CEREBRAL PALSY

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Defenition

- (CP) refers to a heterogeneous group of conditions involving permanent motor dysfunction that affects muscle tone, posture, and movement .
- The motor impairment results in limitations in functional abilities and activity, which can vary in severity.
- It can affect the cortex and basal ganglia and cerebellum .
- Although the disorder itself is not neurodegenerative, the clinical expression may change over time as the central nervous system matures.

Epidemology and etiology

- CP is the most common and costly form of chronic motor disability that begins in childhood.
- This can happen prenataly and during pregenancy and after birth .
- prevalence is much higher in premature low birth weight infants (leading to peri-natal asphyxia), and twin births, congenital malformations .
- Most children with CP, except in its mildest forms, are diagnosed in the first 18 months of life when they fail to attain motor milestones or show abnormalities such as asymmetric gross motor function, hypertonia, or hypotonia.
-

Comorbidities

- basically they have motor disorders, but they may or may not have other symptoms:
- learning difficulties 60%
- epilepsy 30%
- ophthalmoplegia , squint 30%
- hearing problems 20%
- speech problems pain 60%
- bladder problems 60%
- sleeping disorder 20%

Risk factor

Table 10-10	Risk Factors for Cerebral Palsy
PREGNANCY AND BIRTH	
Low socioeconomic status	
Prematurity	
Low birth weight/fetal growth retardation (<1500 g at birth)	
Maternal seizures/seizure disorder	
Treatment with thyroid hormone, estrogen, or progesterone	
Pregnancy complications	
Polyhydramnios	
Eclampsia	
Third-trimester bleeding (including threatened abortion and placenta previa)	
Multiple births	
Abnormal fetal presentation	
Maternal fever	
Congenital malformations/syndromes	
Newborn hypoxic-ischemic encephalopathy	
Bilirubin (kernicterus)	
ACQUIRED AFTER THE NEWBORN PERIOD	
Meningitis	
Head injury	
Car crashes	
Child abuse	
Near-drowning	
Stroke	

- **Prematurity** : periventricular leukomalacia (PVL), intraventricular hemorrhage (IVH) , bronchopulmonary dysplasia (BPD)
- **Multiple twins** : death of a co-twin greatly increases the risk of CP .
- **Intracranial hemorrhage (ICH)**
- **Stroke**
- **Brain malformation .**

- **Intrauterine infection :**
  [TORCH infections]
  Maternal chorioamnionitis

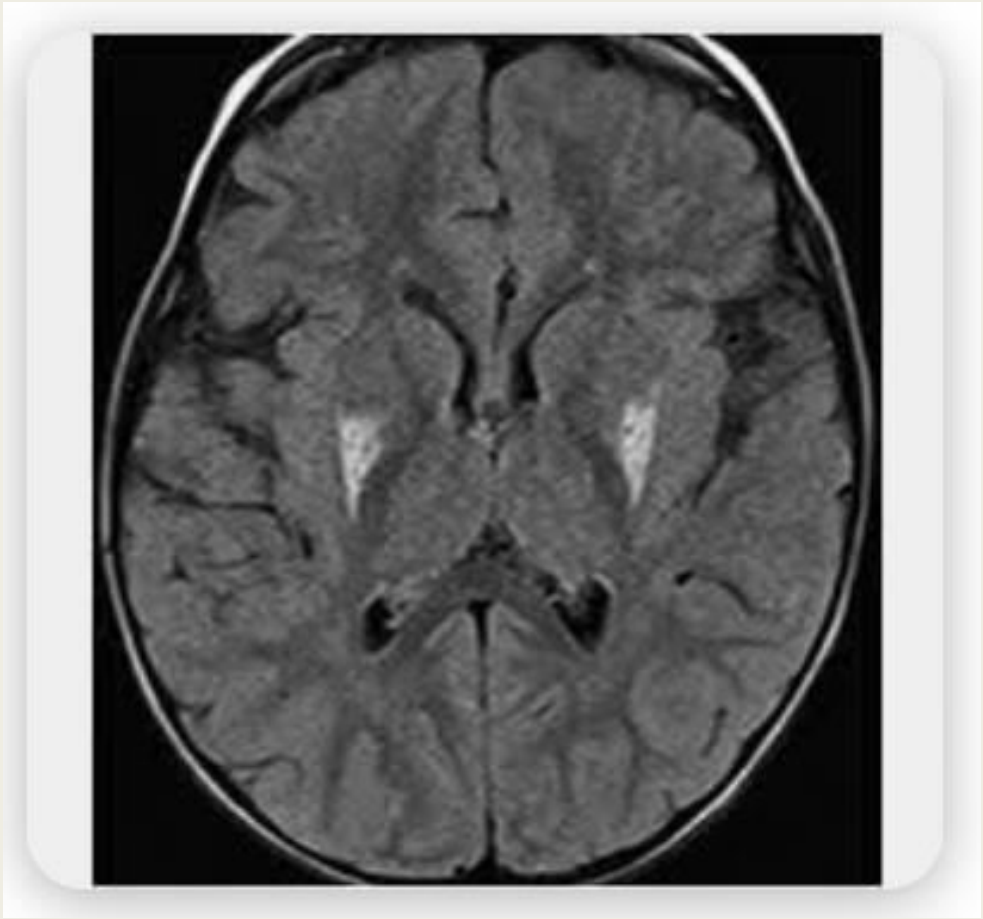
- **Genetic Susceptibility :**

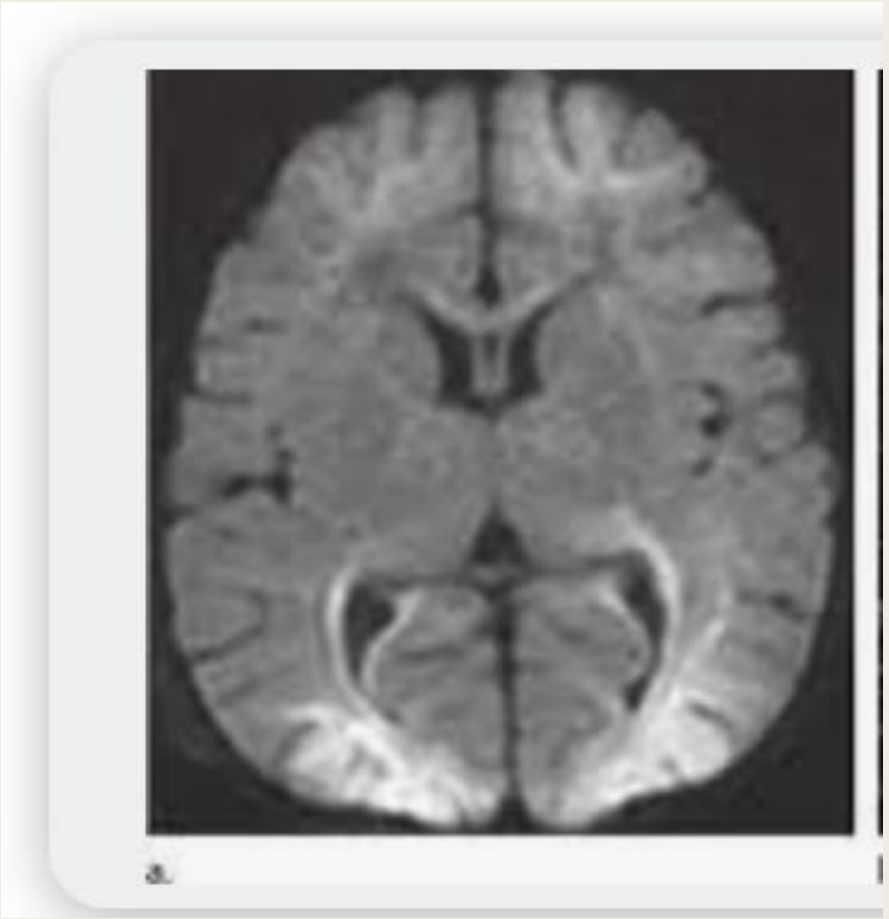
✓ The aggregation of CP in groups with high consanguinity and observations of increased familial risk for CP suggests a genetic contribution to CP risk.

✓ Several genetic polymorphisms have been associated with susceptibility for CP , However, only the association with prothrombin G20210A mutation was confirmed by a subsequent large study.

Perinatal hypoxic-ischemic encephalopathy

- Neonates with severe intrapartum hypoxia-ischemia may have seizures, coma, hypotonia, dysfunction of other organ systems, a persistently low Apgar score, and evidence of profound metabolic acidosis.
- on MRI two main patterns of injury is seen : central [ganglia thalamic predominant pattern] , and perphiral [watershed predominant] or it can be global .





CLASSIFICATION

CLASSIFICATION

- Spastic 75-80%
  1- MONOPLEGIA (Rare)
  2- DIPLEGIA
  3- HEMIPLEGIA
  4- QUADRIPLEGIA

- Dyskientic 10-15%
  1- Chorioathetoide
  2- Athetoid
  3- Dystonic
- Ataxic 1%
- Mixed 10%

Spastic

- Most common type (80%)
- the problem in the cerebral cortex
- stiff and difficulty moving

SPASTIC ~ 70% cases

↳ tight/stiff muscles

↳ can't inhibit

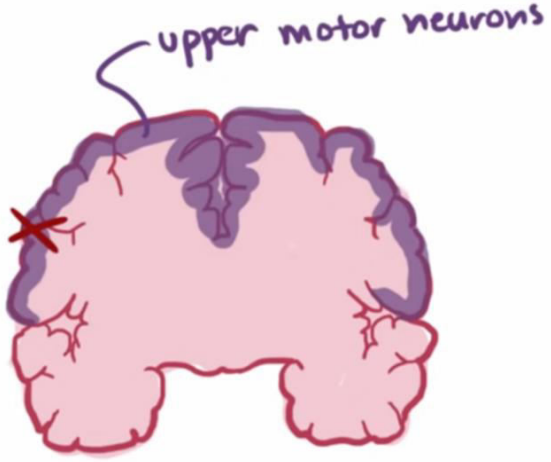
HYPERTONIA

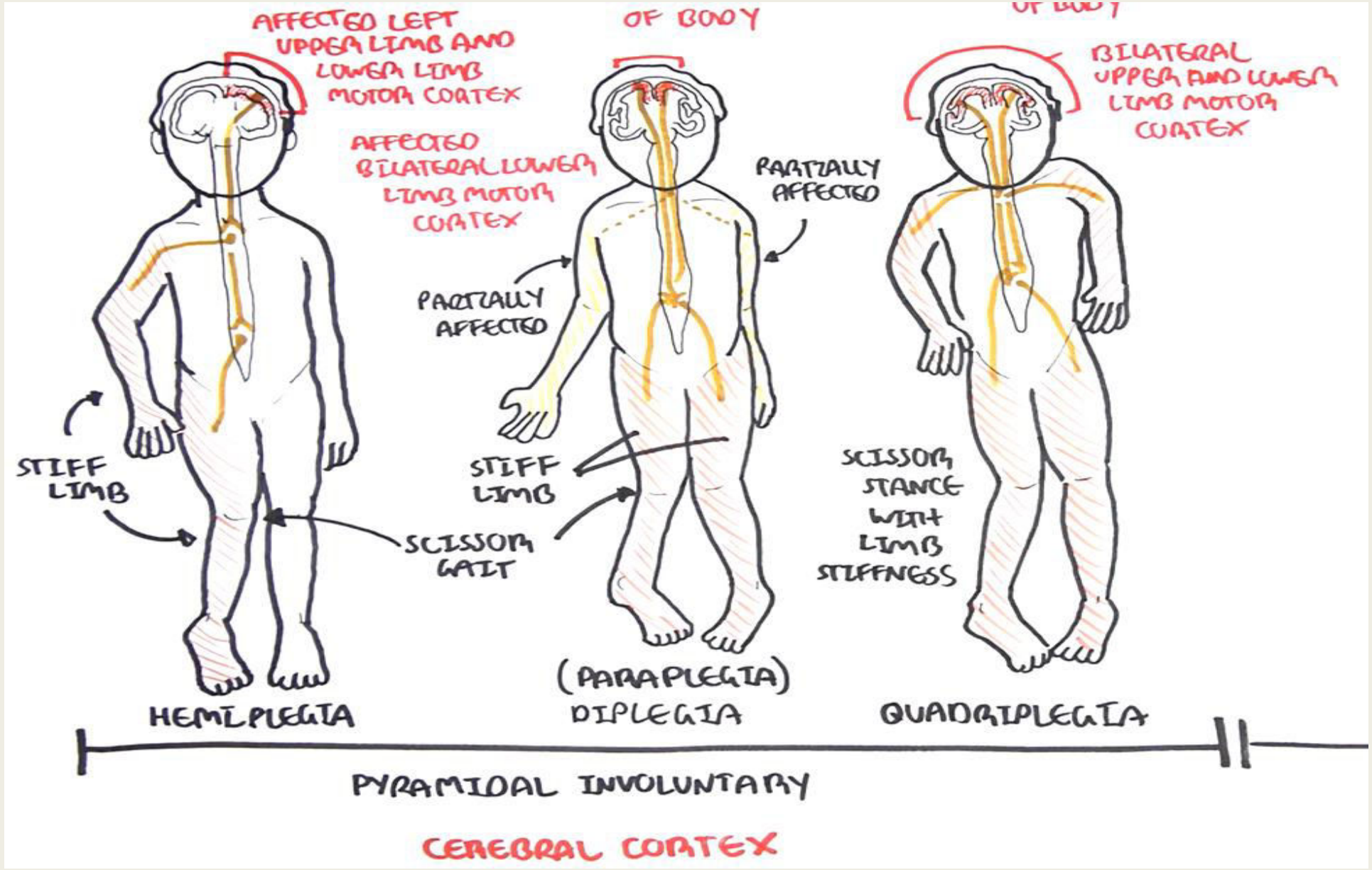
abnormal increase — muscle activity

LESION
(abnormality)

* ability to receive **GABA** impaired

GABA: inhibitory




AFFECTED LEFT UPPER LIMB AND LOWER LIMB MOTOR CORTEX
OF BODY
BILATERAL UPPER AND LOWER LIMB MOTOR CORTEX
AFFECTED BILATERAL LOWER LIMB MOTOR CORTEX
PARTIALLY AFFECTED
PARTIALLY AFFECTED
STIFF LIMB
STIFF LIMB
SCISSOR GAIT
SCISSOR STANCE WITH LIMB STIFFNESS
HEMIPLEGIA
(PARAPLEGIA) DIPLEGIA
QUADRIPLEGIA
PYRAMIDAL INVOLUNTARY
CEREBRAL CORTEX

Spastic hemiplegia

- 21 to 40% of CP cases.
- Typically affect term infants of normal birth weight.
- One side of the body is affected.
- The arm typically is more affected than the leg.
- Infants with spastic hemiplegia have decreased spontaneous movements on the affected side
- Show hand preference at a very early age
- The arm is adducted at the shoulder

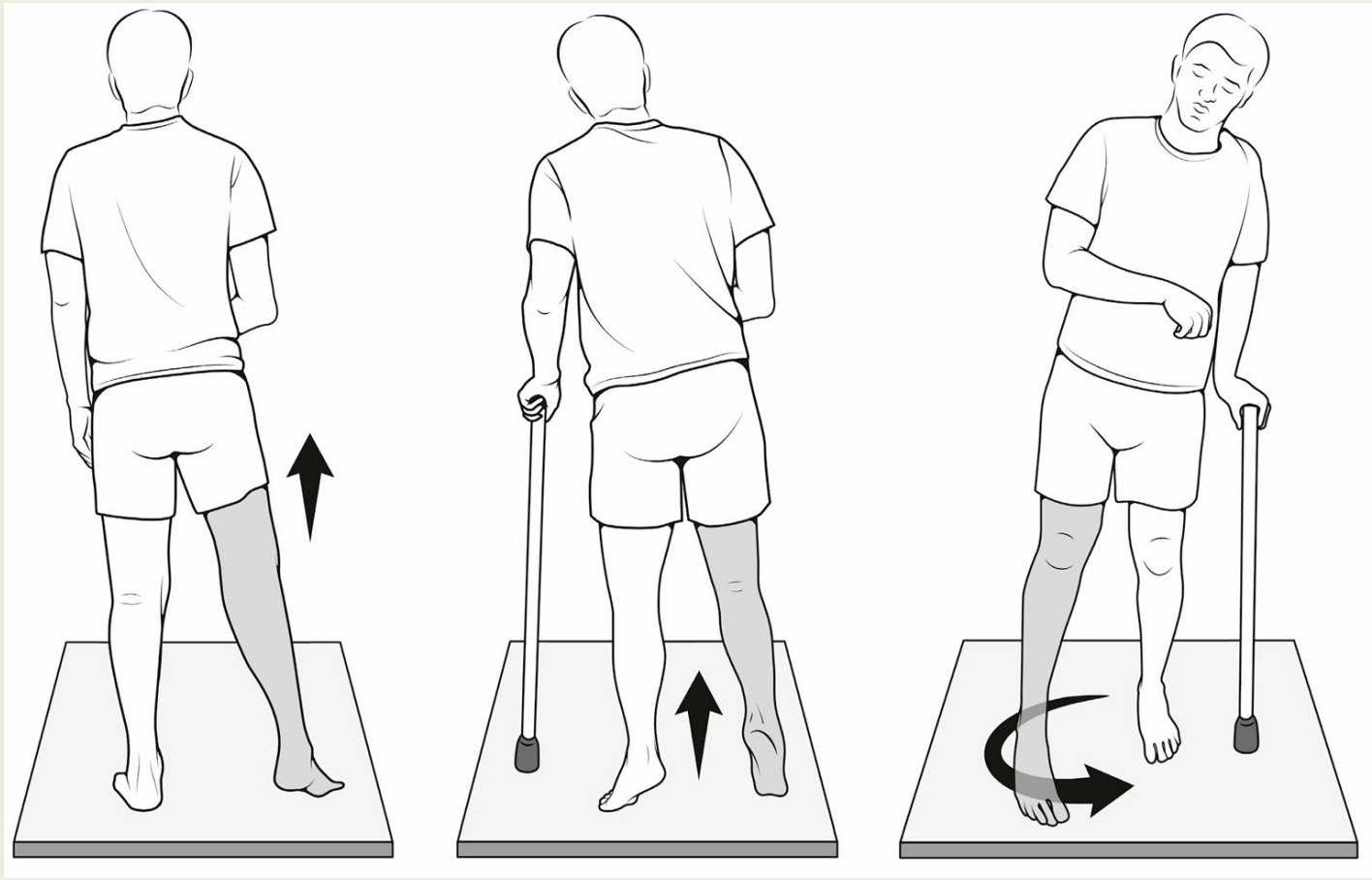
and flexed at the elbow, the forearm is pronated,
and the wrist and fingers are flexed with the hand closed.

- The hip is partially flexed and adducted,

and the knee and ankle are flexed;
the foot may remain in the equinovarus or
calcaneovalgus position.

- In mildly affected patients, postural abnormalities are more apparent during walking or running; however, unless severe intellectual disability is present, independent walking usually occurs at the appropriate age or is only slightly delayed.
- Delayed walk 18-24 months
- Circumductive gait
- Tip toe walk affected leg
- Equinovarus deformity of the foot
- DTR exaggerated

- Examination of the extremities may show growth arrest, particularly in the hand and thumbnail
- Ankle clonus and a Babinski sign may be present, the deep tendon reflexes are increased, and weakness of the hand and foot dorsiflexors is evident
- circumduction gait



Spastic diplegia

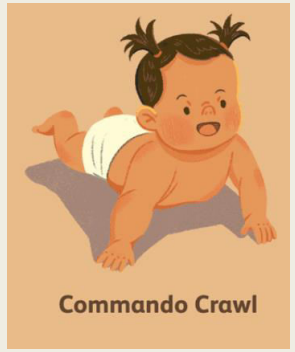
- 13 to 25% of CP cases
- Strongly associated with damage to the immature white matter during the vulnerable period of immature oligodendroglia between 20-34 wk of gestation
- First few months – Hypotonia of the lower limbs with delayed functional maturation
- Affect the lower limb more
- likelihood of seizures is minimal
- often have learning disabilities and deficits in other abilities, such as vision

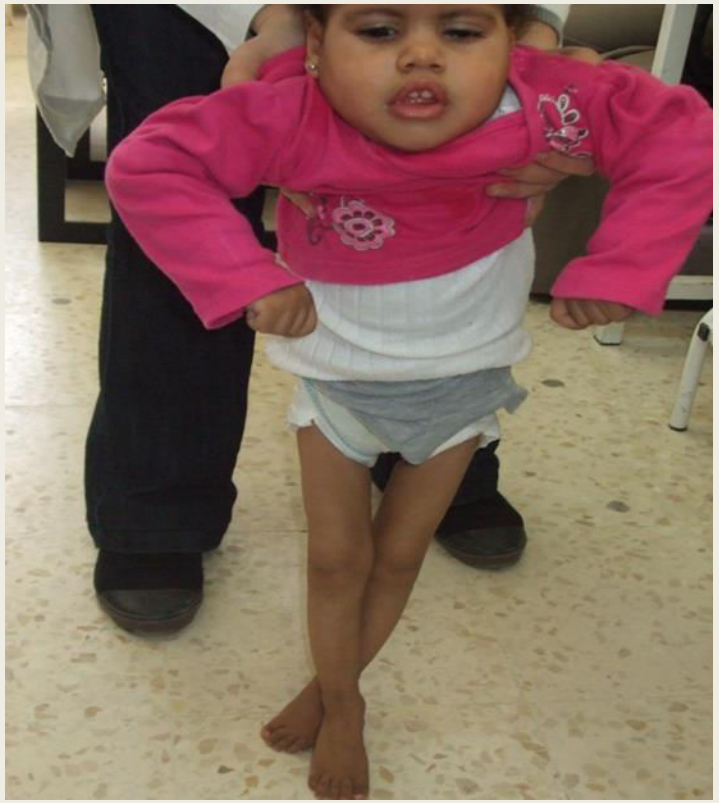
- The first clinical indication of spastic diplegia is often noted when an affected infant begins to crawl

The child uses the arms in a normal reciprocal fashion but tends to drag the legs behind more as a rudder (commando crawl)

- If there is paraspinal muscle involvement, the child may be unable to sit
- the feet are held in a position of equinovarus, and the child walks on tiptoe
- Severe spastic diplegia is characterized by disuse atrophy and impaired growth of the lower extremities and by disproportionate growth with normal development of the upper torso

- Examination of the child reveals spasticity in the legs with brisk reflexes, ankle clonus, and a bilateral Babinski sign
- When the child is suspended by the axillae, a scissoring posture of the lower extremities is maintained
- Commando crawl






BRADLEY

SPASTIC QUARIPLEGIA

- 20 to 43% of CP cases.
- The most severe form of CP
- Most commonly term SGA infants, but can also occur in preterm infants.
- All limbs are affected.
- Moderate or severe psychomotor delay.
- Poor head control..
- Adduction of the thighs results in typical scissoring of the legs.
- By 9 to 10 months of age, infants when pulled to sitting

  are unable to flex the legs and have poor truncal balance.
- Children often are severely handicapped

- Associated :

- Mental Retardation

-chronic respiratory insufficiency

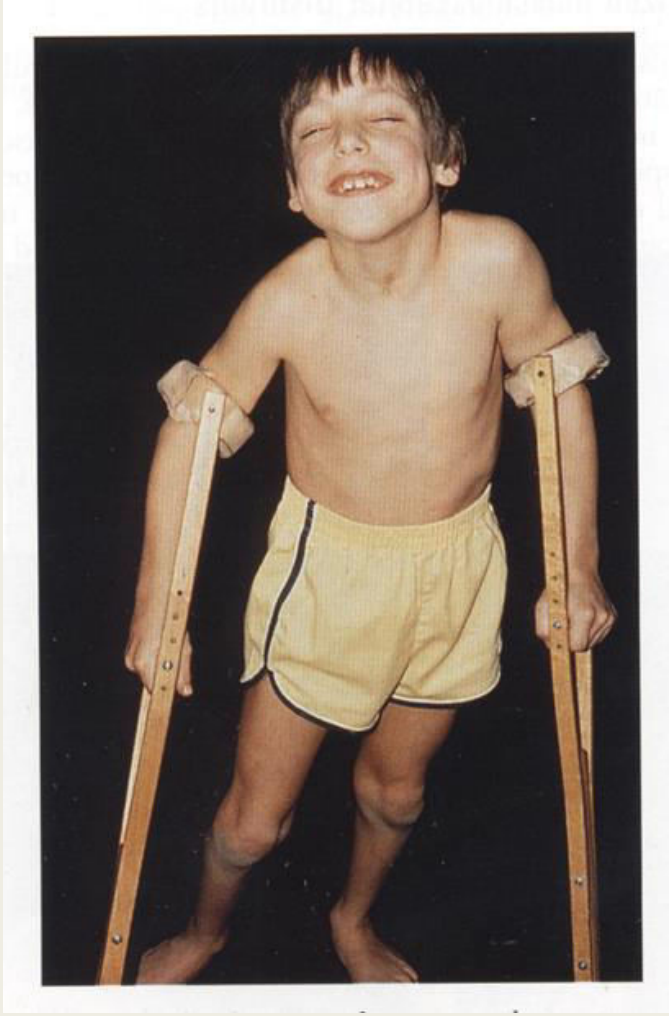
- seizures

- swallowing difficulties

- aspiration pneumonia

- speech

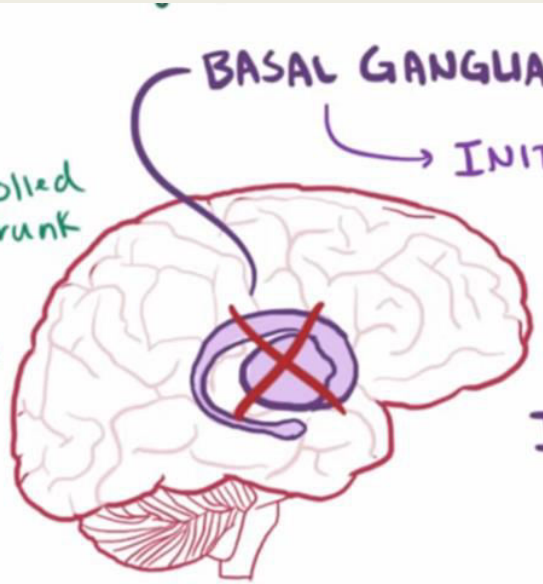
- visual abnormalities



- Athetoid CP, also called choreoathetoid, extrapyramidal, or dyskinetic CP, is less common than spastic CP and makes up approximately 15–20% of patients with CP.
- Affected infants are characteristically hypotonic with poor head control and marked head lag and develop variably increased tone with rigidity and dystonia over several years.
- Unlike spastic diplegia,
Upper limbs are generally more affected in extrapyramidal CP

DYSKINETIC (ATHETOID)

- Dystonia ~ random, slow, uncontrolled movements in limbs / trunk
- Chorea ~ random, "dance-like" movements



BASAL GANGLIA

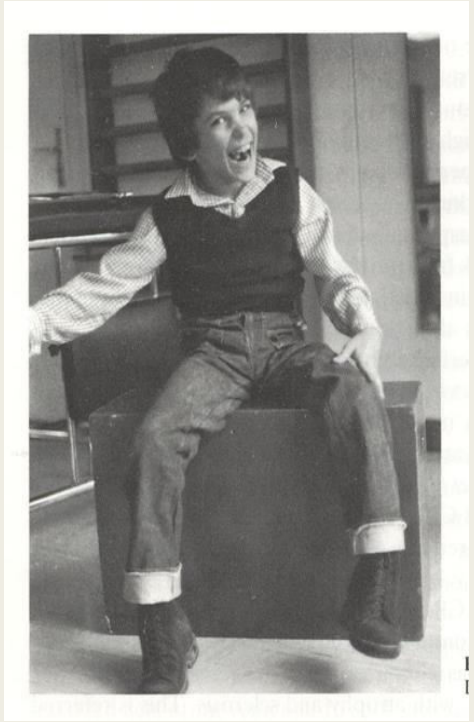
→ INITIATE & ~~PREVENT~~ movements

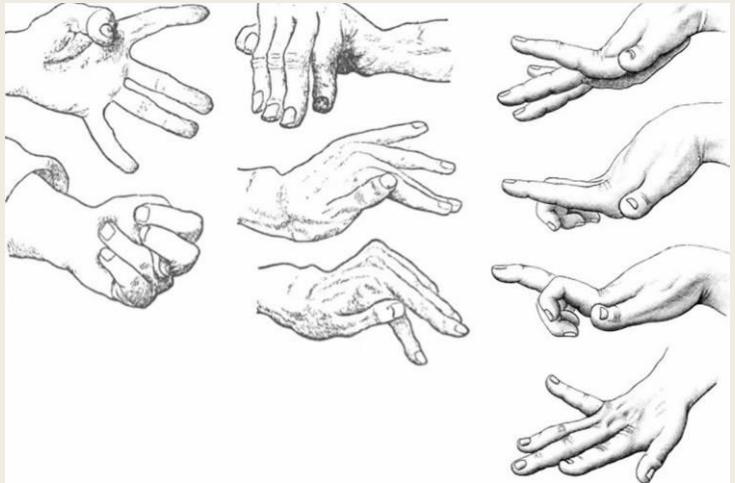
INVOLUNTARY MOVEMENTS

Dyskinesia

- Most cases are caused by severe perinatal asphyxia resulting in injury to the thalamus, basal ganglia, hippocampus, reticular formation, and/or cerebellum.
- Predominantly term infants.
- In early infancy:
  - Reduced spontaneous movement.
  - Persistence of primitive reflexes
  - Involuntary grimacing
  - Drooling
  - Delayed psychomotor development

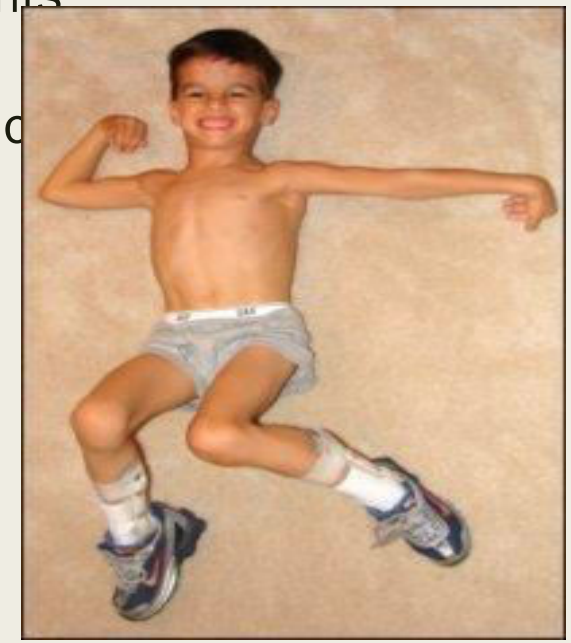
- Age 2 to 3 years:
  - Involuntary movements are apparent
  - Abnormal posturing:
  - Extension patterns in the supine position
  - Flexion with shoulder retraction in the prone position
  - Head usually is persistently turned to one side
  - Variable degree of dysarthria and intellectual disability.





Choreoathetotic CP:

- Chorea consists of rapid, irregular, unpredictable contractions of individual muscles or small muscle groups that involve the face, bulbar muscles, proximal extremities, and fingers and toes.
- Athetosis consists of slow, smooth, writhing movements that involve distal muscles.
- Movements may be induced or accentuated by emotic or change in posture.
- Athetosis is most apparent during reaching
- Stress, excitement, or fever may exacerbate chorea
- Primitive reflexes often are retained
- Oropharyngeal difficulties occur commonly
- Speech affected
- Seizures are common
- ASSOCIATED; Asphyxia and hyperbilirubinemia



Dyskinetics (dystonia):

- Dystonia — The second category of dyskinetic CP is characterized by dystonia, although tension and persistent neonatal reflex patterns often occur.
- Repetitive, patterned, twisting, and sustained movements

of the trunk and limbs that may be either slow or rapid.

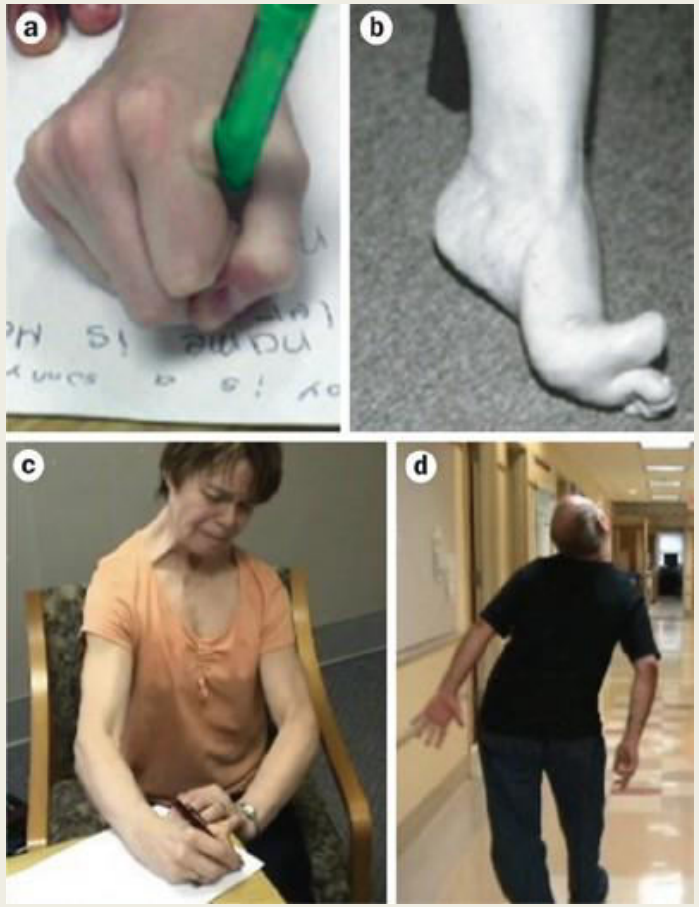
- "Tension," a sudden involuntary increase in tone

affecting both flexor and extensor muscles,

may occur during attempted movement or with emotion

- Tendon reflexes are normal or may be difficult to elicit.
- Clonus and extensor plantar responses are absent.
- Affected patients usually are severely disabled in

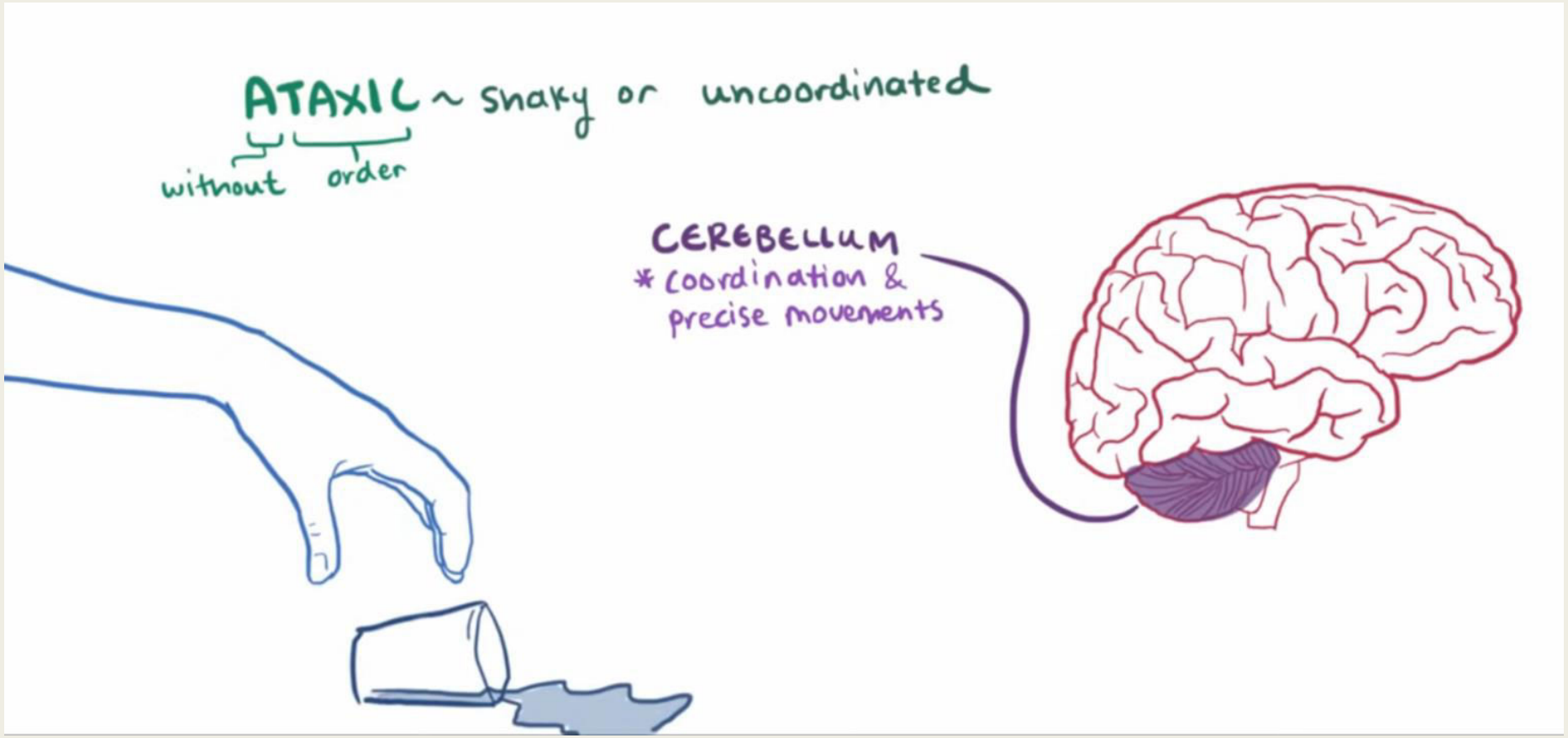
all four limbs, the trunk, and pharyngeal muscles.

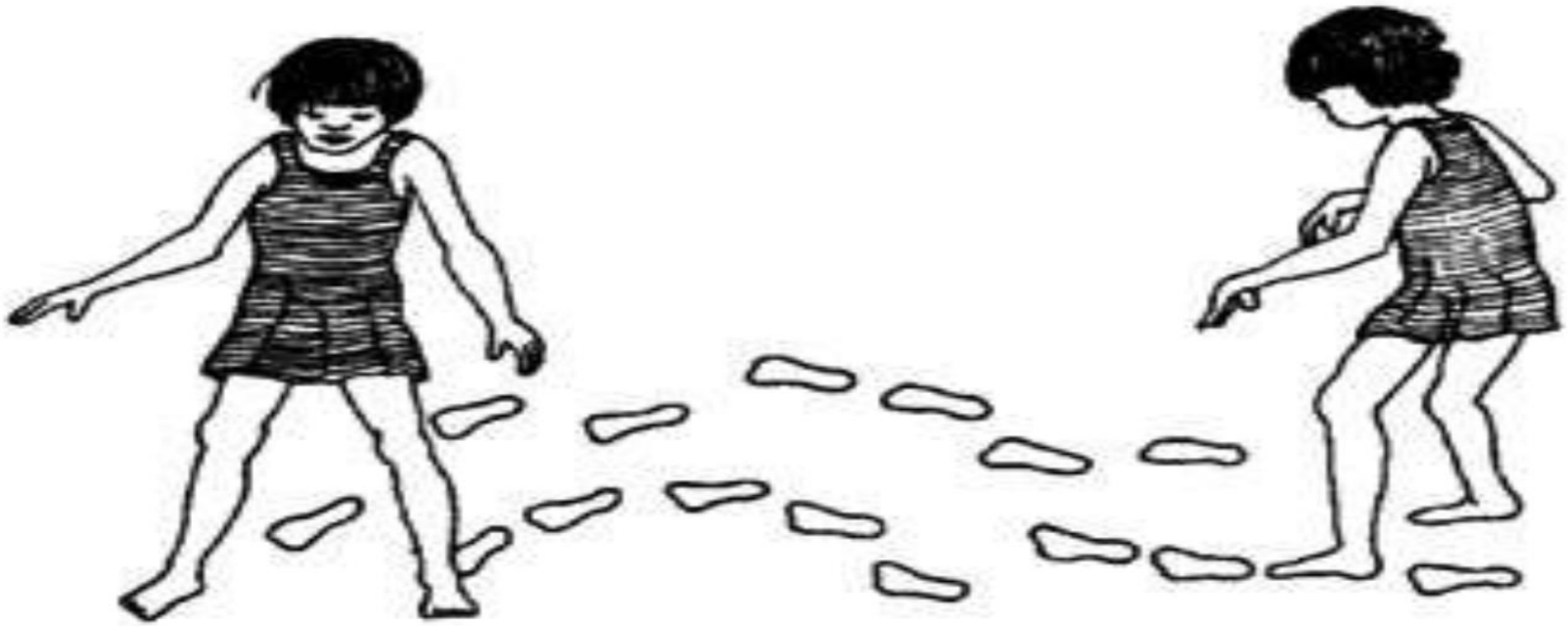


ATAXIC CP

- 4 to 13% of CP cases
- Most cases are caused by early prenatal events.
- Some cases have genetic causes, including:
- Cerebellar hypoplasia , Granule cell deficiency,

  Joubert syndrome.
- Acquired cases --- hydrocephalus
- Term infants.
- Hypotonia and incoordination
- Motor milestones and language skills typically are delayed.
- Ataxic movements.
- Ataxia usually improves with time.
- Speech typically is slow, jerky, and explosive.
- Wide based gait.


ATAXIC ~ shaky or uncoordinated
without
order
CEREBELLUM
* coordination & precise movements



To keep her balance the child with ataxia walks bent forward with feet wide apart. She takes irregular steps, like a sailor on a rough sea or someone who is drunk.

MIXED FORMS CP

- Presence of athetoide movement in one limb and hemiplegia in the other or presence of ataxia and spasticity.
- Functional analysis of individual patient is the bases of therapy.

Associated comorbidities are common and include pain (in 75%), cognitive disability (50%), hip displacement (30%), seizures (25%), behavioral disorders (25%), sleep disturbances (20%), visual impairment (19%), and hearing impairment (4%).

MOTOR SYNDROME (APPROX. % OF CP)	NEUROPATHOLOGY/MRI	MAJOR CAUSES
Spastic diplegia (35%)	Periventricular leukomalacia Periventricular cysts or scars in white matter, enlargement of ventricles, squared-off posterior ventricles	Prematurity Ischemia Infection Endocrine/metabolic (e.g., thyroid)
Spastic quadriplegia (20%)	Periventricular leukomalacia	Ischemia, infection
	Multicystic encephalomalacia Cortical malformations	Endocrine/metabolic, genetic/developmental
Hemiplegia (25%)	Stroke: in utero or neonatal Focal infarct or cortical, subcortical damage Cortical malformations	Thrombophilic disorders Infection Genetic/developmental Periventricular hemorrhagic infarction
Extrapyramidal (athetoid, dyskinetic) (15%)	Asphyxia: symmetric scars in putamen and thalamus Kernicterus: scars in globus pallidus, hippocampus Mitochondrial: scarring of globus pallidus, caudate, putamen, brainstem No lesions: ? dopa-responsive dystonia	Asphyxia Kernicterus Mitochondrial Genetic/metabolic

APPROACH AND DIAGNOSIS

- Diagnosis is primarily **clinical** supported by **imaging**.
- Examination includes *assessment of active and passive range of motion*, *motor power*, *selective voluntary motor control*, *muscle tone*, and *sensation of the limbs*.
- Further testing is individualized based on the history and examination findings.

- The **goals** of the history and physical examination are to:
  1- Identify the clinical features and suspected classification of the type of CP, which may provide clues as to the underlying etiology and have implications regarding the likelihood of associated conditions.
  2- Rule out clinical suspicion of a progressive or neurodegenerative condition.
  3- Establish treatment goals and priorities.

- The diagnosis of CP is typically made be in the first 18 months, through earlier diagnosis is becoming increasingly common, when they fails to attain motor milestone or show abnormalities such as asymmetric gross motor function, hypertonia, hypotonia.
  Presentation generally points to a specific CP subtype, through there is substantial overlap in the clinical features of these subtypes.
- Motor milestones: not sitting by 8 months, not walking by 18 months, early asymmetry of hand function (hand performance) before 18 months.
  - definite hand preference before 18 months, suggests a one-sided muscle weakness and is s red flag for hemiplegia.
- Neurobehavioral signs.
- Developmental reflexes:delay in the disappearance or exaggeration of a developmental reflex may be an early indication of motor disaability.
- Motor tone and posture: persistent or asymmetric fisting may be present, poor head control .

History

1. Review of **perinatal history**, which can identify risk factors for CP. Look for the risk factors mentioned earlier.
2. Review of the newborn **screening results**, including newborn hearing test results and ophthalmologic evaluation if the child was in the NICU .
3. Assessment of **development** (delay in milestones)(motor, cognitive, primitive reflexes, speech) **(key diagnostic factor) .**
4. Poor feeding/frequent vomiting.
5. Irritability.
6. Screening for attention, behavioral, communication, and/or cognitive concerns .
7. Assessment of growth .
8. Evaluation of motor tone, posture, and coordination .

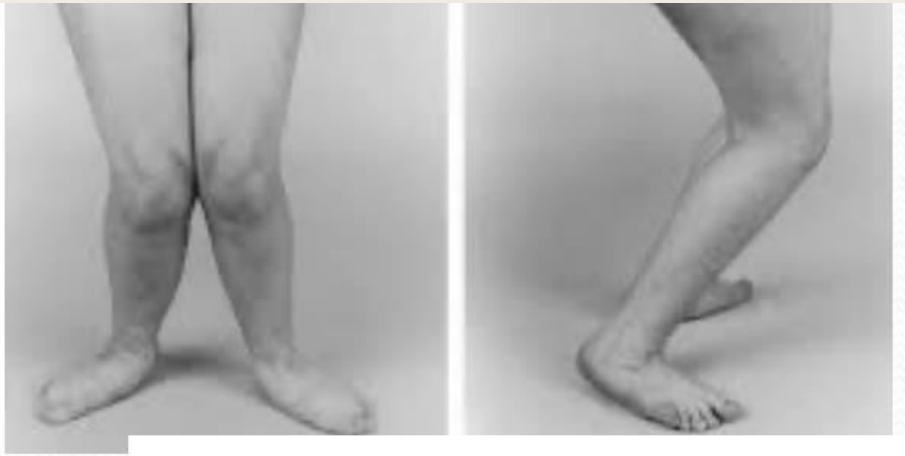
- Review of the **family history** – Relatives with any of the following should raise suspicion for a genetic cause of CP :
- Intellectual disability/developmental disabilities
- Seizures
- CP
- Neuromotor/movement disorders
- Neurobehavioral disorders
- Joint contractures/stiffness
- Thromboses/vascular accidents
- Congenital anomalies
- Infertility
- Recurrent miscarriages
- Stillbirths
- Adult-onset neurodegenerative conditions

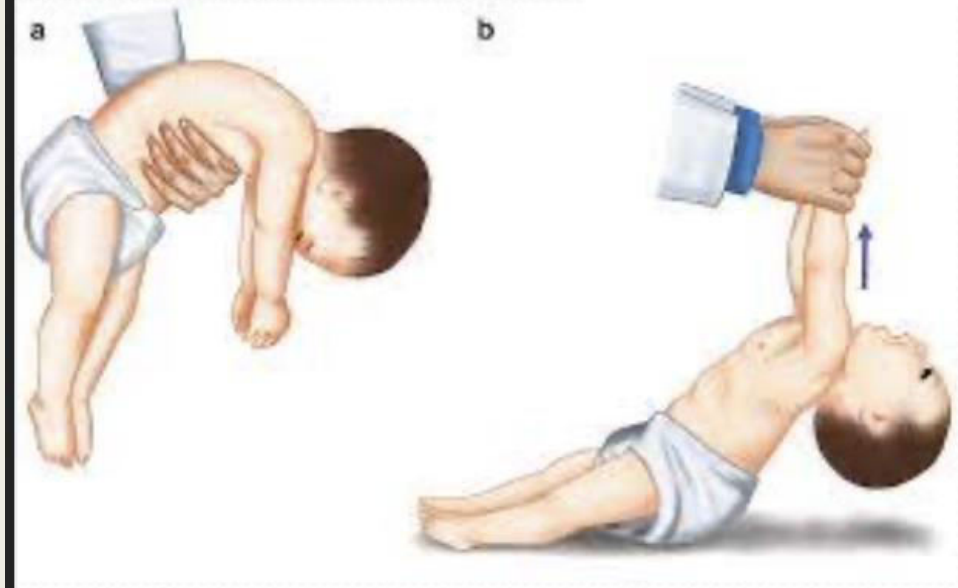
- **Physical examination :**

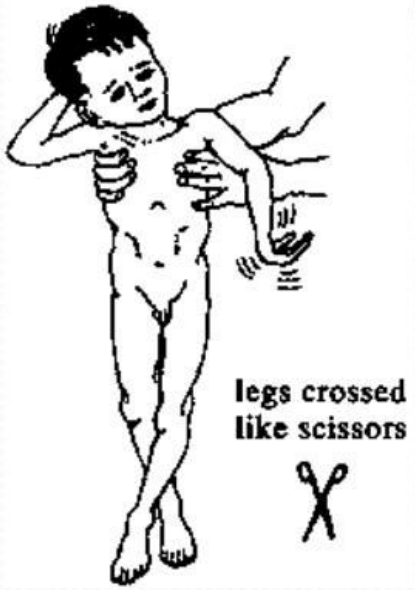
  1. Motor development exam as Poor head control after 3 months in cp patients .
  2. Growth parameters (**essential** and may facilitate early identification of children with microcephaly, macrocephaly, and growth impairments)
  3. Neuromotor exam : ventral suspension (c shape), using one side of the body or only arms to crawl, primitive reflexes, deep tendon reflexes

- look for :
  1. spasticity/clonus
  2. Floppy or limp body posture
  3. Toe walking/knee hyperextension
  4. Scissoring
  5. Crouched gait: excessive dorsiflexion –Contractures
  6. Muscle weakness
  7. Joint instability/dislocation
  8. Ataxia
  9. Chorea, athetosis

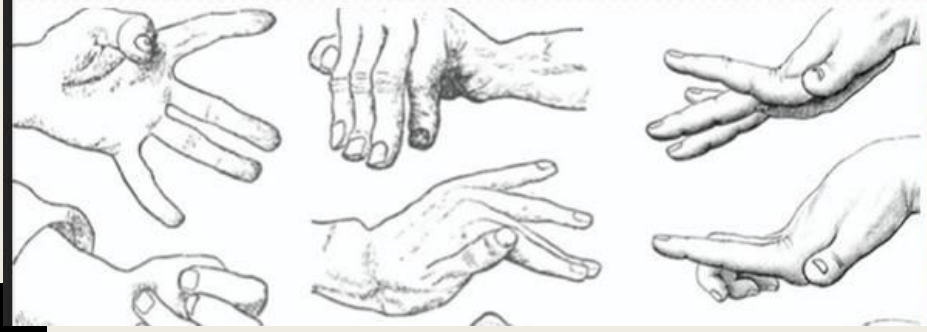

Gait
hip flexion
knee flexion
ankle
dorsiflexion

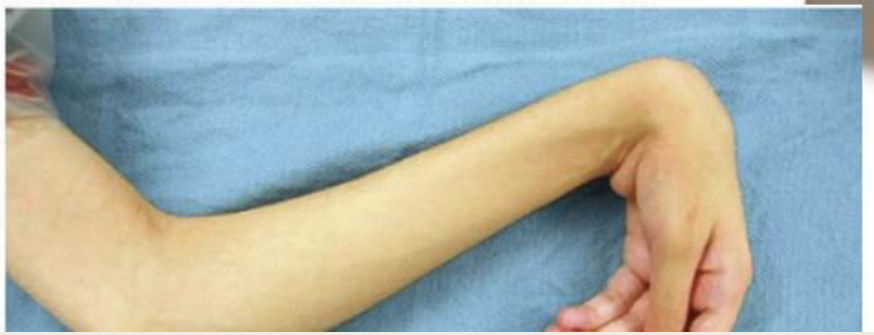


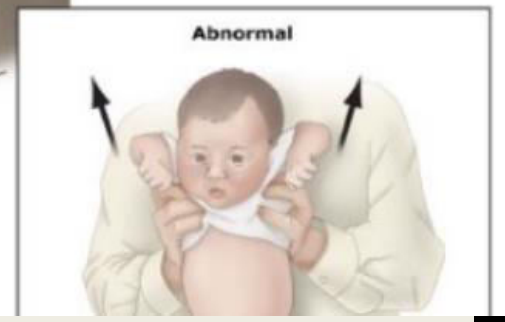

a
b

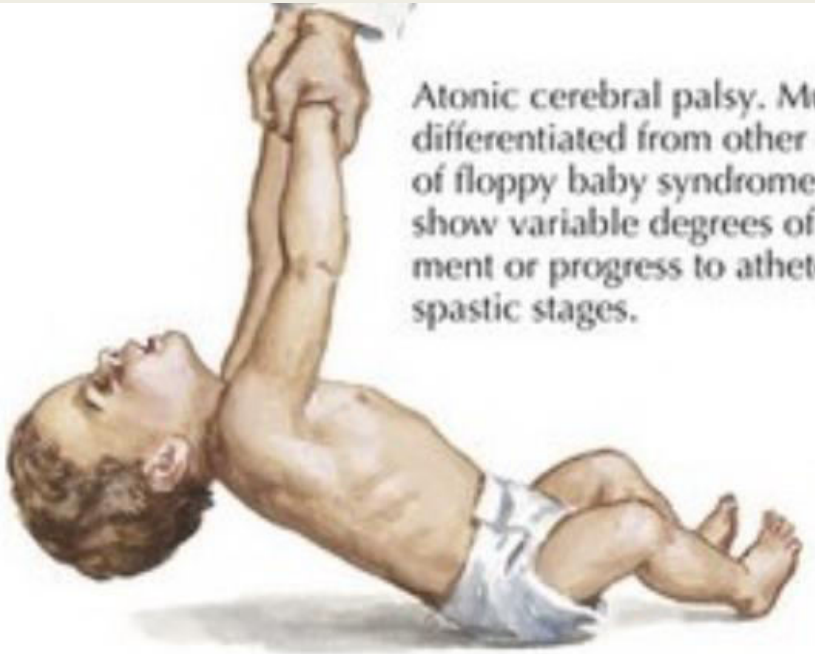

legs crossed
like scissors


Normal

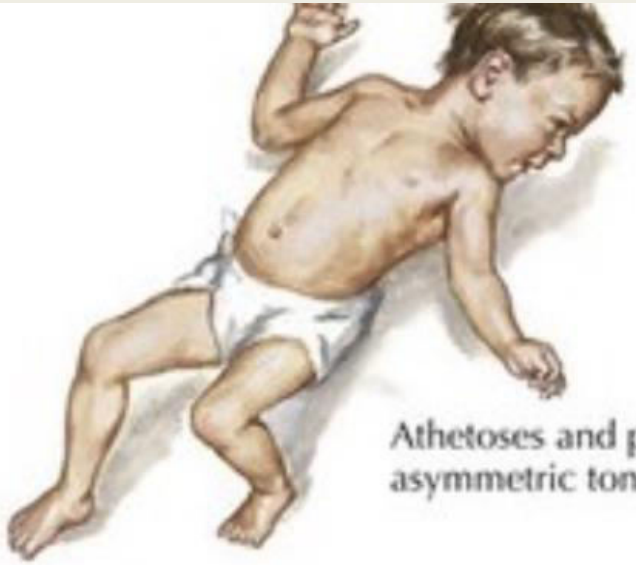





Abnormal

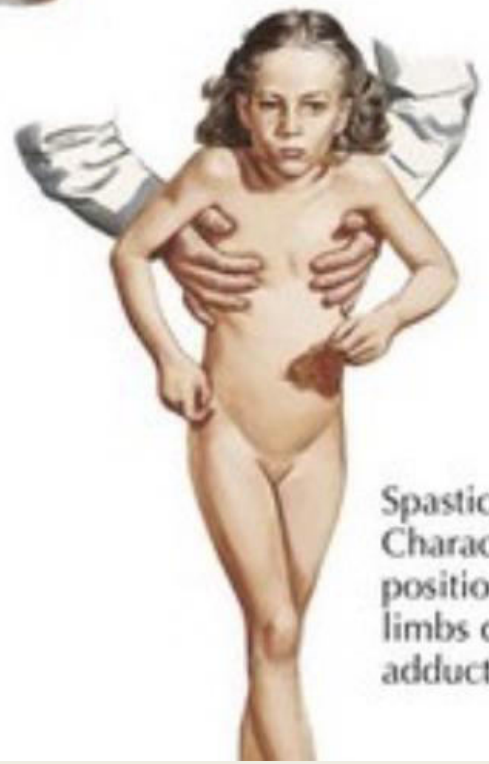


Atonic cerebral palsy. Must be differentiated from other causes of floppy baby syndrome. May show variable degrees of improvement or progress to athetoid or spastic stages.



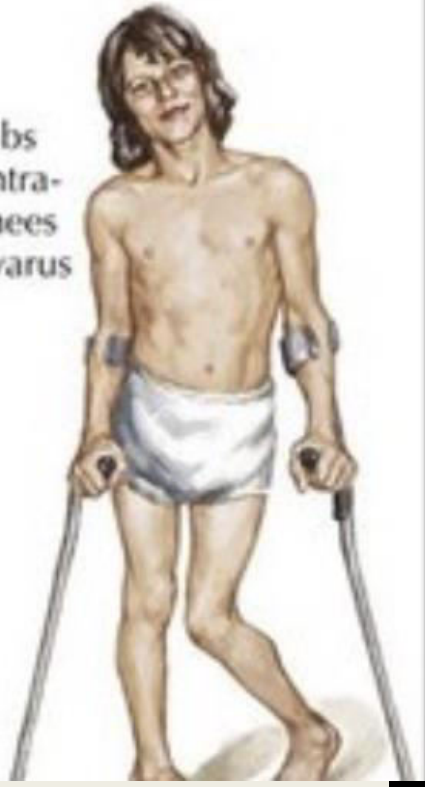
Athetoses and persistent asymmetric tonic reflex

Hemiparesis on right side. Hip and knee contractures and talipes equinus.



Spastic quadriparesis. Characteristic "scissors" position of lower limbs due to adductor spasm.

Diplegia (lower limbs more affected). Contractures of hips and knees and talipes equinovarus (clubfoot).



Diagnostic tests

- The diagnosis of CP is made clinically. No specific test confirms or excludes the diagnosis of CP. However, a diagnostic evaluation should be performed in all children with CP to identify the underlying cause of CP when possible and to exclude other conditions.

Diagnostic tests

1. **Neuroimaging:**

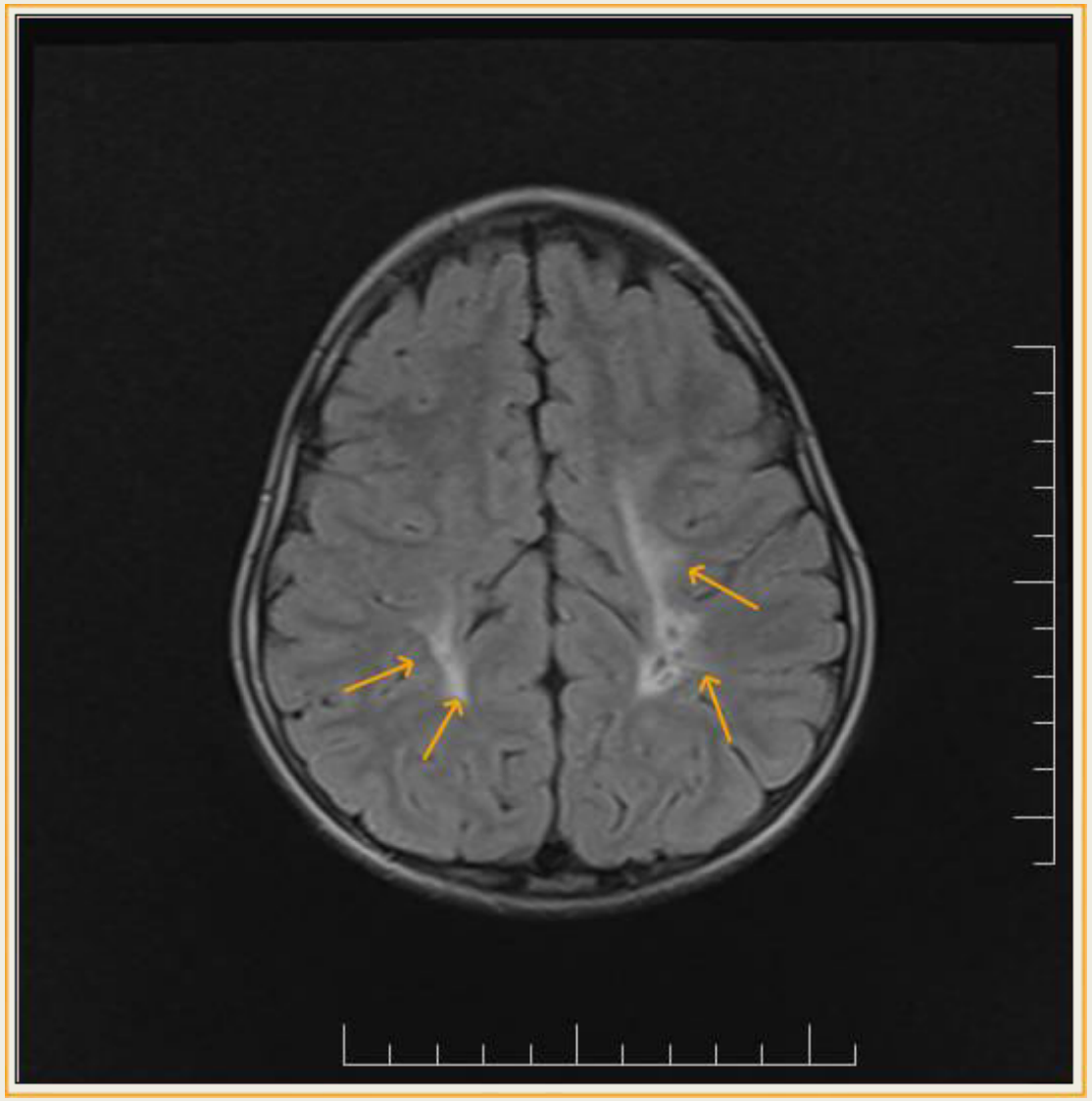
- MRI: We obtain neuroimaging, typically with magnetic resonance imaging (MRI) of the brain for all children with suspected CP.

The timing of when to perform the MRI depends on the clinical circumstances. Early imaging is warranted in severely affected children and those with concerning findings (eg, considerable motor asymmetry). For children with subtle clinical findings, waiting until approximately two years of age to account for myelination may be a better option since subtle findings may be missed on earlier imaging

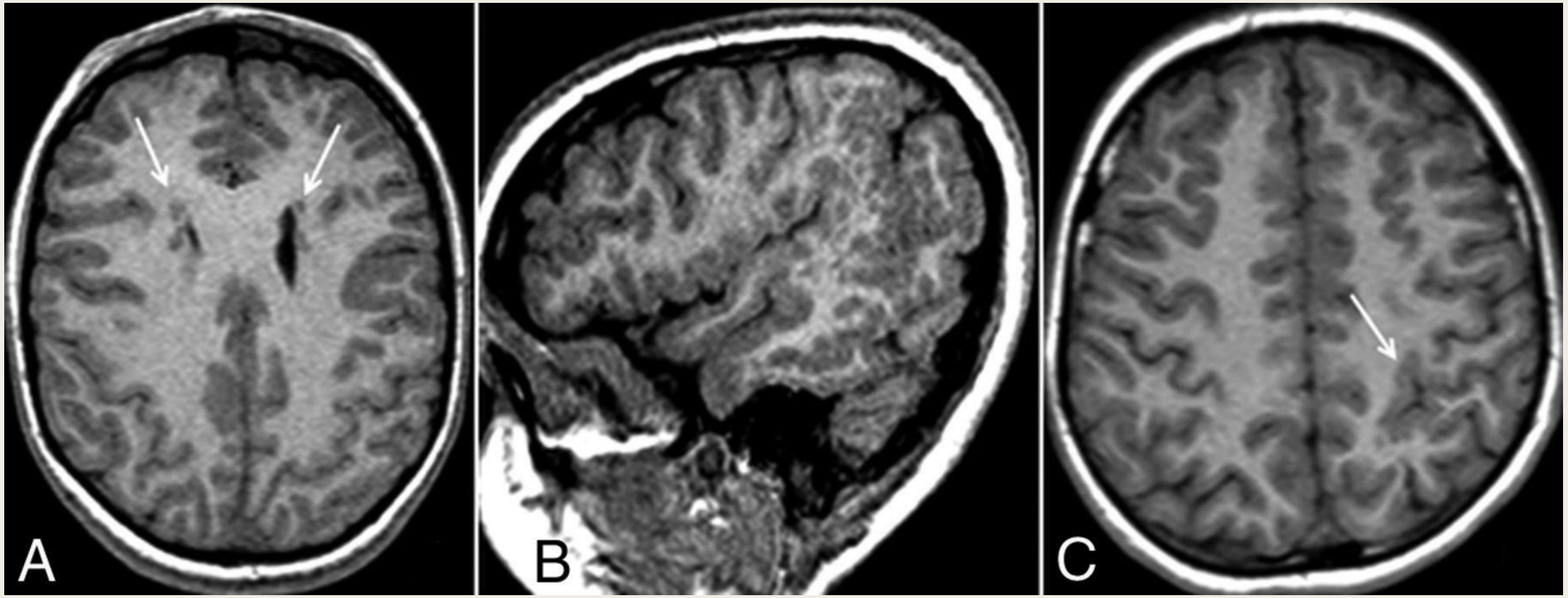
Cont.

- The MRI is abnormal in 85 to 90 percent of children with CP.

- MRI abnormalities in patients with CP include evidence of hypoxic ischemia (eg, periventricular leukomalacia), cortical malformations, and lesions of the basal ganglia



Leukomalacia



Cortical malformations

Cont.

- CT

Of course, MRI is preferred over computed tomography (CT) because it generally has a higher diagnostic yield and can be helpful in determining both the etiology and the timing of insult (prenatal, perinatal, postnatal). At the same time, CT exposes the developing brain to high doses of radiation.

However, CT can be helpful in urgent settings such as intracranial hemorrhage

- Cranial ultrasound

is a neuroimaging modality that can be used in neonates and young infants who have an open anterior fontanel abnormal findings on cranial ultrasound should generally be followed up with MRI. Ultrasonography can identify hemorrhage, periventricular leukomalacia

2. Metabolic and genetic testing

Metabolic and genetic testing is appropriate in the following circumstances:

- ❑ If there are features in the history or clinical examination that are atypical for CP, or otherwise suggestive of a genetic or metabolic abnormality (eg, history of progressive rather than static encephalopathy.
- ❑ If brain imaging reveals a developmental brain malformation
- ❑ If dysmorphic features are present
- ❑ If no etiology is identified by history, physical examination, and neuroimaging

Cont.

- A number of metabolic disorders may present with symptoms resembling CP.
- Initial laboratory testing for metabolic diseases includes serum concentrations of glucose, creatine kinase, ammonia, lactate, and pyruvate; plasma amino acid analysis;
- Genetic disorders were historically thought to be uncommon causes of CP but recent studies have detected potentially disease-causing genetic variants in as many as one-third of CP patients who lack an otherwise identified etiology

3. Other tests

- *Thrombophilia testing* may be appropriate for select children with hemiplegic CP or MRI evidence of cerebral infarction and a strong family history of thrombotic disease.
- *EEG* is only necessary if patient has seizures
- *Lumbar puncture* –is not routinely necessary -may occasionally be warranted to evaluate for rare causes of seizure disorders (eg, glucose transporter GLUT1 deficiency) or movement disorders , based upon the clinical findings

Cont.

- *Examination of the placenta*

can provide clues to the etiology of CP. It may indicate infection or chronic pathologic changes due to ischemia.

Pathologic findings associated with asphyxia include chronic ischemic change, meconium staining, nucleated red blood cells, intravillous hemorrhages.

Screening for associated conditions

Children with CP should be evaluated for other conditions that are commonly associated with CP

- *Intellectual disability* occurs in approximately %50 of patients with CP. Infants and children with CP should undergo developmental surveillance using standardized screening tools.
- *Seizures* occur in approximately 45%
- *Vision impairment* like strabismus, refractory errors, and other vision disorders are common in children with CP, occurring in 30%-50%. The risk is greater in preterm infants.

Screening for associated conditions

- *Hearing impairment* 5% are of CP patients are deaf.

All infants should be screened for hearing loss in the newborn period. In addition to newborn screening, children with CP should have at least one formal audiologic assessment by 24 to 30 months of age or sooner if necessary

- *Speech and language impairment*

Speech and language problems such as aphasia and dysarthria, occur in approximately 40%-50%of children with CP, and approximately 25% are nonverbal.

- *Growth failure:* Patients with CP often have growth failure, which is primarily due to poor nutrition, Children with quadriplegic CP are at increased risk for poor growth compared with those with diplegic or hemiplegic CP

Differentials

- Conditions that can mimic cerebral palsy include neurodegenerative disorders, inborn errors of metabolism, developmental abnormalities of the spinal cord, neuromuscular disorders, movement disorders, and neoplasms.
- Few examples of how to differentiate:

*Spastic diplegia or quadriplegia — Spasticity may occur in urea cycle disorders and other neurodegenerative disorders. These disorders may be distinguished from CP based upon their progressive clinical course and through metabolic and genetic testing.

*Muscle weakness — In infants with muscular dystrophy or myopathy, muscle weakness may be mistaken for hypotonia, which often occurs in infants with CP. Generally, in children with CP, the

complications

A variety of complications can accompany cerebral palsy, including:

- ***Mental health and Cognitive impairment*** is present in two-thirds of patients with cerebral palsy. Neurosis and psychosis also can occur.
- ***Markedly reduced bone*** *mass* in nonambulatory adults and children Can cause osteopenia, osteoporosis, fractures and scoliosis.
- ***Spasticity and Contractures***

Spasticity prevents the stretching of muscles and tendons. Consequently, they do not grow at the same rate as lengthening bones, forming contractures and difficulty with ambulation and fine/ gross motor movements.

- ***Pain*** is created by hip dislocations, repetitive use syndromes, and degenerative joint disease.

complications

- **Gastrointestinal problems** (e.g., vomiting, constipation, or bowel obstruction) Caused by delayed gastric emptying, abnormal autonomic control of gastrointestinal mobility, immobilization, inadequate oral intake, and prolonged colonic transit.
- **Pulmonary disease.**
- **Hearing, speech and vision impairment.**

MANAGEMENT

- Cerebral Palsy Management -

- *Main Goals Of Management :*

Reduce abnormalities of movements and tone to optimize normal psychomotor development (Medications, Surgeries)

Psychological development, Communication and Education

Optimal treatment of medical comorbidities

- Cerebral Palsy Management -

- Management should include :

  Multidisciplinary Team

  - Neurodevelopmental paediatrician
  - Paediatrician Neurologist
  - Physical medicine and Rehabilitation specialist
  - Speech pathologist and Developmental psychologist

  Teach some daily activities, Exercise and Adaptive equipment's

  Use some SPASTICITY and DYSTONIA DRUGS

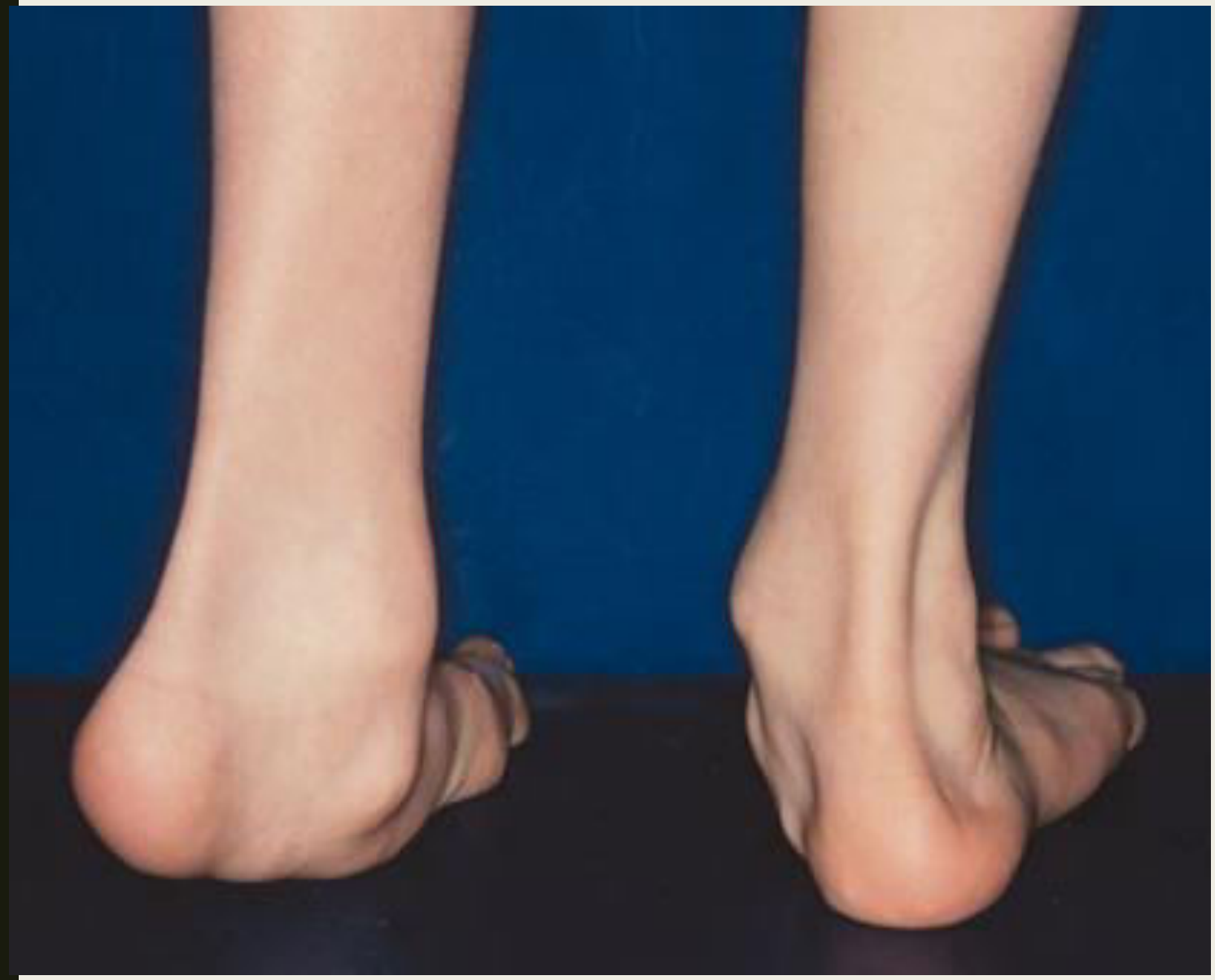
- Cerebral Palsy Management -

- *First thing we should educate patient family some exercises and how to work with their child in daily activities* *(Feeding, dressing, bathing and playing in ways that limits the effect of their muscle tone)*

- *The goal of these exercise – Prevent development of contractures*

  especially TIGHT ACHILLES TENDON

- *Some of these exercises may require adaptive equipment as Brace and Wheel Chair*



Achilles' Tendon Contracture

Tendon and muscle tightness, this tightness may shorten the muscles and tendon over time

- Cerebral Palsy Management -

- *Physical and Occupational therapy :*

  Both are useful for promoting mobility and enhance use of upper extremities for doing daily activities and decrease possibility of having muscle and tendon contracture – Important role in promote range of motion and coordination .

  Occupational therapy mainly targeted Fine motor skills and improve ability for self-care .

  Occupational and physical therapy should started early and continued by parents or caregivers .

- Cerebral Palsy Management -

- *Adaptive Equipments :*

  These includes braces, orthotics, standers and mobility devices

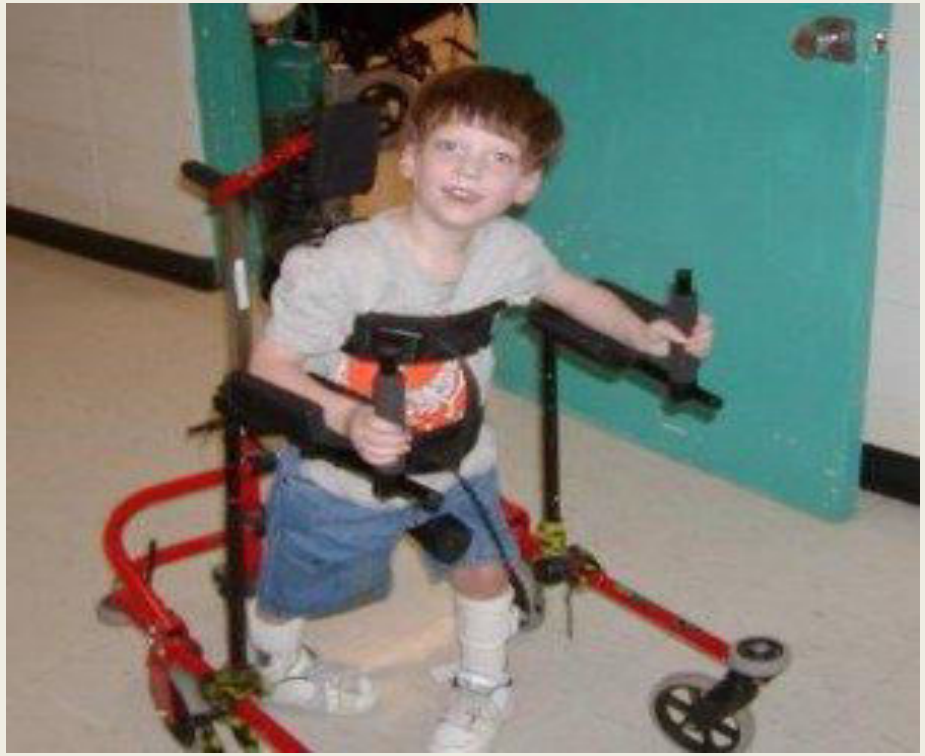
  Help promote function, mobility and participation

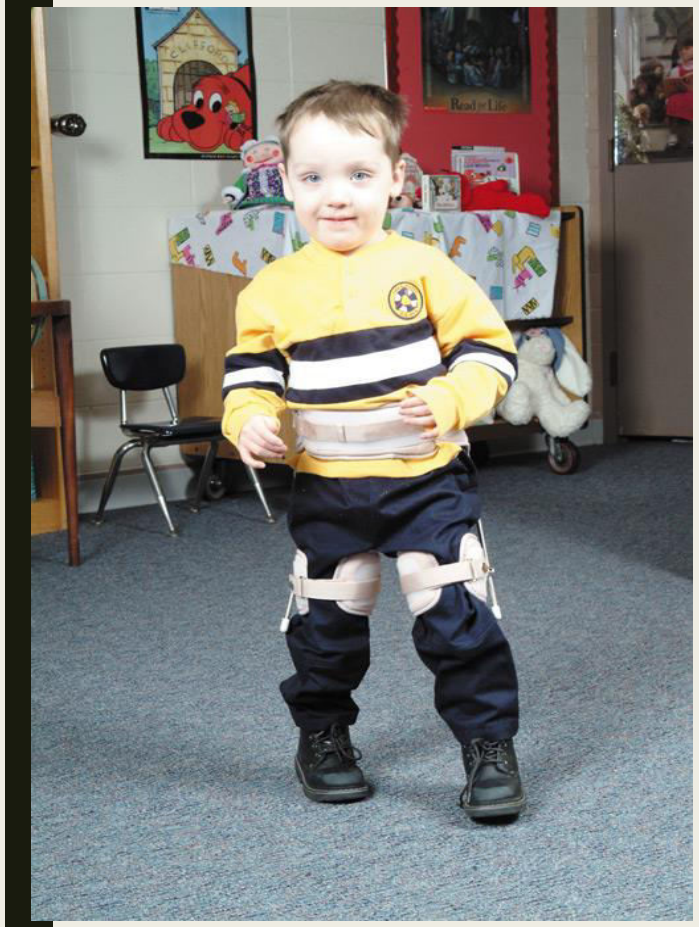
  Also they enhance musculoskeletal alignment and prevent contractures

Ankle-foot orthotics are most commonly used but also orthotics for knee hip and hands all of them are useful

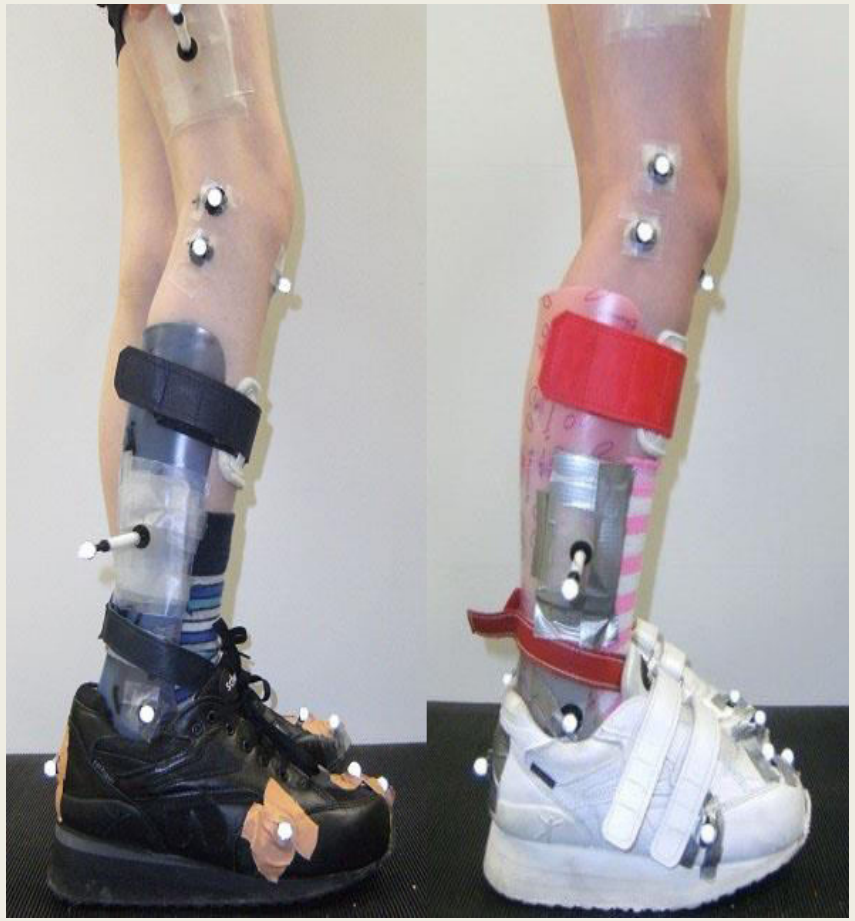
Some braces are worn for specific function as Walking, promote joint alignment or weight bearing

- *For the children who are unable to ambulate independently or for long distances – Mobility Devices Are critical .*
- *Wheelchairs can be introduced as early as two years of age .*
- *Over time, if the child had cognitive or motor abnormalities to steer chair independently – Electronic Version may be useful .*





Hip Orthotics



Ankle-foot Orthotics

Hand Orthotics

- Cerebral Palsy Management -

- *Pharmacological Management :*

1) Anti-Spasticity Drugs (mainly Benzodiazepines and Baclofen) :

Oral Diazepam (0.01 – 0.3 mg/kg divided BID or QID)

- Patient may develop dependence and should discontinued slowly

Baclofen (0.2 – 2 mg/kg divided BID or QID)

- Used in sever forms, may potentiate seizures by lowering threshold

Dantrolene (0.5 – 10 mg/kg given BID)

- Acts peripherally, limited use – Hepatotoxicity and fatal hepatitis

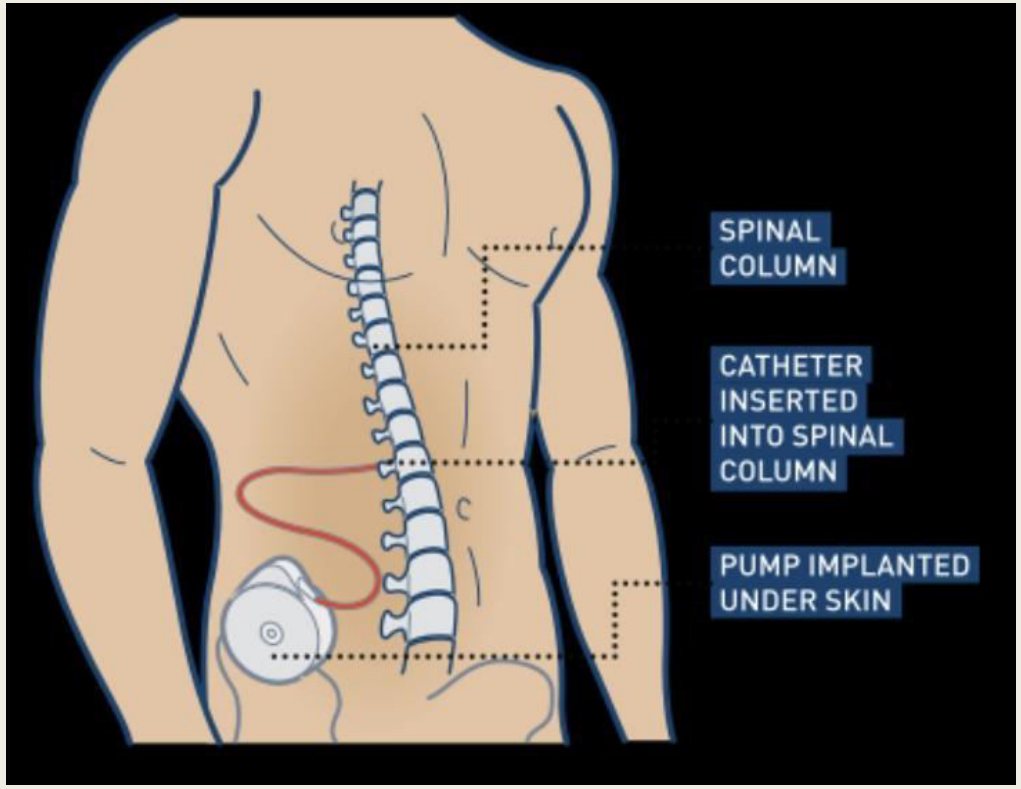
- *Intrathecal Baclofen :*

  Used in the cases of sever spasticity, delivered with implanted pump – Delivers baclofen to spinal cord via catheter into thecal space .

  More effective than oral form because it delivers the drug directly into spinal cord – Reduces neurotransmission of afferent fibers

  Direct delivery to spinal cord overcomes problem of CNS side effects that caused by large oral doses that penetrate BBB

- Baclofen withdrawal can be caused by Failure of pump, failure to refill drug, leakage or breakage in catheter .
- Withdrawal symptoms – Exaggerated rebound spasticity, multiple organ failure and Rhabdomyolysis



- *Botulinum Toxin Injection :*

Injected into specific muscle group for the treatment of spasticity

MOA – The most potent NEUROTOXIN, produce paralysis by blocking presynaptic release of Ach to neuromuscular junction .

One of the symptoms that present in CP patient – DROOLING, which present in 10% - 30% of cases – So, we inject toxin into salivary glands

REDUCES THE SEVERITY OF DROOLING

2) Dystonia Medications :

Levodopa (0.5 – 2 mg/kg/day)

- to treat dystonia or DOPA-Responsive dystonia

Artane (0.25 mg/day, Trihexyphenidyl)

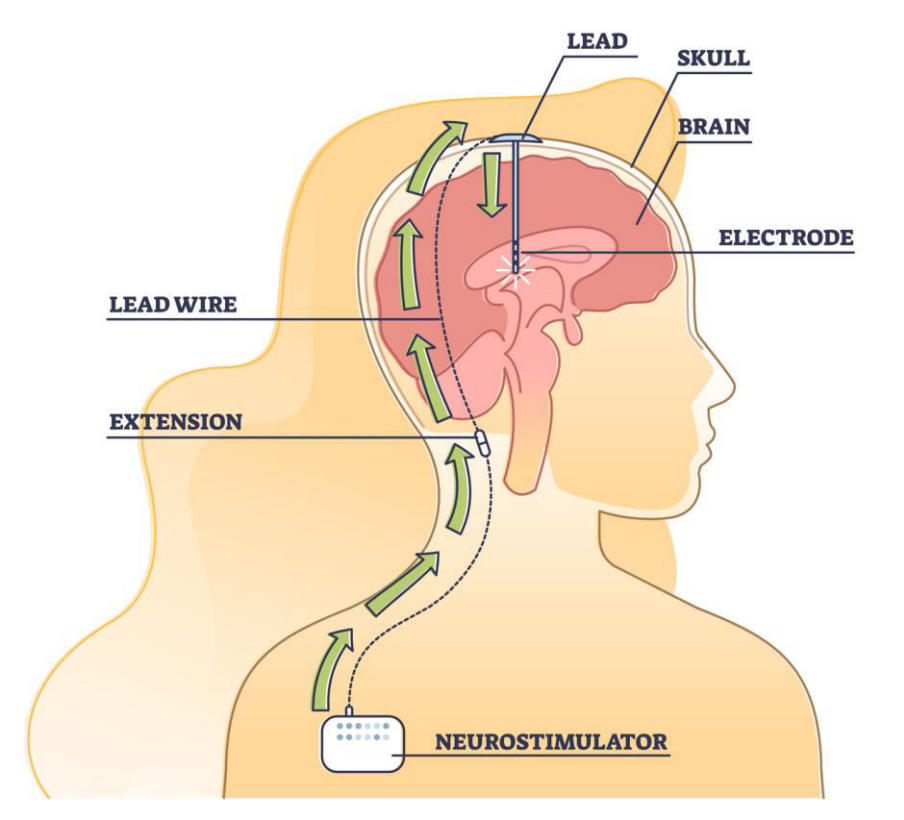
- Treat dystonia and can increase use of upper extremities and vocalization

Tetrabenazine (12.5 – 25 mg divided BID or TID)

- Useful for hyperkinetic movements disorder (Athetosis)

* DOPA-RESPONSIVE DYSTONIA = SEGAWA DISEASE
* Give trial of LEVODOPA to assess if it is Dopa-responsive or if it is not (if responsive = Dramatic Response)

- In the case of REFRACTORY Patients – Deep brain stimulation can be useful !
- In DBS – Electrical stimulation by electrodes placed in the posteroventral lateral globus pallidus Decrease extrapyramidal movement disorder !



- Cerebral Palsy Management -

- *Surgical Management :*

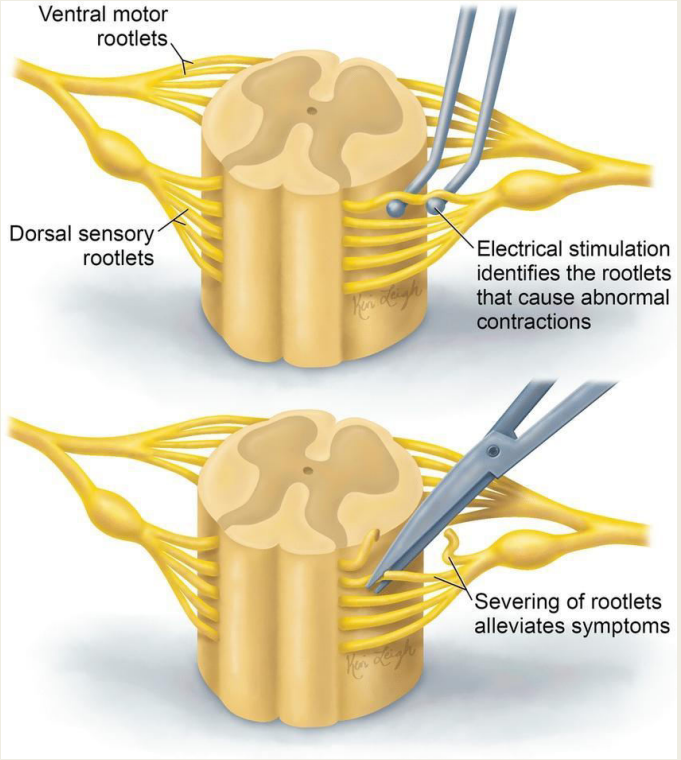
1) Rhizotomy Procedure

One-time irreversible procedure done in the cases of sever refractory spasticity to enhance care and relieve pain .

In this procedure, Roots of spinal cord divided produce considerable improvement in cases of sever spastic diplegia with little or no basal ganglia involvement

this procedure should be combined with physiotherapy and should have neuro-workup prior to operation – cause patient mostly will have Post-OP weakness, so he will need long-term and aggressive physiotherapy

Also, Ankle-foot orthoses are recommended POST-OP for at least 6 month to protect weak plantar flexors from overstretch



2) Adductor tenotomy or Psoas transfer and release

Used if the patient had marked spasticity of lower extremities or evidence of hip dislocation – Reduce muscle spasm around hip girdle !

* In case of tight heel cord – Tenotomy of Achilles tendon or use injection of botulinum toxin

3) Gastrostomy

Used in cases with significant eating, drinking and swallowing difficulties ! Risk of aspiration pneumonia, dehydration and poor weight gain increase in CP – So, this procedure protective against it

- Conclusion –

** For child with generalized spasticity – ORAL ANTISPASTICITY usually the first line of treatment*

** Localized or Segmental spasticity – BOTULINUM INJECTIONS*

** Spastic Diplegia who have mild to moderate impairment with no significant weakness and wiling to POST-OP rehabilitation – RHIZOTOMY maybe combine with ANTISPASTIC THERAPY to improve gait*

* *In case of severely affected child, who have significant side effects of oral anti-spastic medications or who do not achieve required response with max dose then INTRATHECAL BACLOFEN may achieve better control or RHIZOTOMY procedure can be used as an alternative option !*

* *Learning and attention deficit disorder and mental retardation should be assessed and managed by Psychologist and educator*
* *Nystagmus, optic nerve atrophy are common in CP patients, Ophthalmologist should be included in assessment*
* *Lower urinary tract dysfunction should be assessed and treated if present*

THANK YOU

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