Evaluation of the deformity in club foot by somatosensory evoked potentials

M. F. Macnicol, R. D. Nadeem

From the Murrayfield Hospital, Edinburgh, Scotland

Somatosensory evoked potentials (SSEPs) measure the conduction pathways from the periphery to the brain and can demonstrate the site of neurological impairment in a variety of locomotor conditions.

SSEPs were studied in 44 children (64 feet) with surgically corrected club feet. Four children had unreproducible responses, 18 showed abnormal recordings and 22 showed normal responses. In a further 31 feet (21 children) subjected to motor electrophysiological tests, 16 (52%) were abnormal.

Overall, 44 of 95 feet (46%) showed abnormal SSEPs or motor electrophysiological tests.

Neurological abnormality was related both to the severity of the deformity and the surgical outcome. It was seen in 38% of feet with grade-2 and in 53% of feet with grade-3 deformity. A fair surgical result was obtained in 36% of feet with a conduction deficit and in only 6% with no abnormality. These results suggest an association between neurological abnormality as demonstrated by SSEPs or motor electrophysiological studies and the severity of deformity in club foot and its response to surgical treatment.

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In most children with club foot a definite neurological deficit is hard to demonstrate, although atrophy of the calf and sluggish or absent peroneal function is seen clinically. A few have conditions such as spinal dysraphism or central core disease.¹ Abnormal muscle development or a disproportion of type-1 muscle fibres have also been implicated.² It is debatable, however, whether the alterations are primary or secondary.³

Neurological testing is difficult in the infant. Nerve-conduction studies, measurement of the H-reflex (spinal

monosynaptic reflex) and electromyography⁴ have produced conflicting reports. Histochemical studies of muscle, ligament or neural tissue have given varying data (see Table IV).

We report what we believe to be the first application of somatosensory evoked potentials (SSEPs) in the investigation of children with surgically corrected club feet. Duckworth et al⁵ used SSEPs to assess abnormalities of the spinal cord in children with spina bifida, and the technique is established for monitoring spinal procedures⁶⁻¹², and reconstruction of the pelvis^{13,14} and of the lower limb.¹⁵

Patients and Methods

A total of 65 children (44 boys and 21 girls) with grade-2 and grade-3 clubfoot deformity, as described by Harrold and Walker,¹⁶ and which had been treated surgically, was investigated in a paediatric neurophysiology laboratory. The studies were undertaken in a room at constant temperature, but with no electrical shielding or deadening of sound. Each child had a comprehensive neurological examination and their height and weight were recorded. The surgical outcome¹⁷ was classified using a scoring system modified from Laaveg and Ponseti¹⁸ and McKay.¹⁹ The score for each case was subtracted from 130 points, and the result described as follows: excellent, 115 to 130; good, 100 to 114; fair, 85 to 99; and poor, less than 85.

Surgical correction had been undertaken in 76 feet in children aged between three and six months, after a preliminary period of strapping. In some children plaster was also used after the initial strapping before operation. In the remaining 19 feet (13 children) surgery had been carried out after the age of six months, since the children had been referred from other hospitals with a recurrent deformity. The mean age at operation in this group was 3 years 8 months (1 to 10).

The studies were undertaken in children when they were aged between two and 16 years (mean, 7.4) and at least two years after surgical release.

We recorded SSEPs using scalp electrodes fixed with collodion and adhesive tape to keep the impedance below 2 and 4 kohms. Electrical pulses of 0.1 ms duration were delivered at 5 Hz to the skin over the posterior tibial nerve at the ankle using a saddle-type bipolar stimulator with the

M. F. Macnicol, MCh, FRCP, FRCS Ed(Orth), Consultant Orthopaedic Surgeon

R. D. Nadeem, MCh, FRCS Ed(Orth), Research Fellow
Royal Hospital for Sick Children, Sciennes Road, Edinburgh EH9 1LF, UK.

Correspondence should be sent to Mr M. F. Macnicol.

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cathode placed 3 cm proximal to the anode. The intensity of the current was increased to produce twitching of the abductor hallucis muscle or, if no twitch could be elicited, to at least three times the perceived sensory threshold. A stimulus was also applied to the skin over the common peroneal nerve at the neck of the fibula. Twitching of the extensor digitorum brevis muscle (EDB) was used as a guide to the adequacy of the stimulus.

A current of 10 to 20 mA was sufficient to cause twitching in the distal, reference muscle and a minimum of two traces was recorded to ensure reproducibility. Specified peak components of the waveforms, the absolute latencies and interpeak intervals were measured in accordance with normative data obtained in children by Gilmore et al.²⁰

The output of each electrode was fed to a Medelec sensory evoked potential system (Medelec Sapphire; Oxford Instruments, Surrey, UK) for amplification, filtered using a 30 to 1500 band pass and averaged over 1000 to 2000 responses. The computer was programmed to reject any input potential exceeding 22.5 V. All the responses were replicated at least once to check the reliability of the data which were stored on a floppy disk for subsequent analysis.

In group 1 (22 children, 28 club feet) SSEPs were measured in both the posterior tibial nerve, for which there is good normative data²⁰ and the common peroneal nerve, for which there is no reliable information in the normal child. The reference muscle EDB was used to confirm a stimulus-induced twitch. There were three types of response: normal (both latency and wave form), abnormal (delayed latency and/or conduction times) and unrecordable (reproducible responses could not be elicited).

Although the unrecordable responses may represent a complete absence of conduction, we did not include these recordings in the abnormal group as artefact could not be entirely ruled out.

A second group of 22 children with 36 club feet was assessed over the posterior spinous processes at L1 and C7 to trace the level of abnormality in the spinal cord.

Because of difficulties in recording the common peroneal response, and the uncertainty regarding normative data for it, the measurements of the SSEP in this group were confined to the posterior tibial nerve. The corticospinal tracts for the posterior tibial and common peroneal nerves are adjacent in the spinal cord and therefore the study was designed to identify any alteration in conduction over these segments of the cord.

In a third group of 21 children with 31 club feet (group 3) conventional motor electrophysiological studies such as nerve-conduction studies, measurement of the H (mono-synaptic) reflex at L5/S1 and electromyography (EMG) were undertaken. Ideally, SSEPs would also have been carried out, but the combined studies were considered too demanding.

The H-reflex was assessed by stimulating the posterior tibial nerve at the popliteal fossa with electropulses of short

duration and low intensity. Surface recording electrodes were placed on the distal triceps surae and the reflex was measured.

EMG was performed with a concentric intramuscular needle recording from the tibialis anterior, the peronei and the gastrocnemius. Evidence of denervation included spontaneous fibrillation, positive sharp waves, increased amplitude of motor action potentials and recruitment patterns. Normal values for nerve conduction and EMG were established from a control group of healthy, age-matched children.

The studies were supervised by a neurologist and an orthopaedic surgeon. The tracings were assessed by two neurologists separately, and the orthopaedic surgeon. Parents gave informed consent for the investigations which were undertaken in compliance with standards set by the local Ethical Committee.

Results

Responses of the common peroneal nerve. In group 1 responses were elicited in nine children (11 club feet). All these feet had been graded as moderate (grade 2) before operation and conduction was noted to be slowed compared with the normal side in unilateral cases. Lack of normative data made it impossible to quantify the conduction defect more accurately. SSEPs in the peroneal nerve could not be measured in the remaining 17 club feet in 13 children. Most of these feet had presented with severe deformity (grade 3).

Responses of the posterior tibial nerve. A total of 30 club feet (22 children) in groups 1 and 2 showed a normal response. In 25, the responses were reproducible at all levels, but in five group-2 feet the peripheral and spinal responses were normal, but the cortical responses were not reproducible. Since the peripheral and spinal responses and conduction times were normal, these were considered normal.

The latency of the posterior tibial nerve was prolonged in 13 club feet in group 1. In group 2, 13 club feet had prolonged spinal conduction times and a further child with bilateral club feet had prolonged peripheral conduction times on both sides. Hence, 28 of the 64 club feet in groups 1 and 2 had abnormal results.

Only six club feet in groups 1 and 2 did not give reproducible results. Two children with bilateral club feet had an unreproducible result on both sides at all levels, and two feet in two children with unilateral deformity had normal results on the unaffected side. Since the results were unreproducible on the side of the club foot we have not included them in the abnormal results.

Motor electrophysiological studies. In these studies 16 club feet showed abnormal findings. We divided the results into three patterns:

1) Six feet had prolonged conduction velocity of the common peroneal nerve. Of these, four in two children with bilateral deformities also had prolonged conduction

velocity of the posterior tibial nerve. All six feet had low amplitudes of the H-reflex suggesting peripheral demyelination neuropathy.

2) Eight feet had normal conduction velocity of both the common peroneal and the posterior tibial nerves but had a prolonged H-reflex suggesting an abnormality at the spinal level.

3) Only two feet in one child with bilateral involvement had an abnormality of the EMG with positive sharp waves at rest on both sides, suggesting denervation changes. There was no evidence of electrophysiological abnormality.

Table I details the abnormal results in the three groups. Almost half of the club feet (46%) showed clear neurological deficits. Table II gives the level of the deficit. In four children tracings were difficult to reproduce. The use of SSEPs in children aged under five years posed problems both in the interpretation of results and co-operation by the child. There was also greater variability in the waveform of the scalp-recorded potentials as has been noted previously.^{20,21}

The case notes of the children with abnormal SSEP tests (28 feet) were reviewed by a separate observer who found that 12 (43%) were previously grade 2 and 16 (57%) previously grade 3. Combining the results for the SSEP and motor electrophysiological studies, 16 grade-2 feet (38)

Table I. The proportion of club feet with neurological deficit identified by either measurement of the SSEP or motor electrophysiological studies

Neurological test	Number of feet studied	Number of feet (%) with a neurological deficit
SSEP	64	28 (44)
Motor electrophysiological	31	16 (52)
Total	95	44 (46)

Table II. The anatomical level of detectable neurological abnormality in the 95 club feet

Level of neurological deficit	Neurological tests		Number of feet (%)
	SSEP	Motor electrophysiological	
Spinal	13	8	21 (22)
Peripheral	2	6	8 (9)
Mixed	13	0	13 (14)
Muscles	0	2	2 (2)

Table III. The surgical outcome according to the severity of the deformity and neurological deficit associated with the 95 clubfeet

Neurology	Functional outcome			Total
	Excellent	Good	Fair	
Deficit				
Grade 2	14	2	0	16
Grade 3	3	9	16	28
No deficit				
Grade 2	22	4	0	26
Grade 3	13	9	3	25

Fig. 1

The influence of measurable neurological deficit on the functional outcome after surgical release.

Fig. 2

The severity of the presenting structural deformity related to the outcome after surgery.

Table IV. Summary of previous reports in the literature

Normal	Abnormal
Electrophysiological tests	
Tonniss ²⁴	Abbruzzese et al ²⁷
Attenborough ²⁵	Peretti and Surace ²⁸
Bill and Versfeld ²⁶	Koczocik-Przedpelska and Marciniak ²⁹
Trontelj and Pavlovic ³⁰	
Feldbrin et al ⁴	
Histochemistry (muscle, ligament or spinal cord)	
Flinchum ³¹	Bechtol and Mossman ³⁷
Wiley ³²	Isaacs et al ³⁸
Irani and Sherman ³³	Ippolito and Ponseti ³⁹
Settle ³⁴	Handelsman and Badalamente ²
Henkel, Woods and Arnold ³⁵	Swart ²³
Kaplan ³⁶	Fukuhara, Schollmeier and Uthoff ⁴⁰
Gray and Katz ³	

were neurologically abnormal and 28 grade-3 feet (53%) were abnormal.

Table III summarises the surgical outcome of the 95 club feet in relation to the severity of the preoperative deformity and the presence or absence of a neurological deficit. When there was no measurable neurological deficit 94% of feet achieved a good or excellent outcome. In the presence of a neurological abnormality this proportion fell to 64%. Figure 1 shows this difference in terms of the number of feet reviewed and the effect of demonstrable neurological deficit on the functional outcome after surgical release.

Discussion

Our study forms part of a review of the results of a standardised, single-stage lateral posteromedial release using the transverse Cincinnati incision.¹⁷

It has often been observed that the peroneal muscles are slow to recover, or are non-functioning after operation. This lack of muscle balance is a potential cause of recurrence. We believe that neurological testing is of value in conjunction with evaluation of the function of the foot, muscle balance and mobility of the tarsal joints, and radiography of the talocalcaneal and calcaneocuboid relationships²² in deciding whether reoperation is needed. It can also help to decide whether an ankle-foot orthosis is indicated.

We consider that this is the first use of measurement of the SSEPs to evaluate the neurological deficit in club foot. A combination of measurement of the SSEP and motor electrophysiological studies revealed that 44 of 95 club feet (46%) were neurologically abnormal, and this may be an underestimate. The clinical severity of the deformity was related to the deficit, with 38% of grade-2 feet and 53% of grade-3 feet being abnormal neurologically. Deficits were found at different levels of the neuraxis (spinal cord, peripheral nerve and muscle). A mixed picture was seen in 13 of the 44 abnormal studies. A fair surgical outcome was more likely in grade-3 deformity with a neurological deficit. The proportion of fair results altered from three in 25 neurologically normal feet (12%) to 16 in 28 feet (57%) showing neurological abnormality (Table III).

One problem in accepting the role of neurological testing in club foot has been the divergent findings in the literature (Table IV). These studies were discussed by Feldbrin et al.⁴ Our study has demonstrated that SSEPs can be effective in defining the neurological deficit in the older child. Ideally, these tests should be carried out before operation in the neonate. We had hoped to undertake measurements under general anaesthesia immediately before the surgical operation. Practical difficulties in obtaining recordings in the infant mean that, at present, the published electrophysiological results have only been undertaken after operation. This introduces a possible source of error. It can also be difficult to obtain co-operation in the conscious child under the age of five years. These factors may make neurological evaluation unattractive in the young child.

We believe that the use of SSEP testing could be restricted to the assessment of grade-3 (severe) club-foot deformity since neurological deficit has a pronounced effect on that group (Fig. 2). It is important to recognise the influence of muscle imbalance in the aetiology and prognosis of club foot.⁴ In some cases it may result from an infection of the spinal cord at the L5/S1 level.²³

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References

1. Dubowitz V, Sharrard WJ. Congenital clubfoot with ? central core disease of muscle. *Proc Roy Soc Med* 1968;61:1258-60.
2. Handelsman JE, Badalamente MA. Neuromuscular studies in club-foot. *J Pediatr Orthop* 1981;1:23-32.
3. Gray DH, Katz JM. A histochemical study of muscle in club foot. *J Bone Joint Surg [Br]* 1981;63-B:417-23.
4. Feldbrin Z, Gilai AN, Ezra E, et al. Muscle imbalance in the aetiology of idiopathic club foot: an electromyographic study. *J Bone Joint Surg [Br]* 1995;77-B:596-601.
5. Duckworth T, Yamashita T, Franks CL, Brown BH. Somatosensory evoked cortical responses in children with spina bifida. *Dev Med Child Neurol* 1976;18:19-24.
6. Nash CL Jr, Brown RH. Current concepts review. Spinal cord monitoring. *J Bone Joint Surg [Am]* 1989;71-A:627-30.

7. Dawson E, Sherman J, Kanim L, Nuwer M. Spinal cord monitoring: results of the Scoliosis Research Society and the European Spinal Deformity Society survey. *Spine* 1991;16:Suppl 8:361-4.
8. Kalkman CJ, Been HD, Ongerboer de Visser BW. Intraoperative monitoring of spinal cord function: a review. *Acta Orthop Scand* 1993;64:114-23.
9. Robinson LR, Slimp JC, Anderson PA, Stolor WC. The efficacy of femoral nerve intraoperative somatosensory evoked potentials during surgical treatment of thoracolumbar fractures. *Spine* 1993;18:1793-7.
10. Trammell TR, Friedlander JK, Reed DB. The effects of lower limb ischaemia on somatosensory evoked potentials during spinal surgery: report of two cases and review of the literature. *Spine* 1993;18:413-5.
11. Helmers SL, Carmant L, Flanigin D. Anterior neck recording of intraoperative somatosensory-evoked potentials in children. *Spine* 1995;20:782-6.
12. Noorden MHH, Lee J, Gibbons CER, Taylor BA, Bentley G. Spinal cord monitoring in operations for neuromuscular scoliosis. *J Bone Joint Surg [Br]* 1997;79-B:53-7.
13. Vrachas M, Gordon RJ, Mears DC, Krieger D, Sclabassi RJ. Intraoperative somatosensory evoked potential monitoring of pelvic and acetabular fractures. *J Orthop Trauma* 1992;6:50-8.
14. Baumgaertner MR, Wegner D, Booke J. SSEP monitoring during pelvic and acetabular fracture surgery. *J Orthop Trauma* 1994;8:127-33.
15. Makarov MR, Delgado MR, Birch JG, Samchukov ML. Intraoperative SSEP monitoring during external fixation procedures in the lower extremities. *J Pediatr Orthop* 1996;16:155-60.
16. Harrold AJ, Walker CJ. Treatment and prognosis in congenital club foot. *J Bone Joint Surg [Br]* 1983;65-B:8-11.
17. Macnicol MF. The surgical management of congenital talipes equinovarus (club foot). *Curr Ortho* 1994;8:72-82.
18. Laaveg SJ, Ponseti IV. Long-term results of treatment of congenital club foot. *J Bone Joint Surg [Am]* 1980;62-A:23-31.
19. McKay DW. New concept of and approach to clubfoot treatment: section III evaluation and results. *J Pediatr Orthop* 1983;3:141-8.
20. Gilmore RL, Bass NH, Wright EA, et al. Developmental assessment of spinal cord and cortical evoked potentials after tibial nerve stimulation: effects of age and stature on normative data during childhood. *Electroencephalogr Clin Neurophysiol* 1985;62:241-51.
21. Onishi H, Yamada T, Saiko T, et al. The effect of stimulus rate upon common peroneal, posterior tibial and sural nerve somatosensory evoked potentials. *Neurology* 1991;41:1972-7.
22. Macnicol MF, Flocken LE. Calcaneocuboid malalignment in club-foot. *J Pediatr Orthop* 1999;8:257-60.
23. Swart JJ. Clubfoot: a histological study. *SA Bone Joint Surg* 1993;3:17-23.
24. Tonnis D. Electromyographische und histologische untersuchungen zur Frage der Entstehung des angeborenen Klumpfußes. *Z Orthop Ihre Grenzgeb* 1969;105:595-615.
25. Attenborough CG. Early posterior soft-tissue release in severe congenital talipes equinovarus. *Clin Orthop* 1972;84:71-8.
26. Bill PLA, Versfeld GA. Congenital club foot: an electromyographic study. *J Pediatr Orthop* 1982;2:139-42.
27. Abbruzzese M, Cabella G, Pastorino P, Sacco G. Electromyographic study of congenital club foot. *Riv Neurobiol* 1972;18:221-32.
28. Peretti G, Surace A. Club-foot, classification, etiology and pathogenesis. *Ital J Orthop Traumatol Suppl* 1976;2:11-37.
29. Koczocik-Przedpelska J, Marciniak W. Determination of the incidence of neurogenic muscular lesions in congenital foot deformities. *Chir narzadow Ruchu Ortop Pol* 1979;44:259-63.
30. Trontelj JV, Pavlovic V. Calf-tendon jerks are altered in congenital equinovarus deformity. *Dev Med Child Neurol* 1992;34:966-71.
31. Flinchum D. Pathological anatomy in talipes equinovarus. *J Bone Joint Surg [Am]* 1953;35-A:111-4.
32. Wiley AM. Club foot: an anatomical and experimental study of muscle growth. *J Bone Joint Surg [Br]* 1959;41-B:821-35.
33. Irani RN, Sherman MS. The pathological anatomy of club foot. *J Bone Joint Surg [Am]* 1963;45-A:45-52.
34. Settle GW. The anatomy of congenital talipes equinovarus: sixteen dissected specimens. *J Bone Joint Surg [Am]* 1963;45-A:1341-54.
35. Henkel HL, Woods CG, Arnold M. Die Muskulatur beim angeborenen Klumpfuß: Morphologische, und histometrische untersuchungen der Mm gastrocnemius, flexor hallucis und tibialis posterior. *Z Orthop Ihre Grenzgeb* 1971;108:604-32.
36. Kaplan EB. Comparative anatomy of the talus in relation to idiopathic clubfoot. *Clin Orthop* 1972;85:32-7.
37. Bechtol CD, Mossman HW. Clubfoot: an embryological study of associated muscle abnormalities. *J Bone Joint Surg [Am]* 1950;32-A:827-38.
38. Isaacs H, Handelsman JE, Badenhurst M, Pickering A. The muscles in club foot: a histological, histochemical and electron microscopic study. *J Bone Joint Surg [Br]* 1977;59-B:465-72.
39. Ippolito E, Ponseti IV. Congenital club foot in the human fetus: a histological study. *J Bone Joint Surg [Am]* 1980;62-A:8-22.
40. Fukuhara K, Schollmeier G, Uthoff HK. The pathogenesis of club foot: a histomorphometric and immunohistochemical study of fetuses. *J Bone Joint Surg [Br]* 1994;76-B:350-7.