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Sensory conduction in medial plantar nerve

Normal values, clinical applications, and a comparison with the sural and upper limb sensory nerve action potentials in peripheral neuropathy

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SUMMARY A method for recording the medial plantar sensory nerve action potential at the ankle with surface electrodes is described. Normal values in 69 control subjects are given and compared with the sural sensory nerve action potential in the same limb in the same subjects. Clinical applications were studied in 33 patients. The procedure may be applied in the diagnosis of L4–5 nerve plexus or root lesions, lesions of the sciatic, posterior tibial, and medial plantar nerves, and is a more sensitive test than other sensory nerve action potentials in the diagnosis of peripheral neuropathy.

Peripheral neuropathies may have some predilection for sensory nerve fibres in the lower extremities (Mavor and Atcheson, 1966), and there is some evidence to suggest that measurement of the sural sensory nerve action potential (SAP) may be a more sensitive test than upper limb SAPs in this situation (Di Benedetto, 1970, 1972; Burke *et al.*, 1974) but no comparisons with other SAPs in the lower limbs are available. The sural SAP tests only the sensory post-ganglionic S1 root distribution. It is unhelpful in L4–5 nerve root or plexus lesions, and in lesions of the lateral popliteal and posterior tibial nerves as the sural nerve is formed by variable contributions from both of these nerves.

The medial plantar nerve is a branch of the posterior tibial nerve whose skin distribution includes the plantar and medial aspects of the big toe. The latter is said to be within the L4–5 sensory root distribution (Foerster, 1933).

Initial attempts to obtain the medial plantar SAP in normal subjects by recording with surface electrodes at the ankle and stimulating the big toe did not give consistent responses (Mayer, 1963; Mawdsley and Mayer, 1965). Using averaging techniques requiring 40 to 200 stimuli Mavor and Atcheson (1966) recorded the response in 12 normal subjects. A series of 33 posterior tibial nerves studied with needle electrodes giving normal values in two age groups for the SAP at the ankle has been reported (Behse and Buchthal, 1971). Data in a control series with surface electrodes and in patients with peripheral nerve disease are lacking.

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Methods

ANATOMY

The posterior tibial nerve at the ankle, just below the medial malleolus, gives origin to the medial plantar and lateral plantar nerves and to calcaneal branches (Hollinshead, 1958). The medial plantar nerve passes forward along the medial side of the foot accompanying the medial plantar artery, one of the terminal branches of the posterior tibial artery. The posterior tibial pulse, located near the medial malleolus, is thus a very convenient anatomical landmark (*Gray's Anatomy*, 1949). It supplies the muscle abductor hallucis, the medial anterior two-thirds of the sole, the plantar skin of toes 1, 2, and 3, the medial side of the fourth toe, and their nail beds. Stimuli were applied to the medial and lateral digital nerves of the big toe, the former originating usually from the medial terminal branch of the medial plantar nerve, the latter from its lateral terminal branch.

RECORDINGS

These were made with the subject lying on a couch, the leg supported by pillows. The skin was cleaned with alcohol. Sural nerve SAPs were recorded antidromically. Stimulation was carried out using a constant voltage stimulator set to deliver 0.1 ms rectangular electric pulses through Ag:AgCl button electrodes covered with gauze soaked in normal

saline. Supramaximal stimuli were delivered at a point close to the tendo Achilles, 140 mm proximal to the lateral malleolus of the ankle. The recording electrodes were rectangular Ag: AgCl bars covered in saline-soaked gauze, mounted on perspex 40 mm apart. The proximal electrode was placed inferior to the lateral malleolus of the ankle, and the distal one along the track of the nerve. Where it was difficult to elicit responses, the recording electrodes were connected to the stimulator, the point of lowest sensory threshold taken as being the course of the nerve.

Medial plantar SAPs were recorded orthodromically in the same limb as the sural SAP. Supramaximal shocks were delivered through two ring electrodes of Ag: AgCl covered in saline-soaked gauze, encircling the hallux, which was separated from the other toes by a strip of plastic sheet. The same recording electrodes as used for the sural SAP were employed. The distal electrode was placed over a point at which the posterior tibial artery could be palpated, close to the medial malleolus of the ankle. The proximal one usually rested on the medial edge of the tendo Achilles (Fig. 1). Initial difficulty in obtaining a response in a number of cases was due to the electrodes being placed too far distally.

Recordings were made using a Medelec MS6 electromyograph. The AA6 amplifier was set to a deflection of 10 μV/cm, with a time constant of 5 ms and a HF cut at 3.2 kHz. Thirty or 32 responses were averaged on an AVM6 or AV6 averager.

The ambient temperature was 21–26°C, and draughts were excluded. Apart from keeping the limb covered as much as possible with blankets, further precautions were not taken. Limb temperature was measured by placing a thermistor probe on the medial and plantar aspect of the foot, towards the end of the procedure (range 27°C–33°C).

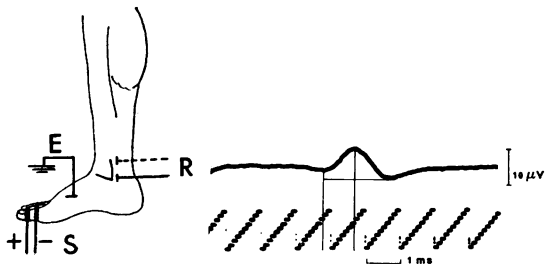

Fig. 1 *Positioning of surface recording electrodes (R), earth (E), and stimulating ring electrodes (S). On the right the averaged medial plantar sensory action potential after 32 sweeps is shown (normal male aged 13 years). Vertical lines show measurements of latency to onset (3.8 ms) and peak (4.7 ms). Amplitude is 8.1 μV. Time scale of the staircases is 1 ms with ten 0.1 ms steps each. Negativity upwards in this and all following figures. Stimulus always at onset of sweep except in Fig. 2.*

MEASUREMENTS

Measurements of amplitude, latency to onset, and latency to peak were made as shown in Fig. 1. With SAPs of around 1.0 μV care was taken to ensure that the deflection seen was reproducible on two or three successive averaging runs and, if necessary, the potentials were scaled up by a factor of two or more for greater ease of measurement, by overriding the automatic scaling of the averager display (Fig. 2).

Fig. 2 *Small medial plantar SAP (1.4 μV).*

The variation attributable to measuring the photographic record of small potentials was checked by asking 10 colleagues who had experience of EMG to measure a 1.1 μV potential. The mean value was 1.1 μV with a range of 0.7–1.8 μV (SD=0.4 μV). Statistical calculations were made according to Snedecor and Cochran (1967).

Subjects

CONTROLS

There were 29 males and 40 females, their ages ranging from 13 to 81 years. These comprised 17 consecutive healthy volunteers, 39 consecutive patients referred to an EMG clinic, but without clinical or electrical evidence of peripheral nerve disease, and 13 patients with either isolated peripheral nerve lesions in the upper limb and no clinical or EMG evidence of peripheral neuropathy, or isolated peripheral nerve lesions in one lower limb but a normal contralateral limb. Individuals with a history of trauma to the ankle or foot requiring immobilisation of the joint for more than a few days, or with hallux valgus deformity were excluded.

PATIENTS

The clinical histories of the 33 patients were carefully reviewed and all of them examined personally. Clinical criteria for peripheral neuropathy were a history of paraesthesiae, numbness or weakness in the limbs or both, and on examination, wasting, weakness, depressed, or absent tendon reflexes and distal sensory loss in the limbs. Individual nerve lesions were

diagnosed on clinical evidence of involvement of the appropriate muscles and the area of sensory loss. All other available EMG and nerve conduction data excluding the medial plantar and sural SAPs were taken into consideration.

Results

CONTROL SUBJECTS

The medial plantar SAP usually consisted of a negative wave sometimes preceded or followed by a positive wave. Figures 1 and 2 illustrate 8.1 μV and 1.4 μV potentials.

The values in 69 control subjects for amplitude, latency to onset of SAP, latency to peak and sensory nerve conduction velocity (measured to onset) for the medial plantar and sural SAPs are shown in Tables 1 and 2. They are arranged by decades of age. The medial plantar SAP was absent only in three control subjects, aged 81, 71, and 60 years. The sural SAP amplitudes in these three subjects was 3.9 μV, 2.5 μV, and 4.2 μV respectively. The sural SAP was present in all control subjects. There is a statistically significant correlation between amplitude and age for both the sural and medial plantar SAPs (Fig. 3). There was no relationship between maximal sensory conduction velocity and age for both SAPs (Fig. 4). There was a significant correlation between the amplitude of the sural SAP and that of the medial plantar SAP (r=0.5, $P<0.001$) (Fig. 5).

PATIENTS

The 33 patients with their individual diagnoses and values for amplitude and maximal sensory conduction velocity for medial plantar and sural SAPs are listed in Tables 3, 4, and 5.

Peripheral neuropathy

There were 13 patients in this group. The clinical features are summarised in Table 3, and the electrical data in Tables 4a and b. Nine patients had definite clinical and electrical evidence of peripheral neuropathy (cases 1, 2, 3, 5, 7, 8, 9, 12, 13). Case 4 had a clinical diagnosis of Guillain-Barré syndrome but no electrical evidence of peripheral neuropathy except for the absent medial plantar SAP. Case 10 had only electrical evidence of peripheral neuropathy. We considered that cases 6 and 11 had clinical and electrical evidence suggestive of a mild peripheral neuropathy.

All the patients had absent medial plantar SAPs. A comparison with other SAPs and mixed nerve action potentials (NAP) is shown in Table 4b.

Because the medial plantar SAP was absent in three

Table 1 *Medial plantar sensory nerve action potential in 69 control subjects*

		Amplitude (μV)			*Latency to onset (ms)*			*Latency to peak (ms)*			*SCV max. (m/s)*		
Age group (yr)	*Number of nerves*	*Mean*	*SD*	*Range*	*Mean*	*SD*	*Range*	*Mean*	*SD*	*Range*	*Mean*	*SD*	*Range*
10–19	8	3.4	2.3	1.3–8.1	5.0	0.9	3.9–6.2	5.9	0.9	4.5–7.2	36.1	6.8	33–50
20–29	11	3.0	1.1	1.7–5.5	4.7	0.5	4.1–5.5	5.5	0.6	4.4–6.5	34.9	2.6	32–39
30–39	10	3.1	1.1	1.6–5.7	4.5	0.7	3.6–5.1	5.3	0.7	4.2–6.5	38.6	5.1	31–49
40–49	11	2.0	0.6	1.1–3.3	5.0	0.8	3.7–6.0	6.0	1.1	4.6–8.4	33.9	5.3	28–42
50–59	13	1.7	0.6	1.0–2.8	5.1	0.7	4.2–6.0	6.1	0.7	5.0–7.1	33.5	3.8	28–42
60–69*	10	1.5	0.8	0.0–3.2	4.7	0.8	3.8–6.5	5.6	0.8	4.7–7.6	36.6	7.9	23–50
Over 70†	6	0.6	0.5	0.0–1.2	4.8	0.5	4.0–5.1	5.9	0.1	5.7–6.1	38.5	7.8	30–49
Total	69	2.3	1.4	0.0–8.1	4.8	0.7	3.7–6.5	5.7	1.1	4.2–7.6	35.6	5.6	28–50

*One SAP absent, †Two SAPs absent } excluded for purpose of calculating velocity and latency, but not mean amplitude.
SCV = sensory nerve conduction velocity.

Table 2 *Sural sensory nerve action potential in 69 control subjects*

		Amplitude (μV)			*Latency to onset (ms)*			*Latency to peak (ms)*			*SCV max. (m/s)*		
Age group (yr)	*Number of nerves*	*Mean*	*SD*	*Range*	*Mean*	*SD*	*Range*	*Mean*	*SD*	*Range*	*Mean*	*SD*	*Range*
10–19	8	21.9	8.5	10.7–35.0	2.9	0.4	2.6–3.7	3.7	0.4	3.1–4.7	45.1	3.9	37–48
20–29	11	16.0	3.8	9.2–20.0	3.0	0.3	2.4–3.5	3.7	0.3	3.1–4.0	46.8	5.1	40–58
30–39	10	23.0	9.2	9.2–38.2	3.0	0.4	2.6–3.3	3.6	0.4	2.9–4.5	48.3	6.9	42–61
40–49	11	15.9	5.6	6.0–26.3	3.0	0.4	2.4–3.6	3.8	0.5	3.1–4.6	46.2	6.7	38–56
50–59	13	14.3	8.8	6.0–41.4	3.2	0.4	2.4–3.8	4.0	0.4	3.1–4.7	44.5	5.5	37–58
60–69	10	9.4	3.6	4.2–12.0	3.3	0.6	2.3–4.0	4.1	0.5	3.5–4.9	45.1	7.3	35–58
Over 70	6	7.1	6.2	2.2–17.8	3.2	0.9	2.5–5.0	4.1	0.7	3.6–5.7	45.5	9.7	28–56
Total	69	15.6	8.3	2.2–41.4	3.1	0.5	2.3–5.0	3.9	0.5	2.9–5.7	45.8	6.3	28–61

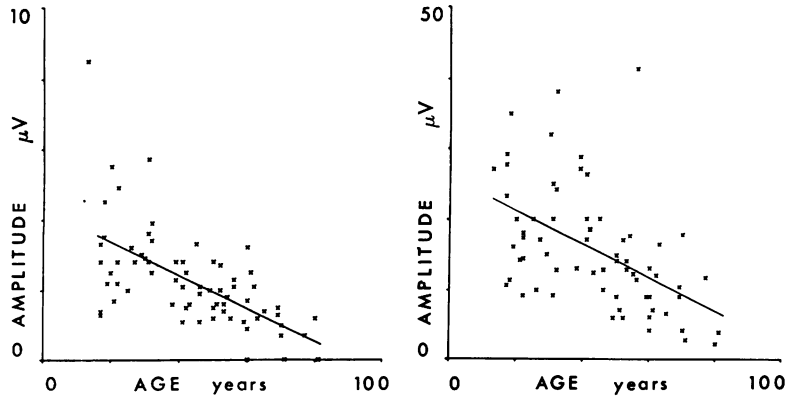

Fig. 3 *Regression curves of SAP amplitude on age in 69 controls.*
Left Medial plantar SAP: r=0.63 (P<0.001), y=4.32−0.047×age.
Right Sural SAP: r=0.51 (P<0.001), y=25.8−0.238× age.

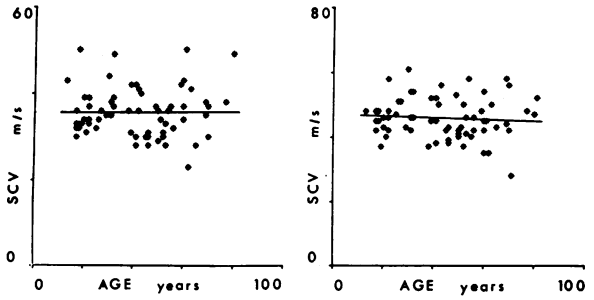

Fig. 4 *Regression curves of maximal sensory conduction velocity on age.*
Left Medial plantar SAP: n=66, r=0.00 (not significant).
Right Sural SAP: n=69, r=−0.08 (not significant).

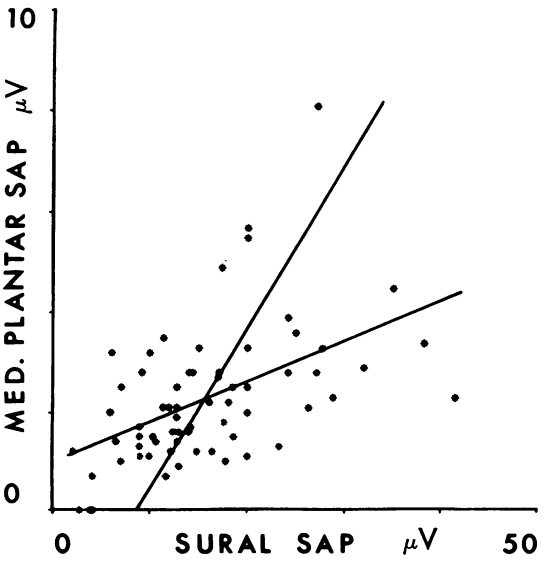

Fig. 5 *Regression curves of medial plantar SAP amplitude on sural SAP amplitude and vice versa in 69 controls. r=0.50 (P<0.001). x=8.68+3.07 y; y=0.99+ 0.08 x.*

subjects aged 60 years or more in the control series, the seven patients with peripheral neuropathy who were under 60 years of age are now considered (SW, SD, MP, VC, VE, KG, MF). The medial plantar SAP was the only SAP absent in all of them. The sural SAP was absent in two, small in two, borderline in one (KG) and normal in two (SD, VC). In the last three patients the lateral popliteal NAP was of normal amplitude. The median SAP was absent in two, small in two and normal in three of the seven cases. The ulnar SAP was absent in four, small in one, borderline in one and not done in one patient (Table 4a). Thus the medial plantar SAP in this small group was a better indicator than median, ulnar and sural SAPs and lateral popliteal NAP of involvement of the peripheral nerves in patients with a diagnosis of peripheral neuropathy made on clinical or electrophysiological grounds.

Preganglionic L5–S1 root lesions

The three patients had normal sural and medial plantar SAPs (Table 5).

Mrs AB (A7643) had weakness in the muscles supplied by L5 and S1 nerve roots, and sensory impairment within similar root distribution in the

Table 3 *Main clinical features in 13 patients with a diagnosis of peripheral neuropathy*

Patient	*Age*	*Sex*	*Wasting*	*Weakness*	*Reflexes*	*Sensory impairment*	*Others*	*Diagnosis*
1 SW GOS403266	12	M	No	No	Depressed or absent in legs	Distally lower limbs	Acanthocytes Abnormal blood lipids	Abetalipoproteinaemia
2 SD A91252	14	M	No	Bilat LMN VII, Palate, neck, upper and lower limbs	All absent	Glove and stocking	Extensor plantars CSF protein 1.42 g/l, cells 15/mm³ Good recovery	Myeloradiculo neuropathy Guillain-Barré
3 MP MV7449	27	F	Hands, feet	Distally upper and lower limbs	All absent	Glove and stocking	Biopsy sural nerve, pes cavus, kyphoscoliosis	Familial hypertrophic polyneuropathy
4 VC MV83967	32	F	No	Distally upper and lower limbs	Depressed supinator and absent ankle jerks	Distally hands and feet	Good recovery	Guillain-Barré
5 VE A86520	48	F	Distally upper and lower limbs	Distally all limbs severe	All absent	Glove and stocking		Peroneal muscular atrophy
6 KG A87673	48	M	Distally legs and feet	No	Absent ankle jerks	No	Pes cavus Carcinoma lung	? Peroneal muscular atrophy
7 MF A73600	48	F	No	Proximal lower limbs	Depressed knee and absent ankle jerks	Distally, feet		Aetiology obscure
8 CC A83998	60	M	No	Distally lower limbs	Depressed lower limbs	Glove and stocking		Diabetes mellitus
9 AP A87177	61	M	Lower limbs	Distal upper and lower limbs	Absent lower limbs	No		Diabetes mellitus
10 DK 48900	64	M	No	No	Depressed ankle jerks	Vibration sense toes		Diabetes mellitus, subclinical peripheral neuropathy
11 IW A87746	66	M	No	No	Absent lower limbs			Diabetes mellitus
12 GW A70802	70	M	No	Bilat external ophthalmoplegia Proximal all limbs	All absent	Minimal distally hands, feet (PS, 2 point discrimination)	CSF protein 1.2 g/l, cells 2/mm³ Good recovery	Miller Fisher syndrome
13 ER MV75776	83	M	No	No	Absent supinator and ankle jerks	Feet (all modalities)		Diabetes mellitus

PS = position sense, LMN = lower motoneurone paresis.

Table 4a *Electromyographic and nerve conduction data in 13 patients with a diagnosis of peripheral neuropathy*

	EMG	*Motor conduction*				*Sensory action potentials*									*Mixed nerve action potentials*		
						Upper limbs				*Sural*				*Med plantar*			
	Denerv	*Nerve*	*DML*	*MCV*	*MAP*	*Nerve*	*Ampl*	*Onset*	*Peak*	*Ampl*	*Onset*	*Peak*	*SCVmax*		*Nerve*	*Ampl*	*Velocity to onset*
SW		Lat popl	2.7	54	4.6	Median	7.0	1.9	2.9	5.0	2.6	3.4	60	Absent	ND	ND	ND
						Ulnar	3.3	1.4	2.2								
SD		Lat popl	13	31		Median	10.7	2.1	2.7	19.3	2.6	3.2	53	Absent	Lat popl	5.0	57
		Ulnar	6.9	55	1.6	Ulnar	Absent										
MP	Yes	Median	9.0	14		Median	Absent			Absent				Absent	ND	ND	ND
						Ulnar	Absent										
VC		Lat popl	5.9	50	2.0	Median	11.0	2.6	3.3	13.6	3.1	4.5	45	Absent	Lat popl	2.1	47
VE	Yes	Femoral	11.3			Median	Absent			Absent				Absent	Median	Absent	
						Ulnar	Absent										
KG		Lat popl	5.6	41	1.6	Median	13.0	2.4	3.1	5.0	3.0	3.8	46	Absent	Lat popl	2.6	45
						Ulnar	6.0	2.4	3.1								
MF	No	Lat popl	5.2	29	3.5	Median	7.0	2.8	3.5	2.3	3.7	4.5	37	Absent	ND	ND	ND
						Ulnar	Absent										
CC	No	Lat popl	4.2	38	1.5	Median	3.0	2.6	3.0	4.2	3.9	4.6	36	Absent	Lat popl	Absent	
						Ulnar	3.0	2.5	3.0								
AP	Yes	Lat popl	5.2		0.14	Median	3.0	3.0	3.9	4.0	6.0	7.7	23	Absent	Lat popl	Absent	
		Median	3.7	52													
DK		R. Lat popl	7.1		0.2	Median	4.0	3.3	4.0	Absent				Absent	ND	ND	ND
		L. Lat popl	4.7	40	1.9	Ulnar	2.9	2.8	3.4								
IW		Lat popl	4.9	36	3.0	Median	7.0	3.2	3.9	7.0	3.9	4.8	35	Absent	Lat popl	1.4	34
GW	No	Lat popl	6.0	40	3.0	Median	Absent			Absent				Absent	Lat popl	Absent	
						Ulnar	Absent										
ER		Lat popl	11.5	37	0.2	Median	15.0	2.8	3.5	2.0	7.4	9.0	18	Absent	ND	ND	ND
Normal		Median	2.9–5.0	51–67		Median	9–45	1.7–3.2	2.5–4.0	See Table 2				See Table 1	Median	> 20	
values		Ulnar	2.9 ± 0.39	49–66	> 4.3	Ulnar	7–28	1.4–2.6	2.2–3.4								
		Lat popl	3.4–6.8	36–64	> 2.20										Lat popl	2.0–15.5	53 ± 3.85
		Femoral	3.7 ± 0.45														

EMG electromyography; DML distal motor latency (ms); MCV motor nerve conduction velocity (m/s); MAP compound muscle action potential (mV) with distal stimulation; Ampl amplitude in μV; Latency to onset and peak in ms; SCV max sensory nerve conduction measured to onset in m/s.; Lat Popl (lateral popliteal nerve) surface electrode on extensor digitorum brevis (motor) or surface or needle electrodes at the head of the fibula (NAP); median nerve surface or needle electrodes in abductor pollicis brevis (motor) and surface electrode at wrist (SAP) or elbow (NAP); ulnar nerve surface electrode on abductor digiti minimi (motor) or wrist (SAP). For median and ulnar SAPs stimulation was given to fingers II and V respectively. Normal values: either the range or mean ± one standard deviation are quoted. ND not done. Denerv = denervation.

Table 4b *Comparison of upper limb SAPs, lower limb SAPs, and NAPs in 13 cases of peripheral neuropathy*

	Median SAP	*Ulnar SAP*	*Sural SAP*	*Medial plantar SAP*	*NAP*	
					Lat. popliteal	*Median*
Absent	3	5	4	13	3	1
Small*	6	3	2	0	1	0
Borderline†	0	1	4	0	0	0
Normal	4	0	3	0	3	0
Not done	0	4	0	0	6	12
Totals	13	13	13	13	13	13

*Small lateral popliteal NAP: less than 2.0 μV
†Borderline SAP: up to 1 μV less than the lower range of normal

Table 5 *Medial plantar and sural sensory nerve action potentials in 33* patients*

Diagnosis	*Patient*	*Age (yr)*	*Sex*	*Medial Plantar SAP†*		*Sural SAP†*		*Remarks*
				Amplitude (μV)	*SCV max (m/s)*	*Amplitude (μV)*	*SCV max. (m/s)*	
Peripheral neuropathy	1 to 13	See Table 3		See Table 4		See Table 4		See Table 3
Lateral popliteal	14 AJ	13	M	3.0	37	18.7	56	Pressure palsy
nerve palsy	15 DL	18	M	2.8	50	11.4	40	Trauma
	16 LWB	22	F	2.2	34	20.0	40	Pressure palsy
	17 GS	45	M	2.4	32	24.0	41	? Pressure palsy
	18 MB	54	M	3.0	31	6.0	41	Aetiology obscure
	19 HG	60	M	1.7	34	9.0	42	Aetiology obscure
Pre-ganglionic	20 AB	44	F	3.2	33	19.1	51	L5–S1 disc prolapse
L5–S1 root lesion	21 BW	32	F	2.9	41	12.8	48	L4–5, L5–S1 disc prolapses Arachnoiditis
	22 JB	50	F	1.2	33	28.0	38	? L4–5 disc prolapse
Sciatic nerve lesion	23 MC	29	M	Absent		Absent		Fractured femur
Posterior tibial nerve	24 PH	36	F	Absent		Absent		Fractured tibia and fibula Right sural nerve lesion also
lesion	25 FR	47	F	Absent		31.0	63	Collagen disease
Medial plantar nerve lesion	26 SAK	35	M	Absent		31.4	35	Tuberculoid leprosy
Trauma ankle	27 RW	50	F	Absent		14.0	43	Medial plantar on opposite side 1.5 μV
	28 KW	50	F	1.3	36	7.8	33	Fracture
	29 GC	66	M	Absent		12.8	50	Fractured tibia and fibula on right
Hallux valgus	30 DC‡	63	F	0.7/2.1	22/26	8.5/15.7	38/38	Bilateral
	31 AB	63	F	Absent		9.2	52	
	32 GC	66	M	Absent		17.0	50	Same patient as 29 but left side
Psychiatric	33 JF	39	F	1.3	42	30.3	52	Compensation
	34 MC	52	F	4.0	28	32.0	47	Compensation

*Patient GC appears twice (cases 29 and 32)
†Values given correspond to affected limb only except for case 30
‡Values for right and left side respectively are given

right leg and foot. Both ankle jerks were present. A large lateral disc protrusion at L5–S1 on the right was confirmed at operation on 30 April 1976 (Professor V. Logue) which revealed that the right S1 root ran over the middle of the protruded disc.

Mrs BW (A89168) had been operated on in 1967 for a protruded L5–S1 disc. A myelogram in February 1976 showed a prolapsed L4/5 disc and signs of arachnoiditis. Examination in July 1976 showed wasting and weakness in the lower limbs with bilateral foot drop, absent knee and ankle jerks and sensory impairment up to L1 dermatome, more dense distally in the feet. At operation both L5 roots were described as 'very tight', the bulging L4/5 disc was removed, and decompression of the L5 and S1 roots was carried out to the foramina (Mr L. Symon). The electrical findings are summarised in Fig. 6.

Mrs JB (MV 84092) developed pain in the left leg and a left foot drop, with weakness of dorsiflexion, inversion and eversion of the foot, but no reflex or sensory abnormality. Electromyography of the left tibialis anterior showed a reduced interference pattern with normal units firing at high rates and no fibrillation. The maximum motor nerve conduction velocity (MCV) of the left lateral popliteal nerve was normal and there was no significant decrement in the size or

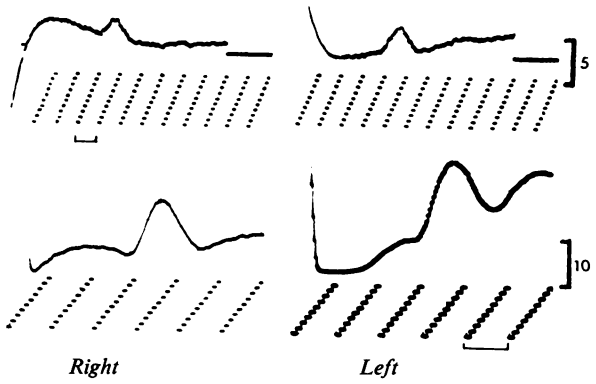

Fig. 6 *Pre-ganglionic L5 and S1 bilateral root lesions (see results). Mrs BW 32 years. Upper traces show normal medial plantar SAPs and lower two traces normal sural SAPs. Calibration vertical lines 5 μV (for upper traces) and 10 μV (for lower traces), staircase 1 ms. EMG showed denervation in right vastus medialis, both tibialis anterior, and both medial gastrocnemius muscles. Distal latency to tibialis anterior was normal.*

dispersion of the evoked muscle action potential of extensor digitorum brevis with stimulation above the fibula. Both lateral popliteal NAPs were normal in amplitude and in velocity to onset and peak. Radiographs showed scoliosis concave to the right in the lower lumbar spine.

Lateral popliteal nerve palsy

There were six patients in this group, all presenting with unilateral foot drop. Three of them were initially suspected of having an L5 root lesion. All had normal medial plantar SAPs and met one or more criteria of lateral popliteal nerve palsy—that is, abnormal NAP, decrement of the compound muscle action potential in the region of the head of the fibula, slowing of motor conduction velocity. Four had EMG evidence of denervation in the appropriate muscles. Additional evidence for the diagnosis came from follow-up in two of the patients (MB, LWB). One of these also had a negative myelogram (MB). Another (DL) (see Fig. 7) at surgical exploration (Mr D. Brooks) was found to have traumatic damage to the nerve without visible division.

Sciatic nerve lesion

MC (case 23, Table 5) had wasting of the right thigh, leg, and foot, weakness of right hamstrings, thigh adductors, and leg and foot muscles and an absent right ankle jerk. The sensory loss and main electrical findings are shown in Fig. 8.

Posterior tibial nerve lesions

PH (A86218, case 24, Table 5) sustained a compound fracture of the tibia and fibula on the right. She had

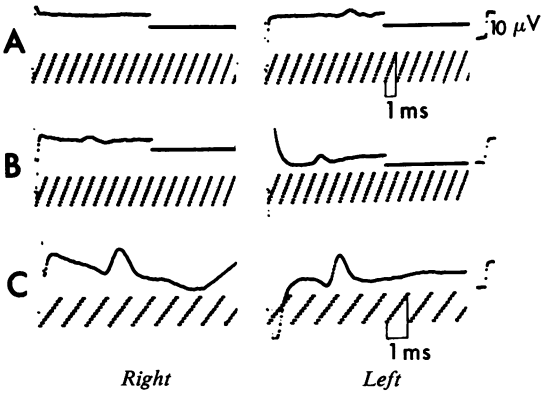

Fig. 7 *Traumatic right lateral popliteal nerve palsy. Mr DL (RNOH 257225) 18 years. (A) Absent lateral popliteal NAP on the right. (B) Normal medial plantar SAPs on both sides. (C) Normal sural SAPs on both sides. Calibration 10 μV. Staircases 1 ms. Note that the magnitudes of the left lateral popliteal NAP and both medial plantar SAPs are similar.*

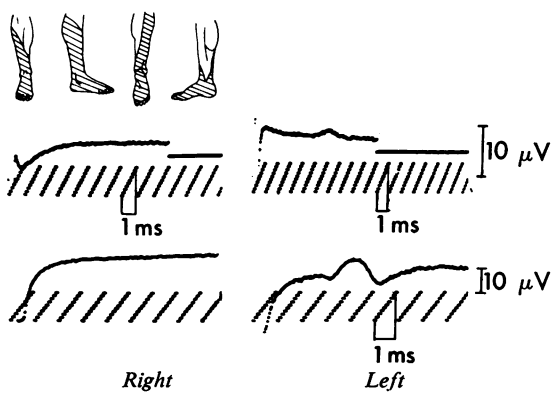

Fig. 8 *Traumatic right sciatic nerve lesion. Mr MC 29 years. Area of complete sensory loss is shown in inset. Upper traces: absent right medial plantar SAP. Lower traces: absent right sural SAP. Calibration 10 μV. Staircases 1 ms. EMG showed complete denervation in right tibialis anterior and medial gastrocnemius. Right lateral popliteal nerve was unexcitable.*

orthopaedic surgery and was left with sensory loss and wasting of the foot (Fig. 9). Power of flexor hallucis longus and gastrocnemius was normal. The main nerve conduction findings are shown in Fig. 9.

Tarsal tunnel syndrome FR (MV 38211, case 25, Table 5) had a diagnosis of mononeuritis multiplex and collagen disease supported by skin, nerve, and muscle biopsies (Dr R. Barnard) in 1971. She had had a painful left ulnar palsy in 1956, bilateral lateral popliteal and right ulnar nerve palsies with erythematous patches in both legs in 1967, and from 1967 to 1971, and again in 1974, small painful ulcers in the toes bilaterally during the winter. She had recovered well from all of these episodes. Repeated ESR and LE cells studies and estimation of cryoglobulins were normal. Cold agglutinins titre in 1971 was 1/8. In November 1975 she developed severe pain radiating from the right heel to the sole, initially worse over the medial side of the big toe. By March 1976 it was present only after prolonged walking. She was left with diminished sensation to pinprick, temperature, and light touch in the sole, particularly in the plantar surface of the toes. There was no obvious wasting of the small muscles of the right foot, but adduction and abduction of the toes were better performed on the left. Power of flexor hallucis longus and gastrocnemius was normal (March 1977). The main electrical findings are shown in Table 6.

Medial plantar nerve lesion

This Indian patient (MV 83670, case 26, Table 5) presented with a one year history of numbness of the right big toe extending to the medial side of the foot and sole. He had areas of sensory loss in all modalities (Fig. 10), those in the right leg and left buttock being associated with chronic granulomatous skin lesions, and clinical thickening of the right radial, right and left ulnar, and right superficial peroneal nerves. A diagnosis of tuberculoid leprosy was confirmed at the Hospital for Tropical Diseases. Figure 10 shows the main electrical findings. The sensory loss on the dorsum and outer side of the big toe was interpreted as due to involvement of distal sensory fibres of the

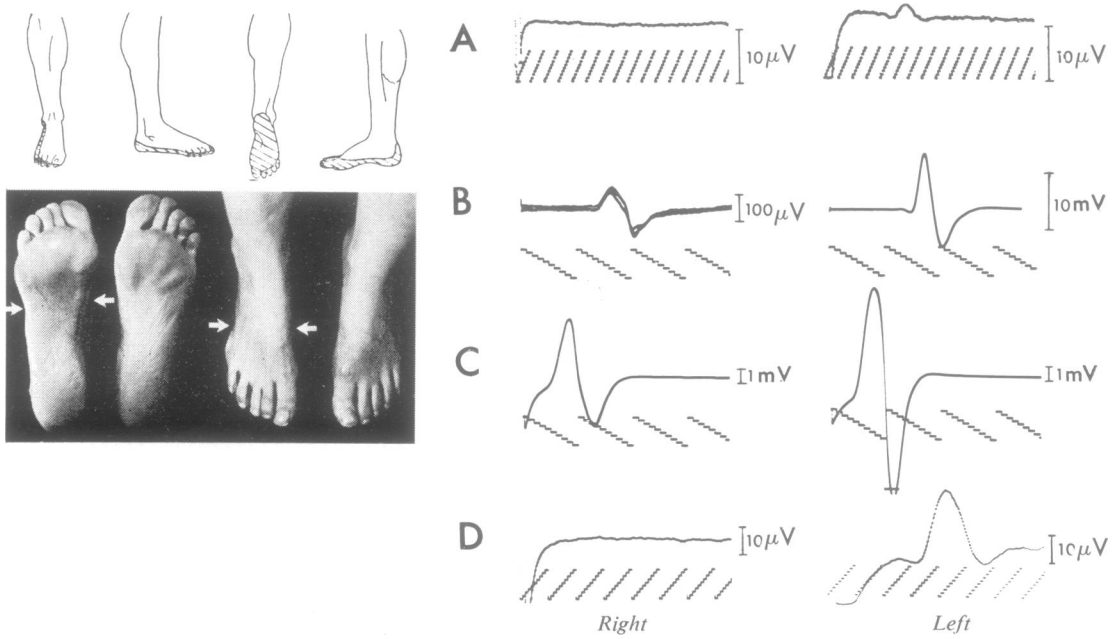

Fig. 9 *Traumatic right posterior tibial and sural nerve lesions. Mrs PH 36 years. Area of sensory loss and wasting of right abductor hallucis and abductor digiti quinti in right foot (arrows) are shown on left. Note on the affected side: (A) absent medial plantar SAP; (B) very small MAP of abductor hallucis (stimulation at popliteal fossa)—note different calibration on right and left; (D) absent sural SAP; normal MAPs of both extensor digitorum brevis muscles are shown in C. Time scales: staircases 1 ms (A,D) and 10 ms (B,C). Temperature in both feet 29–30°C. MCV of both lateral popliteal and both posterior tibial nerves, DML to both abductor hallucis muscles (7.2 and 6.1 ms) and both superficial peroneal SAPs (1.4 and 1.6 μV) were all normal. EMG showed denervation in right abductor hallucis and abductor digiti minimi (but not in tibialis anterior nor gastrocnemius).*

Table 6 *Nerve conduction findings in a case of tarsal tunnel syndrome (female, 46 years, collagen disease)*

Test	*Right*	*Left*	*Normal*	
Medial plantar SAP	Absent	1.2 μV	1.1–3.3	(This series)*
DML to abductor hallucis	6.6 ms	4.0 ms	7.3±1.7	(Mayer, 1963)†
			4.4±0.9	(Goodgold *et al*, 1965)‡
			5.3±0.82	(Johnson and Ortiz, 1966)
DML to abductor digiti quinti	5.6 ms	5.3 ms	4.7±1.0	(Goodgold *et al*, 1965)‡
			4.0–7.5	(Kaeser, 1965)
			5.8±0.84	(Johnson and Ortiz, 1966)
MAP (ankle-abductor hallucis)	1.4 mV	5.3 mV		
MAP (ankle-abductor digiti quinti)	0.7 mV	2.1 mV	2.0–9.2	(Kaeser, 1965)
Posterior tibial nerve MCV (knee-ankle)	44 m/s	39 m/s	36–58	(Kaeser, 1965)

Abbreviations see Table 4a.
T° medial side of the feet 32°C.
* Aged 40–49 years †Aged 36–50 years ‡Aged 19–40 years.
EMG of right abductor hallucis showed fibrillation at two sites. Right sural median and ulnar SAPs and right ulnar and lateral popliteal MCVs were normal.

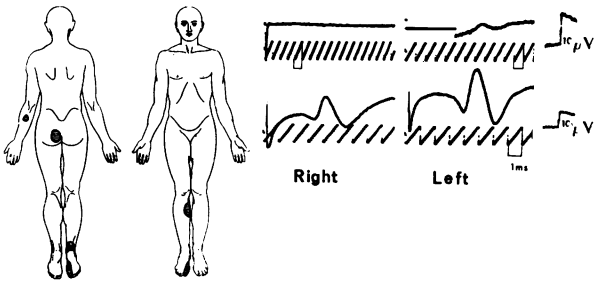

Fig. 10 *Right medial plantar nerve lesion. Leprosy. Mr SAK 35 years. Areas of sensory loss (pain, touch, temperature) are shown on left (see text). Note sensory loss in right foot. Upper traces show an absent right and normal left medial plantar SAP and lower traces normal sural SAPs. Calibration 10 μV. Staircases 1 ms. Distal motor latency to right abductor hallucis was slightly prolonged (8.0 ms). The following were normal: motor nerve conduction velocity of right ulnar, right lateral popliteal, and right posterior tibial nerves, compound muscle action potentials of right extensor digitorum brevis and right abductor hallucis, right median and ulnar SAPs.*

musculocutaneous (superficial peroneal) nerve and the medial terminal branch of the anterior tibial nerve (deep peroneal).

Trauma to the ankle (Table 5)
None of the three patients had clinical evidence of a posterior tibial nerve lesion.

Hallux valgus (Table 5)
All three patients had clinically normal sensation in the big toe.

Psychiatric
Both cases had compensation pending (Table 5).

JF (A80242, case 33) had chronic pain in the back and left leg after a road traffic accident, limitation of straight leg raising, and sensory impairment in the right leg and foot. There was no weakness or abnormality in reflexes and radiographs of the spine; myelography and a spinal venogram did not show significant abnormality. She had improved temporarily on antidepressant drugs.

MC (A87195, case 34) had a fall at work with bruising of the right ankle and six months later complained of pain and numbness in the right ankle and foot. She had a bizarre area of decreased sensation in the right ankle and medial side of the foot resembling the area previously bandaged. Motor conduction in both posterior tibial nerves was normal.

Discussion

The medial plantar SAP was recorded at the ankle with surface electrodes, using averaging techniques, in subjects under 60 years of age and was a useful diagnostic test in lesions of the appropriate nerves. It was a more sensitive indicator than other SAPs of peripheral neuropathy and was also useful in the differential diagnosis of foot drop.

It was possible to measure the medial plantar SAP in all of the 52 control subjects under 60 years of age. In three subjects aged 60 years or over in whom it was not measurable, the sural SAP (calf–ankle) amplitude

was below the lower limit of normal of 8 μV and 6 μV in the two other series reported with a similar antidromic technique (Di Benedetto, 1970; Burke *et al.*, 1974).

Two practical disadvantages of recording the medial plantar SAP at the ankle have been put forward. First, its small size which makes the use of averaging techniques mandatory (Di Benedetto, 1970) and, second, that it is a time-consuming method requiring averaging of up to 250 stimuli (Mavor and Atcheson, 1966) or 500 stimuli (Behse and Buchthal, 1971). However, most standard EMG equipment includes an averaging device, and with the averaging technique described here, recording time was from around one to three minutes when two or three 'runs' were used to confirm the reproducibility of the results with small potentials (around 1.0 μV). The single most important factor in achieving a satisfactory record was careful positioning of the surface electrode at the point where the posterior tibial pulse could be palpated.

There is no strictly comparable series of controls in the literature but the present data have been arranged to fit the age groups reported in the only two previous studies with amplitude values for the medial plantar SAP at the ankle (Tables 7 and 8). The close correspondence between our means and ranges and those described in these two previous reports is striking. The similarity of results using needle and surface electrodes is interesting as the latter are usually less time-consuming. A comparative summary of previous reports on sensory conduction in this nerve is given in Table 9.

The sural SAP amplitude was, in general, much greater than the medial plantar SAP, but potentials of less than 6 μV were seen in five of our 69 control subjects (7.2%), all of whom were over 60 years old. A statistically significant correlation between ampli-

Table 7 *Medial plantar SAP*

	Age (yr)	*Number*	*Mean amplitude (μV)*	*Range*	*SD*	*Absent*
Mavor and Atcheson (1966)	15–35	12	3.8	?	?	0
This series (1977)	15–35	25	3.1	1.4–5.7	1.1	0

Table 8 *Medial plantar SAP*

	Age (yr)	*Number*	*Mean amplitude (μV)*	*Range*	*SD*	*Absent*
Behse and Buchthal (1971)	A. 15–30	23	3.6	?	2.8	?
This series (1977)		19	2.9	1.4–5.5	1.1	0
Behse and Buchthal (1971)	B. 40–65	10	1.6	?	1.8	?
This series (1977)		32	1.8	0–3.2	0.7	1

Table 9 *Sensory conduction in medial plantar nerve (big toe—ankle)*

Author	*Electrodes*	*Averaging techniques*	*Controls*					*Pathological series (number)*
			Number	*Age (yr)*	*Amplitude (μV)*	*SCV max. (m/s)*	*Absent (number)*	
Mayer (1963)	Surface	No	64 subjects	10–80	No consistent responses			No
Mawdsley and Mayer (1965)	Surface	No	105 subjects	20–70	Rarely recorded			Yes (76)
Mavor and Atcheson (1966)	Surface	×20–250	12 subjects	15–35	1.4–5.8 (mean 3.8)	38.4 SD 4.3	0	No
Lovelace *et al.* (1969)	Surface	×up to 400	?	23–42	?	?	0	Yes (15)
Behse and Buchthal (1971)	Needles	×20–500	33 nerves	15–30 40–65	3.6 SD 2.6 1.6 SD 1.8	46.1 SD 3.5 37.0 SD 3.8	?	No
Present series (1977)	Surface	×32	69 subjects	13–81	0–8.1 (Mean 2.3 SD 1.4)	28–61 (Mean 36 SD 5.6)	3 aged over 60 yr	Yes (33)

tude and age was found (Fig. 3) and has been reported before for sural SAP (Burke *et al.*, 1974) and medial plantar SAP (Buchthal *et al.*, 1975). However, in spite of this correlation, the possible hazard of using regression curves as a means of predicting normal values, without due regard to the range of values, is stressed. For example, in our series the greatest amplitude for the sural SAP was seen in the sixth decade (41.4 μV). The smallest medial plantar SAPs in the second and third decades (1.3 μV and 1.7 μV) were smaller than the largest potentials in the sixth or even seventh decades (2.8 μV and 3.2 μV). The wide range makes prediction of amplitude from age of no practical value in the individual case.

There are few studies on the histology of the digital nerves of the foot in healthy subjects. The results of Garven *et al.* (1962) cannot be taken too far as they counted the number and size of myelinated fibres only in selected fascicles. However, in their three control subjects aged 3, 28, and 70 years the findings in the sciatic nerve were comparable, but there was the smallest number of myelinated fibres and an absence of fibres over 8.0 μm in diameter in the medial digital nerve of the big toe in the subject aged 28 years. These findings are consistent with the variation in amplitude of the medial plantar SAP previously mentioned.

We did not find a significant relationship between maximal sensory conduction velocity and age for both the sural and medial plantar nerves (Fig. 4). This is in agreement with Burke *et al.* (1974) for the sural SAP but not with the results of Behse and Buchthal (1971). The difference may lie in the fact that only the last authors constantly monitored the surface temperature of the leg to make certain it was within the range of 36–37°C during the recordings.

Previous studies (Di Benedetto, 1972; Burke *et al.*, 1974) have suggested that measurement of the sural SAP is a more sensitive test than of the median SAP for the diagnosis of peripheral neuropathy, but neither the clinical nor electrical criteria used for accepting such a diagnosis were given.

This study is based mainly on an analysis of amplitude of the SAPs recorded by surface electrodes. The method allows potentials of not less than 0.7 μV to be discriminated and reliably recorded. It would have been possible to increase the number of sweeps averaged and thus increase significantly the resolution. However, it is not practical to prolong recordings for service work. Furthermore, it is not certain that doing so would increase the diagnostic yield of the procedure. For example, in a detailed study of the median SAP in 84 cases of polyneuropathy, Buchthal and Rosenfalck (1971) reported no cases in which abnormalities of sensory conduction velocity were not associated with an abnormal amplitude. From their results it is clear that amplitude of the SAP was a more sensitive measurement than abnormalities of the conduction velocity of its components. Seventy-nine of the 84 patients had an abnormal amplitude, and only 64 had abnormalities of sensory conduction velocity of components of the median SAP.

The amplitude of SAPs can be within the 'normal range' in some cases of peripheral neuropathy (Buchthal and Rosenfalck, 1971). But the range of normality is wide, and follow-up for as long as 10 months may be necessary in an individual case to show retrospectively that the amplitude of a SAP recorded at the onset of the disease is small for that individual, although still within the 'normal range' (Guiloff, 1977).

In our 13 cases of peripheral neuropathy the medial plantar SAP was a more sensitive indicator than the median or sural SAP. The sural SAP was not a more sensitive indicator than the median SAP. Gilliatt and Willison (1962) found that the lateral popliteal NAP (Gilliatt *et al.*, 1961) was a more sensitive indicator than upper limb SAPs in patients with diabetic peripheral neuropathy affecting the lower limbs. In this series the medial plantar SAP was also a more sensitive indicator than the lateral popliteal NAP.

The fact that the medial plantar SAP may be absent when the median and sural SAPs are present is probably largely related to the resolution of the technique used. Since the medial plantar SAP is a smaller potential, a fall in the number of nerve fibres (or slowing of conduction in them) similar to that in a nerve with a larger potential could be reflected in an unmeasurable SAP, while in a larger nerve it would still be measurable. Alternatively, the pathological process in peripheral neuropathy might affect the longer fibres in the lower limbs earlier or more severely or both, or the pathology might be generalised but of irregular distribution, so that some nerves may be electrically affected and others not, as has been described for acute idiopathic polyneuritis (McLeod *et al.*, 1976). On the present data it is not possible to decide. For example, SD (case 2, Table 3) had a fall of 38% in the amplitude of the sural SAP within 24 days and the medial plantar one was absent on both occasions. This late fall could be related to each of these possibilities.

Proximal post-ganglionic lesions can be associated with an abnormal SAP in the corresponding distribution—for example, cervical rib syndrome with C8–T1 nerve root compression may result in an absent ulnar SAP (Gilliatt *et al.*, 1970). Post-ganglionic L4–5 root lesions are not frequent and were not seen in this series but an abnormal medial plantar SAP would be expected under these circumstances. However, patients with unilateral foot drop are often

referred to departments of applied neurophysiology. Post-ganglionic L4–5 root and plexus lesions should be excluded electrically, and the medial plantar SAP can be a convenient screening test in this situation. Eight cases presented with unilateral foot drop in this series, six had a lateral popliteal nerve palsy and two pre-ganglionic L5–S1 root lesions. All had normal medial plantar SAPs.

The medial plantar SAP was absent in the four cases of sciatic, posterior tibial, and medial plantar nerve lesions. In the patient with a sciatic nerve lesion the sural SAP was absent as well. Posterior tibial nerve lesions would not necessarily be expected to be associated with an abnormal sural SAP, as the sural nerve receives a contributory branch from the lateral popliteal nerve. The absence of the sural SAP in PH (case 24, Table 5) could be related to damage to the sural nerve or to its two contributory branches sustained either at the time of trauma or subsequent operation.

An important application of this technique is as an aid in diagnosing distal lesions of the posterior tibial and lesions of the medial plantar nerves. The latter is exemplified by the patient who had leprosy (Fig. 10). The posterior tibial nerve may be entrapped at the flexor retinaculum (Kopell and Thompson, 1960; Goodgold *et al.*, 1965), the so-called tarsal tunnel syndrome (Keck, 1962; Lam, 1962). FR (Table 6) displayed the features of this syndrome and had an absent medial plantar SAP on the affected side. The finding of prolonged distal motor latency to abductor hallucis and abductor digiti quinti has been helpful in diagnosing this condition but both may be normal (Goodgold *et al.*, 1965; Johnson and Ortiz, 1966; Edwards *et al.*, 1969). In our patient these measurements were within the normal range but distal latency to abductor hallucis was comparatively longer on the affected side. In the carpal tunnel syndrome sensory conduction in the median nerve may be affected when motor conduction remains normal (Goodman and Gilliatt, 1961). We suggest that the medial plantar SAP should be recorded in patients suspected of having the tarsal tunnel syndrome.

The technique helped to exclude a posterior tibial nerve lesion in two medico-legal cases with sensory loss in the foot.

Conclusion

The medial plantar sensory action potential (SAP) can be readily recorded with surface electrodes at the ankle using an averaging technique. It was unmeasurable ($< 0.7\ \mu V$) in only three out of 16 subjects aged 60 years or over in a group comprising 69 control subjects. It declined with increasing age as did the sural SAP and its amplitude correlated well with that of the latter in the same limb in the same subjects.

In 13 cases of peripheral neuropathy its measurement was a more sensitive diagnostic test than sural and median SAPs and lateral popliteal mixed nerve action potential. It may be useful in patients with foot drop, helping to show or exclude electrically involvement of L4–5 post-ganglionic sensory fibres at nerve root or plexus level. The cases of traumatic sciatic and posterior tibial nerve lesions, tarsal tunnel syndrome, and lepromatous medial plantar nerve lesion described exemplify its value in testing specifically the sensory fibres of these peripheral nerves.

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