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Cerebral Palsy Rehabilitation

Department Of PMR

Team approach





Associate manifestations & complications

- Mental Retardation
- Epilepsy
- Feeding, Nutrition, and Growth
- Bladder Dysfunction
- Bowel Dysfunction
- Sleep Disturbances
- Drooling
- Hearing Loss
- Visual Abnormalities
- Orthopaedic Abnormalities





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Goals of Rehabilitation

- Improve mobility level of child
- Prevent deformity and contractures
- Educate parents regarding home management of child
- To train child in activities of daily living (ADL)
- ► To improve social participation of affected child.

Rehabilitation plan



Each patient and his or her family provides

a separate challenge



Prognosis

- Bleck(1987) has described tests for prognosis of walking in children over 1 year
- Neck righting reflex, asymmetrical and symmetrical tonic neck reflex, Moro reflex – all should disappear by 1 yr
- Children who retain more than two primitive reflexes after 1 year.
- Cannot sit unsupported by 4 year.
- Cannot walk unaided by 8 years are unlikely to ever walk independently.

NON-PHARMACOLOGICAL MANAGEMENT



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Basic needs

- Ability to communicate with others
- Ability to cope with ADLs
- Independent mobility
- Basic needs for a non walking child is straight spine with level pelvis, painless hip and knee and plantigrade feet

Physical therapy

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- Passive ROM exercises
- Stretching exercises
- Strength training
- Neurodevelopmental therapy (NDT)/ Bobath technique:

aims to improve gross motor function and postural control by facilitating muscle activity through key points of control

assisted by the therapist.



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Physical therapy

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- Constraint-induced movement therapy holds promise in improvements of upper limb dysfunction
- Involved placing a full arm cast on the unimpaired upper limb for 21 consecutive days, accompanied by intensive training of impaired hand for 6 hours each day.
- Functional electrical stimulation and biofeedback can be helpful in training specific muscles

Occupational therapy

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- The primary goal is to help patient gain more independence
- Children work on fine motor skills by grasping and releasing toys
- Practice handwriting skills and hand-eye coordination

Basic tasks such as bathing, dressing, brushing teeth, and eating are also addressed

They also work on fitting the child for special devices that help them function. This may include utensils, dressing devices, wheelchairs, bathing seats



Orthosis

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- Goals include reduction of abnormal tone, avoidance of deformity, and facilitation of normal movement patterns
- AFOs are used in case of dynamic equinus setting the ankle in neutral to slight dorsiflexion promotes heel strike and limits knee recurvatum.
- Hinged AFOs can be used with plantar flexion stop

Ankle Foot Orthosis







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FLOOR REACTION ORTHOSIS

Ankle set at neutral dorsiflexion and molded anteriorly to just below the patella this limit crouch gait secondary to hamstring spasticity by creating an extension moment at knee



Orthosis

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- Posterior leaf spring orthoses are thinned posteriorly to simulate push-off at the end of stance phase
- KAFO & HKAFO do not improve gait and they add weight to already weak muscles
- But they prevent deformity and facilitate standing

Long leg Tone inhibiting orthosis (with abduction mechanism in c/o adductor spasticity) and serial casting help in cases of correctable deformities



Assistive technology

- Power wheelchairs as young as 20 to 36 months can learn to use
- Posterior postural walkers
- Ultrasonic detectors can be incorporated for cortical blindness
- Augmentative communication devices



NON-SURGICAL MANAGEMENT OF CEREBRAL PALSY

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General consideration

Factors to be considered before spasticity treatment:

- Whether to treat at all
- Distribution (focal/regional/global) 2.
- Degree of spasticity 3.
- Family support & ability 4.
- Physical access to medical care 5.

Treatment options

- Oral antispasticity medications
- Nerve & motor point block
- Botulinum toxin
- Intrathecal baclofen





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Oral medications

Baclofen

- Diazepam
- Clonazepam
- Dantrolene
- Tizanidine

Baclofen

- MOA: Activates GABA-B & decreases excitatory neurotransmitter release.
- Dose:- starting dose 1 mg/kg/d (bid). 2 to 7 yr: max 40 mg, 8 to 11: max 60 mg, >12 yr: max 80 mg (divided doses).

S/E: sedation, confusion, paraesthesia, weakness.

Rapid withdrawal: rebound spasticity, fever,

hallucination, seizure.

Caution: renal& hepatic compromise.



Diazepam

- MOA: Acts centrally on GABA-A & facilitates GABA mediated inhibition.
- **Dose:** <12 yr: 0.1—0.8 mg/kg/d,
 - >12 yr: 6—30 mg/d. (divided tid or qid)
- **S/E**: sedation, memory loss.
- Rapid withdrawal: anxiety, insomnia, seizures.
- **Caution**: Hepatic compromise

Clonazepam

MOA: Same as Diazepam

Dose:- <10 yr: 0.1—0.2 mg/kg/d,</p>

>10 yr: max 20 mg/d. (divided bid/tid)

▶ S/E: more sedation than Diazepam.



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Tizanidine

MOA: Alpha 2 agonist, facilitates action of glycine spinally & supraspinally.

Dose: 0.2–0.3 mg/kg/d (tid/qid).

Advantage: Reduces spasm at night, helpful for sleep

S/E: hypotension, drowsiness.

Caution: Ciprofloxacin, hepatic compromise.

Nerve & motor point block

Nerve block: Peri-neural injection targeting a motor/mixed nerve to impair its function.

Motor point block: Intramuscular injection lower down

the nerve trunk (below sensory branches) to create a motor block with minimum sensory involvement.



Chemical denervation agents

- ▶ Phenol (5-7%): Denature protein of nerve tissue.
- Alcohol (45-100%): Dehydrates nerve tissue & causes sclerosis of myelin sheath.
- Site: Motor nerve/point located by electrical nerve stimulator.

Disadvantage of phenol/alcohol

- Needs precise location of injection.
- Pain & dysesthesia very common.
- Overdose & accidental i.v injection may lead to depression convulsion cordioversaular colleges

depression, convulsion, cardiovascular collapse.





Botulinum toxin

- A protein and neurotoxin produced by the anaerobic bacteria Clostridium botulinum.
- MOA: Blocks neurotransmitter (Ach) release at the peripheral cholinergic nerve terminals
- Reduction in muscle tension improved passive and active range of motion, facilitates stretching techniques (casting and splinting).
- **Dosing** is based on amount of tone present, patient's prior response to injections, residual function of the spastic muscles, and potential impact of excessive tonal reduction

Intrathecal baclofen

- ITB therapy significantly reduced severe spasticity which did not respond to oral medications and botulinum toxin treatment.
- It might have reversible adverse effects or catheter-related complications and spasticity reduction did not

always induce functional improvements.



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SURGICAL MANAGEMENT



Four basic principles

- Although the central nervous system injury, by definition, is nonprogressive, the deformities caused by abnormal muscle forces and contractures are progressive.
- The treatments currently available correct the secondary deformities only and not the primary problem.
- The deformities typically become worse during times of rapid growth. For some patients, it may be beneficial to delay surgery until after a significant growth spurt to decrease the risk of recurrence.
- Operative or non-operative treatment should be done to minimize the impact it has on the patient's socialization and education.

Operative Treatment

Operative treatment of deformities:

- (1) correct static or dynamic deformity
- (2) balance muscle power across a joint
- (3) reduce spasticity (neurectomy)

(4) stabilize uncontrollable joints

Often, procedures can be combined—an adductor tendon release can be done at the time of pelvic osteotomy for hip subluxation.



Procedure	Indication	Possible complications
Hip		
Z-lengthening of Psoas tendon	Flexion deformity of the hip over 20 degree, shifting the centre of gravity forwards if primarily due to Psoas	 Decrease in hip flexion pull Compensatory caliper gait with increased pelvic rotation
Adductor tenotomy	Scissoring gait causing instability	Asymmetrical abduction deformity with pelvic tilt
Adductor tenotomy with Obturator neurectomy	do	do

Procedure	Indication	Possible complications
Knee surgery		
Medial hamstring lengthenig	Flexion deformity of the hip over 30 degree during the gait	Patella alta, Knee recurvatum and Secondary rotation of tibia
Lateral hamstring lengthening (Biceps)	Insufficient correction after medial release (over 30 degree)	Patella alta & Knee recurvatum
Distal rectus release	Caliper gait (lack of flexion at swing phase)	Decrease of extension pull inducing relapse of flexion at stance phase



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Procedure	Indication	Possible complications
Ankle & Foot Surgery		
Tendo Achilles (Z- lengthening)	Equinus Deformity	Progressive crouch gait
Selective gastrocnemius fascial lengthening using the Strayer or Baker surgical techniques	do	

