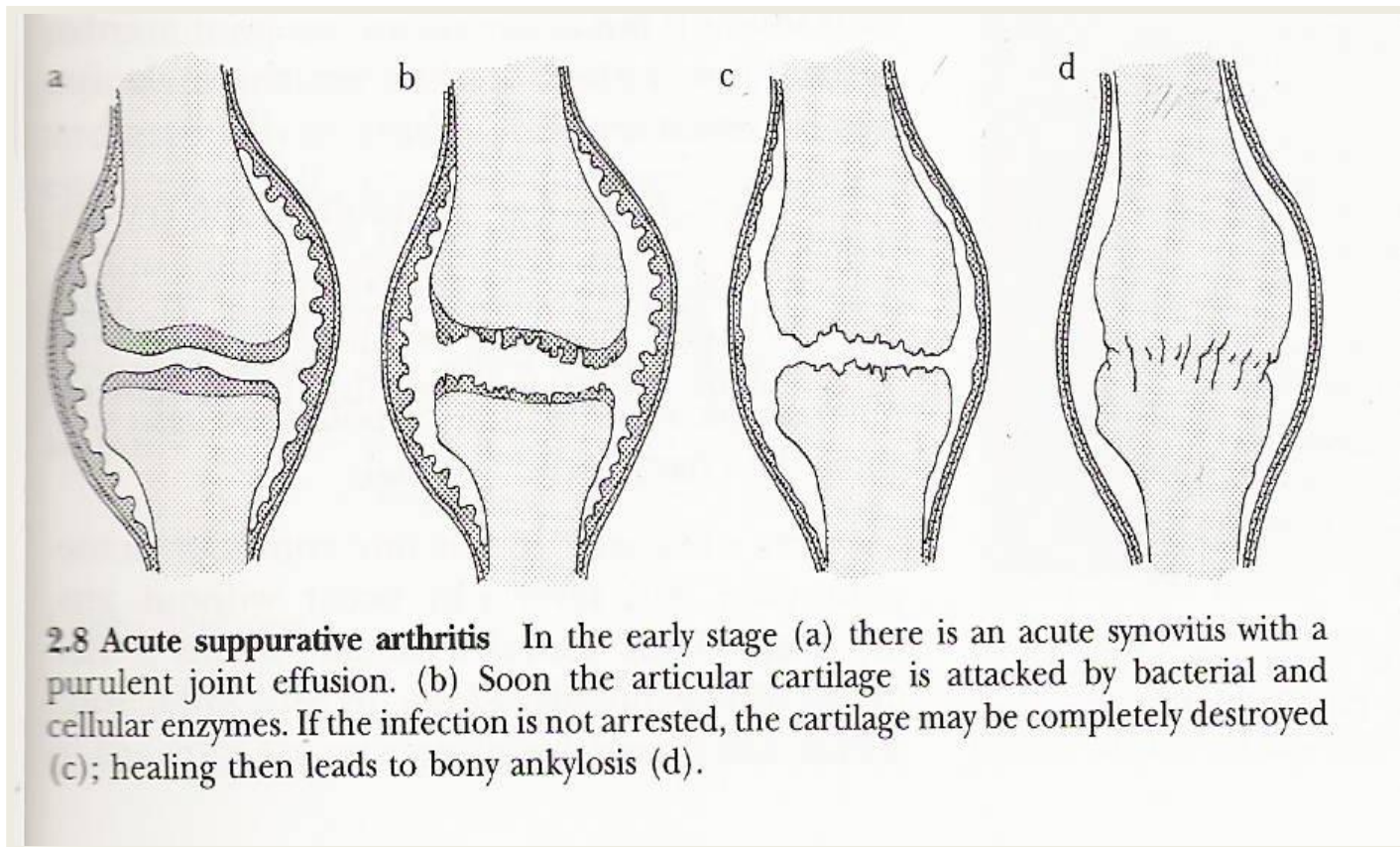
- Establishment of infection:
 - Direct inoculation,
 - Haematogenous spread
 - Contiguous infected foci.
 - Transphyseal vessels/ due to intra articular nature of joints



Septic arthritis

- Microbiology:
 - Neonates- group B strep, staph aureus, Neisseria gonorrhoea.
 - Infants < 2yr- staph aureus, Kingella kingae, Hemoph. influenza.
 - Children 3yrs and older- staph, salmonella, strep pyogenes.

Septic arthritis



Septic arthritis

- Clinical picture:

- **HIGH INDEX OF SUSPICION**

- Depends on age.

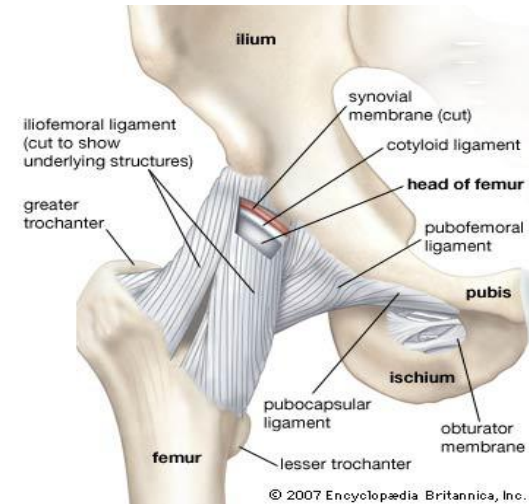
- Pseudo paralysis.

- Failure to thrive.

- Systemic signs/look for heart valve vegetations and lung abscesses.

- Refusal to bear weight.

- Decreased ROM



Septic arthritis

- Diagnosis relies on clinical impression, microbiology, blood infective markers and radiology combined.
- Kocher criteria:
 - History of fever(oral $>38,5^{\circ}$)
 - Non weight bearing
 - ESR >40 mm/h
 - WBC count $>12000/\text{mm}^3$
 - Presence of 1=10%, 2=35%,3=73% and 4=93%.
- **Joint aspiration and analysis of joint fluid remains gold standard.**

Septic arthritis

- Treatment Principles:
 - Resuscitation
 - Drainage
 - Antibiotics
 - Cloxacillin high dose
 - Change according to cultures
 - Immobilization
 - Monitoring of treatment

Osteogenesis Imperfecta

- Brittle bone disease
- Type 1 collagen problem
 - Blue sclerae
 - Triangular facies
 - Macrocephaly
 - Hearing loss
 - Defective dentition (dentogenesis imperfecta)
 - Barrel chest
 - Scoliosis
 - Limb deformities
 - Fractures
 - Joint laxity
 - Growth retardation



Osteogenesis Imperfecta

- **Sillence classification**

<u>Type</u>	<u>Inheritance</u>	<u>Sclerae</u>	<u>Features</u>
1	Autosomal Dominant	Blue	Mild affliction, Multiple fractures, Good prognosis, Teeth may be involved
2	Autosomal Recessive	Blue	Lethal, Severely affected
3	Autosomal Recessive	Normal	Fractures at birth, Short stature
4	Autosomal Dominant	Normal	Milder form, Teeth may be involved

Osteogenesis Imperfecta

- **MANAGEMENT:**
 - Fractures
 - Splints- short period as possible to prevent cycle of immobilization resulting in Osteoporosis and further fractures due to poor quality bone.
 - Exercise
 - Manual fracturing followed by splinting and upright mobilization.
 - Surgical correction.
 - Bisphosphonates

Osteogenesis Imperfecta

- **Indications for surgery**
 - Multiple fractures.
 - Correcting deformities
 - To enable walking and independence.
 - Weight bearing limbs.



Neurofibromatosis

- Type 1 Peripheral type
- Type 2 Central type
- Also known as von Recklinghausen disease
- Autosomal dominant inheritance
- Disorder of neural crest origin

Neurofibromatosis

- **Diagnostic criteria**

- **Six or more café au lait macules over 5 mm in greatest diameter in prepubertal individuals and over 15 mm in greatest diameter in postpubertal individuals**
 - Two or more neurofibromas of any type or one plexiform neurofibroma
 - Freckling in the axillary or inguinal regions (Crowe's sign)
 - Optic glioma
 - Two or more Lisch nodules (iris harmartomas)
 - A distinctive osseous lesion such as sphenoid dysplasia or thinning of long bone cortex with or without pseudoarthrosis
 - A first-degree relative (parent, sibling, or offspring) with NF1 by the above criteria
- The criteria are met in an individual if two or more of the features listed are present.



Neurofibromatosis



- **Diagnostic criteria**

- Six or more café au lait macules over 5 mm in greatest diameter in prepubertal individuals and over 15 mm in greatest diameter in postpubertal individuals
 - **Two or more neurofibromas of any type or one plexiform neurofibroma**
 - Freckling in the axillary or inguinal regions (Crowe's sign)
 - Optic glioma
 - Two or more Lisch nodules (iris hamartomas)
 - A distinctive osseous lesion such as sphenoid dysplasia or thinning of long bone cortex with or without pseudoarthrosis
 - A first-degree relative (parent, sibling, or offspring) with NF1 by the above criteria
- The criteria are met in an individual if two or more of the features listed are present.



Neurofibromatosis



- **Diagnostic criteria**

- Six or more café au lait macules over 5 mm in greatest diameter in prepubertal individuals and over 15 mm in greatest diameter in postpubertal individuals
 - Two or more neurofibromas of any type or one plexiform neurofibroma
 - **Freckling in the axillary or inguinal regions (Crowe's sign)**
 - Optic glioma
 - Two or more Lisch nodules (iris hamartomas)
 - A distinctive osseous lesion such as sphenoid dysplasia or thinning of long bone cortex with or without pseudoarthrosis
 - A first-degree relative (parent, sibling, or offspring) with NF1 by the above criteria
- The criteria are met in an individual if two or more of the features listed are present.

Neurofibromatosis

- **Diagnostic criteria**

- Six or more café au lait macules over 5 mm in greatest diameter in prepubertal individuals and over 15 mm in greatest diameter in postpubertal individuals
- Two or more neurofibromas of any type or one plexiform neurofibroma
- Freckling in the axillary or inguinal regions (Crowe's sign)
- Optic glioma
- Two or more Lisch nodules (iris harmartomas)
- **A distinctive osseous lesion such as sphenoid dysplasia or thinning of long bone cortex with or without pseudoarthrosis**
- A first-degree relative (parent, sibling, or offspring) with NF1 by the above criteria

- The criteria are met in an individual if two or more of the features listed are present.



Bring everything together!!

Examination of the Neonate

- Family History

- Osteogenesis imperfecta
- Neurofibromatosis
- Muscle diseases
- Scoliosis
- Haemophilia

- Birthing History

- Prenatal problems
- Gestation
- Type delivery
- Perinatal problems
- Ventilation
- Complications
- Transfusions
- Infections
- Injections

Examination of the Neonate

- Special efforts to gain confidence of baby
 - Mothers lap
 - Bottle/dummy
 - Warm hands
 - Talking
- Observation extremely valuable because baby unable to locate or describe problem
 - Movements of limbs

Examination of the Neonate

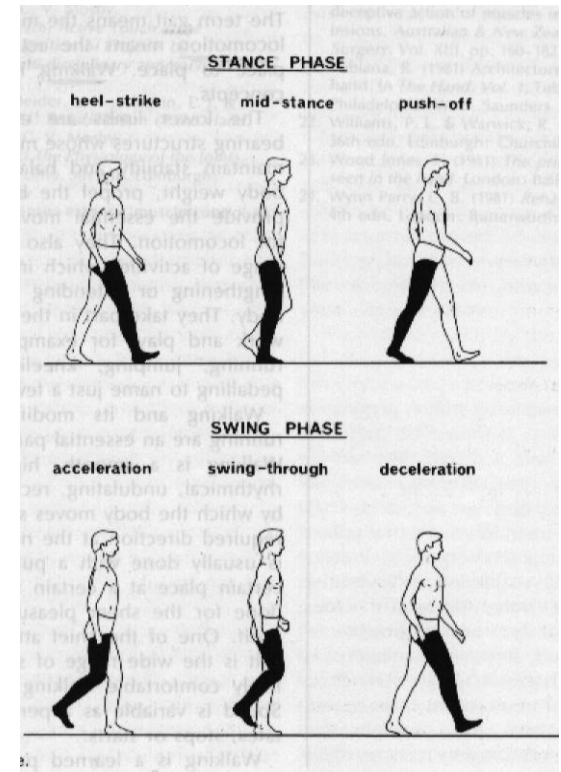
- General state of health.
- Systemic evaluation.
- Remember which conditions are commonly related to newborns.
 - Brachial plexus
 - Hip dysplasia
 - Spinal defects
 - Recurvatum of the knees
 - Syndromic features
 - Packaging defects.
- Arrange F/U examination if needed.

Normal Gait

- Gait tends to be variable and irregular until age 7.
- Functional tasks involved:
 - Forward progression
 - Body balance and limb length adjustments
 - Upper body support.

Normal Gait

- Gait Cycle involves 2 cycles:
 - Stance phase
 - Heel strike.
 - Foot flat.
 - Midstance.
 - Push-off.
 - Swing phase
 - Acceleration.
 - Midswing.
 - Deceleration.
- 60% of time spent in stance phase and 40% spent in swing phase.



Normal Gait

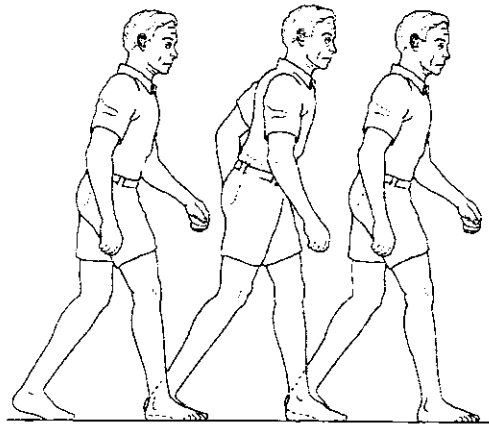
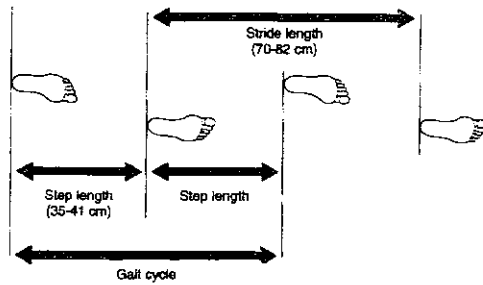


Figure 1
Gait cycle, stride length, and step length.



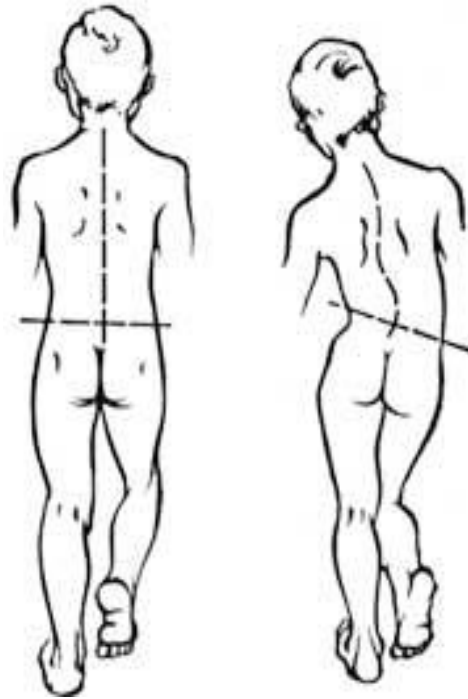
Prerequisites for normal gait

- Stability in stance
- Clearance of foot in swing
- Preposition of foot in swing
- Adequate step length
- Energy conservation

Abnormal gait

- Short leg
- Painful
 - characteristic of antalgic limp is shortened stance phase on affected side thus shorter step length
- Neuromuscular - weak, spastic
- Stiff joint
- Trendelenburg
 - allows opposite side of pelvis to tilt downward during stance on weakened side

Trendellenburg



Examination of the limping child

- General overview of posture.
- Walk without shoes, with shorts.
- Observe from front, sides and back.

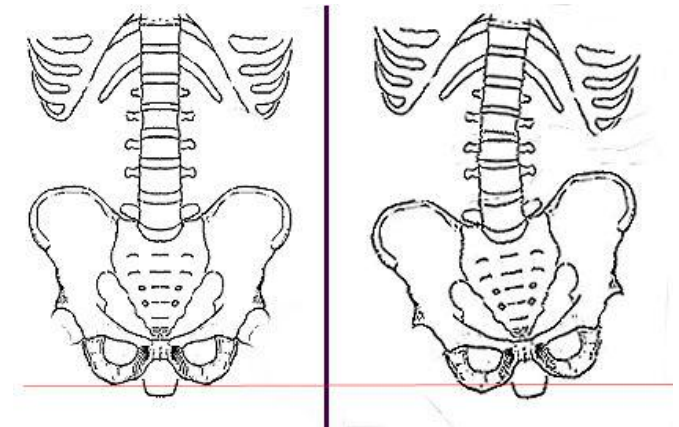
Examination of the limping child

- General examination

- Spine

- Hip

- **Pelvic obliquity/abdominal examination.**
 - Bryant's triangle.
 - Thomas test.
 - Galleazi test
 - Leg length discrepancy
 - Ab and Adduction contractures.
 - ROM-IR/ER

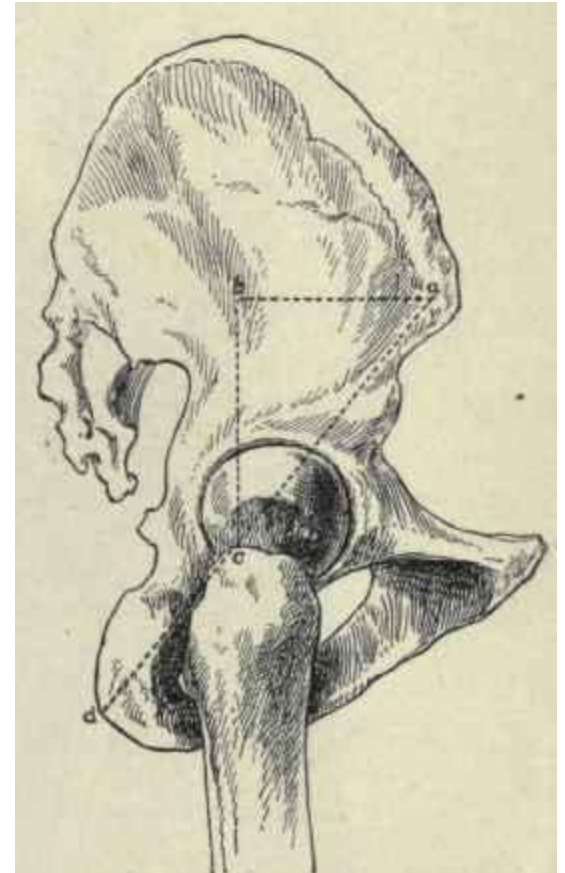


- Knee

- Foot

Examination of the limping child

- General examination
 - Spine
 - Hip
 - Pelvic obliquity/abdominal examination.
 - **Bryant's triangle.**
 - Thomas test.
 - Galleazi test
 - Leg length discrepancy
 - Ab and Adduction contractures.
 - ROM-IR/ER
 - Knee
 - Foot



Examination of the limping child

- General examination
 - Spine
 - Hip
 - Pelvic obliquity/abdominal examination.
 - Bryant's triangle.
 - **Thomas test.**
 - Galleazi test
 - Leg length discrepancy
 - Ab and Adduction contractures.
 - ROM-IR/ER
 - Knee
 - Foot



Examination of the limping child

- General examination
 - Spine
 - Hip
 - Pelvic obliquity/abdominal examination.
 - Bryant's triangle.
 - Thomas test.
 - **Galleazi test**
 - Leg length discrepancy
 - Ab and Adduction contractures.
 - ROM-IR/ER
 - Knee
 - Foot



**Galeazzi
Test**
Difference
in
knee
height

Examination of the limping child

- General examination
 - Spine
 - Hip
 - Pelvic obliquity/abdominal examination.
 - Bryant's triangle.
 - Thomas test.
 - Galleazi test
 - **Leg length discrepancy**
 - Ab and Adduction contractures.
 - ROM-IR/ER
 - Knee
 - Foot



Examination of the limping child

- General examination
 - Spine
 - Hip
 - Knee
 - Alignment.
 - Swelling/effusion
 - Areas of tenderness
 - Joint line tenderness.
 - Ligaments.
 - Foot

Examination of the limping child

- General examination
 - Spine
 - Hip
 - Knee
 - Foot
 - Remember the thorn!!



Abnormal gait

- Toddlers
 - DDH
 - Mild CP
 - Toddlers fracture
 - Juvenile rheumatoid arthritis
 - Discitis

Abnormal gait

- Children 4-10yrs
 - Perthe's
 - Septic arthritis/Osteomyelitis
 - Synovitis
 - Leg length discrepancy
 - DDH

Abnormal gait

- Children 11 – 16yrs
 - SUFE
 - Hip dysplasia
 - Tumor
 - Tarsal coalition
 - Iatrogenic

The End!!

Thank You