Motor Electrophysiological Studies in Congenital Talipes Equinovarus

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Background: Neurological changes in children with congenital talipes equinovarus are seen with variable frequency. Present study aims to detect the neurological deficit in children with congenital talipes equinovarus (clubfoot).
Methods: Motor electrophysiological studies were performed on 21 children (31 club feet) aged 2-12 years with grade 2 and 3 congenital talipes equinovarus previously treated by lateral-posteromedial release.
Results: Nerve conduction, H-reflex and electromyographic abnormalities were found in 10 children. This suggests that it is a heterogeneous problem with 5 children showing axonal neuropathy, 4 children peripheral neuropathy and one child denervation changes. The neurological deficit probably contributed to the severity of the deformity as 7 of 11 children with grade 3 deformity had deficit ($P<0.05$).
Conclusion: We support the concept that abnormal neurology is a prime factor in the development of club foot deformity.
Key Words: Children, Congenital Talipes Equinovarus, Clubfoot, Nerve Conduction Studies, Electromyography, Neruopathy & Neurological Deficit.

Introduction

Many theories prevail regarding the aetiology of congenital talipes equinovarus (CTEV, clubfoot) including hereditary, environmental and neuro-muscular. The later of these has yet to gain unanimous support, despite clear evidence of calf size disparity, decrease in foot size and peroneal muscle wasting.[1-4] Muscle changes in club foot have been studied previously.[5-9] The neurological component of these changes has never been well delineated. Previous electromyographic studies in club foot have included neurological and myogenic alterations as well as normal findings.[10-15] We report the findings of motor electrophysiological studies to define a neuromuscular cause in the pathogenesis of CTEV.

Patients and Methods

Motor electrophysiological studies were performed on 21 children with structural club feet between ages of 2 and 12 years. There were 15 boys and 6 girls, both feet were effected in 10 children. The severity of the deformity was recorded at the time of neonatal assessment and classified into 3 grades,[16] grade 1 mild deformity (postural club foot), and grade 2 moderate and 3 severe deformity (structural club foot). Ten children had grade 2 and 11 had grade 3 CTEV deformity. All 21 children had lateral-posteromedial release by the Cincinnati incision.[17] Each child was assessed by a comprehensive neurological examination and no abnormalities were detected. This included full motor (tone, power, reflexes, clonus and gait) and sensory (superficial and deep sensations) examination. In addition calf girth, leg length, capillary return and temperature of toes, height and weight were charted. The children with spinal or other recognizable neuromuscular disorder were excluded from the study.

The following techniques were used to measure the neuromuscular status of the spine and lower limbs.

Nerve Conductions Studies

Nerve conduction studies were performed to assess the functional integrity of the axon fibers, the myelin sheath and the neuromuscular junction in the tibial and the common peroneal nerves. The conduction and amplitude of compound muscle action potentials were measured to distinguish between axonal degeneration and segmental demyelination. The tibial nerve was examined by stimulation of the nerve with a short square pulse delivered at the popliteal fossa or ankle. Surface electrodes were attached to the abductor hallucis muscle and the amplitude, latency and conduction velocity were measured. Similarly, the common peroneal nerve was examined using the same technique with stimulation of the nerve at the fibular neck, recording were measured from extensor digitorum brevis muscle.

H-Reflex Studies (Monosynaptic Reflex L5-S1)

In these studies the action potentials have H-shaped waves that is why it is called H-reflex or waves. H-reflexes were recorded by stimulating the tibial nerve at the popliteal fossa which innervates the gastrocnemius-soleus with current pulses of long duration and low intensity. The recordings were measured on the lower part of the triceps surae using surface electrodes. This monosynaptic reflex is the electrical equivalent of ankle jerk reflex, provoked by the stimulating of afferents in the mixed nerve instead of mechanical muscle stretch. The H-reflex is lost or latency delayed in the process that affects the L5-S1 nerve root.

Electromyography (EMG)

Electromyography was carried out with a concentric intra-muscular needle to look for the evidence of denervation (spontaneous fibrillation, positive sharp waves, increased amplitude of motor unit's action potential and recruitment pattern). Values were recorded from the tibialis anterior, the peronei and the gastrocnemius muscles. One muscle was chosen from each compartment of the leg. The EMG was not done on the tibial posterior muscle because if was difficult to be certain about the exact location of the needle as the muscles is in the deep part of the posterior compartment of the leg.

All the studies were performed on a Medlec sapphire machine (Oxford Instruments, Surrey, UK). Normal values of nerve conduction studies and electromyography were established form a group of 30 children (20 boys and 10 girls) between the ages of 2-12 years with no history of neurological disease in our paediatric neuro-physiology laboratory. The parents gave their informed consent of the investigations, which were undertaken in the compliance with standards set by the local ethical committee.

Surgical correction of club feet had been taken between the ages of 3 to 6 months by lateral-posteromedial release by the Cincinnati incision after preliminary period of strapping or plaster. The neurological studies were undertaken between the ages of 2 to 12 years. The two 2-year old children had been operated upon at the age of 3 months and therefore all the children were assessed a minimum of 2 year after surgical release.

Results

The clinical neurological examination was normal in all the 21 children. The motor electorphysiological studies showed that 10 children (48%), 6 with bilateral club feet had abnormal findings (table1). The results fell into 3 groups according to the positive findings.

Group I: 4 children had prolonged conduction velocity of the common peroneal nerve. Out of these 4 children the 2 with bilateral club feet had prolonged conduction velocity of the posterior tibial nerve as well. All these children also have low amplitudes of the H-reflex. These findings suggest peripheral neuropathy.

Group II: 5 children, 3 with bilateral club feet had normal conduction velocity of both the common peroneal and the posterior tibial nerve but had a prolonged H-reflex suggesting an abnormality at the spinal level.

Group III: Only one child with bilateral clubfeet had an electromyographic abnormality with positive sharp waves at rest in tibialis anterior and peroneal muscles on both sides, suggesting denervation changes, without any evidence of electrophysiological abnormality.

Detail results of all the children are shown in table 2.

Table 1: Results of motor electrophysiological studies.

Type of Abnormality	Number of children Laterality			Number of children Grade of Deformity	
	Uni	Bilat	Total	2	3
Prolonged nerve conduction and H reflex	2	2	4	2	2
Normal nerve conduction of both common peroneal and posterior tibial nerve with prolonged H -reflex	2	3	5	2	3
Electromyographic abnormality (EMG)		1	1		1
None	7	4	11	6	5

Key: Uni (Unilateral), Bilat (Bilateral)

Table 2: Detail results of motor electrophysiological studies.

No.	Age	Sex	Side	Severity of the Deformity	Operations	NCS TN	NCS CPN	H-reflex	EMG
1	12	F	R	2	1	P	P	N	N
			L	2	1	P	P	N	N
2	7	M	R	2	1	N	N	N	N
			L	2	1	N	N	N	N
3	6	M	R	3	2	N	N	P	N
			L	3	2	N	N	P	N
4	5	M	R	3	1	N	N	N	N
			L	3	1	N	N	N	N
5	4	F	L	3	2	N	N	N	N
6	7	M	R	3	2	N	N	N	A
			L	3	2	N	N	N	A
7	2	M	R	3	1	N	N	N	N
8	2	M	L	2	1	N	N	P	N
9	6	F	R	2	1	N	N	N	N
10	3	M	L	3	3	N	N	N	N
11	5	M	R	3	3	N	N	P	N
			L	3	3	N	N	P	N
12	5	F	R	3	3	N	N	P	N
			L	3	3	N	N	P	N
13	4	F	R	2	1	N	N	N	N
			L	2	1	N	N	N	N
14	6	M	R	3	2	N	P	N	N
15	4	M	L	2	1	N	N	N	N
16	4	M	R	2	1	N	P	N	N
17	5	M	R	3	2	N	N	N	N
18	3	M	L	2	1	N	N	N	N
19	5	F	R	3	2	P	P	N	N
			L	3	2	P	P	N	N
20	6	M	R	2	1	N	N	N	N
			L	2	1	N	N	N	N
21	7	M	L	3	2	N	N	P	N

NCS TN (Nerve conduction studies of tibial nerve, NCS CPN (Nerve conduction studies of common peroneal nerve), EMG (electromyography), M (male), F (female), R. (right), L (Left), N (Normal), P (prolonged), A (abnormal).

Discussion

The aetiology of congenital talipes equinovarus (clubfoot) is probably multifactorial but association of the deformity with a number of paediatric neurological conditions suggests that abnormal neurology may be a major causative factor in the development of this deformity. The children included in this study had normal neurological examination and there was no obvious neurological cause of their deformity. Abnormalities of the peripheral nerves, spinal cord and EMG were noted in 48% of these children.

Several electrophysiological studies have been described to find the neurological abnormality in the club foot (CTEV). Except for the H-reflex, standard diagnostic techniques do not evaluate the spinal cord pathology. Trontelj and Pavlovcic (1992)[18] found altered calf tendon jerks in all the 27 patients with unilateral club foot. Feldbrin et el. (1995)[15] performed nerve conduction, H-reflex and EMG studies on 52 children and reported abnormalities of peripheral nerves and or spinal cord in 83% of the patients. Only one of their patients had degenerative EMG pattern as in our study.

Histochemical and electronic microscopic examination by lsaacs et al. (1977)[6] of 60 club feet showed hypertrophy, predominance of type l fibers and loss of direction and grouping. Handelsman and Bedalamente (1981)[8] studied 90 muscle biopsies by electron microscope from 13 club feet and identified the differences in muscle fiber with a high proportion of type I fibers compared with the control. There was also increased amount of fibrosis and reduced excursion of these muscles. All these ultrastructural abnormalities are compatible with neurogenic aetiology. This indicates that minor degree of muscle imbalance of neural origin during the period of rapid skeletal growth in the early intrauterine period might produce disproportionately severe deformities.[19,20]

All children were older than 2 years of age and had had previous surgery. It is possible that inclusion of the older patients undergoing surgical procedure may alter the eleltromyographic findings as a result of contractures, fibrosis, or chronicity of the disease process. Feldbrin et al. (1995)[15] repeated their studies with at least one year between examination in 13 patients and found no significant improvement or deterioration in these patients, suggesting that the neurological pathology did not vary with age.

The neurological deficit was related to the severity of the deformity ($p < 0.05$) as 3 out of 10 children with grade 2, and 7 out of 11 children with grade 3 CTEV deformity had neurological deficit. Out of the 7 patients with grade 3 deformity, 5 had a second lateral-posteromedial release and 2 had a third extensive club foot release suggesting that presence of the neurological deficit may be a cause of recurrence of the deformity. This indicates that abnormal innervation is a significant factor in the development of this peripheral deformity. Pathology is most likely at the spinal level but more research is needed to confirm that anterior horn cell dysplasia has a role in the pathogenesis of club foot.[21] This will explain the clinical observation of peroneal muscle weakness and calf wasting in structural club foot.

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