Nerve Conduction Studies in Leprosy [1]

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Electrophysiological studies have been employed by several workers in the investigation of patients with leprosy ([1, 3, 6, 9, 11, 12, 13, 15, 16]). Dubois and Rademecker ([6]) reported chronaxie studies on the muscles of patients with leprosy, and electromyography was subsequently employed by others to study the extent of denervation in affected subjects ([1, 13]). In recent years, motor conduction ([9, 11, 12, 15, 16]) and sensory conduction studies ([3, 12, 15]) have demonstrated that marked slowing of conduction may occur in affected nerves, and that there may be significant involvement of nerves which appear normal clinically.

The present study was undertaken in order to evaluate electrophysiological technics in the diagnosis of leprosy in the Aboriginal population of the Northern Territory of Australia. Motor and sensory conduction studies have been performed on both clinically affected and clinically unaffected nerves of patients with leprosy, and also on a group of patients with clinically enlarged nerves in whom the diagnosis was initially in doubt.

MATERIALS AND METHODS

Case material. The subjects, Aborigines from the Northern Territory of Australia, were divided into three groups:

Group I. Thirty control subjects.

Group II. Thirty-six subjects with an established diagnosis of leprosy, matched for age with the control group. Twenty patients suffered from tuberculoid leprosy, ten from the dimorphous form, five from lepromatous leprosy, and one was indeterminate.

Group III. Twenty-seven subjects in whom one or more palpable nerves had been detected on routine examination, but in whom no definite diagnosis had been established. Electrophysiologic studies were performed in order to determine whether or not the clinically palpable nerve was abnormal.

Electrophysiologic studies. *Motor conduction.* The responses to supramaximal stimulation of the median, ulnar, lateral popliteal and posterior tibial nerves were recorded, using surface electrodes strapped over the abductor pollicis brevis, abductor digiti minimi, extensor digitorum brevis and abductor hallucis muscles respectively. Measurements of latency and of muscle action potential amplitude were made on photographic records. The median nerve was stimulated at the wrist, in the ante-cubital fossa and in the axilla; the ulnar nerve was stimulated at the wrist, 3-4 cm below the elbow, just above the medial epicondyle and in the axilla; the lateral popliteal nerve was stimulated at the ankle and at the neck of the fibula; and the posterior tibial nerve was stimulated at the ankle and in the popliteal fossa. Distances between sites of stimulation were measured on the skin and the conduction velocity in the fastest conducting fibers was then calculated for each nerve in the conventional manner. In the case of the ulnar nerve the velocities were calculated for the below-elbow to wrist segment (distal), above-elbow to wrist (elbow) and for the axilla to above-elbow segment (proximal).

Sensory action potentials. Sensory action potentials were recorded with surface electrodes from the median and ulnar nerves at the wrist on stimulating the index and little fingers respectively ([4]). The sensory action potential was recorded from the sural nerve with a pair of subcutaneous needle electrodes placed 4 cm apart in the mid-calf, at a distance of 12 cm proximal to the stimulating cathode situated between the Achilles tendon and the lateral malleolus ([17]). The sensory action potential was recorded from the radial nerve above the lateral epicondyle

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TABLE 1. *Nerve conduction studies in leprosy.*

Clinically Affected Nerves			
Nerve	Total number	Abnormal	%
Ulnar	23	18	78
Median	2	1	50
Lateral popliteal	19	17	89
Posterior tibial	13	11	85
Sural	13	12	92

([5]). Measurements of amplitude, and latency to peak of the responses were made from photographic records.

Mixed nerve action potentials. Mixed motor and sensory nerve action potentials were recorded from the median, ulnar, lateral popliteal and posterior tibial nerves. The median nerve was stimulated at the wrist, and also at a site 10 cm above the wrist, and the response was recorded with surface electrodes placed in the ante-cubital fossa. The ulnar nerve was stimulated at the wrist and above the elbow, and the responses were recorded with surface electrodes above the elbow and in the axilla, respectively. The lateral popliteal nerve was stimulated at the ankle, and the response recorded with needle electrodes positioned alongside the nerve at the neck of the fibula ([8]). In the case of the posterior tibial nerve the response was recorded at the popliteal fossa with surface electrodes following stimulation at the level of the medial malleolus, and also at a site 16 cm proximal to the medial malleolus.

The recording electrodes were connected to a Tektronix FM 122 preamplifier and displayed on the upper beam of a Tektronix 564 oscilloscope. Photographic records were made on Polaroid film. The stimulus was a square wave of 0.2 to 1.0 msec duration, and of up to 500 v amplitude derived from a Disa Ministim.

RESULTS

Group I. Control subjects. Nerve conduction studies were performed on 30 control subjects whose ages ranged from 14 to 50 years (mean 24; S.D. 11). The results are summarized in Tables 2-5 and 7-10. In the group of 30 patients, there were 4 median and 2 ulnar nerves in which the motor conduction studies were abnormal when compared with the control values for Caucasian subjects. These values were excluded from the control group since each fell more than three standard deviations from the mean calculated for the remaining values. The mixed nerve action potential could not be recorded from four lateral popliteal nerves and from three posterior tibial nerves. These values were retained in the group since zero amplitude fell within three standard deviations of the mean of each group.

Group II. Subjects with leprosy. Clinical and electrophysiological studies were performed on 36 subjects with leprosy whose ages ranged from 12 to 54 years (mean 29;

TABLE 2. *Results of motor and sensory conduction studies on control subjects, and on clinically affected nerves of patients with leprosy.*

Motor Conduction Studies										
		Ulnar					Median			
		Latency	Amplitude	Velocity			Latency	Amplitude	Velocity	
				Prox.	Elbow	Distal			Prox.	Distal
		(msec)	(mv)	(m/sec)	(m/sec)	(m/sec)	(msec)	(mv)	(m/sec)	(m/sec)
Control	Mean	2.6	7.5	64.5	59.5	65.0	3.7	6.9	66.0	61.0
	S.D.	0.34	2.2	5.8	4.9	7.2	0.4	2.6	6.7	7.1
	Range	1.9-3.3	3.8-12.5	52-79	52-79	52-88	2.4-4.3	1.6-11.6	52-78	46-69
	No.	30	30	29	30	29	28	30	27	30
Clinically affected	Mean	3.1	4.2	55.6	50.8	54.2	Responses recorded from only 2 nerves			
	S.D.	1.1	3.7	16.7	11.7	12.8				
	Range	2.0-6.0	0-11	22-77	20-64	12-68				
	No.	17	21	15	16	17				
p		0.05	0.10	0.05	0.01	0.01				

TABLE 3. *Results of motor and sensory conduction studies on control subjects, and on clinically affected nerves of patients with leprosy.*

		Motor Conduction Studies					
		Lateral Popliteal			Posterior Tibial		
		Latency (msec)	Amplitude (mv)	Velocity (m/sec)	Latency (msec)	Amplitude (mv)	Velocity (m/sec)
Control	Mean	4.5	4.9	49.0	5.6	4.9	50.0
	S.D.	0.6	2.5	6.0	1.2	2.5	8.9
	Range	2.7-5.6	1.2-9.9	35-59	2.8-8.8	3.6-20.0	34-63
	No.	30	30	30	30	30	18
Clinically affected	Mean	5.2	1.9	42.9	6.8	0.9	47.6
	S.D.	1.7	2.0	7.1	4.2	1.2	8.7
	Range	2.8-9.0	0-6.6	26-49	4.0-18.0	0-3.6	37-60
	No.	12	17	12	9	10	9
p		N/S	0.001	0.01	N/S	0.001	N/S

S.D. 12). The electrophysiologic studies have been considered in two groups: a) results of studies on clinically affected nerves, b) results of studies on nerves which were clinically normal.

Clinically affected nerves. The results of nerve conduction studies on clinically affected nerves in subjects with established leprosy have been compared with those of control subjects (Tables 1-5).

Ulnar nerve. In 18 of the 23 nerves (78%), one or more of the studies fell outside the control range. All five subjects in whom conduction studies were normal suffered from tuberculoid or dimorphous leprosy, and the only abnormality of the nerve was that it appeared to be clinically enlarged; there was no motor or sensory neurological deficit.

The mean amplitude of the muscle action potential was reduced in the 23 nerves studied, and the mean terminal latency was increased. In six cases no response was recorded on supramaximal stimulation of the ulnar nerve. There was significant slowing of motor conduction in the proximal, elbow and distal segments, and in three nerves the conduction velocity in the upper arm was more than 20 m/sec slower than in the forearm indicating more severe involvement of the proximal segment. In three nerves the conduction velocity in one segment was less than 30 m/sec. Two of these patients suffered from lepromatous leprosy and one from the dimorphous form. The mean amplitudes of the sensory action potentials and mixed nerve action potentials were reduced. The mean latency of the response was significantly increased only in the case of the mixed nerve action potential of the proximal segment of the ulnar nerve. The values for four parameters of ulnar nerve function are illustrated in Figure 1.

Median nerve. Only two nerves were clinically affected. In one subject, conduction studies were within the control range, and in the other subject the mixed nerve action potential was absent in the arm and in the forearm. The patient with normal conduction studies suffered from tuberculoid leprosy, and there was objective impairment of clinical function of the median nerve.

Radial nerve. The motor function of the radial nerve was not clinically abnormal in any of the subjects. It was not possible to assess accurately sensory function.

Lateral popliteal nerve. One or more of the studies on motor conduction fell outside the control range in eight of the nineteen nerves studied. In nine of the eleven other nerves the mixed nerve action potential was absent, by contrast with the control group in which the mixed nerve action potential was absent in only four of thirty nerves examined. One of the patients with normal conduction suffered from tuberculoid leprosy and the other from the dimorphous form. In both cases the only clinical abnormality of the lateral popliteal nerve was that it was clinically enlarged.

The mean amplitude of the muscle action potential (Fig. 2) and the mean motor conduction velocity were reduced in the 19

TABLE 4. *Results of motor and sensory conduction studies on control subjects and clinically affected nerves of leprosy patients.*

Sensory Action Potentials (SAP) and Mixed Nerve Action Potentials (NAP)													
		Ulnar						Median					
		S.A.P.		N.A.P.				S.A.P.		N.A.P.			
		Latency	Amp.	Latency Proximal	Amp. Proximal	Latency Distal	Amp. Distal	Latency	Amp.	Latency Proximal	Amp. Proximal	Latency Distal	Amp. Distal
		(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)
Control	Mean	3.2	15.0	3.0	78.4	5.3	59.6	3.5	25.0	3.0	87.8	4.3	61.6
	S.D.	0.44	6.5	1.0	47.6	1.1	27.5	0.5	10.8	0.5	37.4	0.8	25.5
	Range	2.5-4.1	7-26	2.0-6.0	27-183	2.8-6.6	16-120	2.6-4.6	9-52	2.2-4.4	22-155	2.5-5.7	15-128
	No.	28	27	30	30	30	30	29	30	27	27	30	30
Clinically affected	Mean	2.9	5.1	4.1	27.7	5.5	17.1	Responses recorded from only 2 nerves					
	S.D.	0.41	6.3	2.0	31.6	0.9	10.7						
	Range	2.6-3.5	0-22	1.8-8.8	0-110	4.1-7.4	0-88						
	No.	11	23	19	23	14	23						
p		N/S	<0.001	<0.01	<0.001	N/S	<0.001						

TABLE 5. *Results of motor and sensory conduction studies on control subjects and clinically affected nerves of leprosy patients.*

Sensory Action Potentials (SAP) and Mixed Nerve Action Potentials (NAP)											
		Radial S.A.P.		Lateral Popliteal N.A.P.		Sural S.A.P.		Posterior Tibial N.A.P.			
		Latency	Amp.	Latency	Amp.	Latency	Amp.	Latency Proximal	Amp. Proximal	Latency Distal	Amp. Distal
		(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)
Control	Mean	5.8	10.1	6.6	5.8	3.4	29.3	5.2	17.4	8.1	7.8
	S.D.	1.1	3.2	1.3	3.4	0.3	15.7	1.2	9.6	1.2	4.6
	Range	3.4-7.8	4-16	3.4-8.8	0-12	2.8-4.4	5-65	2.4-8.6	5-38	5.2-11.2	0-20
	No.	26	26	26	30	30	30	30	30	27	30
Clinically affected		No nerves were clinically affected		Responses recorded from only 2 nerves		Responses recorded from only 1 nerve		Responses recorded from only 2 nerves			

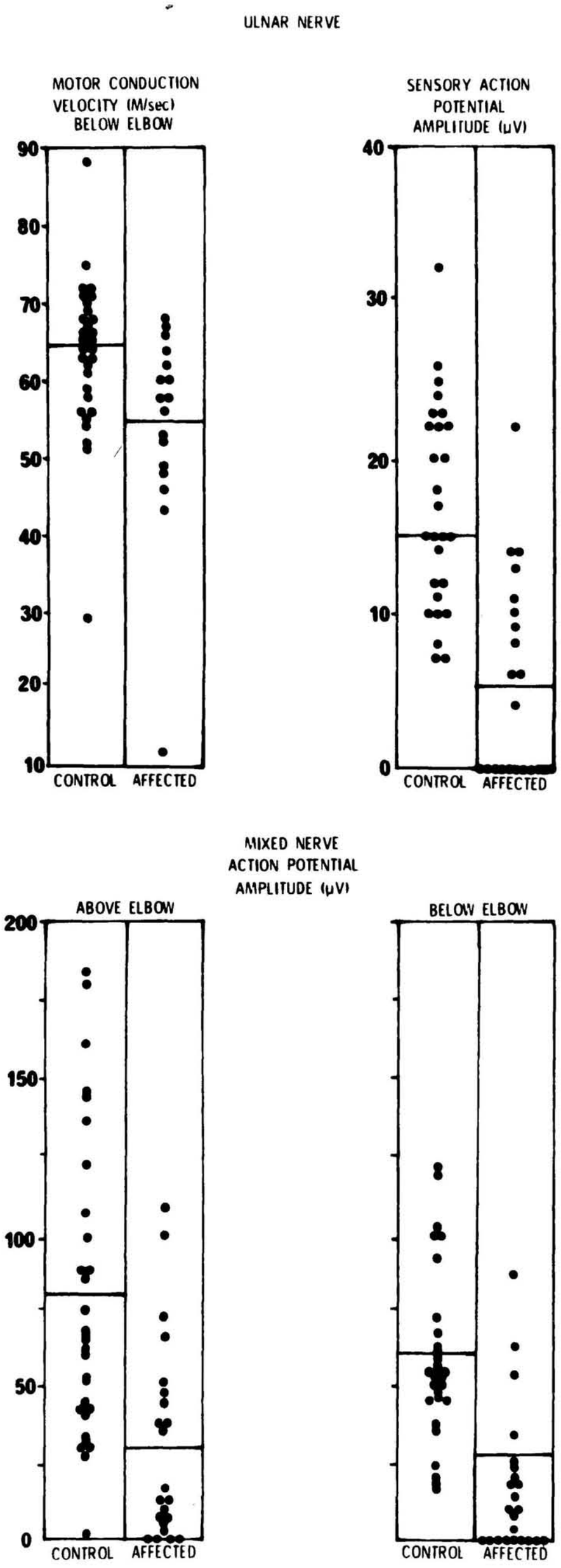

FIG. 1. Motor conduction velocity and amplitudes of sensory and mixed nerve action potential in ulnar nerve of control subjects and in ulnar nerves clinically affected by leprosy. The mean for each group is indicated by a horizontal bar.

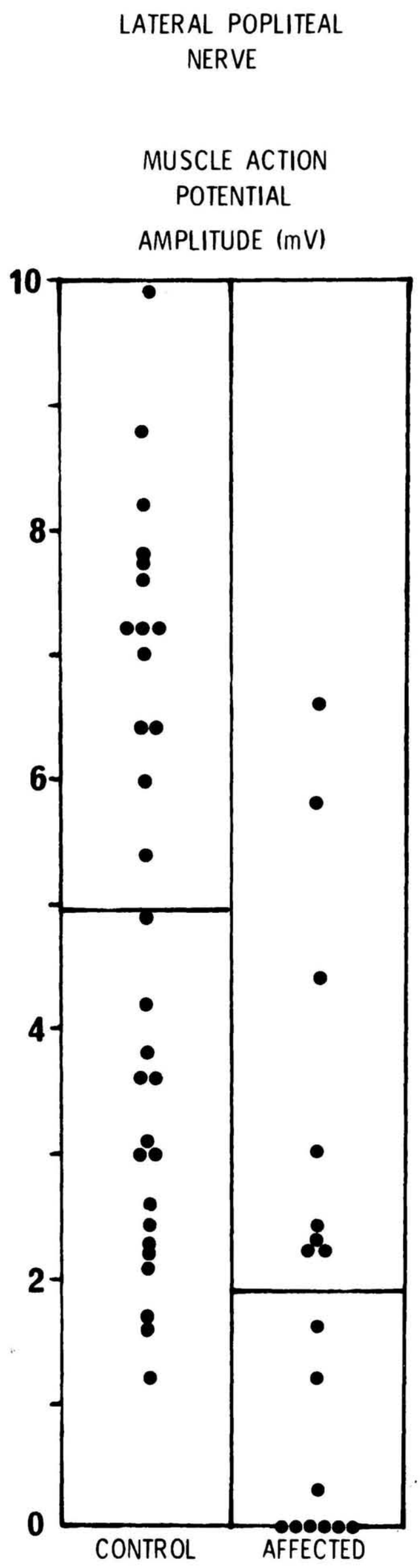

FIG. 2. Amplitude of muscle action potentials recorded following stimulation of the lateral popliteal nerve of control subjects, and of clinically affected lateral popliteal nerves of subjects with leprosy. The mean for each group is indicated by a horizontal bar.

nerves studied. The range of conduction velocities was 26-49 m/sec; no conduction could be recorded in six nerves. The patient in whom conduction velocity was 26 m/sec suffered from lepromatous leprosy.

Posterior tibial nerve. Abnormalities of conduction were detected in 12 of the 13 nerves studied (92%). The patient with normal conduction suffered from tuberculoid leprosy and the only abnormality of the pos-

terior tibial nerve was that it was clinically enlarged.

The mean amplitude of the muscle action potential recorded from the abductor hallucis was significantly reduced but there was no significant reduction in the mean motor conduction velocity. There was no recordable motor conduction in four nerves. The mean amplitude of the mixed nerve action potential recorded from the popliteal fossa on stimulating 16 cm above the ankle was significantly reduced ($p < 0.001$); no response was recorded from nine subjects, by contrast with the control nerves in which a response was recorded from each of the 30 nerves. When the site of stimulation was moved distally to the level of the ankle, a response could only be recorded from two subjects.

Sural nerve. There were 13 clinically affected sural nerves, and an action potential was recorded from only one.

Clinically unaffected nerves. The results of conduction studies on nerves which appeared to be clinically unaffected in patients known to have leprosy are summarized in Tables 6-10.

Ulnar nerve. Abnormalities were demonstrated in 10 of the 13 nerves studied. In one subject, who suffered from tuberculoid leprosy, conduction velocity in the upper arm segment was 17 m/sec. There was no significant difference between the mean values for muscle action potential amplitude and terminal latency in the 13 clinically unaffected nerves when compared with the control values. However, there was significant slowing ($p < 0.01$) of motor conduction in the above-elbow to wrist segment, and the mean values for motor conduction velocity in the proximal and distal segments tended to be reduced but the difference did not reach significant levels. The mean amplitude of both the sensory action potentials and the mixed nerve action potentials was reduced; the amplitude of the sensory action potential was below the control range in six nerves, the amplitude of the distal mixed action potential was below the control range in five nerves and the amplitude of the proximal mixed action potential was below the control range in six nerves.

Median nerve. One or more of the studies was outside the control range in 15 of the 33 nerves examined. There was no significant difference between the mean values for the muscle action potential amplitude and latency, or for motor conduction velocity in the 33 clinically unaffected nerves when compared with control nerves. The muscle action potential could not be recorded in one case and the conduction velocity in the forearm segment was below the control range in four subjects. The mean amplitude of the sensory action potential was reduced and the individual values fell below the control range in 12 subjects. The mean amplitude of the nerve action potential was also reduced. On stimulating 10 cm above the wrist and recording from the elbow, the amplitude of the response was below the control range in seven nerves. On stimulating at the wrist, the amplitude of the response was below the control range in ten nerves. In seven clinically unaffected nerves the sensory action potential was not recordable. The distribution of the values of two parameters is illustrated in Figure 3.

Radial nerve. Abnormalities were demonstrated in 21 of the 36 nerves studied. The mean amplitude of the sensory action potential was significantly reduced.

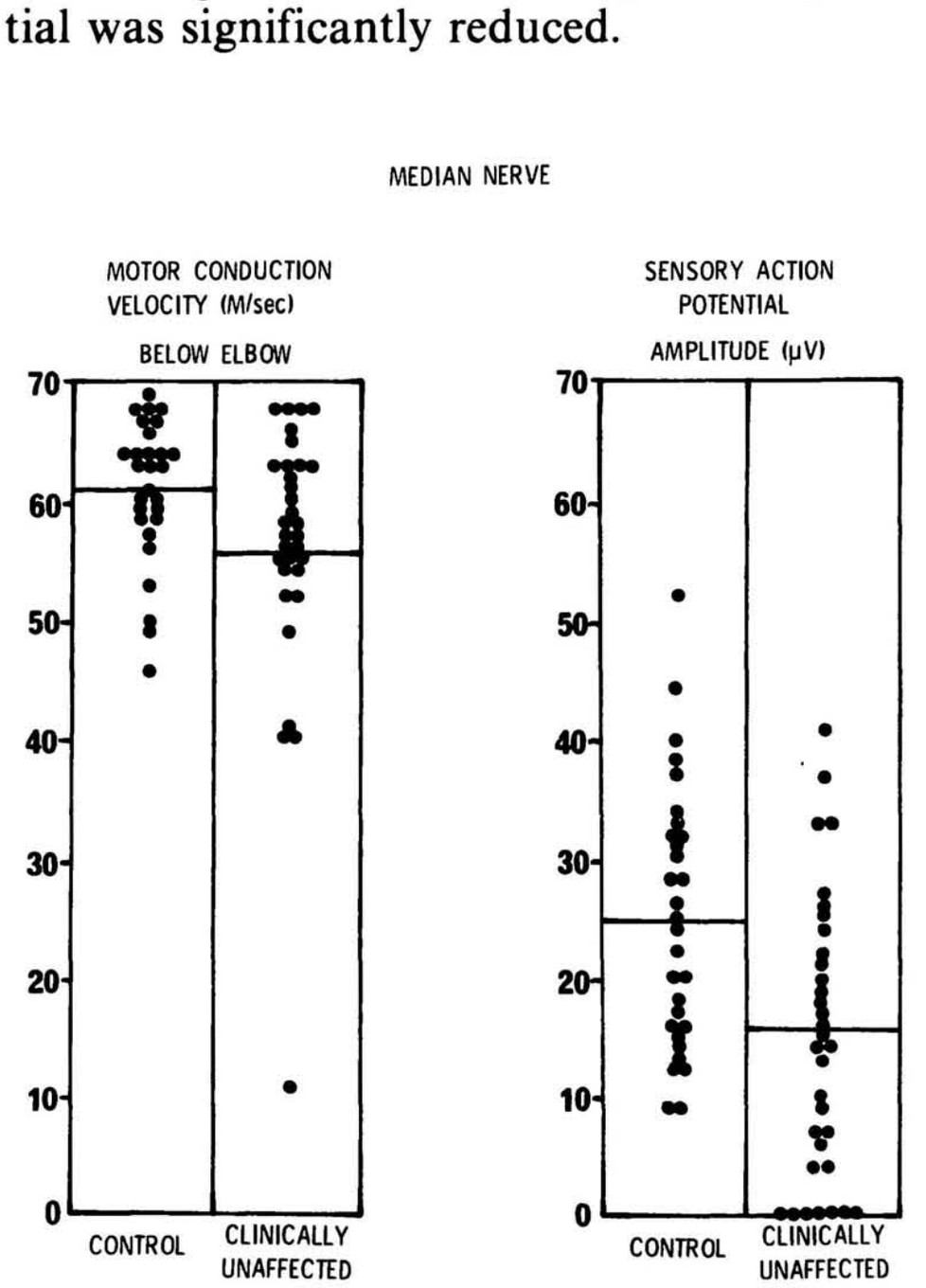

FIG. 3. Motor conduction velocity, and amplitude of sensory action potential in median nerves of control subjects, and in clinically unaffected median nerves of subjects with leprosy. The mean for each group is indicated by a horizontal bar.

TABLE 6. *Nerve conduction studies in leprosy.*

Clinically Unaffected Nerves			
Nerve	Total number	Abnormal	%
Ulnar	13	10	77
Median	33	15	45
Radial	36	21	58
Lateral popliteal	15	10	67
Posterior tibial	23	9	39
Sural	13	7	54

Lateral popliteal nerve. The mean amplitude of the muscle action potential recorded from the extensor digitorum brevis muscle of the 15 nerves studied was not significantly reduced, and the value fell below the control range in only one nerve. The motor conduction velocity of all 15 nerves was within the control range. The mean amplitude of the mixed nerve action potential was reduced ($p < 0.05$). It was absent in nine nerves, by contrast with an absent nerve action potential in four nerves of the control group.

TABLE 7. *Results of motor and sensory conduction studies on control subjects, and on clinically unaffected nerves of patients with leprosy.*

Motor Conduction Studies										
		Ulnar					Median			
		Latency	Amp.	Velocity			Latency	Amp.	Velocity	
				Prox.	Elbow	Distal			Prox.	Distal
		(msec)	(mv)	(m/sec)	(m/sec)	(m/sec)	(msec)	(mv)	(m/sec)	(m/sec)
Control	Mean	2.6	7.5	64.5	59.5	65.0	3.7	6.9	66.0	61.0
	S.D.	0.34	2.2	5.8	4.9	7.2	0.4	2.6	6.7	7.1
	Range	1.9-3.3	3.8-12.5	52-79	52-79	52-88	2.4-4.3	1.6-11.6	52-78	46-69
	No.	30	30	29	30	29	28	30	27	30
Clinically unaffected	Mean	2.8	7.3	58.5	51.9	58.4	3.7	6.3	62.6	56.2
	S.D.	0.63	2.3	14.9	9.2	4.0	0.21	3.3	9.6	11.0
	Range	2.2-4.8	1.0-9.6	17-80	29-61	51-66	2.2-10.5	0-14.4	46-80	11-68
	No.	12	13	13	13	13	33	32	33	33
p		N/S	N/S	N/S	<0.01	N/S	N/S	N/S	N/S	N/S

TABLE 8. *Results of motor and sensory conduction studies on control subjects, and on clinically unaffected nerves of patients with leprosy.*

Motor Conduction Studies							
		Lateral Popliteal			Posterior Tibial		
		Latency (msec)	Amp. (mv)	Velocity (m/sec)	Latency (msec)	Amp. (mv)	Velocity (m/sec)
Control	Mean	4.5	4.9	49.0	5.6	4.9	50.0
	S.D.	0.6	2.5	6.0	1.2	2.5	8.9
	Range	2.7-5.6	1.2-9.9	35-59	2.8-8.8	3.6-20.0	34-63
	No.	30	30	30	30	30	18
Clinically unaffected	Mean	4.3	3.8	52	5.3	6.0	49.0
	S.D.	0.8	2.4	3.7	2.2	4.5	5.0
	Range	3.2-5.8	0-7.3	42-56	3.2-14.0	0-15.0	43-59
	No.	14	15	15	22	23	22
p		N/S	<0.001	N/S	N/S	N/S	N/S

TABLE 9. *Results of motor and sensory conduction studies on control subjects and clinically unaffected nerves of leprosy patients.*

Sensory Action Potentials (SAP) and Mixed Nerve Action Potentials (NAP)													
		Ulnar						Median					
		S.A.P.		N.A.P.				S.A.P.		N.A.P.			
		Latency	Amp.	Latency Amp. Proximal		Latency Amp. Distal		Latency	Amp.	Latency Amp. Proximal		Latency Amp. Distal	
		(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)
Control	Mean	3.2	15.0	3.0	78.4	5.3	59.6	3.5	25.0	3.0	87.8	4.3	61.6
	S.D.	0.44	6.5	1.0	47.6	1.1	27.5	0.5	10.8	0.5	37.4	0.8	25.5
	Range	2.5-4.1	7-26	2.0-6.0	27-183	2.8-6.6	16-120	2.6-4.6	9-52	2.2-4.4	22-155	2.5-5.7	15-128
	No.	28	27	30	30	30	30	29	30	27	27	30	30
Clinically unaffected	Mean	3.2	8.5	3.4	61.3	5.4	35.5	3.1	15.1	4.3	59.5	4.5	37.9
	S.D.	0.4	6.8	1.2	72.7	0.9	24.9	0.4	10.5	2.1	44.1	0.6	28.4
	Range	2.8-4.0	0-20	2.0-6.4	5-265	3.4-6.8	5-76	2.5-4.0	0-37	2.2-9.0	0-150	3.3-6.2	0-106
	No.	10	13	13	13	13	13	27	33	31	32	29	34
p		N/S	<0.01	N/S	N/S	N/S	<0.01	N/S	<0.001	<0.001	<0.05	N/S	<0.001

TABLE 10. *Results of motor and sensory conduction studies on control subjects and clinically unaffected nerves of leprosy patients.*

Sensory Action Potentials (SAP) and Mixed Nerve Action Potentials (NAP)											
		Radial S.A.P.		Lateral Popliteal N.A.P.		Sural S.A.P.		Posterior Tibial N.A.P.			
		Latency	Amp.	Latency	Amp.	Latency	Amp.	Latency Amp. Proximal		Latency Amp. Distal	
		(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)	(msec)	(mv)
Control	Mean	5.8	10.1	6.6	5.8	3.4	29.3	5.2	17.4	8.1	7.8
	S.D.	1.1	3.2	1.3	3.4	0.3	15.7	1.2	9.6	1.2	4.6
	Range	3.4-7.8	4-16	3.4-8.8	0-12	2.8-4.4	5-65	2.4-8.6	5-38	5.2-11.2	0-20
	No.	26	26	26	30	30	30	30	30	27	30
Clinically unaffected	Mean	5.2	3.9	6.9	2.9	3.5	15.0	5.5	13.4	8.2	4.1
	S.D.	1.1	4.4	0.8	4.5	1.1	13.0	1.7	11.5	0.8	5.2
	Range	2.3-7.4	0-11	5.6-8.0	0-18	2.9-7.4	0-66	2.7-10.0	0-46	7.2-9.8	0-20
	No.	21	36	6	15	13	20	20	23	12	23
p		N/S	<0.001	N/S	<0.05	N/S	<0.01	N/S	N/S	N/S	<0.01

Posterior tibial nerve. Abnormalities were demonstrated in 9 of the 23 nerves studied. The mean values for the motor conduction studies did not differ significantly from the control group. There was a significant reduction of the amplitude of the mixed nerve action potential recorded at the knee on stimulating at the ankle ($p < 0.01$); no response was recorded from 11 nerves but it was absent also in three nerves of the control group. On stimulating 16 cm more proximally, there was an abnormally small response in 5 of the 23 nerves.

Sural nerve. The mean amplitude of the sensory action potential was significantly reduced in the 13 nerves studied ($p < 0.01$). A sensory action potential was recorded from all of the 30 control nerves but was absent from seven of the nerves in this group. The latency of the response was prolonged (7.4 m/sec) in another nerve in the group.

Group III. Subjects with clinically enlarged nerves in whom leprosy was undiagnosed. Peripheral nerves are readily palpable in the Aboriginal population, and it is frequently difficult to be certain whether a palpable nerve is clinically abnormal. In order to assess the value of nerve conduction studies in detecting the presence of pathologic changes in clinically enlarged nerves, electrophysiologic studies were performed on 27 subjects in the population at risk who were considered clinically to have one or more enlarged nerves. The group has been followed for a period of three years. In this group of 27 subjects, there were 19 ulnar, 15 lateral popliteal, 3 sural, 2 posterior tibial nerves and 1 median nerve which were judged to be enlarged.

Conduction studies were found to be abnormal in the clinically enlarged nerves of 12 of the 27 patients (44%). In 9 of the 12 patients, a pathologic diagnosis of leprosy was subsequently established. In none of the remaining three patients with abnormal conduction studies and in none of those with normal conduction studies, has leprosy been diagnosed.

DISCUSSION

Nerve conduction studies in the normal Australian Aboriginal population have not been reported previously. Motor conduction velocities are in the same range as those of the white Australian population ([18]) but the upper limits of the range of amplitudes of sensory and mixed nerve action potentials are greater in the Aboriginal subjects. This latter finding is attributed to the fact that the Australian Aboriginal is frequently thin in body build, and that because of the small amount of subcutaneous tissue the nerve trunks are relatively closer to the recording electrodes.

Abnormalities of nerve conduction were usually present in the clinically affected nerves of patients with leprosy. Abnormalities of nerve conduction were found in 18 of the 23 ulnar nerves (78%), 1 of the 2 median nerves (50%), 17 of the 19 (89%) lateral popliteal nerves (assuming an absent lateral popliteal nerve action potential to be abnormal), 11 of the 13 posterior tibial nerves (85%) and 12 of the 13 sural nerves (92%). These findings indicate that although impaired conduction occurs in the majority of nerves which are clinically affected, normal motor and sensory conduction is sometimes present in diseased nerves. Similar observations have been made by other workers ([3, 11, 16]). In all but 1 of the 11 cases in which normal conduction was found in a clinically affected nerve, the only abnormality of the nerve was that it appeared to be clinically enlarged.

In 3 of the 23 ulnar nerves studied, the conduction velocity in the upper arm segment was significantly slower than that in the forearm segment, by contrast with control nerves in which conduction velocities are slightly greater in the proximal segment. The finding is consistent with the fact that the disease commonly affects the ulnar nerve in the lower half of the upper arm ([2]). Slowing of conduction below 30 m/sec was unusual, but occurred in the proximal or distal segments of the ulnar nerve in two cases of lepromatous leprosy, and one case with dimorphous disease. In one patient with lepromatous leprosy there was a conduction velocity of 26 m/sec in the lateral popliteal nerve. Motor conduction velocities which are reduced to such a degree are nearly always associated with segmental demyelination ([7, 14]). Pathologic changes of segmental demyelination have been demonstrated in teased fiber preparations of our own pathologic material in lepromatous and tuberculoid leprosy (to be published) and by other

workers in lepromatous disease.

Abnormalities of motor conduction, and more commonly of sensory conduction, were demonstrated in a large proportion of clinically unaffected nerves of patients with leprosy. One or more studies were outside the control range in 10 of 13 (77%) clinically unaffected ulnar nerves, 15 of 33 (45%) median nerves, 21 of 36 (58%) radial nerves, 10 of 15 (67%) lateral popliteal nerves (assuming an absent lateral popliteal nerve action potential to be abnormal), 9 of 23 (39%) posterior tibial nerves, and 7 of 13 (54%) sural nerves. These observations are consistent with the findings of other workers ([11, 16]), and indicate the widespread involvement of the peripheral nervous system in the disease, and the value of nerve conduction studies in the early detection of leprosy.

It is difficult to decide whether or not a clinically enlarged nerve in a patient at risk is necessarily abnormal and indicates that he is suffering from leprosy. The value of nerve conduction studies in detecting abnormalities was therefore examined in 27 patients with a clinically enlarged nerve in whom the diagnosis was in doubt. Abnormal conduction was demonstrated in 12 subjects, 9 of whom were subsequently proven to have leprosy. It is suggested that when abnormal conduction is found in the clinically enlarged nerve of a patient at risk from the disease, there is a strong probability that he has leprosy and surgical exploration and biopsy of the nerve are indicated. Naturally, other causes of clinical enlargement of nerves, such as trauma, diabetes, acromegaly, and genetically determined hypertrophic neuropathies must be considered. If the nerve is thought to be enlarged, but if conduction studies are normal, a further period of observation is indicated before other investigations or treatments are instituted. It has already been noted, however, that normal conduction may be present in a diseased nerve.

It has been shown in the present study, and by other workers ([16]) that it is possible by electrophysiologic means to localize the segments of a nerve to which the disease is chiefly restricted. Nerve conduction studies are therefore helpful in the selection of suitable patients for the treatment of localized lesions by nerve grafting procedures ([10]).

SUMMARY

Motor and sensory nerve conduction studies have been performed on the peripheral nerves in the upper and lower limbs of 30 control subjects, and 36 subjects with leprosy from the Aboriginal population of the Northern Territory of Australia. Impairment of conduction was demonstrated in the vast majority of clinically abnormal nerves, and a large proportion of nerves which appeared clinically to be uninvolved. In a third group of subjects, abnormal conduction was demonstrated in a significant number of nerves which were considered to be clinically enlarged but in whom the diagnosis was initially in doubt. The majority of these patients were subsequently proven to have leprosy. It is concluded that nerve conduction studies are of considerable value in the diagnosis and management of leprosy.

RESUMEN

Se realizaron estudios de conducción motora y sensorial en los nervios periféricos de los miembros superiores e inferiores de 30 individuos controles y de 36 individuos con lepra, de la población aborigen del Territorio Norte de Australia. Se demostró deterioro de conducción en la gran mayoría de los nervios clínicamente anormales y en una gran proporción de los nervios que clínicamente parecían indemnes. En un tercer grupo de individuos, se demostró conducción anormal en un número significativo de nervios que se consideraban engrosados, pero en los cuales el diagnóstico inicialmente fué dudoso. La mayoría de estos pacientes demostró posteriormente tener lepra. Se concluye que los estudios de conducción nerviosa son de considerable valor en el diagnóstico y manejo de la lepra.

RÉSUMÉ

On a procédé à des études de la conduction nerveuse motrice et sensitive au niveau des nerfs périphériques des membres supérieurs et inférieurs chez 30 individus témoins, et chez 36 patients atteints de lèpre appartenant à la population autochtone des Territoires du Nord de l'Australie. Une détérioration de la conduction a été mise en évidence dans la grande majorité des nerfs présentant des signes cliniques anormaux; par contre, une grande proportion des nerfs apparaissant normaux au point de vue clinique ne présentaient aucune atteinte. Dans un troisième groupe d'individus, des phénomènes de conduction anormale ont été démontrées dans un nombre significatif de nerfs qui avaient été considérés comme épassis au point de vue clinique, mais dont le diagnostic était douteux au début. La ma-

jorité de ces malades se sont révélés ultérieurement atteints de lèpre. On en conclut que les études de conduction nerveuse présentent un intérêt considérable pour le diagnostic et la prise en charge des malades de la lèpre.

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