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The HELP Guide to Cerebral Palsy

Contents

Preface

Drs. Nadire Berker and Selim Yalçın have created an excellent publication, providing an overview of the diagnosis and management of cerebral palsy (CP). This publication is authoritative, comprehensive, extensively illustrated, colorful and engaging. The need for this publication is enormous, as CP is common throughout the world and no affordable management guide has been available to date.

The authors are highly qualified to produce this publication. Dr. Nadire Berker is professor of rehabilitation medicine at the Marmara School of Medicine in Istanbul, Turkey. She has extensive experience in managing the full spectrum of CP from childhood to adult life. Dr. Berker continues a family tradition in rehabilitation medicine that was started in the 1920s by her grandfather who established the specialty in Turkey, and was continued by her mother who headed the department for many years. Dr. Selim Yalcin is associate professor of orthopedics at the same university. Dr. Yalcin is an accomplished clinician who specializes in pediatric orthopedics. He has broad clinical experience, and is a prolific author and producer.

The authors have produced numerous books and videos on cerebral palsy, spina bifida, gait analysis, clubfoot, the use of ultrasonography in orthopedic management, and historical aspects of medicine.

They practice medicine in Istanbul, a city positioned at the crossroads of the world. The economy of Turkey is midway in the economic spectrum of nations of the world. This geography and economic environment provides the authors with an ideal vantage point from which to make management recommendations that are both practical and relevant for most societies of the world.

The authors recommend a balanced approach to management. This approach balances the medical, social, psychological and educational needs of the child and family. The humanitarian, whole-child approach is evident throughout the publication. Global-HELP Organization is pleased to support *The HELP Guide to Cerebral Palsy*. Printed copies are available for those in developing countries for only the cost of postage and in developed countries for a small charge. Please visit our web site at www.global-help.org for details. The book is available through our web site in pdf format; for non-commercial use, it may be downloaded without charge.

Lynn Staheli, MD
Seattle, USA 2004

The Story of the EMCPDM

The authors of this book were among the group of dedicated American Academy of Cerebral Palsy and Developmental Medicine (AACPDM) members who felt the necessity to improve the quality of health care services urgently needed by children with neuromuscular diseases in the Middle East. After long discussions on what to do, they planned to start meetings to provide a regional platform for educating physicians and therapists. Drs. Berker and Yalçın shouldered the responsibility of the first meeting in Istanbul, performing the secretarial work, graphic design, and fund raising by themselves. With seed money donated by the AACPDM, they organized a large meeting at which recent scientific advances in the area of childhood neuromuscular disability and CP were the main topics.

The first Eastern Mediterranean Cerebral Palsy and Developmental Medicine meeting was held in 2002 in Istanbul. More than 300 participants from 18 countries were in attendance. For the 3-day congress in the most modern convention center in Turkey, registration fees were kept to a minimum of $100 and free accommodation was provided to participants from countries with economical problems.

The faculty included renowned physicians and physiotherapists from the United States, Europe and the Middle East, most of whom volunteered to participate and donated their time and efforts. The success of the first meeting led to the next in 2004 in Greece, which also was very successful. The 2006 meeting will be held in Warsaw, Poland with the aim of covering Eastern Europe.

The EMCPDM (www.turkortopedi.net/emcpdm.htm) became a wonderful opportunity for the physicians and therapists of the region to get together, share knowledge and experience, and discuss the latest developments. The group is looking forward to future meetings with proud anticipation.

Dear Reader,

Years of treating children with cerebral palsy (CP) has shown us that a worldwide need exists for a concise, illustrated book to guide health professionals regarding this difficult problem. This book is an attempt to fulfill that need. The *Guide* is intended for use by physicians, residents, medical students and allied health professionals who treat children with CP. We focus on the latest concepts in the treatment of musculoskeletal problems and describe the associated impairments, providing suggestions for further reading. The chapters on total body involvement, diplegia, hemiplegia and dyskinesia include the most common treatments applied for these patients. Note that problems described in one section may occur in other types of CP. We present the most frequently used and accepted treatment methods with scientifically proved efficacy and include references at the end of each chapter.

The illustrations and photographs of patients are from ours and Dr. Leon Root's archives unless stated otherwise. We would like to thank our patients and their families for sharing their problems with us and also for allowing us to use their pictures. We are indebted to Ms. Dory Kelly for helping us with text editing.

Treatment of the child with CP is difficult, often frustrating and sometimes depressing. This is even more pronounced in countries with limited resources for the disabled. We try to provide information on how to proceed in places where resources are limited. An interdisciplinary management approach is the only means to integrate children with CP with the society and lessen the impact of the problem. We hope that readers will benefit from our work and use this guidebook in the treatment of unfortunate millions of patients with CP worldwide.

Nadire Berker and Selim Yalçın
Istanbul, Turkey - 2005

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Dr. Berker has treated disabled children for nearly 10 years. She has pioneered the application of modern methods in pediatric rehabilitation, lectured extensively, and co-authored many books in Turkish on various subjects of childhood disability. nadireftr@yahoo.com

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Leon Root, MD

Dr. Root is one of the leading names in cerebral palsy orthopaedic surgery worldwide. A former president of the AACPDM, he has dedicated his career to cerebral palsy, given many lectures and authored more than 100 articles on the subject.

Lynn Staheli, MD

Dr. Staheli is one of the most prominent names in pediatric orthopaedics. He is the founder and chief editor of the Journal of Pediatric Orthopaedics, author of six books and a wizard of desktop publishing. He founded the Global-HELP organization to create affordable medical textbooks worldwide.

Contributors

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Lana Staheli, PhD is a certified counselor, marriage therapist, and consultant on interpersonal relationships and life strategies. She authored several relationship books. Lana is co-founder and vice-president of Global-HELP.

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Dr. Ganjwala is a pediatric orthopaedic surgeon form India. Teaching is his passion and he conducts workshops and deliver lectures at various institutions. He has published and edited many books on variety of topics for doctors, residents and patient education.

Garen Koloyan, MD

Dr. Koloyan is a pediatric orthopaedic surgeon from Yerevan, Armenia. He has been one of the creators of the EMCPDM and has done pioneer work for disabled children of Armenia and Georgia.

Antigone Papavassiliou, MD

Dr Papavassiliou is the director of Pediatric Neurology at the Pendeli Children's Hospital in Athens, Greece. She has been treating children and adolescents with CP for many years and has devoted a lot of time in teaching physicians and therapists. She has co-authored two medical textbooks on CP and many others for patient education in Greek.

Idil Çilingiroğlu

Ms. Çilingiroğlu is an architect who devoted her time and talent to draw all the illustrations in the section for families.

Foreword

Cerebral palsy (CP) is the most common chronic disability of childhood today. It is ubiqitious and it occurs all around the world. In developed nations, the incidence is about 1 to2 per 1000 births. In spite of improved obstetrical and perinatal care, CP remains with us. As a result of injury to the brain, these children have motor defects which will affect them for their entire lifetime. Treatment often starts when they are infants, and continues throughout their life, even into adulthood. The problems involved are complex; not only do these children have problems of mobility, but they can also have seizure disorders, gastrointestinal system problems, learning and perceptual difficulties, visual problems, hearing problems, and growth deficiency. In spite of all these numerous difficulties, cerebral palsied children can be helped.

What the authors attempt in this book is to divide information for physicians, therapists or other paramedical personnel who are interested and will be treating and taking care of these children for their lifetime. They present the basic understanding of what CP is and the fact that it takes a team to treat them. The child and his parents become the focus of treatment because you cannot treat the child without involving the parents as well. The team has to consist of the physician who will be the captain of the team. That physician can be a pediatrician, orthopaedic surgeon, physiatrist or even a neurologist, but they must take overall control of the child and make sure that all parameters of care are attended to. The team has to also include the occupational therapist, the speech therapist, the physical therapist, teachers and social workers.

Dr. Wallace Salter of Toronto, Canada is fond of saying that after you operate on the child with CP, he still has CP. This may seem discouraging because we cannot cure the CP with our present knowledge, but we can make life better for these children, and that is important. Even small degrees of improvement makes a great difference. Getting a child to walk, be it in crutches, in braces or with a walker, is much better than having him in a wheelchair. Having a child be able to live in a wheelchair, as is true for children with total body involvement, is much better than having him be on a stretcher or in a bed for the rest of his life. These are important things to consider.

The authors have carefully defined the types of CP, the prognosis of CP, therapies that are at present available, the surgical indications and most important of all, the pre and postoperative care that these children must have. They write in a very clear and concise way which provides a ready reference for the interested reader in treating these children.

In my own experience, I have found that working with the cerebral palsied children and their families has been the most rewarding aspect of my medical career. The children and families are deeply grateful to you for whatever you can offer them and particularly, they respond to the fact that you care. And it's with your caring and your ability to help that makes a difference.

Leon Root, MD
Orthopaedic Surgeon
Hospital for Special Surgery
New York, February 2005

Dedication

We would like to thank
Our mothers and fathers, for their guidance and inspiration.

Our children, for their compassion towards those less fortunate than themselves.

Asaf Yalçın   Sabahat Yalçın   Ender Berker   Mustafa Berker

Deniz Yalçın   Deniz Özaras   Güneş Yalçın

General Concepts

Definition

Cerebral Palsy (CP) is a disorder of movement and posture that appears during infancy or early childhood. It is caused by nonprogressive damage to the brain before, during, or shortly after birth. CP is not a single disease but a name given to a wide variety of static neuromotor impairment syndromes occurring secondary to a lesion in the developing brain [A]. The damage to the brain is permanent and cannot be cured but the consequences can be minimized [B]. Progressive musculoskeletal pathology occurs in most affected children.

The lesion in the brain may occur during the prenatal, perinatal, or postnatal periods [C]. Any nonprogressive central nervous system (CNS) injury occurring during the first 2 years of life is considered to be CP.

In addition to movement and balance disorders, patients might experience other manifestations of cerebral dysfunction [D].

CP was first described by the English physician Sir Francis William Little [E] in 1861 and was known as *Little's disease* for a long time. Little thought that this condition was caused by neonatal asphyxia [F]. Later, Sigmund Freud [G] and other scientists challenged Little's idea and proposed that a variety of insults during pregnancy could damage the developing brain. Today, it is accepted that only approximately 10% of cases of CP can be attributed to neonatal asphyxia. The majority occur during the prenatal period, and in most of the cases, a specific cause cannot be identified.
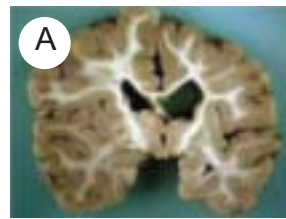
Epidemiology

CP is the most common cause of childhood disability in Western societies. The incidence is 2-2.5/1000 live births. Some affected children do not survive and the prevalence varies between 1-5/1000 babies in different countries. It was previously thought that improvements in perinatal and obstetric care would decrease the incidence of CP. However, the incidence has not declined and the overall prevalence increased during the 1980s and 1990s. This is explained by increased survival of premature and very-low-birth-weight infants and by a rise in the number of multiple births. Even at centers where optimal conditions exist for perinatal care and birth asphyxia is relatively uncommon, the incidence of CP in term babies has remained the same. This has led researchers to consider unknown prenatal causative factors.

Etiology

The etiology can be identified only in 50% of the cases. Certain factors in the history of the child increase the risk of CP. The incidence of CP among babies who have one or more of these risk factors is higher than among the normal population. The clinician should therefore be alerted to the possibility of the presence of CP in a patient with these factors.

The brain lesion cannot be cured but it does not get worse either.
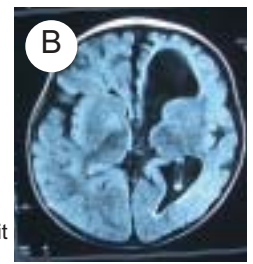
C	Time of brain injury
Prenatal period	Conception to the onset of labor
Perinatal period	28 weeks intrauterine to 7 days postnatal
Postnatal period	First two years of life

D Manifestations of cerebral palsy	
Neurological	*Associated problems*
Muscle weakness	Intellectual impairment
Abnormal muscle tone	Epilepsy
Balance problems	Visual problems
Loss of selective control	Hearing loss
Pathological reflexes	Speech and communication problems
Loss of sensation	Swallowing difficulty
Musculoskeletal	Feeding difficulty, failure to thrive
Contractures	Respiratory problems
Deformities	Incontinence
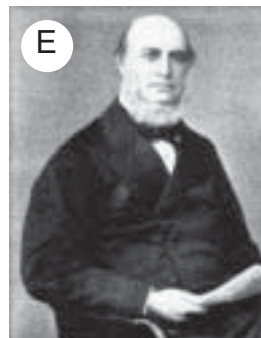
In 1861 William Little described a paralytic condition of children that is recognizable as spastic diplegia.

In his paper, Dr. Little showed a child who appeared to have some adductor spasm with crouch gait and intoeing.
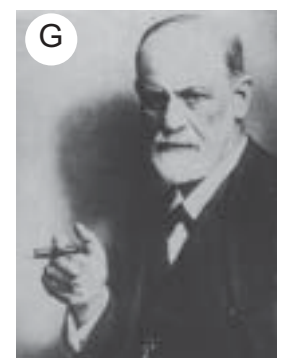
Sigmund Freud challenged Little's opinion on the association between CP and birth trauma.

CP has been defined as a non progressive injury to the immature brain leading to motor dysfunction.
Although the lesion is not progressive, the clinical manfestations change over time (Mercer Rang).

A Risk factors

Prenatal

Risk factors
Prematurity (gestational age less than 36 weeks)
Low birth weight (less than 2500 g)
Maternal epilepsy
Hyperthyroidism
Infections (TORCH)
Bleeding in the third trimester
Incompetent cervix
Severe toxemia, eclampsia
Hyperthyroidism
Drug abuse
Trauma
Multiple pregnancies
Placental insufficiency

Perinatal

Prolonged and difficult labor
Premature rupture of membranes
Presentation anomalies
Vaginal bleeding at the time of admission for labor
Bradycardia
Hypoxia

Postnatal (0-2 years)

CNS infection (encephalitis, meningitis)
Hypoxia
Seizures
Coagulopathies
Neonatal hyperbilirubinemia
Head trauma

Risk factors

Risk factors associated with CP are grouped into prenatal, perinatal, and postnatal factors [A]. Prematurity and low birth weight are the two most important risk factors in developed countries with high standards of obstetrical care. Postnatal risk factors additionally play a major role in other countries.

A clear association exists between premature delivery and spastic diplegia [B]. Low birth weight increases the risk. Rubella, herpes simplex, toxoplasma, and cytomegaloviruses cross the placenta to infect the fetus and have severe effects on the developing CNS. Eclampsia or other severe maternal illness hypothermia, hypoglycemia of the neonate cause a reduction in the levels of oxygen and nutrients available to the fetus or an increase in the levels of toxins or waste products, adversely affecting the developing CNS. Multiple pregnancies or breech presentation also can increase the risk [C]. Excess of bilirubin resulting from the haemolytic disease of the newborn is clearly associated with CP.

Babies who carry these risk factors should be under close supervision by a pediatric neurologist for signs suggestive of neuromotor developmental delay.

Pathological findings in the CNS

Specific brain lesions related to CP can be identified in most of the cases. These lesions occur in regions that are particularly sensitive to disturbances in blood supply and are grouped under the term *hypoxic ischemic encephalopathy*.

Five types of hypoxic ischemic encephalopathy exist [D]; parasagittal cerebral injury, periventricular leukomalacia [E], focal and multifocal ischemic brain necrosis [F], status marmoratus and selective neuronal necrosis.

Approximately 11% of premature babies who survive in neonatal intensive care units develop CP.

In vitro fertilization results in multiple pregnancies and increases the risk of CP.

Periventricular leukomalacia (PVL)
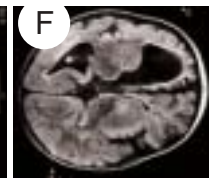
Multifocal ischemic brain necrosis

D Subtypes of hypoxic ischemic encephalopathy

Lesion	Location	Clinical Finding
Parasagittal cerebral injury	Bilateral in superior medial and posterior portions of the cortex	Upper extremities more severely affected than lowers
Periventricular leukomalacia	Bilateral white matter necrosis near lateral ventricles descending fibers of the motor cortex, optic and acoustic radiations	Spastic diplegia and quadriplegia visual and cognitive deficits
Focal and multifocal ischemic brain necrosis	Infarction in a specific vascular distribution (most commonly left middle cerebral artery)	Hemiplegia and seizures
Status marmoratus	Neuronal injury in the basal ganglia	Choreoathetosis or mixed
Selective neuronal necrosis (usually combined with the above)	Lateral geniculate, thalamus and the basal ganglia	Mental retardation, seizures

Clinical findings

Children with CP present with three types of motor problems [A]. The primary impairments of muscle tone, balance, strength and selectivity are directly related to damage in the CNS. Secondary impairments of muscle contractures and deformities develop over time in response to the primary problems and musculoskeletal growth. Tertiary impairments are adaptive mechanisms and coping responses that the child develops to adapt to the primary and secondary problems. One typical example is gastrocnemius spasticity as a primary impairment [B] leading to secondary ankle plantar flexion contracture and knee hyperextension in stance [C] as an adaptive mechanism.

Mechanism of the movement problems

Abnormal muscle tone, disturbance of balance mechanisms, muscle weakness and loss of selective motor control lead to an inability to stretch muscles. Muscle weakness, spasticity, and contractures [D] also result in abnormal skeletal forces which cause bone deformity [E] as the child grows older.

Muscles grow through stretch which occurs during active movement. When the child wants to play, he moves and stretches the muscles. This creates the necessary input for muscle growth. The child with CP cannot play because of pathological tone, weakness, poor selective control and abnormal balance. His muscles are not stretched and do not grow. The distal biarticular muscles are more affected because selective motor control is worse distally and the biarticular muscles [F] are more abnormal than are the monoarticular muscles.

The child with CP has abnormalities of muscle tone and reflexes, shows delay in developmental milestones, and presents with posture and movement problems. When he tries to move, muscle contractions cannot be effectively controlled. This is a result of many factors [G].

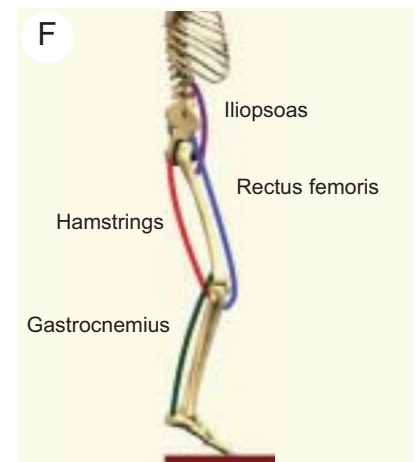| A	Primary impairments
	(due to the brain lesion)
	Muscle tone (spasticity, dystonia)
	Balance
	Strength
	Selectivity
	Sensation
	Secondary impairments
	(due to the primary impairments causing the movement disorder)
	Contractures (equinus, adduction)
	Deformities (scoliosis)
	Tertiary impairments
	Adaptive mechanisms (knee hyperextension in stance)
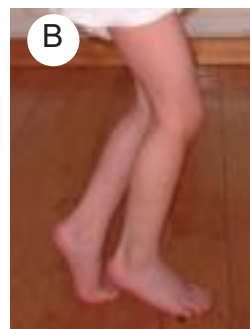
Equinus contracture due to triceps surae spasticity is a secondary impairment.
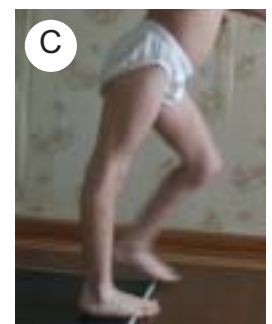
Knee hyperextension is an adaptive response to equinus deformity.

D	Common sites for contracture	
Upper extremity		*Lower extremity*
Pronator		Hip adductor-flexor
Wrist and finger flexor		Knee flexor
Thumb adductor		Ankle plantar flexor

E	Common sites for deformity	
Spine		Scoliosis, kyphosis
Hip		Subluxation, dislocation
Femur & tibia		Internal or external torsion
Foot		Equinus, valgus, varus

Iliopsoas

Rectus femoris

Hamstrings

Gastrocnemius

Biarticular muscles of the lower extremity are most commonly involved.

Causes of the motor problem	G
The muscles are weak and cannot generate the appropriate force necessary for movement.	
Spasticity does not allow the muscle to relax. It causes unnecessary contractions during movement.	
The coordinated contraction and relaxation of many muscles is necessary for a smooth movement. Certain muscles need to relax while others contract. The cerebral centers controlling this complex selective motor control are disturbed in CP. The child is unable to relax certain antagonist muscles and contract the agonists necessary for a specific task.	
Primitive reflexes interfere with the development of gross and fine motor control.	
Advanced postural reactions for balance and equilibrium that are a prerequisite for sitting and walking are either delayed or nonexistent. When the child cannot sustain balance, movement becomes more difficult.	
Apraxia (inability to plan and execute motor function) is present.	
Superficial sensation is generally normal, cortical sensation, proprioception and sensation of movement may be impaired.	

Maturation of the central nervous system
Primitive reflexes   A
Cutaneous
Palmar grasp
Plantar grasp
Rooting
Sucking
Gallant
Labyrinthine
Prone
Supine
Proprioceptive
Symmetric tonic neck reflex
Asymmetric tonic neck reflex
Moro
Foot-hand placement
Advanced (postural/protective) reactions
Head righting
Head and body righting
Protective-antigravity
Forward-lateral-backward reactions
Parachute-protective extension response
Landau
Equilibrium reactions
Voluntary movement
Fine motor
Gross motor
Rolling
Sitting
Standing
Sphincter control

Evolution of CP during infancy and early child-hood

The movement problem associated with CP is not apparent in infancy. It is established during the period of CNS development. Children who are going to have CP show neuromotor developmental delay in infancy. The typical clinical picture is established toward the age of 1 year in a number of these children. Movements become normal as the nervous system matures in some others.

The normal newborn demonstrates primitive reflex movements. These are complex, stereotypical patterns that occur in response to a variety of sensory stimuli. At birth almost all motor behavior is controlled by these primitive reflexes. Within a few months, they are replaced by a more mature set of protective and postural reflexes called advanced postural reactions that position the body segments against each other and gravity. Advanced postural reactions provide the basis for trunk balance and voluntary control of movements. The child gains motor skills as primitive reflexes are supressed and advanced postural reactions are established [A].

Primitive reflexes persist [B-D] and advanced postural reactions [E,F] do not appear in the child with CP. Abnormal movement patterns emerge as the child grows.

Asymmetrical tonic neck reflex (ASTNR): The child lies supine. Turn the head to one side and then the other. The extremities on the face side extend and the ones on the occiput side flex. This is called the fencing position. The reflex is present at birth and disappears at 6 months.

Photo courtesy of G. Koloyan

Foot placement reaction: Hold the child by the axilla and bring the dorsum of the foot against the edge of the table. The child will automatically place his foot on the table top. This is a normal response in all children and is inhibited by age 3 to 4.

Tonic labyrinthine reflex: Put the baby in the prone position, bring the neck to 45º flexion. The response is flexion of the arms, trunk and legs. Put the baby in the supine position and bring the neck to 45º extension. Extensor tone will increase. This reflex is present at birth and disappears at 4 months.

Landau reflex: Suspend the baby horizontally. The neck, trunk and arms extend, legs partially flex. This is an advanced postural reaction which appears at 6 months.

Parachute response: The child lies prone on the table. Lift the child vertically and suddenly tilt forward towards the table. The arms and the legs extend as a protective reaction. This is an advanced postural reaction that appears at 8 months.

The child's ability to achieve head control, sit, crawl, stand, and walk is always delayed. Late achievement of a milestone such as sitting indicates the presence of a motor deficit and the degree of delay correlates with the severity of the problem [A,B].

Babies with CP usually have a period of hypotonicity during the early months of life. Between the ages of 6 to 18 months, muscle tone gradually increases in those who are going to develop spasticity. Fluctuations in tone from hypo- to hypertonicity is a characteristic of developing dyskinetic CP. Athetosis becomes obvious after 18 to 24 months. Ataxia may not be apparent until even later.

Early signs suggestive of CP in the infant are abnormal behavior, oromotor problems and poor mobility [C]. The infant is irritable, too docile, or difficult to handle. He does not suck well, sleeps poorly, vomits frequently and has poor eye contact. Deviant oromotor patterns include tongue retraction and thrust, tonic bite and grimacing. Early motor signs are poor head control [D] with normal or increased tone in the limbs [E], and persistent or asymmetric fisting. Motor development is both delayed and abnormal [F]. Instead of crawling, the child moves by creeping or hopping like a bunny. Hand preference during the first two years of life is a sign of hemiplegic CP.

The clinical picture of CP is established in early childhood as the movement problem becomes prominent [G,H].

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Absent Landau reflex at 11 months is a sign of developmental delay.

B	CP is likely if there is no	
Head control		3 months
Sitting		6 months
Rolling over		6 months
Walking		18 months

Signs suggestive of CP in an infant   C

- Abnormal behavior
  - Excessive docility or irritability
  - Poor eye contact
  - Poor sleep
- Oromotor problems
  - Frequent vomiting
  - Poor sucking
  - Tongue retraction
  - Persistent bite
  - Grimacing
- Poor mobility
  - Poor head control
  - Hand preference before 2 years of age
  - Abnormal tone

Absent traction response indicating poor head control in a 10 month old baby.

Increased tone in the limbs and truncal hypotonia is common in spastic quadriplegia.

Children with increased femoral anteversion and adductor spasticity sit in the W-position to maintain balance.

Pathological asymmetrical posture in a 6 year old child

H   Major deficits in patients with CP

Loss of selective motor control and dependence on primitive reflex patterns for movement

Abnormal muscle tone that is strongly influenced by body posture, position & movement

Imbalance between agonist and antagonist muscles that, with time and growth, leads to fixed muscle contracture and bony deformity

Impaired body balance mechanisms

Sensory loss
  Vision
  Hearing
  Superficial & deep sensation

Associated problems
  Seizures
  Mental retardation
  Behavior problems
  Nutrition
  Constipation

A Partial involvement | Total body involvement

Hemiplegia | Diplegia | Quadriplegia
Spastic

Athetoid | Dystonic
Dyskinetic
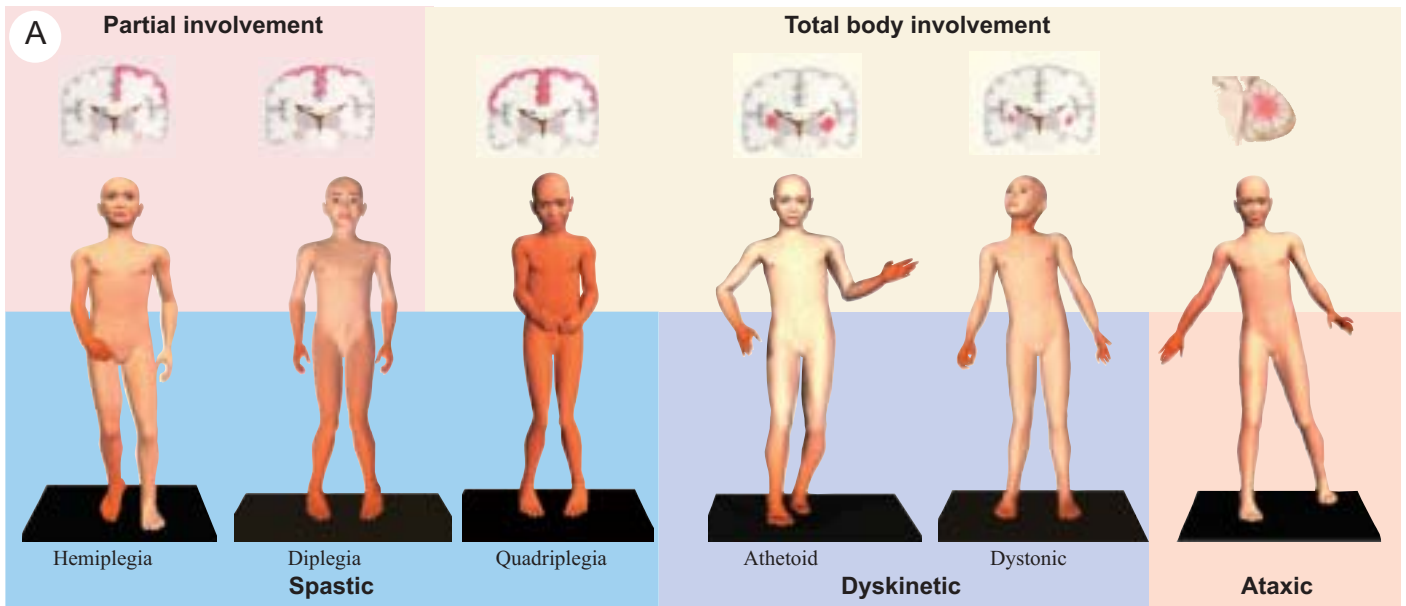
Ataxic

B

Clinical classification	
Tonus	*Lesion site*
Spastic	Cortex
Dyskinetic	Basal ganglia - extrapyramidal system
Hypotonic / Ataxic	Cerebellum
Mixed	Diffuse

C

Anatomical classification	
Location	*Description*
Hemiplegia	Upper and lower extremity on one side of body
Diplegia	Four extremities, legs more affected than the arms
Quadriplegia	Four extremities plus the trunk, neck and face
Triplegia	Both lower extremities and one upper extremity
Monoplegia	One extremity (rare)
Double hemiplegia	Four extremities, arms more affected than the legs

D

All hemiplegic children become independent walkers by the age of 3. Sensory deficits and learning disability add to the movement problem in hemiplegia. Prognosis for independent living is good.

E **F**

Most diplegic children need various treatments to be able to walk independently. Problems in maintaining balance and spasticity interfere with walking. Children who can sit by the age of 2 can usually walk by the age of 4 to 7. Hand dexterity is impaired. Children have difficulty writing or other tasks that need fine motor control. Almost all diplegic children need surgery for contractures and deformities, many use walking aids.

Classification

CP encompasses a spectrum of motor disorders of varying tone, anatomical distribution and severity [A]. Clinicians classify patients to describe the specific problem, to predict prognosis and to guide treatment. Classification is based on the change in muscle tone [B], anatomical region of involvement [C] and severity of the problem. Classification provides a clearer understanding of the specific patient and directs management.

Spastic CP

Spasticity is defined as an increase in the physiological resistance of muscle to passive motion. It is part of the upper motor neuron syndrome characterized by hyperreflexia, clonus, extensor plantar responses and primitive reflexes. Spastic CP is the most common form of CP. Approximately 70% to 80% of children with CP are spastic. Spastic CP is anatomically distributed into three types.

Hemiplegia

With hemiplegia, one side of the body is involved with the upper extremity generally more affected than the lower [D]. Seizure disorders, visual field deficits, astereognosis, and proprioceptive loss are likely. Twenty percent of children with spastic CP have hemiplegia. A focal traumatic, vascular, or infectious lesion is the cause in many cases. A unilateral brain infarct with posthemorrhagic porencephaly can be seen on magnetic resonance imaging (MRI).

Diplegia

With diplegia, the lower extremities are severely involved and the arms are mildly involved [E,F]. Intelligence usually is normal, and epilepsy is less common. Fifty per cent of children with spastic CP have diplegia. A history of prematurity is usual. Diplegia is becoming more common as more low- birth-weight babies survive. MRI reveals mild periventricular leukomalacia (PVL).

Quadriplegia (Total body involvement - tetraplegia)

With quadriplegia, all four limbs, the trunk and muscles that control the mouth, tongue, and pharynx are involved [A and B on opposite page]. When one upper extremity is less involved, the term triplegia is used. Thirty percent of children with spastic CP have quadriplegia. More serious involvement of lower extremities is common in premature babies. Some have perinatal hypoxic ischemic encephalopathy. MRI reveals PVL.

Dyskinetic CP

Abnormal movements that occur when the patient initiates movement are termed dyskinesias [C,D]. Dysarthria, dysphagia, and drooling accompany the movement problem. Mental status is generally normal, however severe dysarthria makes communication difficult and leads the observer to think that the child has intellectual impairment. Sensorineural hearing dysfunction also impairs communication. Dyskinetic CP accounts for approximately 10% to 15 % of all cases of CP. Hyperbilirubinemia or severe anoxia causes basal ganglia dysfunction and results in dyskinetic CP.

Ataxic CP

Ataxia is loss of balance, coordination, and fine motor control [E]. Ataxic children cannot coordinate their movements. They are hypotonic during the first 2 years of life. Muscle tone becomes normal and ataxia becomes apparent toward the age of 2 to 3 years. Children who can walk have a wide-based gait and a mild intention tremor (dysmetria). Dexterity and fine motor control is poor. Ataxia is associated with cerebellar lesions.

Mixed CP

Children with a mixed type of CP commonly have mild spasticity, dystonia, and/or athetoid movements [F]. Ataxia may be a component of the motor dysfunction in patients in this group. Ataxia and spasticity often occur together. Spastic ataxic diplegia is a common mixed type that often is associated with hydrocephalus.

Exceptions

Some children with CP cannot be fitted into these CP groups because they present with many different impairments. Dystonia may be seen in the spastic child, and anatomical classification may not be fully explanatory because clinical findings may overlap. An example is the hypotonic total-body-involved baby who stays hypotonic throughout childhood. Define the pathological abnormalities observed in these children according to the anatomical, and clinical involvement, as described above.

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1997 Russman BS, Tilton A, Gormley ME. 'Cerebral palsy: a rational approach to a treatment protocol, and the role of botulinum toxin in treatment' Muscle Nerve Suppl 6 S181-S193

Total body involved children have various associated impairments. The severity of the motor involvement and the associated problems prevent independent living.

Dystonia is characterised by slow and twisting movements or sustained muscle contractions in the head, trunk and extremities. Children have difficulty in sitting, most cannot walk or talk. Despite the severe movement disorder, intelligence may be normal.

Ataxia is often combined with spastic diplegia. Most ataxic children can walk, but some need walkers.

The movement problem in mixed CP is a combination of spasticity, dystonia and ataxia. Involuntary movements combined with spasticity and muscle weakness may make independent mobility impossible. Upper extremity involvement prevents walker use.

A   Associated problems in CP

Seizures
Visual impairments
Intellectual impairment
Learning disabilities
Hearing problems
Communication problems and dysarthria
Oromotor dysfunction
Gastrointestinal problems and nutrition
Teeth problems
Respiratory dysfunction
Bladder and bowel problems
Social and emotional disturbances

B   Primary impairments due to the neurological lesion

Cortical blindness
Deafness
Intellectual impairment
Epilepsy

Secondary problems - disabilities

Strabismus due to weak eye muscles
Malnutrition due to swallowing deficits

Tertiary problems - handicaps

Loss of binocular vision
Psychosocial problems

C

Primary impairment	Secondary impairment	Deprivation handicap (tertiary impairment)
Eye muscle weakness	Strabismus	Loss of binocular vision
Muscle weakness loss of balance	Impaired mobility	Loss of stimulation & play
Loss of sensation in the hand	Neglect of the involved hand	Failure to develop hand function

Multiple seizures complicate the management of this child. They also lead to mental retardation. Children with mental retardation cannot cooperate with rehabilitation efforts.

E   Visual impairments seen in CP

Pathology	Clinical finding
Damage to the visual cortex	Cortical blindness
Damage to the optic nerve	Blindness
Loss of oculomotor control	Loss of binocular vision
Refraction problems	Myopia

Associated Problems

A number of associated problems occur that increase with disease severity [A]. Cortical blindness, sensory loss, deafness, mental retardation and epilepsy are primary impairments because of the neurological lesion. Disabilities that are secondary to motor deficits are weakness of external eye muscles causing strabismus or difficulties in normal swallowing leading to malnutrition. Malnutrition is an important cause of retarded brain growth and myelination. Lastly, deprivation handicaps occur [B]. The child who cannot move is deprived of peer interaction and stimulation through play. Psychosocial problems develop as a result.

Check for the presence of associated problems and get appropriate referral for treatment. Correct these problems as much and as early as possible to prevent the development of deprivation handicaps [C].

Intellectual impairment

Cognition refers to specific aspects of higher cortical function; namely, attention, memory, problem solving and language. Cognitive disturbance leads to mental retardation and learning disability. The prevalence of moderate, severe and profound mental retardation is 30 to 65% in all cases of CP. It is most common in spastic quadriplegia. Visual and hearing impairments prevent the physician from accurately assessing the degree of intellectual impairment. Children with intellectual impairment need special education and resources to stimulate the senses for optimal mental function.

Epileptic seizures

Seizures affect about 30 to 50% of patients [D]. They are most common in the total body involved and hemiplegics, in patients with mental retardation and in postnatally acquired CP. Seizures most resistant to drug therapy occur in hemiplegics. Seizure frequency increases in the preschool period. Electroencephalograms are necessary for the diagnosis of seizure disorder.

Vision problems

Approximately 40 % of all patients have some abnormality of vision or oculomotor control [E]. If there is damage to the visual cortex, the child will be functionally blind because he will be unable to interpret impulses from the retinas. In severe cases, the optic nerves may also be damaged [F]. Loss of coordination of the muscles controlling eye movements is very common [G]. The child cannot fix his gaze on an object. In half of the cases, binocular vision does not develop. Myopia is a concomitant problem. Screen for visual deficits because some are preventable and they contribute to the movement problem.

Total body involved blind child

Strabismus interferes with binocular vision in this child. Rehabilitation of visual problems may be possible in CP to a certain extent.

Hearing

Sensorineural hearing loss is seen in 10 % of children [A]. Children born prematurely are at high risk for hearing loss. It is generally not diagnosed early because of other handicaps. Test all babies for hearing loss because appropriate hearing devices prevent many future problems resulting from loss of hearing ability.

Communication problems and dysarthria

Dysarthria refers to speech problems. The child has difficulty producing sound and articulating words. Dysarthria occurs in 40% of patients. The causes are respiratory difficulties due to respiratory muscle involvement, phonation difficulties due to laryngeal involvement, and articulation difficulty due to oromotor dysfunction. Spasticity or athetosis of the muscles of the tongue, mouth and larynx cause dysarthria. It is important that every child is provided with an alternative means of communication as early as possible to avoid further disability [B,C].

Oromotor dysfunction

Sucking, swallowing, and chewing mechanisms are impaired [D]. Drooling [E], dysarthria and inability to eat result in failure to thrive, delayed growth and nutrition, poor hygiene [F,G] and impaired socialization.

Gastrointestinal problems and nutrition

There is a general deficiency of growth and development. Children with dyskinesia and spastic quadriplegia fail to thrive [H,I]. This is related to inadequate intake of food [K], recurrent vomiting with aspiration secondary to gastroesophageal reflux and pseudobulbar palsy. Difficulties in swallowing (dysphagia), hyperactive gag reflex, spasticity or loss of fine motor control impair feeding. Gastroesophageal reflux and impaired swallowing cause aspiration pneumonia. Many children with CP have high basal metabolic rates. Increase in basal metabolic rate coupled with feeding difficulties cause malnutrition. Malnutrition may be severe enough to affect brain growth and myelination in the first 3 years of life. There is immune system suppression and increased risk of infection.
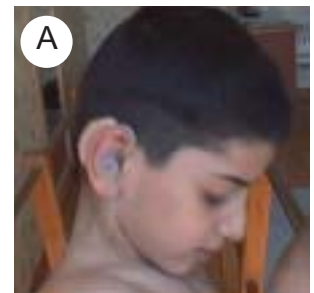
This severely involved diplegic child with hearing impairment has been using hearing aids ever since he was a baby.

Communication aids range from advanced computer systems to simple picture boards. Children with adequate mental function learn to use these to interact with their environment.

Mouth hygiene is poor and dental caries is common. Obtain regular dental care.

G	Teeth problems
Dentin	Primary or hyperbilirubinemia
Malocclusion	Spasticity
Tooth decay	Feeding, swallowing problems
Gingival hyperplasia	Antiepileptic drug use

D	Oromotor dysfunction
	Drooling
	Dysarthria
	Inability to chew
	Inability to swallow

Spastic quadriplegic child with malnutrition

I	Reasons for failure to thrive
	Inadequate food intake
	Recurrent vomiting
	Aspiration
	High basal metabolic rate

K	Causes of inadequate food intake
	Difficulty chewing and swallowing
	Hyperactive gag reflex
	Spasticity of oropharyngeal muscles
	Loss of selective control of oropharyngeal muscles
	Gastroesophageal reflux

Drooling and strabismus coexist in this child. Drooling is caused by oromotor dysfunction and is a difficult problem to treat. Consider oral medications and botulinum toxin in management.

The severely involved mixed quadriplegic child seen above was 14 years old and weighed only 15 kgs. He could not speak, chew or swallow. He had drooling and gastroesophageal reflux. After his esophageal ulcers bled twice, he had a gastrostomy tube inserted 2 years ago. He has been steadily gaining weight ever since.

Respiratory problems

Aspiration in small quantities leads to pneumonia in children who have difficulty swallowing. Premature babies have bronchopulmonary dysplasia. This leads to frequent upper respiratory tract infections. Respiratory muscle spasticity contributes to the pulmonary problems.

Bladder and bowel dysfunction

Loss of coordination of bowel and bladder sphincters results in constipation and/or incontinence. Enuresis, frequency, urgency, urinary tract infections and incontinence are common problems [A]. The causes are poor cognition, decreased mobility, poor communication and neurogenic dysfunction [B]. Urodynamic assessment has demonstrated bladder hyperreflexia, detrusor sphincter dyssynergia, hypertonic bladders with incomplete leakage and periodic relaxation of the distal sphincter during filling.

Constipation is a common but overlooked phenomenon. It causes distress in the child, increases spasticity and results in poor appetite. It is a result of many factors, including poor diet and decreased mobility. Establishing a routine for bowel training and encouraging upright posture help reduce constipation.

Psychosocial problems

A diagnosis of CP is extremely stressful for the family and the child when he grows up. This causes various reactions ranging from denial to anger, guilt and depression. Coping with the emotional burden of disability is easier if the family has strong relationships, financial security, and supportive members of the community. The child and the family need to find ways to connect to each other. A healthy relationship between the mother and the child forms the basis of future happiness.

Prevention or appropriate treatment of associated problems improves the quality of life of the child and the family [C].

A Urinary problems	Causes of urinary problems
Enuresis	Poor cognition
Frequency	Decreased mobility
Urgency	Decreased communication skills
Urinary tract infections	Neurogenic dysfunction
Incontinence	

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Physical Examination and Making the Diagnosis

Physical examination of a child with movement problem has two basic purposes [A]. First, physical examination accompanying a detailed history enables an accurate diagnosis. Second, it allows the treating physicians to define the impairments and disabilities, determine the functional prognosis and set treatment goals in children with CP. These then help devise a treatment plan for each child.

It is difficult to identify the cause of CP. When faced with a motor disorder in the child, the physician must be careful to rule out conditions that are results of genetic defects, such as hereditary spastic paraplegia, that are similar to CP. A detailed history and physical examination help the clinician exclude these rare syndromes and prevent expensive and extensive work-up.

Physical examination of the child with CP is not easy. It is a three-way relationship between the child, the physician and the family [B,C]. Adjustment problems can cause fear, distrust, confusion, and anxiety in the family and in the child. This disturbs their capability to understand the problem and cooperate with the treatment team. The physician must be willing to deal with anxious, confused, frustrated and unhappy families and frightened children. The examination cannot succeed unless the physician gains the parents' confidence and trust. Parents will trust a physician who takes a genuine interest in their child.

History

History is a key component in evaluating the child [D]. It provides valuable information for diagnosis. In children with a definite diagnosis, the timing of achievement of developmental milestones and the presence of associated impairments help to decide a functional prognosis. The physician gains insight into the parents' expectations and disappointments from previous treatment procedures. Knowledge of previous botulinum toxin injections, physiotherapy, surgical procedures, outcomes, complications, and psychological burden are key issues when making a treatment plan [E].

History taking provides the time and room to build a sense of understanding between the family and the physician. The goal is to make the child and the family comfortable so that the clinical examination will be accurate.

Goals of physical examination in a child with movement disorder	A
Establish an accurate diagnosis	
Differentiate CP from progressive causes of childhood neuromotor disability	
Classify the type and severity of involvement	
Define the musculoskeletal impairment (spasticity, balance, weakness, contractures and deformities) and decide on ways of treatment	
Evaluate associated impairments and get appropriate treatment	
Determine functional prognosis	
Set treatment goals	
Devise a treatment plan	
Evaluate the outcome of previous treatment procedures	
Assess the changes that occur with treatment as well as with growth & development	

A lot of information can be gained by watching the child move. The child is generally upset and frightened when he first enters the doctor's office. He must have some time to get used to his surroundings. In the meantime, the doctor should calmly talk and play with him while watching him. The room must be spacy, well-lit and friendly to calm the upset child. Play is a wonderful opportunity to establish a relationship.

Examination of the child with CP may be difficult and frustrating. Playing with the child and using simple objects as toys helps the physician to establish a better contact with the parents and the child.

D	A detailed history provides knowledge about
	Risk factors
	Timing of achievement of developmental milestones
	The presence of associated impairments
	Progression of child's capabilities
	Insight into the family's resources

E	Key points in history		
Health of parents	Development and present status of	Convulsions
Hereditary factors		Emotional development
Siblings	Head balance & control	
Pregnancy	Sitting	Social and recreational activities
Labor and delivery	Crawling	School placement
Rh factor	Walking	Parental attitude
Birth weight	Feeding	Braces
Condition at birth	Dressing	Medication
Neonatal history	Toilet care	Previous treatment
Age disability recognized and symptoms noted	Speech	Reason for referral
	Mental status	
	Hearing	
	Vision	
	Handedness	
	Drooling	

Try to be friends with the older child and the adolescent. Talk to them alone and if necessary perform the examination when members of the opposite sex are not present. Do not treat older children like babies.

Examination begins with observation. Do not touch the child before you watch her move.

Examine the young and frightened child in his mother's lap. Evaluate tonus abnormalities when the child is comfortable.

Guide the parents to help you during the clinical examination.

Start the examination by giving the child something to play with. A ball or a balloon will help to test upper extremity function.

Let the child sit in the mother's lap. Provide the older child with a chair of his or her own. Remember to smile at and talk to the child. Generally the parents provide the information. Older children should answer for themselves [A,B]. The parents can help fill in the details later. Including the child in the conversation builds trust. If the parents trust the physician, they will be more open-hearted when expressing their expectations and disappointments.

The child and the parents will remember less than 20% of the information provided. Provide them with a written summary of the results and the treatment plan for future reference.

Clinical Examination

Observing the child's movements is the initial and a crucial part of the examination. Observe before you touch [C]. If the child is young, apprehensive or tearful, let him or her stay on mother's lap while you watch and talk to the mother. As the child adapts to the environment, slowly place him or her on the examination table or on the floor and watch him or her move around. If the child cries a lot and does not cooperate, continue while he or she is in the mother's lap [D].

Tools required for the examination are very simple: toys, small wooden blocks, round beads or pebbles, triangular, circular and square shaped objects, a few coins, objects with different textures and a tape measure.

Perform a neurological, musculoskeletal and functional examination, although not necessarily in that order [E]. Every physician develops his or her own style and sequence of examination over the years [F,G].

Examination outline
Neurological examination
Skull, head circumference
Spine
Mental status
Cranial nerves
Vision - hearing - speech
Motor system
Muscle tone
Muscle power
Muscle bulk
Degree of voluntary control
Reflexes
Involuntary movements
Sensory examination
Sphincters
Developmental milestones
Musculoskeletal examination
Range of motion
Deformities, contractures
Posture
Functional examination
Sitting
Balance
Gait
Hand function

Neurological examination

Neurological evaluation of the infant and the child requires adequate knowledge of neurological developmental stages [A].

Mental status

Observe the child's orientation and interest in the surroundings. Watch for eye contact, following objects, alertness, and ability to obey simple commands.

Vision and hearing

The diagnosis of visual and hearing loss in infants can be easy. Call the child when he is not looking. Clap your hands or deliberately drop an object to make a noise behind the child and watch the response. If the child does not seem to hear, look in the child's ears for wax or signs of infection. Considering the high incidence of visual and oculomotor problems in cases of CP, all children with a definite diagnosis of neurodevelopmental delay and/or CP should undergo a detailed ophthalmological and audiological examinations during early infancy. The examinations should be repeated at yearly intervals until school age.

Muscle strength and selective motor control

Many children with CP cannot voluntarily contract or relax their muscles in isolation and therefore are unable to move their joints separately. For example, when the child attempts to extend his elbow, he involuntarily moves his whole arm. Lack of selective motor control makes it impossible to determine muscle strength using simple manual muscle testing [B]. Observe muscle strength by watching the child perform certain tasks, such as throwing or hitting a ball.

Reflexes

Evaluate the persistence of primitive reflexes and the absence of advanced postural reactions [C,D]. The presence of primitive reflexes beyond 6 months of age is a sign of poor prognosis [E].

Muscle tone and involuntary movements

The child must be calm for assessment of muscle tone. Place the head in neutral position because turning or flexion can trigger tonic neck reflexes and interfere with muscle tone. Spasticity is the resistance felt while moving the joint through a passive range of motion. Use the modified Ashworth or Tardieu scales to grade spasticity. Also record tremor, chorea, athetosis, dystonia and ataxia [F].

A	Normal developmental stages of the child
Age (months)	*Milestones*
1	Lifts head
3	Good head control, follows, laughs, smiles
5	Reaches and grasps objects
6	Propped sitting
8	Independent sitting, equilibrium reflexes
9	Gets to sitting position, presents parachute reflex
10	Pulls to stand, cruises
12-14	Walks, first words
18	Removes clothes, uses spoon
24	Uses two word phrases, throws overhand
30	Knows full name, puts on clothing
36	Jumps, pedals tricycle, learns rhymes
48	Hops, plays with others

Children who do not have selective motor control are unable to dorsiflex the ankle without moving the hip or knee; but they can produce this response in a gross flexion movement of the lower extremity. This is called pattern response. When the child flexes the hip against resistance, ankle dorsiflexion becomes apparent.
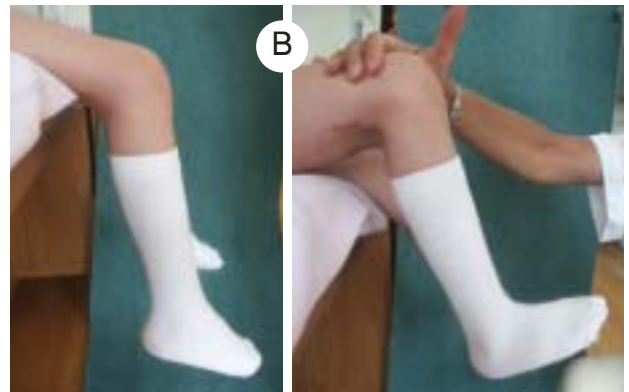
This spastic quadriplegic child still has not developed protective extension and is severely delayed.

This 8 month old baby has difficulty maintaining head control on traction response indicating developmental delay.

E	Signs of poor prognosis	
	Present	*Absent*
	ASTNR	Parachute response
	STNR	Neck righting reactions
	Moro	
	Extensor thrust	
	Stepping reflex	

F	Differences between spasticity & dystonia	
	Spasticity	*Dystonia*
Examination	You feel	You see
Tendon reflexes	Increased	Generally normal
Clonus	Present	Absent
Pathological reflexes	Present	Rare

A   Musculoskeletal examination

- Joint range of motion (ROM)
- Deformities
- Contractures
- Balance
- Posture
- Sitting
- Gait

B

Deformities are not apparent in many young children when they lie supine. Bring the child to erect position to demonstrate dynamic deformities.

C

Spinal deformity	Occurs in
Scoliosis	Total body involved spastic and dystonics
Kyphosis (thoracolumbar)	Patients with no sitting balance
Kyphosis (lumbar)	Patients with hamstring contractures
Hyperlordosis (lumbar)	Ambulatory patients with hip flexion contractures

D

E

Inspect the back when the child is sitting. Visual examination is sufficient to detect scoliosis or kyphosis.

Musculoskeletal examination

The musculoskeletal examination [A] reveals contractures and deformities that interfere with mobility. Perform the examination in a comfortable room with adequate space and props to attract the child's attention. Control spasticity by relaxing the child.

Range of motion

Examine range of motion in a slow and smooth manner because sudden stretch of the muscle will increase spasticity, creating the false impression of a fixed joint contracture.

Most young children do not have fixed deformities. The hip and knee joints can be moved through a full range of motion when the patient is prone or supine. However, the child will demonstrate hip flexion and adduction, knee flexion or extension and ankle equinovarus or valgus in the erect position when weightbearing. This is dynamic deformity caused by spasticity, impaired motor control and weakness of muscles [B].

Severe dynamic deformity caused by spasticity is difficult to differentiate from contracture. Stretch slowly, reassure the child and provide a relaxed and calm atmosphere in which to assess muscle tone.

Back assessment

Spinal deformity [C] associated with CP might be postural or structural and includes scoliosis, hyperkyphosis, and hyperlordosis. Patients lacking sitting balance often exhibit a long postural kyphosis. Lumbar hyperlordosis occurs in ambulatory patients with hip flexion contractures, whereas lumbar kyphosis occurs in patients with hamstring contractures. Inspect the back for scoliosis and kyphosis with the patient standing and in forward flexion. Examine the back of the nonambulatory child [D,E] while he or she sits in the wheelchair. Have the child bend forward as you check for any paramedial elevations indicating lumbar spine involvement or rib elevations showing thoracic spine involvement. Note sitting balance and pelvic obliquity, if present. Contracture and/or limb length discrepancy also contribute to spinal asymmetry.

Pelvic obliquity

Pelvic obliquity is the abnormal inclination of the pelvis in the frontal plane. It is commonly associated with scoliosis and hip instability in the nonambulatory child. Check for sitting balance in the child with scoliosis and hip dislocation.

Limb-length discrepancy

Measure actual lower limb lengths from the anterior superior iliac spine to the medial malleolus. Measure from the trochanter to the knee joint line and from there to the medial malleolus if knee flexion contracture is present [F,G].

F

G

Limb length discrepancy is a sign of pelvic obliquity or hip instability. Actual limb length discrepancy may occur in hemiplegic CP due to unilateral growth asymmetry. Repeat the measurements for limb length in the upright position if you wish to prescribe shoe lifts.

Hip assessment

Measure passive and active hip range of motion. Check for flexion and adduction contractures. Evaluate flexion contracture with the Thomas test.

The Thomas test is based on the fact that a hip flexion contracture is compensated by an increase in lumbar lordosis. If the lumbar spine is fixed in the neutral position, lordosis is prevented and hip flexion contracture becomes apparent. The child lies supine on the examination table. Bring both legs up to the chest to stabilize the lumbar spine and decrease the lordosis [A]. Flex the hip and feel that the pelvis is straight. Then, keep one leg in flexion while extending the other until resistance in hip extension is felt or movement in the pelvis occurs [B]. The extended leg should be flat with the knee in full extension. If knee flexion contracture is present, extend the knee beyond the table. The angle between the thigh and the examination table gives the degree of the flexion contracture of the hip [C,D].

Test for adduction contracture Evaluate range of abduction with the hips in flexion [E] and in extension [F]. If abduction is limited when the hips are extended but better when they are flexed, the adduction contracture is caused by gracilis and medial hamstring spasticity. If hip abduction is limited in both extension and flexion, the cause is hip adductor spasticity.

The Ely test shows rectus femoris tightness. The rectus femoris flexes the hip and extends the knee, crossing both joints so that when the hip is in extension, it is difficult to flex the knee if the rectus is tight. With the child lying prone [G], stabilize one hip in extension and bring the lower leg quickly into flexion [H]. If the buttock rises off the table, it is a sign of spastic or tight quadriceps muscle [I].
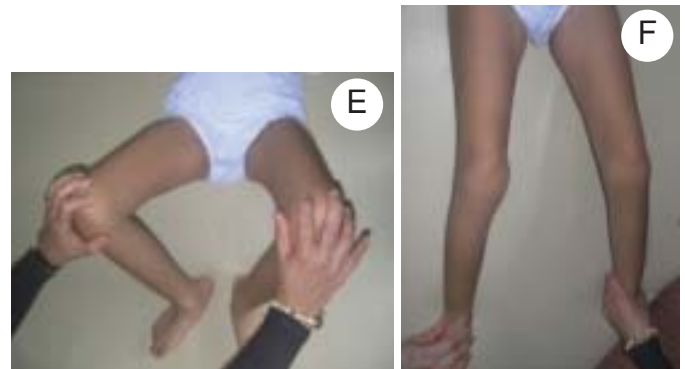
Use the Ely test to demonstrate rectus femoris spasticity and hidden flexion contracture of the hip. Most children are unhappy in the prone position so they will have increased muscle tone. Be careful not to mistake increased tone from actual contracture.

If the leg is brought into flexion swiftly, the Ely test will demonstrate rectus femoris spasticity. Do the test once more, slowly, in order to differentiate rectus femoris tightness from spasticity.

Test for hip rotation Test in prone position with the knee in flexion. Excessive internal rotation suggests persistent femoral anteversion [J,K].
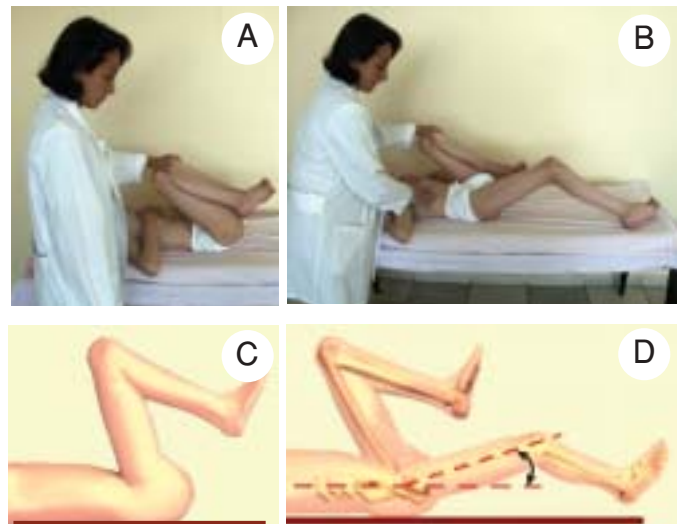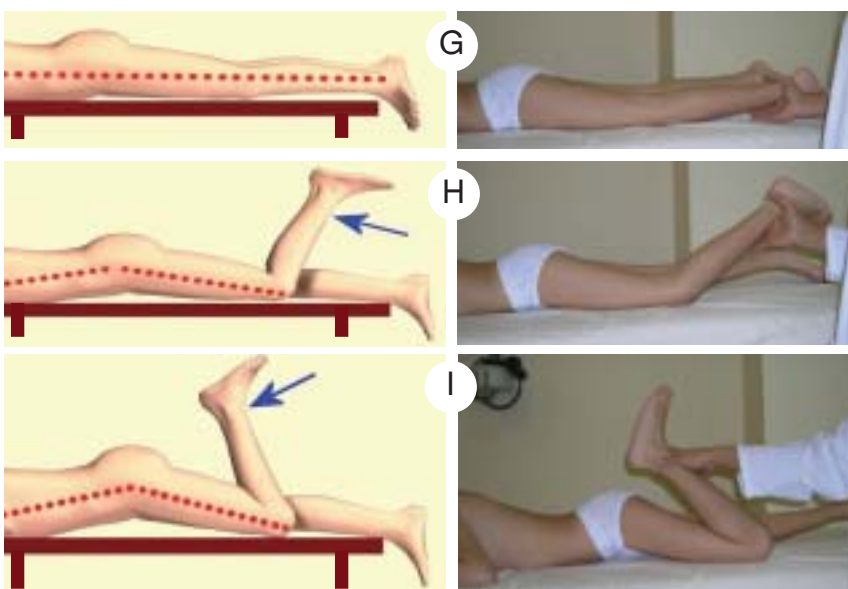
The Thomas test shows flexion contracture of the hip. Do not hyperflex the pelvis when flexing the hip.
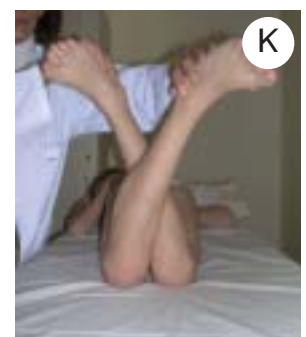
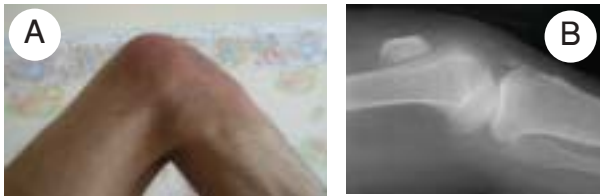Test adduction contracture in both flexion and extension.

Test for hip rotation: Excessive internal rotation with the patient in prone position
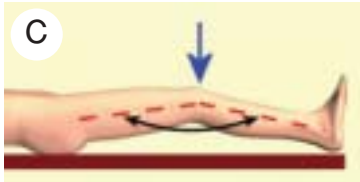
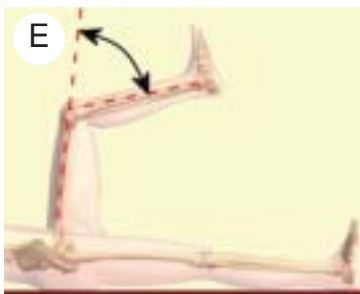The Ely Test demonstrates rectus femoris tightness and hip flexion contracture.

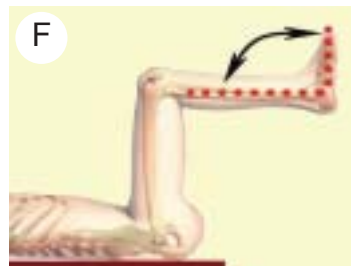Test for hip rotation:
Excessive external rotation

Evaluation of patella position
Look for a high riding patella (patella alta) which is common in cases of rectus femoris spasticity.

Test for posterior capsule tightness
1. Extend the child's legs on the examination table.
2. Force the knees and the hips in full extension.
3. The back of the knee should touch the table.
4. Limitation indicates tight posterior capsule.

The popliteal angle
Stabilize one leg on the table, then flex the other hip to 90°. Extend the lower leg until you feel resistance. Measure the angle from either the tibia and the line of full extension or the 90° position to full extension. Popliteal angle shows the amount of hamstring contracture.

Knee assessment

The patella position   Evaluate the patella position with the child supine and sitting. The patella slides up in children with severe quadriceps spasticity [A,B].

Posterior capsule tightness   Extend the leg. If it does not extend fully, slowly force the knees and hips into full extension. Limitation indicates posterior capsule tightness [C].

Popliteal angle Measure the popliteal angle to test for hamstring contracture [D,E].

Foot and ankle assessment

Evaluate contractures and deformities of the ankle and subtalar joints and toe deformities.

Test for triceps (gastrocnemius/soleus) contracture   The gastrocnemius muscle is shortened and the soleus is normal in most children. Use the Silfverskiöld test to assess triceps surae tightness. 1. Lie the patient in supine position. 2. Measure ankle dorsiflexion first with the knee in flexion [F] and then in extension [G]. If the ankle dorsiflexion is greater when the knee is flexed, the gastrocnemius is shortened and the soleus is normal. If dorsiflexion is unchanged with the knee in flexion or extension, then both gastrocnemius and soleus are contracted. Always hold the foot in slight inversion while performing this test.

Test for tibial torsion Examine tibial torsion with the patient in the prone position. Evaluate the thigh-foot angle with the knee flexed to 90 degrees [H].

Evaluation of posterior tibialis, anterior tibialis and peroneal muscles A spastic posterior tibialis muscle causes hindfoot varus [I]. A spastic anterior tibialis muscle also causes varus and must be carefully evaluated in mono- and hemiplegic patients. A spastic peroneus or gastrocnemius muscle may cause a valgus deformity [J].

Foot deformities Pes valgus, pes varus and hallux valgus [K,L] occur in ambulatory children.

The Silfverskiold test: (The S test) shows spasticity in the triceps muscle.
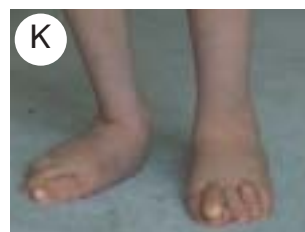

Examine external tibial torsion in the prone position with knees flexed to 90 degrees. The angle of the feet relative to the thigh (the thigh-foot angle) shows the degree of external tibial torsion.

Pes equinovarus is common in hemiplegia.

Gastrocnemius spasticity is another cause of pes valgus.

Pes valgus and hallux valgus generally occur together.

Striatal toe is seen in patients with extensor hallucis longus spasticity.

Upper extremity examination

Examination for the hand and upper extremity consists of observation and evaluation of joint range of motion, the presence of contracture, muscle strength, and sensation [A]. Testing wrist and finger muscle contracture requires a detailed examination [B,C,D,E].

Spasticity of intrinsic hand muscles causes flexion contracture [F,G,H] of the metacarpophalangeal (MCP), proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints. Superficial flexor tightness causes PIP joint limitation whereas deep flexor tightness causes DIP joint limitation. The most common deformity is thumb in palm deformity.

Using local anesthetic blocks to test contractures It is difficult to assess each hand muscle separately. Inject local anesthetic to the nerve to block the muscles innervated by that nerve. Then check for muscle tightness. If the muscle does not relax, fixed contracture is indicated. Blocking the spastic muscles with a local anesthetic unmasks active contraction in the antagonist muscles. Block the median nerve at the wrist to relax the wrist and finger flexors. This allows you to see activity in wrist and finger extensors. Antagonist muscle function is important when considering reconstructive surgery.

Using dynamic electromyography to test contractures Dynamic electromyography identifies which muscles are active and when they are active. Actively contracting muscles can be used for transfers. Transfers are more effective if the transferred muscle group fires in phase with the recipient muscle group.

Try to recognize adaptive responses so as not to interfere with them. Efficient hand grasp depends on balance between flexor and extensor muscles. Wrist flexors are dominant and the finger extensors are weak in the hand with spasticity. When the child wants to grasp objects, he brings the wrist into flexion by releasing his finger flexors. The child then locks the object in the palm by bringing the wrist into extension. This is not a strong grasp, but an adaptive mechanism that is valuable to the child. Flexor releases will lead to loss of hand grasp in children with this adaptive response.

Lack of sensation is a significant disability. Evaluate stereognosis, two-point discrimination, and proprioception. Stereognosis is the ability to recognize an object by touching it without looking at it. This ability requires the synthesis of multiple sensory inputs at the cortical level.

Consider using age-appropriate simple tasks to test upper extremity and hand function [I]. These include combing the hair, brushing teeth, putting food to mouth, drawing a triangle and throwing a ball.

The appearance of the hand is important especially for older girls with hemiplegia since the hand is a social tool for communication. Evaluate the appearance of the hand and the patient's satisfaction with it.

A	Examination of the upper extremity
	Joint range of motion
	Presence of contracture
	Muscle strength
	Coordination
	Sensation
	Function
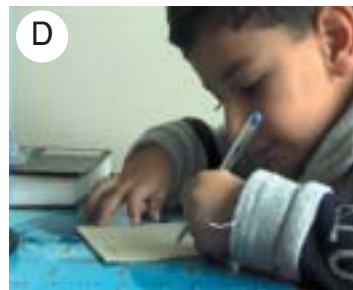
1. The small child sits in the mother's lap and the older in a comfortable chair.
2. A desk or a table is necessary for testing fine motor control and coordination. Children without sitting balance use their regular wheelchair with a tray placed on their lap.
3. Watch active shoulder and elbow movements while the child plays with a plastic or wooden cylindrical toy to detect movement disorders and spasticity.
4. When the child grasps the object, observe the gross motor function of the hand, the quality of grasp and release and the ability to cross the midline. For wrist and finger fine movements, small plastic beads or toys are better.

E: Courtesy of M. Carlsson

Volkmann angle demonstrates wrist flexion contracture.

H	Flexion contracture
1. Measure flexion contracture of the wrist with the wrist in full flexion and the fingers in full extension.	
2. Slowly and gently pull the wrist into extension while keeping the finger joints in extension.	
3. The angle of wrist with the forearm is the angle of flexion contracture.	
4. Then evaluate the PIP & DIP joints separately to determine the spastic muscle group.	

Note the speed and precision of movement and the quality of grasp and release. Observe how much the child uses his involved extremity.

A   Classification of sitting ability

Hands-free sitter (Independent sitter)	can independently come to a sitting position, does not need hands to sit up and can sit in a normal chair without losing his balance.
Hand-dependent sitter	uses hands for support when sitting, needs a chair with side supports to be able to use his hands for eating or writing.
Propped sitter	has to be brought to a sitting position by someone else, needs external support and sits in a reclining position when strapped into the seat.

This classification applies particularly to quadriplegic children as a guide to the use of seating aids.

Hands-free sitter

Hand-dependent sitter

Propped sitters needs external support to sit.

The Romberg sign: the child cannot maintain his balance with eyes closed.

Functional examination

Sitting

Evaluate sitting [A] to decide whether the child needs support [B-E]. Children with adequate sitting balance are more functional.

Balance

Balance and equilibrium reactions are prerequisites for walking. Evaluate balance in all children. Push the standing child gently from the front, back, and side to see whether he or she can promptly regain balance. Assess deficiency of balance and equilibrium using the Romberg sign, unilateral standing balance test and the hop test.

Romberg sign [F,G] shows whether the child can maintain balance. If the child sways and cannot keep his balance with feet held together and eyes closed (positive Romberg's sign), then there is sensory ataxia. If the Romberg sign is negative in the ataxic child, the ataxia is of cerebellar origin.

Unilateral standing balance test [H,I] Reveals inability to maintain balance in less severely involved children. A 5 year old should be able to stand on one foot for 10 seconds. Failure in the unilateral standing balance test explains why children sometimes show excessive trunk leaning when walking.

Hop test Boys can hop on one leg for five to 10 times from age 5 years and girls from age 4 years onwards. Inability to perform single-leg hop is another sign of poor balance and neuromuscular control.

Mobility

A crucial part of the examination is the observation of the child's walking pattern [J]. Video recordings of the child's movement also guide treatment. Ask the family to obtain photographs or video recordings of their child to understand how the child functions at home. Computerized gait analysis is possible in advanced centers. The nonambulatory child is placed on the floor to assess his mobility [C-F on opposite page]. The child may roll, creep, crawl or 'walk on all fours'.

Unilateral standing balance test: The child is able to stand on her right foot but tends to fall when she stands on her left foot. This is a sign of unilateral balance disturbance.

J   Classification of ambulation

Community ambulators	are free to ambulate in the community independently with or without orthotics or assistive devices.
Household ambulators	walk independently indoors using braces and assistive devices. They need a wheelchair for outdoor mobility.
Therapeutic ambulators	walk as part of a therapy session for short distances with a helper. They need a wheelchair at all other times.
Nonambulators	use the wheelchair for mobility.

A	Functional scales used in CP		
Scale		*Ages*	*Measures*
Gross Motor Function Measure		Birth to 5 years	Change in gross motor function over time compared to normal children
The Pediatric Evaluation of Disability Inventory		6 months to 7 years	Functional status and functional change
Wee Functional Independence Measure		6 months to 7 years	Level of independence in 6 different areas
The Movement Assessment of Infants		Birth to 12 months	Gross and fine motor performance of infants

Functional scales

Different scales are used to assess the functional status of patients with CP [A]. Some are descriptive and compare the child with normal age-matched peers whereas a few of them measure change over time that occurs with growth and treatment. Functional tests identify babies and children who have delayed gross or fine motor development and record the progress of those children under treatment. Quality of life is measured with scales such as the Child Health Questionnaire and the Care and Comfort Measure.

Study, skill and experience render the application and scoring of most of these scales easier. Instructional courses are required to apply the Wee Functional Independence Measure (WeeFIM) and the Pediatric Evaluation of Disability Inventory (PEDI). These scales are expensive and not very beneficial for the patients but necessary for medical research. Many require at least an hour to administer which makes it difficult to use in outpatient clinics. The busy clinician must decide on his or her methods of assessing the functional status of the child and the effect of treatments. The Gross Motor Function Measure (GMFM) and the Gross Motor Classification System (GMFCS) have been the most helpful to date and they are freely available for use.

Gross Motor Function Measure (GMFM)

The GMFM was developed to measure changes in gross motor function over time in children with CP. It compares the child with normal children of the same age. The GMFM is a reliable scale to evaluate gross motor function. It measures the child's skill in lying, rolling, sitting, crawling, kneeling, standing, walking, running, and jumping, but it does not measure the quality of movement. It can be used for children from birth to 5 years of age.

Gross Motor Function Classification System (GMFCS)

The Gross Motor Function Classification System (GMFCS) [B] was developed to create a systematic way to describe the functional abilities and limitations in motor function of children with CP. The emphasis is on sitting and walking. The purpose is to classify a child's present gross motor function. Five levels are used in the GMFCS from very mild to very severe. The levels are based on the functional limitations, the need for assistive technology and wheeled mobility. The quality of movement is not very important. Because motor function depends on age, separate scales are used for different age bands. Classification at 2 years allows one to predict prognosis at age 20 years.

Gross Motor Function Classification System (GMFCS)		B
Level	*Ability*	
1	Walks without restrictions	
2	Walks without assistive devices but limitations in community	
3	Walks with assistive devices	
4	Transported or uses powered mobility	
5	Severely limited dependent on wheelchair	

This ten year old girl with mixed CP can use a walker for ambulation. A closer look at her knees shows callosities on both patellae. This indicates that her primary means of mobility is crawling on all fours.

Reciprocal movement is the ability to move one extremity after the other while crawling or walking. It is a sign of good motor control.

A Progressive disorders resembling CP

Glutaric aciduria Type I

Arginase deficiency

Sjögren – Larsson syndrome

Metachromatic leukodystrophy

Lesch - Nyhan syndrome

Joubert syndrome

Chiari Type I malformation

Dandy - Walker syndrome

Angelman syndrome

Gillespie syndrome

Marinesco - Sjögren syndrome

Ataxia - Telangiectasia

Hexoaminidase A and B deficiency

Behr syndrome

Serotendinosus xanthomatosis

B Nonprogressive disorders resembling CP

Mental retardation

Deprivation

Malnutrition

Non-motor handicaps (blindness)

Motor handicaps (spina bifida, myopathies)

Early differential diagnosis in developmental disability

| C	Cerebral palsy	Mental retardation
Risk factors	Often positive	Mostly absent
Complaints	Irritable, sleepless baby	Easy baby
Milestones	Delayed	Delayed
Examination	Delayed growth or negative	Negative or a syndrome
Muscle tone	Increased	Hypotonia
Primitive reflexes	Persist	Normal disappearance
Postural reflexes	Delayed appearance	Delayed appearance
Focal signs	Appear	Absent

Radiographies are the standard evaluation method for hip instability. A three dimensional CT scan provides valuable data in the preoperative evaluation for hip reconstruction.
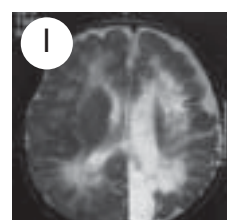
Evaluate scoliosis with radiographies
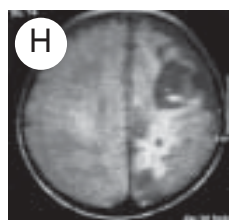
The GMFCS is an important tool for physicians and therapists treating children with CP. It is easy to use; classifying a child takes 5 - 15 minutes. Physicians and therapists from various disciplines can easily use this scale for their patients. Therefore, it provides a basic understanding of the level of involvement of a child for all those involved in caring for the child. The use of the GMFCS is becoming increasingly common in CP clinics as a universal tool for communication with colleagues, determining the prognosis and planning treatment.

Some scales and suggestions for further reading are provided in the appendix.

Differential diagnosis

One needs to distinguish CP from progressive disorders of childhood [A]. It may not be always necessary to find the exact cause because this does not change the management for most children (with the exception of inborn errors of metabolism that can be cured). Mental retardation syndromes, attention deficit disorder, autism and non-motor handicaps such as blindness and emotional disorders also cause motor delay [B,C]. Cognitive problems are prominent in all these syndromes except for blindness. On the contrary, motor problems are predominant in CP. All children with suspected motor delay should be seen by a pediatric neurologist to assess for differential diagnoses.

Imaging studies

Imaging studies enable the physician to define the type and location of the brain lesion and to differentiate progressive neurological syndromes.

Radiology

The primary indication to perform radiography in cases of CP is to monitor hip instability. Obtain baseline spine and hip radiographs in every child and follow the hip at risk with hip radiographs [D]. Measure the Reimer's index which is the percentage of femoral head coverage by the acetabulum. Three-dimensional CT is useful when planning hip reconstruction [E]. Clinical examination is sufficient to diagnose and follow-up scoliosis. Measure the Cobb angle in children who are candidates for surgery [F]. Obtain radiographs of the extremities for patients if you plan osteotomies. Standing radiographs of the feet help if there are varus/valgus deformities.

Cranial ultrasonography (USG)

Cranial USG [G] can help in the differential diagnosis of the infant when the fontanelle is open. It is easy and it does not require sedation as does MRI. Cranial USG evaluates the ventricles, basal ganglia and corpus callosum. Periventricular white matter ischemic injury and intraventricular haemorrhage are apparent on real-time cranial ultrasonograms.

Cerebral computerized tomography (CT)

CT is helpful in the diagnosis of intracranial bleeding in the newborn, it may be helpful in evaluating congenital malformations and PVL [H] but in these and other lesions MRI is superior.

Cranial magnetic resonance imaging (MRI)

MRI is the best method for diagnosing lesions in the white matter after 2 to 3 weeks of age [I,K]. At present, MRI and

Cranial ultrasound

Porencephaly on cranial CT

Periventricular leukomalacia on MRI

Porencephaly on MRI

ultrasonography are the only methods to show periventricular leukomalacia in an infant from 1 week of age. No biochemical methods are available to identify high-risk infants at birth.

Electroencephalography (EEG)

EEG measures electrical activity on the surface of the brain. It is a necessary tool in the diagnosis and follow-up of seizure disorders.

Explanation of the diagnosis to parents

The diagnosis takes time. The child must be at least 1 year old before a definite diagnosis can be made, especially in cases in which the lesion occurs during the prenatal or perinatal stage (approximately 80% of all CP cases). The infant with a cerebral dysfunction shows signs of neurodevelopmental delay at initial examinations. Tone abnormalities such as spasticity and dystonia, or disorders of movement such as ataxia appear generally at or after 12 months of age, when it will be possible to name the movement disorder as CP.

Wait until the childhood period for a definite diagnosis. Provide those babies who show delayed development with an adequate exercise program to stimulate the CNS. In the eyes of the society the diagnosis of CP labels the child as handicapped or abnormal. Be cautious about the diagnosis. Avoid initial use of the term *cerebral palsy* to the parents because of the stigma. Instead, describe the child's symptoms and why they occur. Parents are anxious to learn about their child's problems and need definite answers. Evaluate the child's condition and prognosis well in order to provide satisfactory answers without raising false hopes or breaking all hope [A].

The diagnosis is difficult in the infant. The extent and severity of involvement that a child will have in the future is impossible to assess when he is a baby. Do not talk about the child's problems in detail. This causes disappointment in the parents who start to feel bad about their child. Slowly prepare parents for the possibility of damage to the brain. Let the child and the family bond together early in infancy. Be very cautious when predicting the future because the outcome is variable.

How to explain the diagnosis to the parents?

For a baby:

Your baby has had an injury to his brain. He is developing a little slower than his peers. He may have problems with movement. The outlook depends upon how serious or how extensive the brain damage is. Because he is a baby, right now it is difficult to tell what the future outlook will be eventually. We have to follow him carefully, he has to have therapy and we will see how he will develop. All children develop along a certain pathway. Children who have this brain injury that we call cerebral palsy also develop along a certain pathway but they do it more slowly. If a normal child sits at the age of 6 months, he may not sit to a year and a half; if a normal child walks at one year, he may not walk until he is 3 or 4 years old. We will see how he will improve as he grows older.

For a child:

Your child has difficulty controlling his movements. This is because of an injury in the movement center in the brain. The brain injury is permanent. It does not get worse, but it never really gets better. Regardless, the child deserves anything we can do for him to achieve whatever potential he has. The most important things are talking, being able to eat and to take care of himself, then mobility and walking. We can help the child move better by relaxing his muscles, teaching him exercises and giving him various braces. He may need injections or surgery in the future to relieve the stiffness. Our goal is to make sure that he becomes part of the society. We must make sure that he is able to communicate, take care of himself and go from one place to another. Even if his walking may be impaired he can still have a full life.

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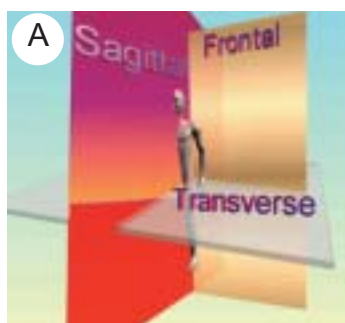
Three planes of human motion:
Sagittal: flexion / extension
Frontal: abduction / adduction
Transverse: rotation

Double support

Right foot stance
Left foot swing

Double support

Left foot stance
Right foot swing

C	Phases of gait
Stance phase	60%, foot on the ground
Swing phase	40%, foot in the air
Period of double support	11%, both feet are on the ground
Period of single support	80%, one foot is on the ground
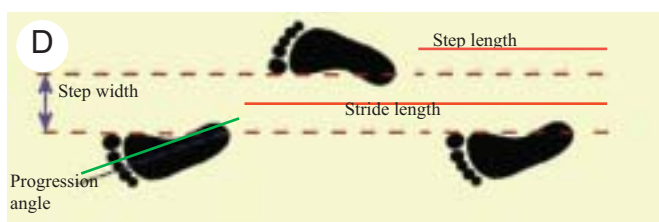
Step length: distance from the point of contact of one foot to the point of contact of the other foot.
Stride length: distance from the initial contact of one foot to the initial contact of the same foot.
Cadence: number of steps taken per minute.
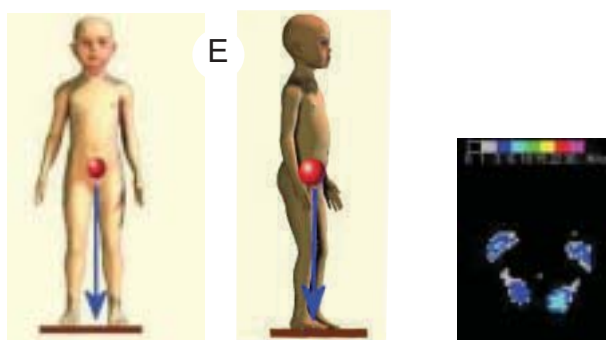
Center of mass and center of pressure
The center of mass (COM) is in front of the second sacral vertebra in a standing adult. A vector perpendicular to the ground drawn from this center of mass represents the force exerted by the body on the ground. This vector must coincide with the center of pressure (COP) of the body. The COP is a point on the ground between the feet. When the COM is over the COP, the person is balanced. When the COM is outside the COP, the person loses balance.

The body weight exerts a force to the ground. The ground responds by a force of equal magnitude and in opposite direction to the body. This ground reaction force creates rotatory forces about the joints called external moments. Muscles contract and produce internal moments to keep the body stable.

Gait
Some children with CP cannot walk. Others have walking difficulty. This is generally the basic reason for seeking medical advice in CP and probably one of the most difficult to affect. To understand the gait pathology associated with CP, first understand normal gait. Walking is one of the most important functions of the human musculoskeletal system. Efficient walking requires complete coordination of the brain, spinal cord, peripheral nerves, muscles, bones and joints.

Normal gait
Walking consists of a series of movements that are repeated in a cyclical pattern. These movements are analyzed in three planes [A]. The gait cycle is defined as the period of time from the moment when one foot strikes the ground to the moment when the same foot strikes the ground again [B]. The gait cycle is divided into the stance and swing phases. The stance phase consists of 60% of the total cycle and represents the time period when one foot is in contact with the ground. The swing phase consists of 40% of the cycle and represents the period when the foot is not touching the ground [C]. In the gait cycle, an 11% period occurs during which both feet are in contact with the ground. This is the double support phase. Every person has a comfortable walking speed according to age and gender. In a healthy adult male this is approximately 80 meters per minute [D]. Normal walking requires balance, propulsion, shock absorption and energy consumption. The person must maintain balance, propel the body forward, absorb the shock created by the body weight and spend as little energy as possible.

Balance
Balance depends on the relationship between the center of mass and the center of gravity [E]. The person has balance in stance, loses it during swing and recovers it during double support. Ligaments provide static stability and muscles provide dynamic stability. Inability to maintain upright balance is an important cause of walking difficulty. During walking a person must be able to shift and support the body weight over the extremity in stance, preserving lateral balance. Children with CP lack this ability and cannot shift their weight over the leg on the ground during stance. They have a tendency to fall toward the swinging limb. This deficiency in maintaining lateral balance is the reason many diplegics and some hemiplegics walk with a sideways lurch. Weak abductors cause a sideways lurch too. Provide the patient with crutches, canes and walkers to help maintain balance.

Inability to maintain balance also creates a tendency to walk very fast, as if running. This phenomenon resembles an inexperienced rider on a bike. Inexperienced riders go fast to maintain their balance whereas experts can ride slowly without falling.
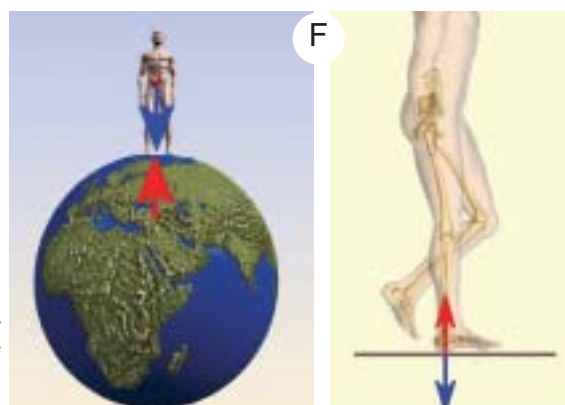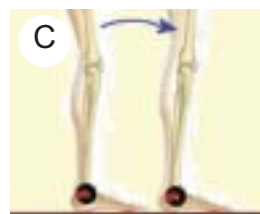
A walking person moves his body forward over the stance leg by a series of movements called rockers.

Heel (first) rocker: The period from the moment the heel strikes the ground to the moment the forefoot touches the ground. The pivot of motion is the heel.

Ankle (second) rocker: The period from the moment the forefoot touches the ground to midstance. The pivot of motion is the ankle joint.
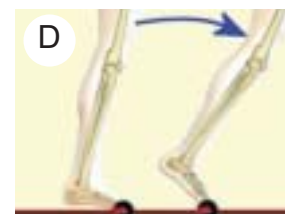
Forefoot (third) rocker: begins with heel rise and finishes with toe off. The pivot of motion is the metatarsophalangeal joint.

Propulsion

The body moves forward during swing and stance phases. Hip and knee flexion are necessary to clear the swinging leg off the ground. The knee must extend at the end of swing for an adequate step length. The body moves forward also over the stance leg by a series of movements called rockers [A-D]. Forward progression is disturbed in CP. The muscles cannot produce the necessary force for moving the body forward. The swinging leg cannot clear the ground because of inadequate hip and knee flexion. Step length is short because of limited knee extension. The body weight cannot move over the stance leg because of muscle weakness and contractures that disturb the rocker mechanism.

Muscle function during gait

The body mass exerts a force to the ground and the ground responds with an equal force in the opposite direction to the body; this is termed the ground reaction force. The body responds by muscle contraction to sustain balance and stability in the joints [F on opposite page].

Tibialis anterior [E] is active in the first rocker of gait cycle. It allows smooth ankle plantar flexion as the foot comes in contact with the ground. It provides mediolateral stability and foot clearance by active dorsiflexion of the ankle during the swing phase. Weakness contributes to foot drag during swing and to instability during stance.

Quadriceps [F] contracts from initial contact through midstance to allow 15º of knee flexion and contribute to forward progression of body. It contracts at the end of stance to counteract the external flexor moment that the ground reaction force produces at the knee. This is a brief contraction to prevent the swinging knee from flexing too far. Weakness of the quadriceps muscle causes the knee to flex too much during stance, leading to crouch. Spasticity causes inability to flex the knee during swing leading to stiff knee gait.

Hamstrings [G] contract at initial contact to keep the hip and knee stable and at the end of swing to prevent the tibia from going too far into extension. Spasticity causes crouch.

Gastrocnemius-soleus [H] are active during the middle and end of stance, limiting passive ankle dorsiflexion and providing push off. Their weakness causes crouch and spasticity causes equinus.

Shock absorption

Approximately 60% of the body weight is transferred to the extremity in stance in 0.02 seconds during heel strike. The effects of this shock are reduced by muscle action at the ankle, knee and the hip. Ankle dorsiflexors limit ankle plantar flexion and allow a smooth contact with the ground. The quadriceps limits knee flexion and the hip abductors prevent excessive pelvic drop.

Energy consumption

The excursion of the body center of mass [I,K] determines the energy cost of walking. Energy cost is high in patients with CP because of the increased excursion of the body center of mass.
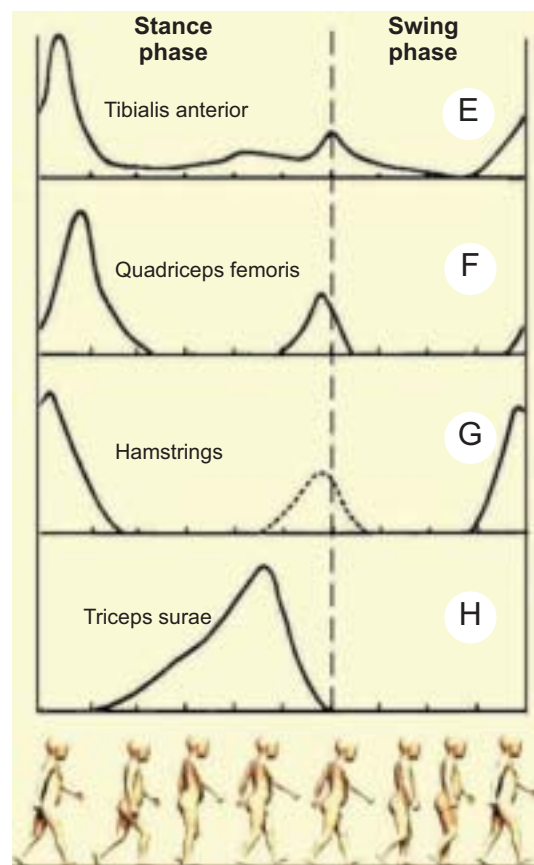
Timing of muscle action during gait
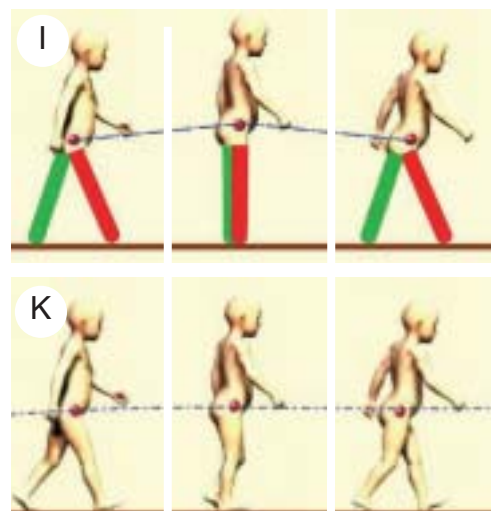
The excursion of the body center of mass is a sinusoidal curve of little amplitude. When the lower extremity joints do not move normally, the excursion increases.
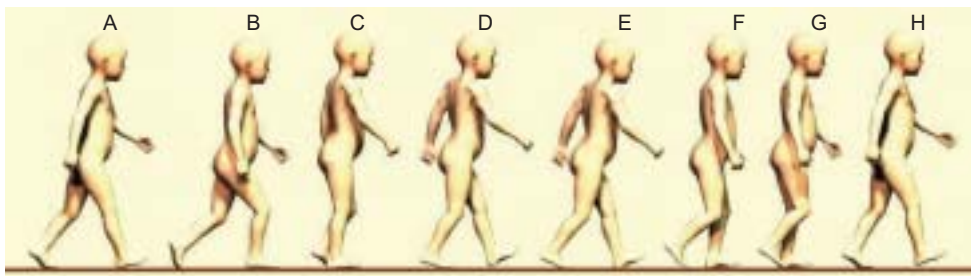
Gait cycle			
Stance phase		*Swing phase*	
A	Initial contact	F	Initial swing
B	Loading response	G	Midswing
C	Midstance	H	Terminal swing
D	Terminal stance		
E	Preswing		

Phases of Gait

The gait cycle is divided into the stance and swing phases. Common problems in stance are positioning the foot during initial contact, too much knee, hip and ankle flexion or knee hyperextension in midstance and poor push-off. Problems in swing include insufficient foot clearance and limb advancement. Loss of stability in stance and inadequate progression in swing occur.


Initial contact
Initial contact begins when the foot strikes the ground. The hip is in 30º flexion, the knee in full extension, the ankle is in neutral and the foot is in supination. At the point of heel strike, the ground reaction force is in front of the hip and the knee. This creates a flexion moment at the hip and an extension moment at the knee. Hip extensors and knee flexors contract to overcome this moment. Tibialis anterior contracts to keep the ankle in dorsiflexion.

Loading response
The person puts his weight on the extremity. The hip begins to extend, the knee is in 20º and the ankle is in 10º plantar flexion. Ground reaction force creates flexion at the hip and knee, plantar flexion at the ankle. Hip and knee extensors and ankle dorsiflexors contract to counteract this force.
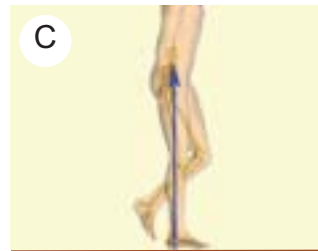

Midstance
Only one foot is in contact with the ground. The hip and knee are in extension, the ankle is in dorsiflexion. Ground reaction force is behind the knee and in front of the ankle, causing flexion at the knee and dorsiflexion at the ankle. Hip abductors, quadriceps and ankle plantar flexors contract.


Terminal stance
The heel begins to lift off the ground. The hip is in 10º extension, the knee starts to flex, the ankle is in plantar flexion to clear the leg off the ground. Ground reaction force is behind the hip but in front of the knee and ankle. Iliopsoas and triceps surae are active.
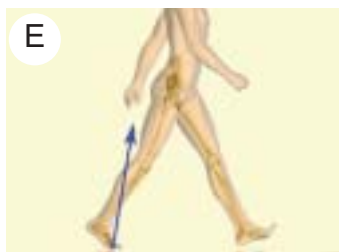
Preswing
The toes lift off the ground. Hip extension, knee flexion and ankle dorsiflexion increase. Ground reaction force is behind the knee producing a flexion moment. Iliopsoas, rectus femoris, triceps surae are active. Rectus femoris contracts to overcome knee flexion.
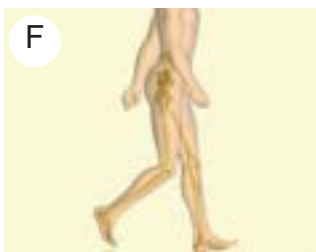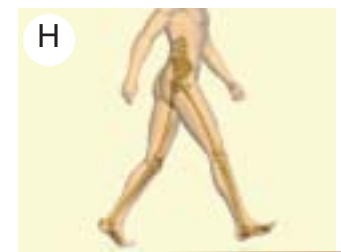

Initial swing
There is flexion at the hip and knee joints and dorsiflexion at the ankle. Hip flexors and ankle dorsiflexors are active, knee flexion is passive.

Midswing
The swing leg passes in front of the stance leg. Flexion of the lower extremity increases to a maximum. Hip and knee flexion is passive due to inertia while ankle dorsiflexors are still active.

Terminal swing
The swing leg prepares for landing. The hip is in flexion, the knee in extension and the ankle is in neutral position. Hamstring muscles contract to limit hip flexion and knee extension. Ankle dorsiflexors contract to keep the ankle at neutral.

Clinical examination of gait

Ambulatory children with CP have various types of pathological gait. Efficient intervention depends on proper evaluation [A] Observation [B] and video recordings [C] are sufficient to understand the abnormality in many cases.

Watch the video in slow motion for a better understanding. Videos are useful to demonstrate the child's progress to the parents. Computerized gait analysis is necessary in the few cases with more complex gait patterns.

Gait analysis

Computerized gait analysis is an objective, standardized, reproducible and quantifiable method to evaluate gait pathology. Computerized gait analysis consists of 5 components [D - I]. Gait analysis helps to decide on the type of therapeutic intervention and to asses the effects of the intervention. It has a role in research, education and therapeutic decision making. Computerized gait analysis has advantages and disadvantages [J].

Gait analysis is useful as a research and education tool. It is an additional aid in decision making for treatment. It requires expensive high technology equipment and educated staff. It shows how the child walks graphically but does not tell how functional the gait pattern is unless it measures the amount of energy consumed during walking. It adds little to the clinical examination and remains more a research tool than part of a routine clinical examination in most countries.

Examination of gait	A
Observation	
Video recording	
Computerized gait analysis	

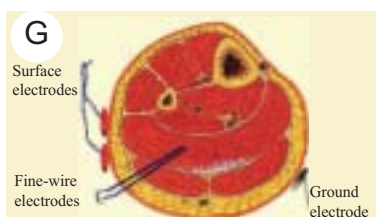| Observation	B
1. The child walks a distance of 10 meters.	
2. Stand at a distance of 3 m., watch the child walk toward you.	
3. Stand at a distance of 3 m., watch from the side.	
4. Look at each joint separately in the order of: L hip, R hip, L knee, R knee, L ankle, R ankle.	
5. Watch balance as the child turns.	
6. Record step length, stride width and any deformities.	
7. Record the gait on video.	
8. Do not overtire the child.	

Kinematic data are obtained from markers placed on the body. As light reflects from the marker, the camera captures it and feeds the information to the computer. Special software processes the data into graphics.
Photos E,I: Courtesy of MotionAnalysis Co.

Forces acting upon the joints are estimated through a complex mathematical equation by first capturing the ground reaction force using force plates. The three dimensions of the ground reaction force vector can be measured separately.

| Components of computerized gait analysis	D
Kinematics: Recording joint motion with markers and cameras	
Dynamic electromyography: Electrophysiological monitoring of muscle activity using fine wire electrodes placed into the key muscles	
Kinetics: Assessment of force vectors using force plates	
Energetics: Evaluation of O_2 consumption and energy cost of walking using gas analysis systems	
Dynamic pedobarography: Pressure changes of the sole of the feet	

Computerized gait analysis		J
Advantages	*Disadvantages*	
Provides quantifiable data	Data interpretation necessary	
Shows moments & powers across the joints	Different laboratories produce different results for the same patient.	
Shows muscle activity during gait	Expensive to start and maintain	
	Difficult in small children	
	Kinetic data not possible below age 4	

Needle electrodes can be placed into deep muscles and skin electrodes are used for superficial muscles in dynamic EMG.
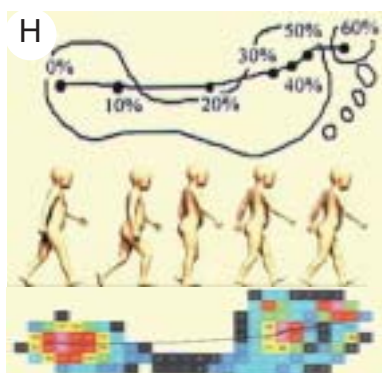
Pedobarograph measures the pressure changes of very small sections of the sole of the foot. It gives a pressure distribution map of the weight bearing foot.

These measures are then fed into a computer system and processed. Analysis of this data by physicians experienced in the field of gait analysis will result in a clearer definition of problems during gait.

Characteristics of gait in children		A
Parameter	*Characteristic*	*Normalizes at age*
Step length	Short	15
Step width	Increased	4
Cadence	Increased	15
Speed	Slow	15
Stance	Longer	4
Muscle activity	Increased	4
Heel strike	None	2-3
Knee flexion	Minimal in stance	2-3
Legs	External rotation during swing	2-3
Arm swing	Absent	4

Jump gait typical for the young diplegic child.

Crouch gait occurs in the growing diplegic child. It is characterized by increased knee flexion and ankle dorsiflexion during stance. Isolated gastrocnemius lengthening or overlengthening weakens push-off and causes crouch. Severe hamstring weakness also causes crouch.

Stiff knee gait may accompany crouch. In this case, the quadriceps and the hamstring muscles are spastic. Stiff knee gait is easily recognized by shoewear due to drag in swing.

Scissoring or crossing over is caused by medial hamstring and adductor muscle spasticity in the young child. Increased femoral anteversion contributes to the problem in the older.

Maturation of walking in children

A child's gait is different from an adult's until adolescence. The toddler walks with wide, short steps. The foot strikes the ground with the whole sole. Stance phase knee flexion is minimal. The legs are in external rotation throughout the swing phase. Reciprocal arm swing is absent. Stance phase is longer in young children compared with swing phase. There is increased muscle activity.

The gait pattern matures as the child grows older [A]. Heel strike begins at approximately 3 years of age. Stance phase knee flexion and external rotation values approach normal limits. Step width narrows and reciprocal arm movements begin at approximately 4 years of age. Cadence, step length and speed reach adult values at approximately age 15 years.

Types of gait in diplegic and ambulatory total body involved children

Stability in stance, progression and foot clearance in swing are necessary for efficient walking. Stability is disturbed in CP because of impaired balance, increased muscle tone leading to contractures and muscle weakness. The common problems in stance are equinovarus, jump knee, crouch knee and internal rotation of the legs. Progression of the body is disturbed because of weakness and contractures as well. The common problems of swing are shortened step length and impaired foot clearance such as that which occurs in stiff knee gait.

The child's walking pattern changes with age. Diplegic children begin standing with the hips, knees and ankles extended and the legs crossed. Later, hip and knee flexion and ankle plantar flexion occur. Crouch occurs as the child grows older. Walking patterns are established at approximately 5 to 7 years of age.

In the sagittal plane, look for three types of pathologically abnormal gait: The jump, the crouch and the stiff knee gait.

Jump gait

The child walks with hips in flexion, knees in flexion and ankles in plantar flexion as if getting ready to jump [B]. This is typical for diplegic and ambulatory total body involved children when they begin to walk. The reason is spasticity of hip and knee flexors and ankle plantar flexors.

Crouch gait

Increased knee flexion and ankle hyperdorsiflexion occur during stance phase [C]. They occur in older children and after isolated triceps lengthenings that have been performed without addressing the spastic hamstrings. Hip flexors and hamstrings are tight, and quadriceps and triceps are weak.

Stiff knee gait

Decreased knee flexion occurs during swing phase [D]. The rectus femoris muscle is spastic and does not allow the knee to flex in initial and midswing phases. Limitation of knee flexion causes difficulty in foot clearance and stair climbing.

These sagittal plane gait patterns coexist with frontal and transverse plane pathologies. Look for scissoring and trunk lurching in the frontal plane.

In the frontal and transverse planes look for scissoring gait and trunk lurching.

Scissoring gait and internal hip rotation

Scissoring gait is defined as crossing over of the legs during gait [E]. The cause is hip adductor and medial hamstring spasticity combined with excessive femoral anteversion.

Trunk lurching

Trunk lurching is an increase in the side-to-side movement of the trunk during walking [A]. It is caused by deficiency of balance. It may become worse after surgery and during periods of rapid growth.

Traps to avoid: Apparent equinus

The cause of toe walking may not be gastrocnemius spasticity, but rather insufficient knee extension in certain children. When the patient is unable to extend the knee because of hamstring spasticity or knee flexion contracture, he or she seems to walk on tiptoe which can be mistaken for pes equinus.

Types of gait in hemiplegic children

Hemiplegic gait is subdivided into four types. With type 1, no active dorsiflexion of the ankle is present, and the foot in equinus. With type 2, a functioning tibialis anterior is present, and the foot is still in equinus because of the spasticity in gastrocnemius. With type 1, even if the gastrocnemius muscle is lengthened, the patient still needs a brace to keep the foot in neutral; however with type 2, lengthening of the gastrocnemius results in a more functional gait because the patient is able to dorsiflex the ankle. The differentiation between the two types of gait can be made using dynamic electromyography, which shows the activity in the tibialis anterior. With type 3, abnormal hamstring or rectus femoris activity is present, causing genu recurvatum or stiff knee, in addition to the problems observed with types 1 and 2. With type 4, in addition to the abnormal knee muscle activity, increased hip flexor and adductor spasticity or contracture are present.

Transverse plane deformities such as tibial torsion and femoral anteversion also might be present [C].

In spite of all technological advances in computerized gait analysis, certain gait abnormalities in CP continue to present difficulties for the clinician. The hints presented in the table help make better decisions for treatment [D].

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The only remedy for trunk lurch is using a mobility device such as a walker or canes. Strengthening the hip abductors may also be helpful.

Distinguish apparent equinus from true equinus. Some children appear to walk in equinus but their ankle is actually in neutral or even dorsiflexed. Hamstring spasticity causes dynamic knee flexion deformity and the child walks as if he has equinus.
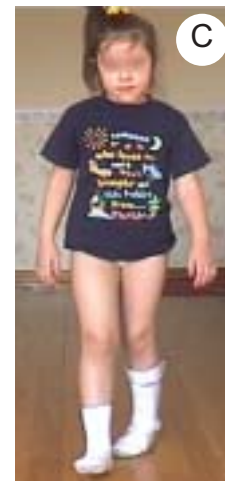
Femoral anteversion leads to intoeing and causes equinus. This type of deformity can also occur in hemiplegic children.

Hints on how to analyse gait [D]
Be familiar with normal child gait, watch children walk
Watch the child many times in different conditions
Record walking with a video camera
Ask the parents for photos and videos recorded at home and outside
Interpret gait data cautiously
Test balance and stability
Test in real life situations (at school, on the street)
Test speed