

Neuromuscular Pediatric Orthopaedics

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1- Non Progressive

- * Cerebral Palsy
- * Myelodysplasia- Spina Bifida
- * Arthrogryposis M C



Cerebral Palsy - CP

Chronic, non-progressive disorder of motor control, following an insult to the cerebral cortex → abnormality of

Posture

Movement

Tone

- * **Single largest disability in children**

- * **1-5:1,000**

- * **Time of occurrence**

- * Prenatal 80%





- * Perinatal 5%

- * Postnatal 15%



Prenatal (infections, drugs
malformations, etc.),
Perinatal (prematurity, anoxia)
Postnatal (injury, infection)

TABLE 33-2 Geographical Classification of Cerebral Palsy

TYPE	DESCRIPTION	INVOLVEMENT
Monoplegia	One extremity involved, usually lower	
Hemiplegia	Both extremities on same side involved Usually upper extremity involved more than lower extremity	
Paraplegia	Both lower extremities equally involved	
Diplegia	Lower extremities more involved than upper extremities Fine-motor/sensory abnormalities in upper extremity	

Quadruplegia All extremities involved equally
Normal head/neck control


















Double hemiplegia All extremities involved, upper more than lower






Total body All extremities severely involved
No head/neck control



 Normal
 Mild involvement
 Severe involvement

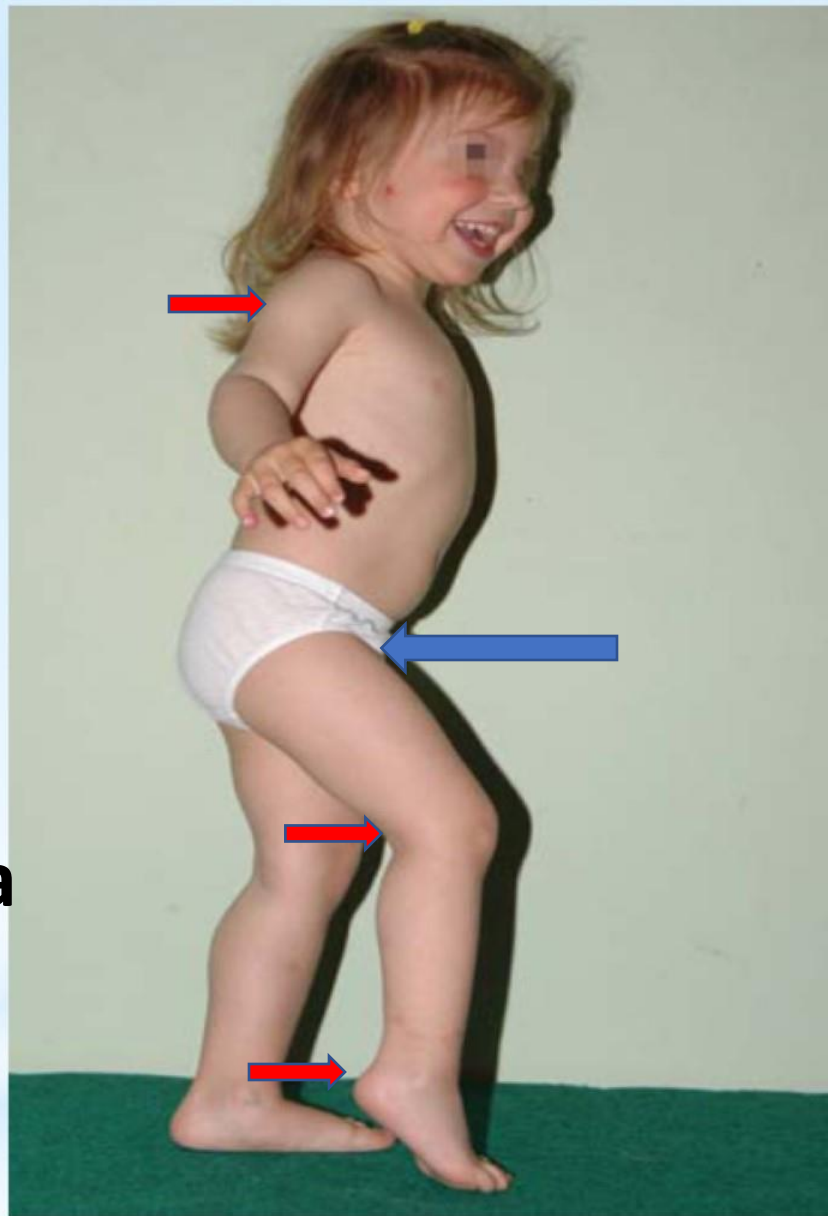
Regional involmment			Global (total body) involmment		
Spastic			Dyskinetic		Ataxia
					
					
Hemiplegia			Athetoid		Ataxic
Diplegia			Dystonic		
Quadriplegia					
Pyramidal			Extrapyramidal		

-  Normal
-  Mild involvement
-  Severe involvement

TYPES OF CEREBRAL PALSY AND AREAS OF BRAIN DAMAGE INVOLVED

- * **Spastic** – Cortical motor tract **70%**
- * **Athetotic** – Extrapyramidal tract
- * **Ataxic** – Cerebellum /balance disturb
- * **Rigid - Mixed**

Hemiplegia



Diplegia.

- 1- Flexion-, Adduction-IR contracture of the hips**
- 2- Flexion contracture of the knees**
- 3- Equinus contractures of the ankles**



Tetraplegic

1- “crossing legs” severe

Hips adduction contracture

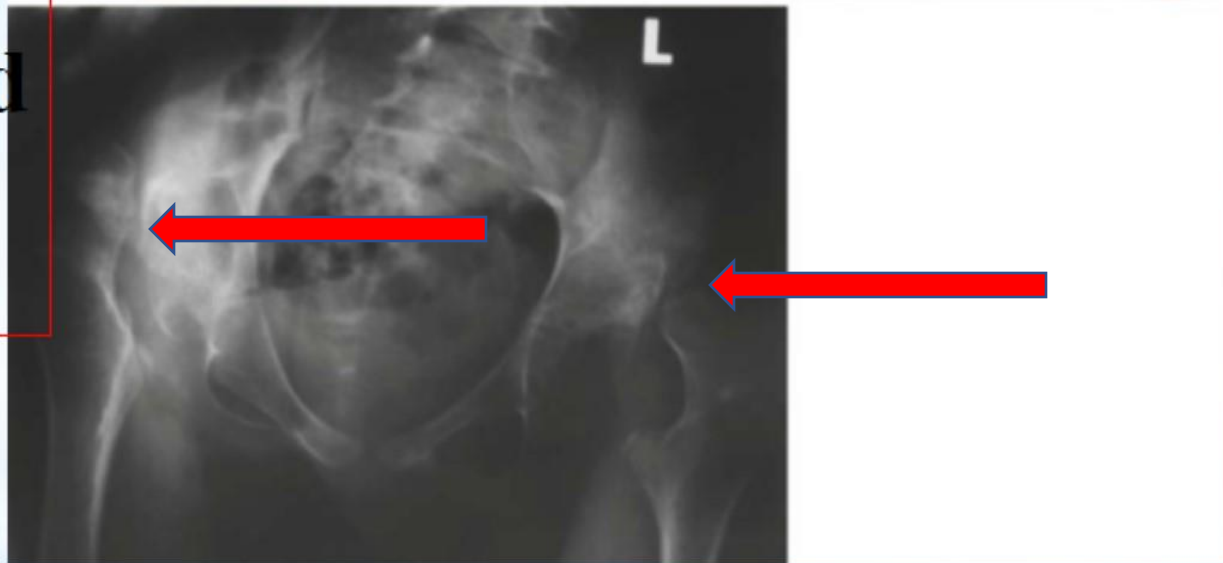
**2-Flexion contracture in
both upper limb**





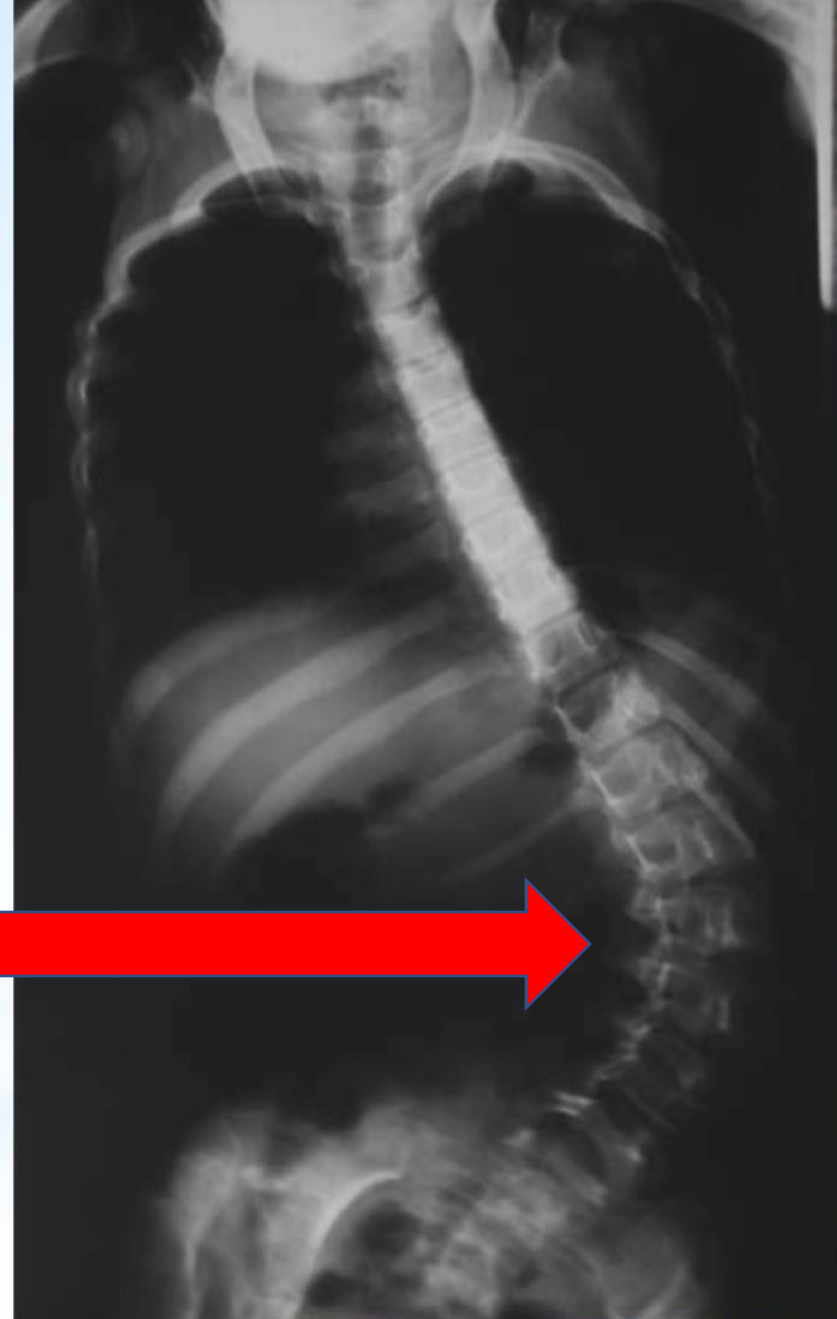
Hips are well developed at birth but due to the adduction contracture, coxa valga, → Subluxation

Severe spastic contraction of the flexor and adductor muscles of the hips → fixed hip dislocation



**Because of the
imbalance of the
paravertebral
muscles → Scoliosis**

C- Shape curve



A 21-year-old non-ambulatory patient with severe spastic Tetraparesis



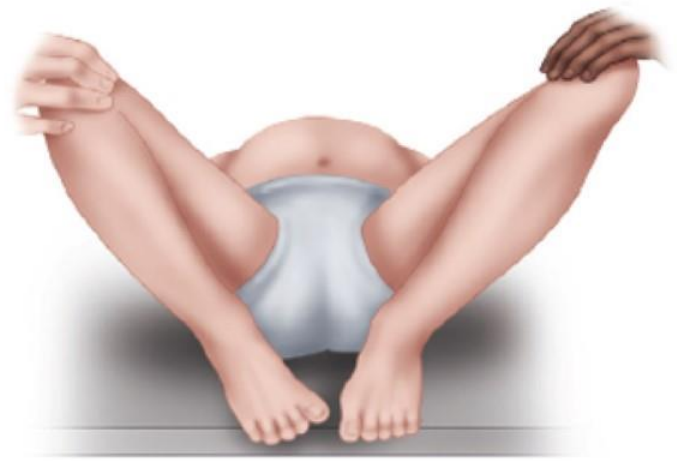
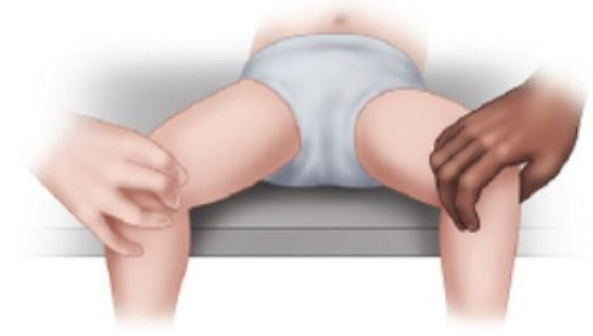
A**B****C****A****B****C**

FIGURE 33-16 Testing for hamstring spasticity and contracture. **A**, Patient is supine with hips extended. Pressure is exerted over knees, forcing them into extension. Flexion remaining in knees is absolute knee flexion contracture. **B**, Knee on side to be tested

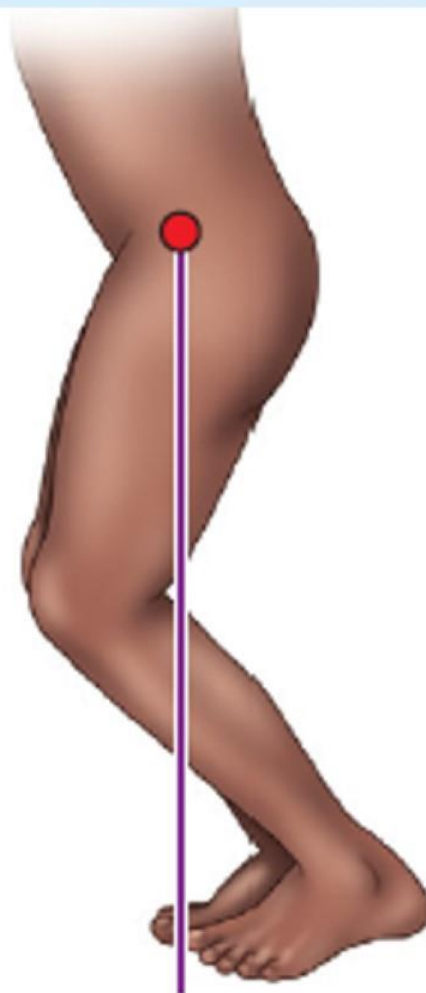


FIGURE 33-7 Typical crouch posture caused by flexion deformities of hips or fixed flexion deformities of knees.

*Myelodysplasia – Spina Bifida

***Group of malformations of spine/post. laminae of vertebra fail to close**

***Occulta 20-25% all births**

***some with no symptoms**

***Cystica - Sac**

***Meningocele – meningeal sac**

***Myelomeningocele – spinal cord too!**

***Has neuro deficits distal to lesion**

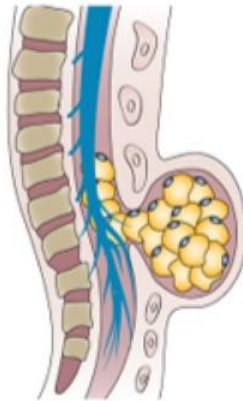
***Hydrocephalus 90%**





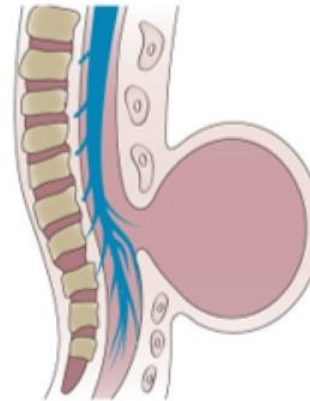
Spina bifida occulta

Closed asymptomatic NTD in which some of the vertebrae are not completely closed



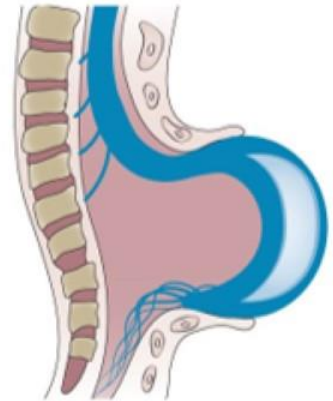
Closed spinal dysraphism

Deficiency of at least two vertebral arches, here covered with a lipoma



Meningocele

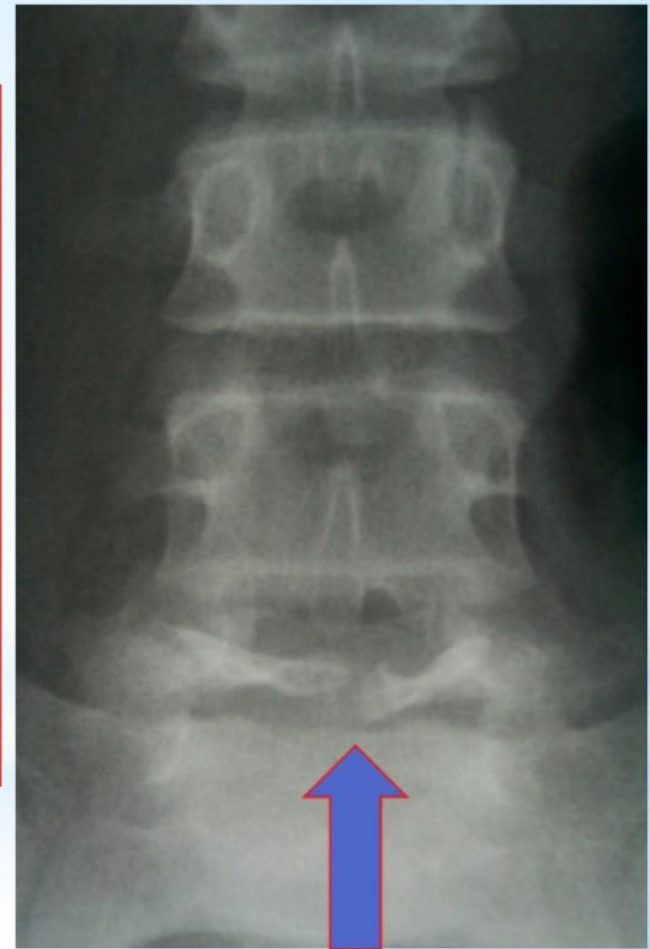
Protrusion of the meninges (filled with CSF) through a defect in the skull or spine



Myelomeningocele

Open spinal cord (with a meningeal cyst)

- * $F > M$ 1: 1,000 births
- * **Etiology:** genetic, ↓ folic acid, valproic acid
- * **Dx:** Clinical exam, X-ray, MRI
- * **Rx:** Neuro, Ortho, Urologic



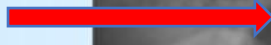
4. Clinical Features

- a) **Paralysis** and loss of sensation below lesion level (ulcerations, fractures)
- b) **Lack of bladder and bowel control**
- c) **Hydrocephalus**
- d) **Hip dislocations, scoliosis and foot deformities (equinovarus most common)**



AFO

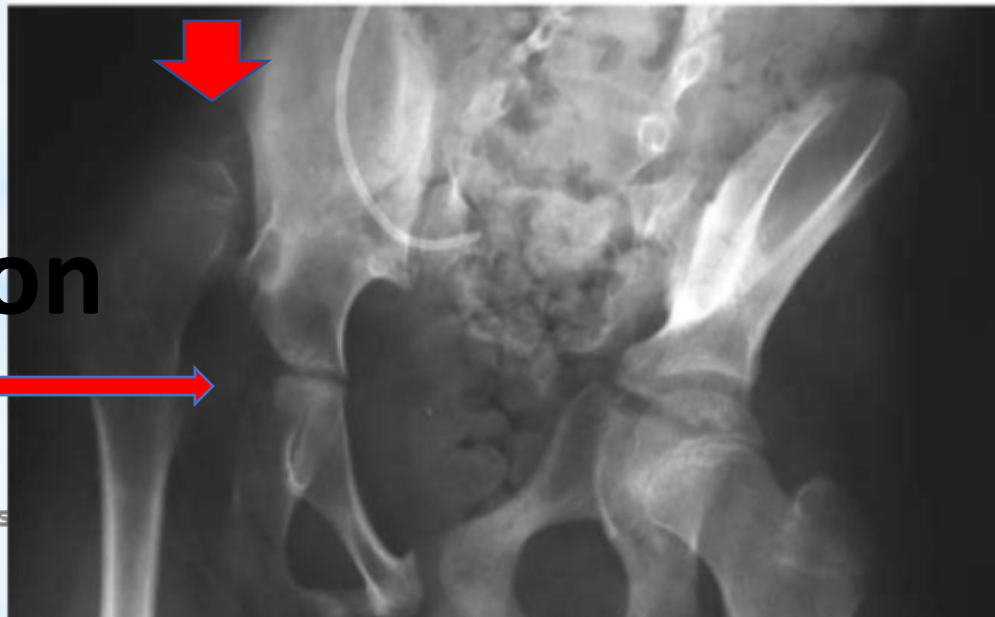
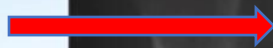
Shunt



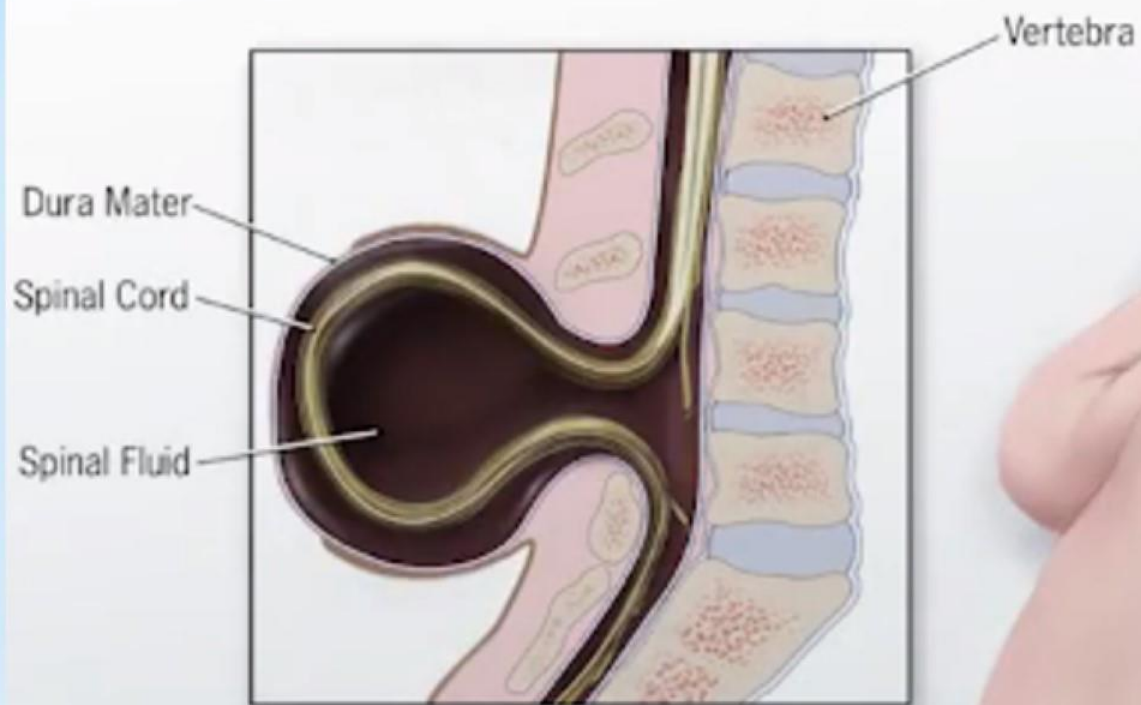
**C-Shape
Scoliosis**



**Hip
dislocation**



Spina Bifida (Open Defect)





Cause of death in myelomeningocele
**Urinary tract infection, renal failure,
meningitis, and sepsis.**

Associated problems

- 1- Hydrosyringomyelia**
- 2- Arnold-Chiari malformation** (caudal displacement of the posterior lobe of the cerebellum)
- 3- Tethered cord**
- 4- Latex hypersensitivity 3.8% to 38%.**

*Multi-disciplinary team

- *Promote mobility, ADLs, diet,
- *Bowel bladder program
- *Latex precautions
- *Neuro status – shunt problems
- *Insensate skin, water temp, braces
- *Multiple ortho corrections



AMC

*Nonprogressive cong. NM syndrome ch.ch by severe joint contractures, muscle weakness, and fibrosis.

***Etiology:** unknown

0.03% general population

***Dx:** clinical exam, X-ray, ml bx





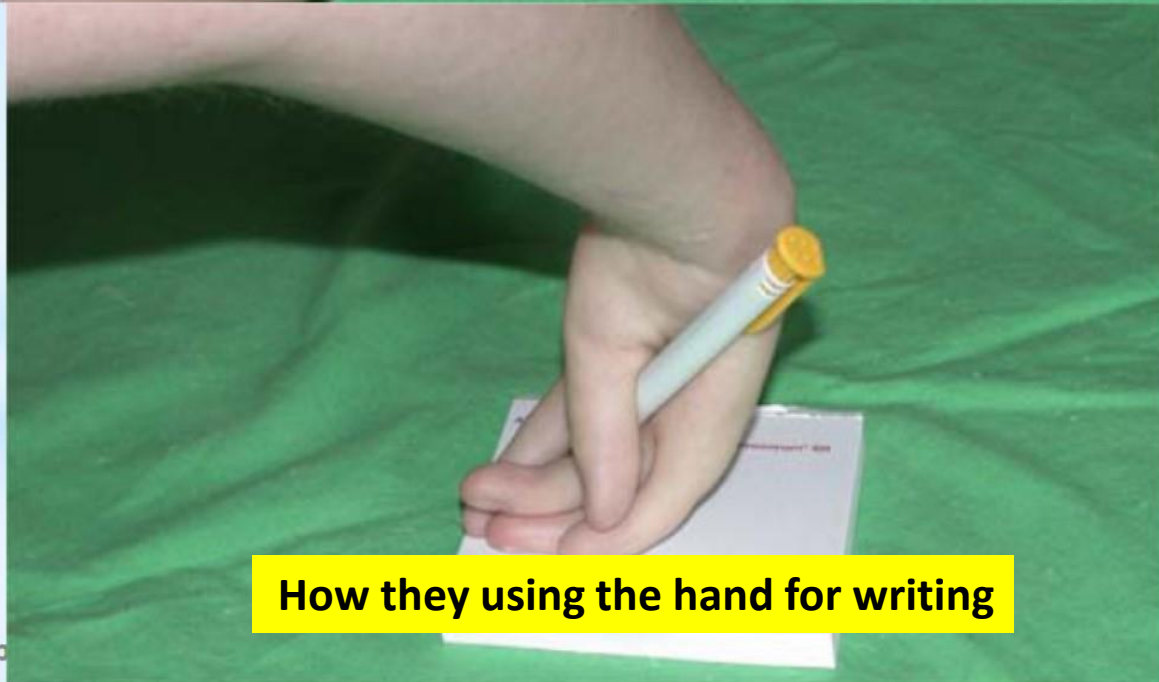
Internal rotation shoulders
Elbow extension with absent crease
Thumb in palm





**Fingers
contracture**

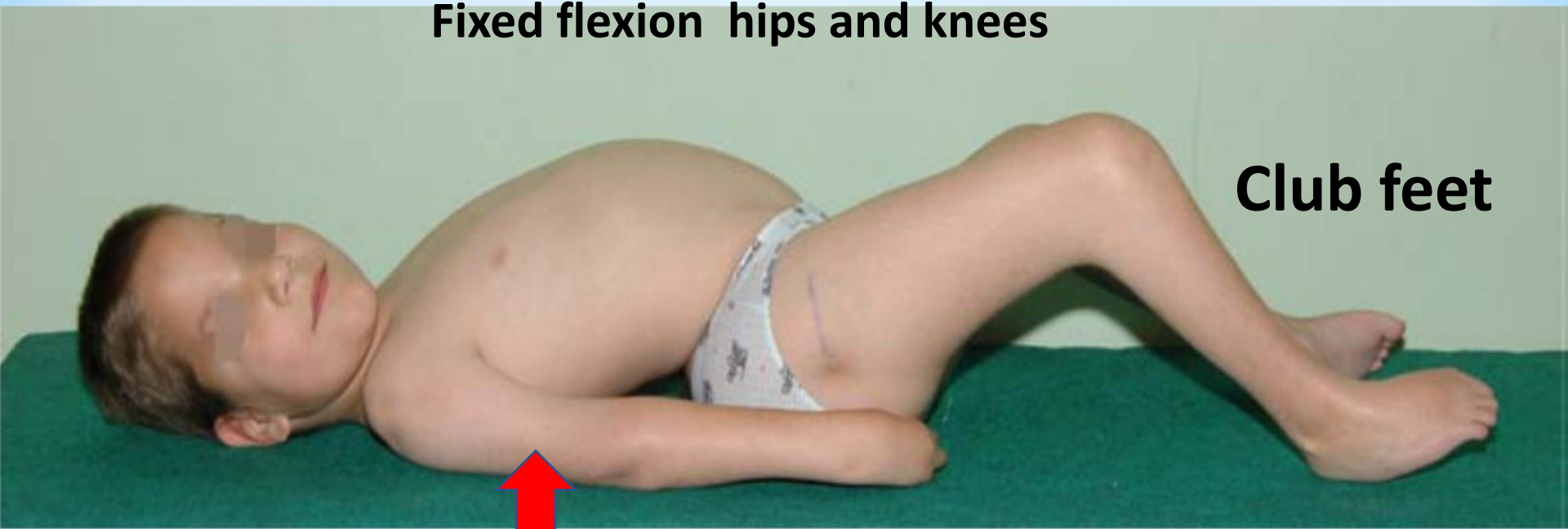
Thumb in palm deformity



How they using the hand for writing

Fixed flexion hips and knees

Club feet



Internal rotation shoulders
Elbow extension with absent crease
Thumb in palm

- * Rx: Aggressive PT at birth
- * Casting, bracing, surgical releases
- * Normal intelligence

2- Progressive

- * **Muscular Dystrophy**
- * **Neurofibromatosis**
- * **Peroneal Muscular Atrophy**
- * **Friedrich's Ataxia**
- * **Polio**

Muscular Dystrophy - Myopathy

- * Progressive hereditary degenerative weakness of skeletal muscles
- * 2-3 of 10,000 males
- * DX: Muscle Bx. ^ CPK , abnormal EMG
- * Gower's sign –climb up legs w/ hands
- * **Duchenne's** most common & severe < 20 yr life span
- * **Becker's** seen at 7yrs < 40 yr life span

1. Duchenne MD

- a) Most common and most severe, **X-linked recessive**, high mutation rate
- b) **Male** to female, 9:1
- c) Onset usually apparent by **age 3**
- d) Difficulty climbing stairs, use arms to rise from chair
- e) **Gower's sign**, waddling gait, toe walking

2. Becker's MD

X-linked recessive

- b) Benign form, onset 5-20, slow progression

Pseudohypertrophy of the calf muscles is apparent in Duchenne's dystrophy due to the degeneration of muscle tissue and accumulation of fat

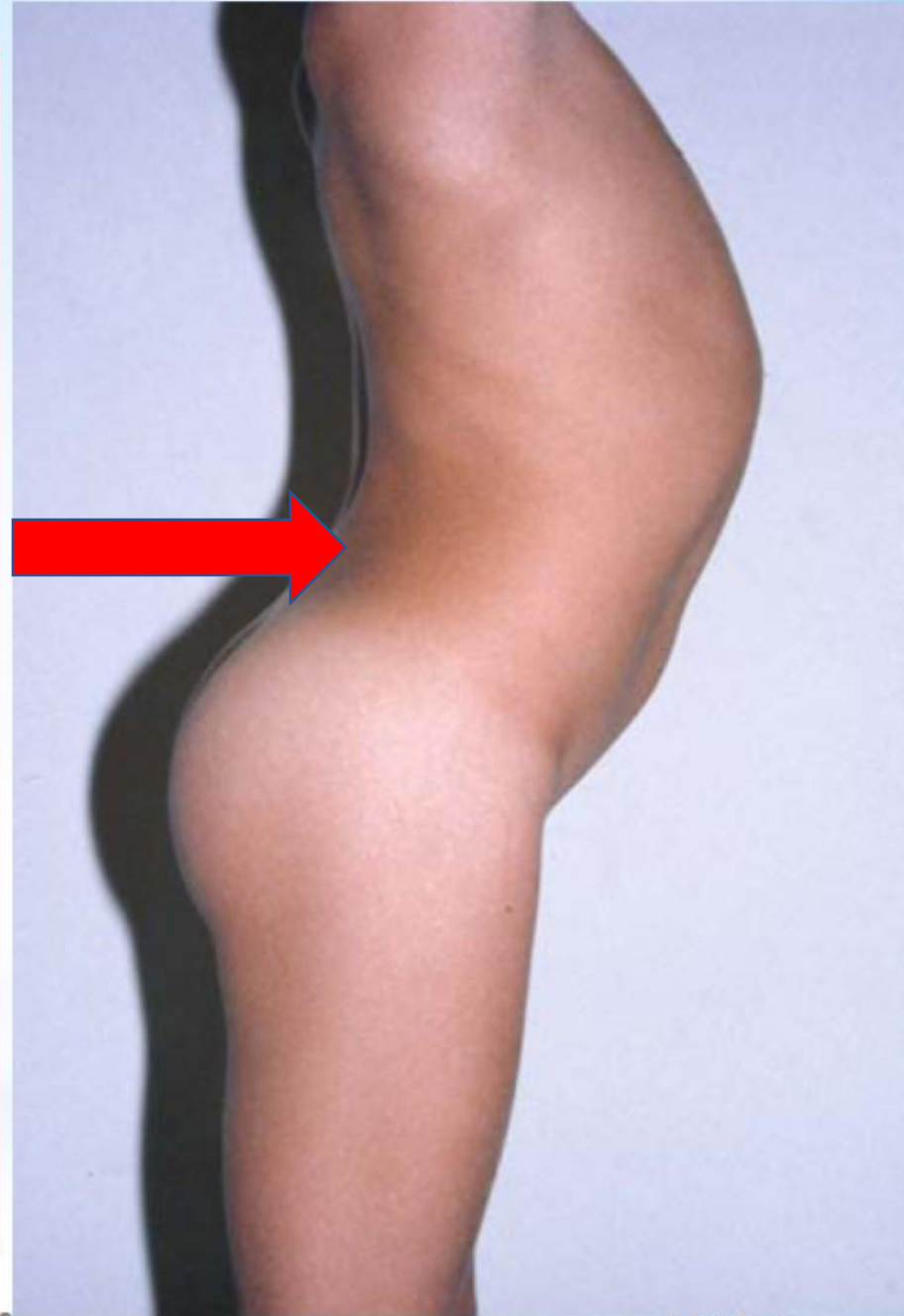


Tip Toes walking

**1-Weakness of the
gluteal musculature
→specific posture in
muscular dystrophy.**

**2-Dorsal shift of the
Trunk**

**3- increased lumbar
lordosis and anterior
tilt of the pelvis**





Gower's sign

Final outcome → Death

- * Respiratory - cardiac complications
- * Malignant hyperthermia



Neurofibromatosis

- * Von Recklinghausen's disease
- * Progressive
- * Tumors in central and peripheral nervous system
- * 1:400,000 births M=F , 4 Stages
- * Dx: café au lait spots >6
- * Lisch nodules in iris



- * Triggered by puberty
- * Spinal deformities 10-15%
- * Scoliosis, pseudoarthrosis
- * Most common PF to cancer for children
- * Elephantiasis
- * Multi system effected



Peroneal Muscular Atrophy

- * **Charcot-Marie-Tooth** – hereditary atrophy of peripheral and motor nerve roots & freq spinal cord
- * Etiol: unknown 2-5 100,000 people M>F
- * Dx: decrease sensation / function
- * Rx: orthotics – surgical releases

Bilateral Pescavus



Poliomyelitis

a viral infection localized in the AHC of the SC and certain brain stem motor nuclei

- * Incidence rising

- = immunization refusal by parents

- = Poor countries

- * Sx: fever, malaise, muscle pain, paralysis, some recover 4 mos-2yrs

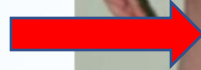
- * Continuous disease activity has been reported in spinal cord segments 20 years after disease onset.

Complications of polio infection

Life-long severe muscle weakness, often in the leg, causing the foot to drop and making walking difficult.

Sensation intact

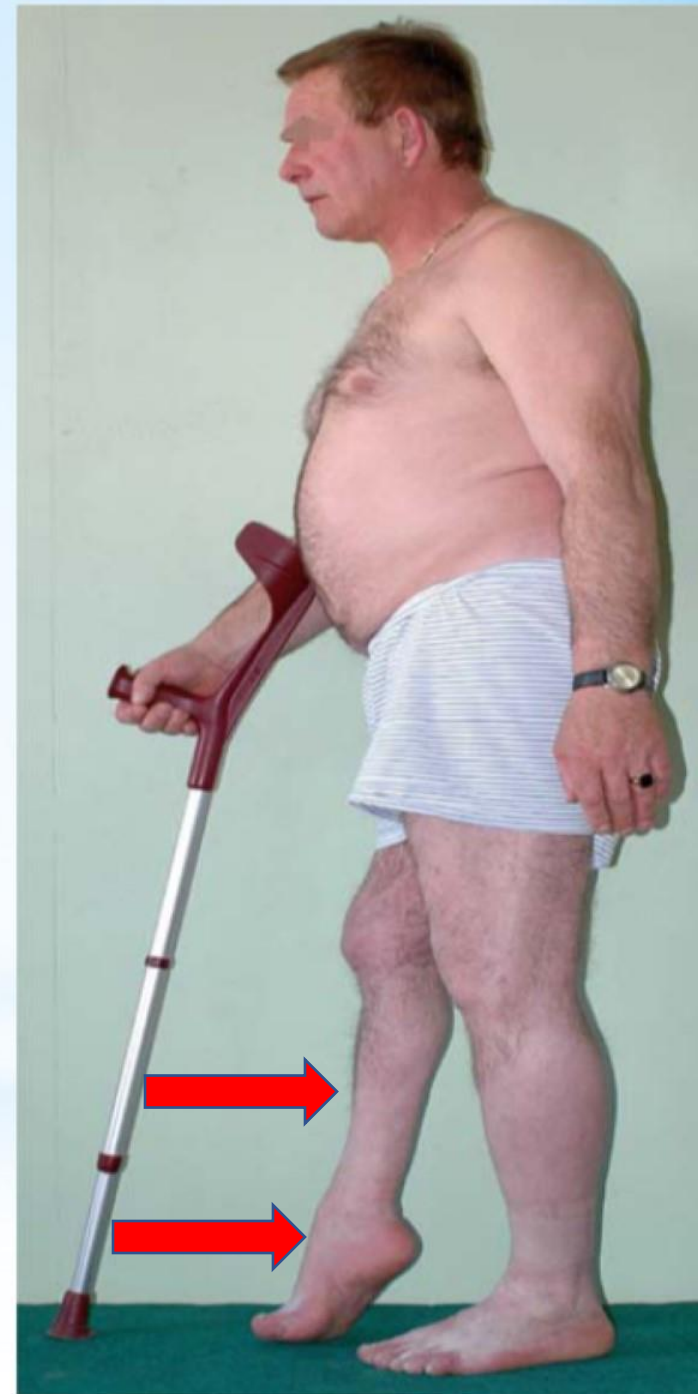
- 1-Paralyzed left leg has atrophied calf muscle**
- 2- LLD**
- 3-weakness of quadriceps muscle**





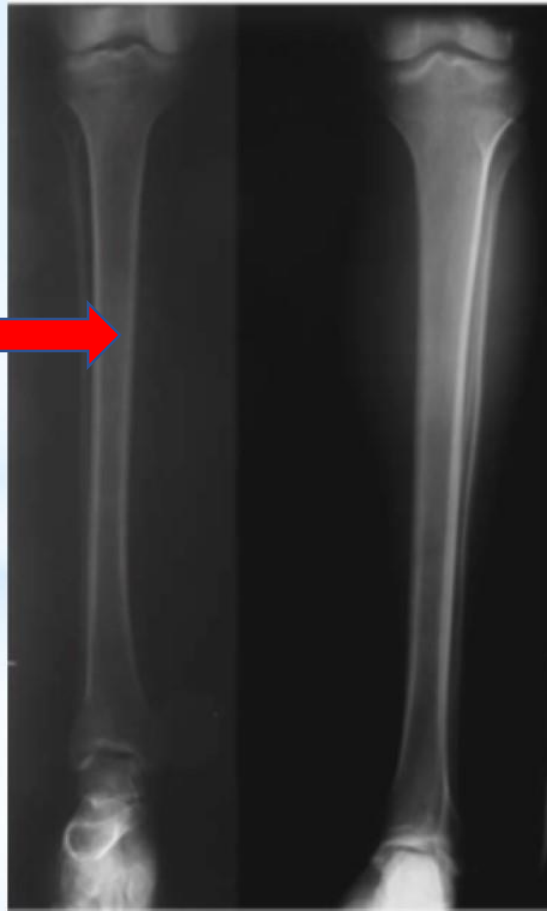
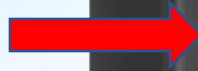
Professor Freih Ab

LLD





Bone atrophy



ORTHOPAEDICS AND TRAUMA

ORTHOPAEDICS AND TRAUMA **MADE EASY**

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A SYNOPSIS
FOR
MEDICAL STUDENTS
AND
JUNIOR ORTHOPAEDIC
TRAINEES

MADE EASY

PROFESSOR FREIH ODEH ABU HASSAN

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The University of Jordan
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دار الفُرْقَان للنشر والتوزيع

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