Rehabilitation Management of Spastic Diplegic CP child with Ambulation Dysfunction.

M J BABAR S AHMED

Department of Physical Medicine and Rehabilitation, Children's Hospital & Institute of Child Health, Lahore.
Correspondence to: Dr Mumtaz J Babar

Rehabilitation management of spastic diplegic cerebral palsy child with ambulation dysfunction. To determine the role of physical therapy and corrective surgery in improving the functional status of walking in spastic diplegia. Outpatient physical medicine and rehabilitation clinic of the Children's Hospital, Ferozepure Road, Lahore. Fifty children with spastic diplegia presenting over a 9 months period. A prospective descriptive study of spastic diplegic children presenting with delay or difficulty in walking during a 10 months period (from June-2000 to March-2001). They were assessed and categorized from level (I to V) according to the Gross Motor Functional Classification Scale. The children under went a rehabilitation program comprising of physiotherapy and bracing alone or corrective surgery with pre and postoperative physiotherapy and bracing. The patients were regularly followed up on a monthly basis to assess the rehabilitation management and functional improvement in ambulation. Result showed that 36(72%) of the children improved I to II level higher than their presenting ambulatory status. Of these 2(4%) improved from level (V to III), 9(18%) from level (V to IV), 19(38%) from level (IV to III) and 6(12%) from level (III to II), 14(28%) of the total cases showed no improvement in their functional status. These finding suggest that the functional level of ambulation in spastic diplegic cerebral palsy children can be improved with proper rehabilitation management.

Key words: Spastic diplegia, rehabilitation management, ambulation dysfunction

Cerebral palsy is a disorder of movement and posture that results from a non progressive lesion or injury of the brain [1,2]. The definition includes a heterogeneous spectrum of clinical syndromes characterized by alteration in muscle tone, deep tendon reflexes, primitive reflexes, and postural reactions[3,4]. These neurologic abnormalities often produce characteristic abnormal patterns of movement that are recognized as the hall marks of cerebral palsy[5,6]. Although the essential diagnostic sign is a motor deficit causing ambulation delay or dysfunction and difficulty in self care activities, there may be other associated symptom complexes of cerebral dysfunction such as mental retardation, seizure disorder, oromotor dysfunction, visual, hearing and pulmonary problems.

Cerebral palsy is the leading cause of childhood disability[7,8]. The reported incidence is approximately 2-3/1000 live births[9,10]. Recent studies report an overall prevalence rate of 1.5-2.0 per 1000 live births[11]. Advances in medical care and technology in the west raised the hopes of reducing morbidity among neonatal survivals. Despite marked improvements in maternal and perinatal care in the advanced countries, the prevalence has remained relatively constant. This is partially explained by higher survival rates for more immature, smaller, and premature infants with medical complication[9,10,12]. Paneth and Kiely[13] concluded that the overall prevalence of cerebral palsy has not changed from 1950s and that school-age rate of 2 per 1000 live births is a reasonable estimate for industrial countries.

The origin of brain injury resulting in cerebral palsy may occur during the prenatal, perinatal or postnatal period. Overwhelming evidence suggests that in approximately 70 to 80%, cerebral palsy is prenatal in origin[11,13-18]. Prematurity remains the most common antecedent of cerebral palsy [2,10,19-21].

The modified neurologic classification system divides the cerebral palsy patients into the spastic (pyramidal), dyskinetic (extra pyramidal), and mixed cerebral palsy types. The spastic cerebral palsy disorders tend to be the most commonly occurring. This group accounts for approximately 75% of the children affected, where as the other 25% is divided into the dyskinetic and mixed types[22]. The spastic group can be further subdivided into the topographic distribution i.e., monoplegia, diplegia, triplegia, quadriplegia and hemiplegia[23]. In spastic diplegia, also known as Little's disease[24], history of prematurity is common (80% evolve into the spastic diplegic type[25]). A more complex multifactorial cause may be present in the term infant, although in 28% no identifiable risk factors are present[26]. There is history of intraventricular hemorrhages, particularly in the 28 to 32 weeks old infants. Magnetic resonance (MR) imaging may show periventricular leukomalacia or post hemorrhagic porencephaly[27,28]. The infants present with early hypotonia followed by spasticity[29]. The developmental delays are more pronounced in the gross motor sector particularly affecting ambulation. The children with spastic diplegia present with delay or difficulty in walking. They have increased lower extremity spasticity, often associated with contractures involving the hip adductors, knee flexors, ankle plantar flexors and invertors. The gait pattern is abnormal with scissoring, hip flexed and adducted, knees flexed with valgus and ankles in equinus.

The rehabilitation management of spastic diplegia requires proper musculoskeletal examination including a static evaluation on the examination table[30] and dynamic evaluation by performing a gait assessment[31]. Depending upon the individual case, physical therapy, bracing or corrective surgery may be indicated. The physical medicine and rehabilitation department of the Children's Hospital, Lahore receives a constant flow of cerebral palsy children with spastic diplegia who are referred for rehabilitation management. In order to assess the efficacy of our rehabilitation program for this group of children a prospective study was designed and carried out.

Material and methods

The study was conducted over a period of 9 months from June-2000 to March-2001. Fifty consecutive children with spastic diplegia presenting with delay or difficulty in walking were included in this study. They were assessed thoroughly and categorized according to a new functionally based system developed by Palisano and colleagues[32] at the Mc Master University. According to this Gross Motor Function Classification System, which is based on the premise of rating a child on functional skills, such as sitting and walking, the patients were classified into one of five groups.

Level – I Walks without restrictions: limitations in more advanced gross motor skills
Level – II Walks without assistive devices: limitation in walking outdoors and in the community.
Level – III Walks with assistive mobility devices: limitation in walking outdoor and in the community.
Level – IV Self mobility with limitations: children are transported or use power mobility outdoor and in the community.
Level – V Self mobility is severely limited even with the used of assistive technology.

After the initial assessment, the children who had potential to improve in functional ambulation, but were limited by increased muscle tone were prescribed systemic use of antispasticity medications such as Lioresal or Ternelin. These medications were used in small increasing doses during the first six to eight week period of intensive physical therapy, in order to get the desired range of motion at the affected joints. Other modalities included the use of ultrasound deep heat followed by gentle passive stretching of soft tissue contractures and use of static splints and braces. Electrical muscle stimulation was also utilized for re-education of weak muscle groups. Tilting and standing tables were used to assist in standing and to improve standing balance and tolerance. Progressive gait training began in parallel bars and was progressed to the use of assistive devices for independent walking.

The children in whom the desired potential was not achieved after the six to eight weeks period of intensive physical therapy as above, surgical intervention was advised. This included operative procedures e.g., adductor tenotomy, hamstring release and tendo-achilles lengthening. These operations were performed in the paediatric orthopediac department of the Children's Hospital. Postoperatively physical therapy and rehabilitation was continued at the physical medicine and rehabilitation department as needed. Patients were regularly followed up on a monthly basis to assess the functional improvement in ambulation. A proforma was used for each patient which was filled out by the principal investigator based on interview and clinical observations.

Results:

The results are presented in the following table:-Table II: Important factors in the rehabilitation of spastic diplegia.

Factors	n= 50
Age of presentation:	
• 0 – 2 years	02 (4%)
• 2 – 5 years	23 (46%)
• 5 – 10 years	20 (40%)
• 10 – 15 years	05 (10%)
Birth history:	
• Prematurity.	08 (16%)
• Caesarian section with birth asphyxia.	08 (16%)
• Full term SVD with birth asphyxia.	21 (42%)
	13 (26%)
Gross motor delay:	
• Inability to stand.	20 (40%)
• Inability to walk.	30 (60%)
Soft tissue contractures:	
• Hip flexion	22 (44%)
• Hip adduction	21(42%)
• Knee flexion.	38 (76%)
• Ankle equinovarus	43 (86%)
Gait abnormalities:	
• Scissoring.	20 (40%
• Hip flexed and adducted.	8 (16%)
• Knees flexed with valgus.	7 (14%)
• Ankle equines	15 (30%)
Use of oral antispasticity medications:	15 (30%)
Use of lower limb braces:	
• Ankle foot orthosis (AFO).	16 (32%)
• Knee ankle foot orthosis (KAFO).	7 (14%)
• Hip knee ankle foot orthosis (HKAFO)	2 (4%)
Use of assistive mobility devices:	
• Paediatric walker with front wheel casters.	40 (80%)
Surgical intervention:	
• Adductor tenotomy	5 (10%)
• Hamstring release.	4 (8%)
• Tendo achilles lengthening.	3 (6%)
Improvement in gross motor function:	36 (72%)
• Level V to III	2 (4%)
• Level V to IV	09 (18%)
• Level IV to III	19 (38%)
♦ Level III to II	6 (12%)
Duration of treatment	
• Upto 28 weeks.	24 (48%)
• Greater than 28 weeks	26 (52%)

Based on the above results the following important observations are made.

- Delayed referral for rehabilitation (after 2 years of age) was commonly noted. Forty three of fifty cases (86%) presented between 2 to 10 years of age when soft tissue contracture had already developed.
- History of full term spontaneous vaginal delivery with birth asphyxia was present in 21(42%) of children while prematurity was present in 8 (16%) of cases only.
- Gross motor delay in standing was present in 20 (40%) of cases and walking in 30 (60%) of cases.
- Most commonly seen soft tissue contractures were knee flexion in 38(76%) and ankle equinovarus in 43 (86%) of cases.
- The most common gait abnormality was scissoring in 20 (40%) of cases and ankle equinovarus in 15 (30%) of cases.
- Lower extremity orthotics were used in 25 (50%) of children, with polypropylene ankle foot orthosis being the most commonly used in 16 (32%) of cases.
- Paediatric walker with front wheel caster was the most commonly used assistive mobility device in 40 (80%) of cases.
- Surgical intervention was required in 12 (24%) of the cases.
- Duration of rehabilitation management period was prolonged (upto 28 weeks or more) in all 50 (100%) of cases.
- 72% of the children improved I to II level higher than their presenting ambulatory status.

Discussion

Delay or difficulty in walking was the presenting problem of all the children with spastic diplegia in our study. The delayed referral for rehabilitation (after 2 years of age) was possibly due to lack of early detection of cerebral palsy in these children and availability of a few special centers for paediatric rehabilitation in Pakistan. Soft tissue contracture in the lower extremities was the most difficult problem to deal with requiring prolonged physical therapy and corrective surgery. Improved functional outcome at discharge was present in the majority (72%) of children with spastic diplegia. The improved status of these children in walking helped several of them to achieve access to special education centers which was previously not possible. The remaining 28%, which showed no improvement, was attributed to more severe form of cerebral palsy with cognitive impairment and lack of consistency in the rehabilitation management program. In the light of above findings we conclude that the functional level of ambulation in spastic diplegic cerebral palsy children can be improved with proper rehabilitation management. Early referral of these children to a rehabilitation facility can prevent several complications and also help in achieving better functional outcome.

References:

1. Bax MCO: Terminology and classification of cerebral palsy. Dev. Med. Child Neurol 1964;6:295.
2. Molnar GE: Cerebral palsy. In Molnar GE (ed): Paediatric rehabilitation. Baltimore, Williams & Wilkins 1985, p481.
3. Blasco PA: Primitive reflexes: Their contribution to the early detection of cerebral palsy, Clin Pediatr 1994;33:388.
4. Harris SR: Early neuromotor predictors of cerebral palsy in low birth weight infants. Dev. Med. Child Neurol 1987; 29:508.
5. Illingworth RS: The diagnosis of cerebral palsy in the first year of life. Dev. Med. Child Neurol 1966;8:178.
6. Bobath K, Bobath B: The diagnosis of cerebral palsy in infancy. Arch Dis Child. 1956;31:408.
7. Nicholson A, Alberman E: Cerebral palsy: An increasing contributor to severe mental retardation? Arch Dis Child 1992; 62:1050.
8. Stanley FG: Cerebral palsy trends: Implications for parinatal care. Acta Obstet Gynaecol Scand. 1994; 73:5.
9. Grether JK, Cummins SK, Nelson KB: The California cerebral palsy project. Pediatr Perinatol Epidemiol. 1993; 6:339.
10. Cummins SK, Nelson KB, Grether JK et al: Cerebral palsy in four northern California counties, births 1983 through 1985. J. Pediatr 1993; 123:230.
11. Pharoah POD, Cocke RWI: A high hypothesis for the aetiology of spastic cerebral palsy-the vanishing twin. Dev. Med. Child Nerol. 1997; 39:292.
12. BhushanVB, Paneth N, Kiely JL: Impact of improved survival of very low birth weight infants on recent secular trends in the prevalence of cerebral palsy. Pediatrics 1993; 91:1094.
13. Paneth N, Kiely J: The frequency of cerebral palsy. In Stanley F, Alberman E (ed): The Epidemiology of Cerebral Palsies. Clinics in Developmental Medicine Series. Philadelphia, JB Lippincott 1984.
14. Nelson KB, Ellenberg JH: Antecedents of cerebral palsy. Am J Dis Child 1986; 315:81.
15. Taft LT: Cerebral palsy. Paediatr rev. 1995; 16:411.
16. Stanley FJ, Blair E: Cerebral Palsy. In Pless IB (ed): The Epidemiology of Childhood Disorders. New York, Oxford University Press, 1994.
17. Stanley FJ: Prenatal determinants of motor disorders. Acta Paediatr 1997; 422(Suppl):92.
18. Stempien LM, Gaebler-Spira D: Rehabilitation of children and adults with cerebral palsy. In Braddom RL (ed): Physical Medicine and Rehabilitation. Philadelphia, WB Saunders, 1996.
19. Ellenberg JH: The Nelson KB: Cluster of perinatal events identifying infants at high risk for death or disability. J.Pediatr 1988:113:546.
20. Batshaw ML, Eicher PS: Cerebral palsy. Pediatr Clin North Am 1993; 40:537.
21. Kuban KC, Leviton A: Cerebral palsy. N Engl J Med 1994; 330:188.
22. Uvebrandt P: Hemiplegic cerebral palsy. Acta pediatr Scand 1988; 345(Suppl):1.
23. Nelson K, Swaiman K, Russman B: Cerebral palsy in Swaiman K(ed): Pediatr Neurology: Principles and Practice. St Louis, Mosby, 1994.
24. Crothers BS, Paine RS: The Natural History of cerebral palsy. Cambridge, Havard Press, 1959.

25. McDonald AD: Cerebral Palsy in children of very low birth weight. Arch Dis Child 1963;38:579.
26. Veelken N, Hagberg B, Hagberg G, Olow I: Diplegic cerebral palsy in Swedish term and preterm children: Difference in reduced optimality, relations to neurology and pathogenetic factors. Neuropaediatrics 1983; 14:20.
27. Okumura A, Hayakawa F. Kato T, et al: MRI findings in patients with spastic cerebral palsy: I. Correlation with gestational age at birth. Dev. Med. Child Neurol 1997; 39:363.
28. Okumura A, Hayakawa F. Kato K, et al: MRI findings in patients with spastic cerebral palsy. Dev. Med. Child Neurol 1997; 39:369.
29. Ingram TT: Pediatr Aspects of Cerebral Palsy. Edinburgh, ES Livingston, 1964.
30. Bleck EE: Orthopedic Management of Cerebral Palsy Philadelphia, MacKeith Press 1987.
31. Jones E, Knapp R: Assessment and management of the lower extremity in cerebral palsy. Orthop Clin North Am 1997; 18:725.
32. Palisano R, Rosenbaum P, Walter S, et al: Development and reliability of a system to classify gross motor function in children with cerebral palsy. Dev. Med. Child Neurol 1997; 39:214.