Personal Practice

Cerebral Palsy

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Cerebral palsy (CP) is a common neurodevelopmental disorder of childhood with a prevalence of 1.5-2.5 per 1000 live births(l). The exact incidence and prevalence figures from our country are not available. Problems related to early diagnosis as well as proper management of these children are often because of a lack of understanding not only by the parents but also by medical personnel. As a result the child with CP is frequently subjected to neglect and a poor quality of life. The present communication attempts to provide an overview of the condition with emphasis on early diagnosis and principles of management.

What is Cerebral Palsy?

Cerebral palsy needs to be considered as a symptom complex rather than a specific disease(l). A recent consensus definition has described CP as "a group of nonprogressive but often changing motor impairment syndromes secondary to lesions or anomalies of brain arising in early stages of its development" (2). It is a static encephalopathy and excludes all progressive neurological disorders. Since motor dysfunction evolves over time as the child grows, it may give an erroneous impression of the disorder being progressive. Defining CP in terms of motor deficits should not undermine the fact that other neurological deficits are frequently associated and may at times be more disabling than the motor deficit itself.

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What are the Etiological Considerations in CP?

A large number of epidemiological risk factors for CP have been identified *(Table* I) and the relative role of prenatal, perinatal and genetic factors has been debated. Adverse intrauterine factors like developmental malformations of brain, neuronal migration disorders, intrauterine infections, *etc.* account for some cases(3). Significant perinatal damage has been reported in 8-10% of cases(4,5). An increased incidence of noncerebral congenital malformations was found in children with CP in the National Collaborative Perinatal Project (NCCP)(4). Genetic factors contribute in about 2% cases of CP(6).

What is the Role of Birth Asphyxia?

The causal role of birth asphyxia in CP has been questioned. It has been suggested that asphyxia may be a consequence rather than cause of the processes that lead to CP(1). In many cohort studies, markers of asphyxia have not correlated with increased numbers of children with CP. However, in the NCPP project 20 minute Apgar of 3 or less was associated with 250 fold increased risk of cerebral palsy(7). Case control studies, in which children with CP have been compared with controls for markers of birth asphyxia have yielded

TABLE I–*Factors Identified in Epidemiologic Studies as Associated with Cerebral Palsy*

Before pregnancy

- History of prior fetal loss
- Unusually short (<3 months) or long (>3yr) interval since previous pregnancy
- Family history of CP

During pregnancy

- Low socio-economic status
- Congenital malformations-cerebral/extracerebral
- Fetal growth retardation, small head circumference
- Twin gestation
- Abnormal fetal presentation

During perinatal period

- Chorionitis
- Premature separation of placenta
- Hypoxic ischemic encephalopathy

an approximate figure of 10% for an asphyxial cause for CP(8,9)

What is the Risk of CP in Premature Babies?

An increased prevalence of CP has been associated with decreasing birth weight or gestational age(10,11), but this association is not absolute The increasing proportion of premature children within the total CP population in epidemiological studies(10,11) is largely because of increasing number of preterm survivors The underlying pathological lesions in these babies are penventncular leukomalacia (PVL) and penventricular hemorrhagic venous rnfarcts (PHVI)(12) Ultrasonographic detection of hypoechoic penventricular areas has been found to be a strong predictor of later development of motor dysfunction in these preterm babies(l)

In majority of the cases of CP the cause is unknown(13) Some important causes of acquired CP include CNS infections, namely menrngoencephalitis, cerebrovascular accident and head trauma(14)

What are the Types of Cerebral Palsy?

CP can be subdivided into several types based on predominant motor pattern(15,16) Classifications based on physiological characteristics (qualitative) and extent of involvement or topography of motor deficits (quantitative) is given in *Table II* These classifications are of limited benefit from the perspective of early diagnosis because these are based on established rather than evolving syndromes(15)

Spastic CP

It is the most common form and accounts for 70-75% of cases It is characterized by upper motor neuron signs, namely, clasp knife hypertonia, exaggerated deep tendon reflexes and extensor plantar responses(3,13,17)

Spastic *Quadnplegm*

These patients are the most catastrophically disabled All four limbs are affected with upper limbs being equally or more affected than lower limbs Vast majority of

TABLE II *Classification of Cerebral Palsy*

Physiological	Topographic
• Spastic	Tetraplegia/ Quadriplegia
• Dyskinetic	Diplegic
Choreoathetoid	Triplegia*
Dystonic	Hemiplegia
• Ataxic	Monoplegia*
• Hypotonic	
• Mixed	

* Very rare and may represent evolving forms

the patients have severe mental handicap, psuedobulbar palsies, microcephaly, growth failure, visual and hearing deficits and often epilepsy. Motor findings include hypertonicity leading to arching of the back and scissoring of legs (either spontaneous or when vertically suspended). Hip subluxation or dislocation may occur because of severe spasticity. Walking is markedly delayed and often the child has toe walking because of tendo-achilles tightening. Arms are internally rotated, elbows extended or lightly flexed and hands fisted. Later flexion contractures develop at ankles, knees and elbows(3,15,17,18).

Spastic Diplegia

In this form of CP, lower limbs are involved substantially more than upper limbs Intellectual involvement is minimal. Growth of lower limbs may suffer whereas upper torso grows normally. It is characteristically seen in preterm babies with penventricular leukomalacia (PVL) (3,17,18).

Spastic Hemiplegia

It refers to involvement of one side of the body. The arm is usually more severely affected than the leg, except m the preterms with penventricular hemorrhagic infarction where the leg may be more affected than the arm Cerebral injury in the region of the middle cerebral artery is the commonest pathology. For reasons not clear, right sided involvement is more frequent than left Since normal development is cephalocaudal, abnormal signs are noticed first in arms than in legs Milder cases are often missed by the parents and even by doctors(3). The following clinical signs help in arriving at early diagnosis *(i)* Poverty of movements and fisting of hand on affected side, (11). Defmite hand preference in children less than 12 months of age, and *(in)* Sittmg and crawling are not much delayed, walking is delayed generally by 2-3 months When child is supine, the affected lower limb may be externally rotated

In severe established cases, the arm is held adducted, flexed and internally rotated at the shoulder, with the elbow flexed, forearm pronated, wrist flexed and thumb adducted The leg is held adducted, semiflexed at knee and plantar flexed at the ankle In longstanding cases asymmetries of limb growth may occur

Dyskmetic CP

Both the dystonic and choreoathetoid forms are included in this. It is caused by damage to basal ganglia and other extrapyramidal structures, often because of kernicterus and perinatal hypoxic brain damage(19). With early and aggressive treatment of neonatal hyperbihrubinemia, kernicterus has been virtually eradicated in the West(15) However, it continues to be an important cause in our country(3). Clinical features are characterized by severe motor disability, with preservation of neonatal reflex patterns. Asymmetric tonic neck response (ATNR) is prominent and postural reflexes appear late. Infants are usually hypotonic with marked head lag, drooling of saliva and feeding difficulties. Athetosis manifests generally after one year of age and tends to coincide with hypermyelinahon of the basal ganglia, a phenomenon called status marmoratus. Reaching for objects leads to flaying of fingers. Overflow movements and facial grimacing are prominent *(Fig 1)*. These are exaggerated with intention, emotion and holding a posture Standing and walking are delayed. Although intelligence is often preserved, presence of severe physical and communicative disabilities give a mistaken diagnosis of mental retardation(3,17)

Ataxic CP

This occurs due to predominant

ig. 1. Child with athetoid CP showing facial grimacing, dystonic posturing of left arm and flaying of fingers.

invovlement of cerebellum. These infants are hypotonic and inactive. Walking is delayed. The gait is ataxic, wide-based and accompanied by exaggerated balancing movement of arms. Cerebeller signs are present, nystagmus is, however, rare(3).

Ataxia may occur in the pure form or may co-exist with spasticity (ataxic diplegia). Ataxic CP needs to be differentiated from heredo-degenerative ataxias(15).

Hypotonic CP

This is extremely rare. In many cases, it may in fact represent an evolving form of dyskinetic or spastic CP. Other causes of hypotonia should be excluded(3).

What are the Associated Problems and their Significance?

Several non-motor disabilities often accompany CP(1,3,17), their frequency varying with the type of CP, being maximum in spastic quadriplegia. Most of these are potentially treatable, namely, seizures, hearing or visual problems, and need to be given due emphasis in the overall management of CP. Some of these problems especially mental retardation, uncontrolled seizures can interfere with rehabilitation activities. *Table III* summarizes these associated disabilities.

Apart from these, orthopedic problems like scoliosis, dislocation of hips and medical problems like neurogenic bladder, feeding difficulties, urinary tract infection and constipation are also seen especially in severe cases and spastic quadriplegia(15,17).

What are the Clinicopathological Correlates of CP?

The subtypes of CP are correlated with distinctive etiologies and pathological lesions *(Table IV).*

How is Diagnosis of CP Made?

There is no gold standard test for diagnosis of CP. The diagnosis of CP is essentially clinical and involves a detailed prenatal, natal and postnatal history and a careful physical and neurodevelopmental examination(3,13,15,17). In severe and long-standing cases the diagnosis of CP is not difficult. Early diagnosis during evolving stages and diagnosis of mild cases calls upon the clinical acumen and experience of the doctor. This is generally required in two settings *(i)* during follow up of 'at risk' neonates, and *(ii)* when infants are

TABLE III *Problems Associated with CP*

	Problem	Frequency (%)	Remarks
1	Mental retardation	50-75	Most often in spastic quadriplegia and ataxic CP
2	Seizures	25-33	Most often in spastic hemiplegia and spastic quadriplegia, least in dyskinetic CP
3	Hearing and speech problems	15-20	Most common in dyskinetic CP and spastic quadriplegia
4	Ocular	50-70	Refractory errors, strabismus
5	Behaviour problems	30 50	Stubbornness, aggressiveness, lack of attention and hyperkinetic behaviour

TABLE IV *Some Clinico pathological Correlates in CP*

CP subtype	Pathology	Underlying etiology
Spastic diplegia	• Periventricular leukomalacia • Periventricular hemorrhagic venous infarction	• Prematurity
Spastic quadriplegia	• Multicystic encephalopathy with cortical atrophy • Selective neuronal necrosis • Parasaggital cerebral injury	• Perinatal/intrauterine hypoxic ischemic events
	• Cerebral malformation(s)	• Genetic
Spastic hemiplegia	• Cerebral injury MCA territory (infarction, necrosis)	• Prenatal events like hypoperfusion, hemorrhage
	• Malformations of cerebrum	• Genetic
Dyskinetic	Basal ganglion • Stauts marmoratus • Bilirubin deposition	• Perinatal asphyxia • Neonatal hyperbilirubinemia (kernicterus)
Ataxic, hypotonic	• Cerebellar lesions • Enlarged ventricles	• Prenatal (genetic)

brought with subtle complaints of delay in acquisition of early developmental milestones

A number of neurologic evaluation protocols and developmental assessment tools have been developed but their ability to predict outcome is somewhat variable Techniques for the neurodevelopmental examination of the newborn, older infant and young child have been described in detail(20,21) Comprehensive neurodevelopmental assessment is time consuming and

may not be possible in a busy neonatal follow up clinic. Infant motor screen is a screening tool used to identify high risk preterm infants in need of a detailed neuromotor assessment(22). We have found it to be highly specific and sensitive for early diagnosis of CP in both preterm and term babies(23).

Certain observations and signs that we have found useful for prediction and early identification of CP are listed in *Table V* (3,17). It may, however, be emphasized that (i) infants particularly preterm babies may show some transient neuromotor abnormalities; it is, therefore, important to do repeated assessments before making a final diagnosis of CP; *(n)* Developmental screening tests should not be used as definitive diagnostic tools. Abnormalities detected on these tests warrant comprehensive neurodevelopmental assessment; *(in)* The nonprogressive nature of the disorder must be ascertained by periodic examinations. Slowly progressive neurodegene-rative disorders can often be misdiagnosed as CP; *(iv)* The dyskinetic, hypotonic and ataxic forms of CP are particularly liable to cause

TABLE V–*Signs Useful in Early Diagnosis of CP*

1. *Warning signs*
- Lack of alertness
- Decreased spontaneous motility
- Stereotyped abnormal movements
- Constant fisting after 2 months of age (*Fig. 2*)
- Poor quality of sleep

2. *Abnormal signs*
- Reduced head circumference or fall in its growth
- Delayed social smile
- Excessive extensor tone, dystonia (*Fig. 3*)
- Primitive reflexes persisting beyond 6 months
- Persistent asymmetric tonic neck response (ATNR) (*Fig 4*)
- Delayed appearance of postural reflexes and developmental milestones
- Persistent asymmetry in posture, movement and reflexes

3. *Associated signs*
- Oculovisual problems: roving eyes, no visual following, persistent squint
- Lack of auditory response

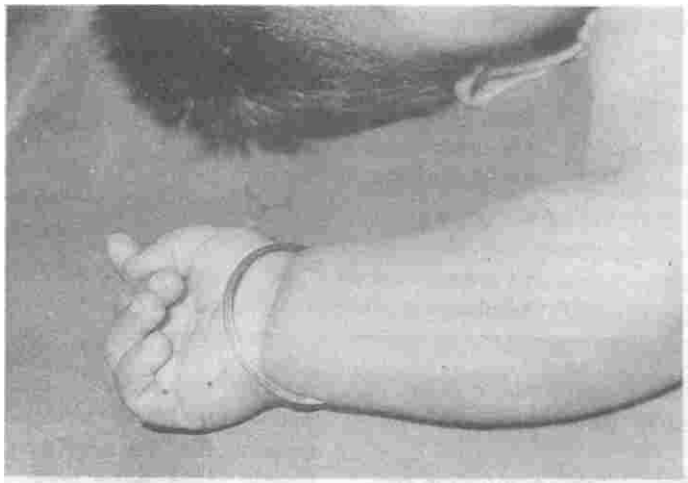

Fig. 2. A one-year-old child with CP showing constant fisting and cortical thumb.

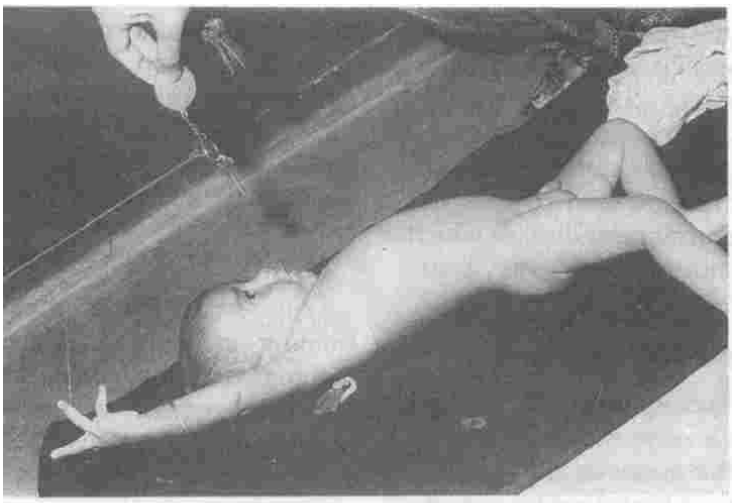

Fig. 3. Excessive extensor tone and dystonia in a 10 month old baby.

Fig. 4. Persistent asymmetric tonic neck reflex in a 1½ year old child.

confusion. Neuromuscular, spinal disorders, and other causes need to be excluded; *(v)* The clinical pattern of CP evolves over a period of time. A hypotonic child may have spasticity or dystonia on follow-up. One should not feel compelled to give a diagnostic label at the first examination and should not hesitate to change it if so needed on subsequent assessment; and *(vi)* Testing of intelligence using conventional tests is often erroneus in children with CP, because of associated motor problems and visual, hearing and speech deficits. IQ estimates are generally lower, hence mental retardation should not be diagnosed based on these(3,17).

What is the **Role of Investigations?**

In infants who are in 'high risk' group and are being followed up closely, investigations would be determined by the events in the prenatal and perinatal period and with a clinical correlation, no further tests at all might be required. In preterm infants ultrasound is useful for picking up PVL and PVHI. The extent of these lesions

correlates with the prognosis. In other circumstances especially when the etiological factors are not clear, neuro-imaging or other genetic or metabolic tests may be required to identify the underlying etiopathological mechanisms and to rule out any progressive neurological disease which might at the onset appear as static encephalopathy(15,17).

General Principles of Management

Appropriate management of a child with CP requires a comprehensive assessment to identify the motor as well as associated deficits This is best done by a mulh-discrplinary team comprising of a neurodevelopmental pediatrician as a team leader, physiotherapist, occupational therapist, clinical psychologist, ENT specialist, ophthalmologist, orthopedic surgeon, teacher and social worker, preferably under one roof The main aim is to assess the functional capacity of the child in various domains and to plan an intervention programme to maximize it. Assessment of the functioning of the family is essential to ensure their involvement in the management plan(3,15,17). The main goals of intervention are (i) to improve function and encourage independence and *(ii)* prevent secondary problems The various aspects of therapy include:

Physiotherapy (PT)

It aims at promoting normal movement patterns and inhibiting the abnormal ones in order to maximize functional motor independence. When started early it helps in preventing contractures and deformities3,15,17). Several techniques have been used, but there is no evidence to support the superiority of one over the other.

Neurodevelopmental therapy using Bobath technique(24) is most popular This essentially involves training of the child through normal sequence of motor development, abolition of primitive and abnormal reflexes, reinforcement of normal postural reflexes and facilitation of normal movements. An eclectic approach individualized to the child's needs is required(3).

Occupational Therapy (OT)

An occupational therapist provides training in daily activities like feeding, bathing, dressing, toilet training, *etc.* and the management of sensory perceptual problems. The activity is broken into its components and these are taught one by one in the required sequence(3,17).

Speech Therapy

If needed, it is provided by the speech therapist Prevention of drooling and development of oral motor function are best done by the co-ordinated efforts of speech and occupational therapists(17).

We emphasize a home based management programme in which parents are taught the correct method of handling and lifting the child and carrying out specific exercises. Multisensory stimulation is provided through peg board, blocks, toys, *etc* to enhance co-ordination and sensory-perceptual integration. It is emphasized that these techniques be incorporated into the pattern of daily life(3).

Adaptive and Mobility Devices

These include side layers, prone boards, standing frames, walkers, tricycles and wheel chairs are often needed to promote skeletal alignment, compensate for abnormal postures or to prepare the child for independent mobility(17).

Simple low cost aids can be made indigenously according to the child's needs Some of these ideas have been well illustrated(25).

Splints, Casts and Calipers

These may be required at times to maintain normal postures and prevent deformity. Specially designed shoes, ankle foot orthoses and calipers may be required to provide stability to the joints in a child who is learning to stand and walk(3,17). The use of dynamic lycra splints that provide flexible support is being evaluated particularly for children with dyskinetic CP(26).

Neurosurgery

Selective dorsal rhizotomy is useful in some children with spasticity especially where lower limbs are predominantly involved with near normal upper limb function(27,28). In this procedure, afferent dorsal rootlets from L_2 to S_2 levels are stimulated to identify abnormal electromyographic response and then selectively severed. However, it needs to be done by an experienced surgeon weighing the risks involved against the possible benefit (a functional goal should be predefined before resorting to surgery). Post-operative intensive physiotherapy is needed to maximize the benefit of surgery.

Continuous intrathecal administration of baclofen using an adjustable subcutaneous pump has been found effective in reducing spasticity in adults with moderate to severe spastic quadriparesis. Experience with children is however limited(29).

Management of Associated Problems

Various members of the team are involved in the management of associated problems. The clinical psychologist helps with management of behavior problems using behavior modification techniques, psychotherapy, family counselling, *etc.* The orthopedic surgeon helps with prevention and correction of musculoskeletal problems, *e.g.,* braces to prevent scoliosis, tendon release and lengthening, *etc*. The audiologist and ENT specialist takes care of hearing and speech problems, provision of hearing aids. The ophthalmologist provides remedies for oculovisual problems-corrective glasses, squint surgery, *etc.* (3,17,30).

High technology devices like electronic feeding devices, computerized speech systems and cochlear implants are available for children with CP in Western countries. In our country, very few can have access to such facilities(31).

Conductive Education

This is a system of management proposed by Andreas Peto Institute Budapest. In essence it involves the 'education' of fairly homogenous groups of motor disabled children by specially trained conductors. All the activities of the child are incorporated into the daily routine and the conductor guides the children through these, generally in a residential setting(32).

Role of Drugs

Drugs have a limited role in the overall management of children with CP(3,17,33).

Antispasticity Medication

These include baclofen, diazepam, tizanidine and dantrolene sodium. Baclofen is commonly used in a starting dose of 1.25-2.5 mg BD orally and increased gradually upto a maximum of 30-60 mg/day. It is not recommended for children with seizures as it may provoke them. Diazepam is another useful drug but excessive drowsiness that it may cause, limits its use.

Recently Botulinum-A toxin (BAT) has been found to be a useful antispasticity agent. Intramuscular injections of BAT into the muscles affected by spasticity produces relaxation of the involved muscles and improves the functional status of the involved

limbs The effect lasts upto 3-6 months(34).

Anhconvulsant Drugs

Their role in the control of seizures cannot be overemphasized Since seizures in children with CP are of various types, and frequently difficult to control, an appropriate anticonvulsant drug is selected and used in doses which will provide reasonable control of seizures

Other drugs include L-dopa and trihexyphemdyl for severe extrapyramidal symptoms, methylphemdate for hyperactivity and aggressive behavior, atropine or benztroprne for sialorrhea and antireflux drugs for gastroesophageal reflux used on an individual basis. There is no role for so-called brain tonics.

Parental Counselling

It is the most important aspect because without parental involvement any intervention programme is unlikely to be successful It is not a one time job but a continuous endeavour to ensure parental acceptance of the problem and their participation in prolonged treatment programmes Considerable understanding and insight are required for proper counselling(35). The basic essentials are (i) Explain the condition of the child in a simple and honest way, with emphasis on the positive aspects of the child, (ii) Explain the need for long term treatment, but set short term goals, *(iii)* Provide encouragement for the attainment of goals; and *(iv)* Help remove the feeling of guilt, and instill positive attitudes.

Education and Training

As the child grows older, the emphasis of rehabilitation must shift from management of motor problems to a child's functioning in school or training programmes These need to be individualized, according to the child's abilities, as determined by detailed assessment Guidance to the parents should be provided regarding the availability of appropriate schools and training institutions The ultimate goal is to help the child achieve his optimal developmental potential and to integrate him as a functional member in the society

Outcome

Majority of the patients with cerebral palsy live to adulthood. Life expectancy of severely affected individuals is significantly less than that of the general population(36). As mentioned earlier, prognosis is worst with spastic quadriplegia, *i.e.*, the more limbs involved, the worse the prognosis Prognosis is also modified by the presence of associated defects, socio-economic status of the family and availability of rehabilitative services(17).

In conclusion therefore, cerebral palsy represents a non-progressive central motor disorder with a wide range and severity of manifestations. With proper understanding and experience it is possible to make an early diagnosis and start early intervention. With a team approach and active parental involvement, a lot can be done for children with cerebral palsy.

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