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The Peroneal and Tibial Nerves in Lepromatous Leprosy
Clinical and Electrophysiologic Observations[1, 2]

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Weakness or paralysis of muscles of the lower extremities is quite common in lepromatous leprosy, and constitutes a major disability in the disease. Whereas sciatic and femoral neuropathy is rare, peroneal and tibial nerve involvement is quite common.

Involvement of peroneal and tibial nerves in lepromatous leprosy commonly results in weakness and wasting of the intrinsic foot muscles, especially the extensor digitorum brevis, as well as the anterior tibial group and the calf muscles. Atrophy of intrinsic muscles of the foot may lead to clawing of the toes and contribute to deformity by reducing soft tissue resiliency on the plantar surface.

Extensive electrical studies of the nerves of the upper extremity in leprosy have been reported over the past seven years (4, 7, 8, 9, 15, 19, 23, 27, 28, 29, 31, 34), but there has been little work on motor nerve conduction in the lower extremities. Jopling and Morgan-Hughes (15) reported impaired conduction of peroneal and tibial nerves of one patient with pure neural tuberculoid leprosy. Rosenberg and Lovelace (23) found slowing of motor conduction velocity of both peroneal and both tibial nerves in a single patient with lepromatous leprosy. Distal latencies were significantly prolonged for the peroneal nerves but were normal for the posterior tibial nerves. Following treatment with sulfones, there was improvement in conduction velocity of the right peroneal nerve (from 20.6 meters/sec to 45.4 meters/sec). Magora *et al* (19) reported peroneal motor conduction studies on 67 lepromatous, 8 tuberculoid, 8 dimorphous, and 20 indeterminate patients. Of 108 peroneal nerves tested, 91 or 84.2% had a normal motor velocity and 17 or 15.8% were abnormal. Similar percentages had normal and impaired distal latencies. During a six-year follow-up period, those patients with clinical courses uncomplicated by reaction had no significant change in conduction velocity whereas slowing occurred in over half the patients with reaction. This slowing could be reversed by corticosteroids or thalidomide in almost all patients. Recently, Sohi *et al* (31), in a group of twelve lepromatous, six dimorphous, five tuberculoid, five neuritic, and two maculoanesthetic patients, have described slowing of peroneal and tibial conduction velocities and distal latencies for all groups. However, the mean values quoted for normal subjects are faster than those from other studies on peroneal and tibial nerves (1, 3, 10, 12, 14, 17, 21, 33).

The results from these studies are difficult to evaluate from several standpoints: first, there have been only a few correlations of conduction velocity with the presence or absence of reaction neuritis in the nerves tested, with the type of leprosy present, and with the clinical examination of such parameters as muscle strength, nerve enlargement, and reflexes; second, the method of patient selection has been uncertain; and third, segmental conduction of nerve trunks has not been done.

It seemed valuable, therefore, to examine clinically a group of patients with lepromatous leprosy who were unselected, and to compare clinical observations with segmental motor nerve conduction velocity and latency studies of the peroneal and tibial nerves.

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MATERIALS AND METHODS

Motor conduction velocity studies of peroneal and tibial nerves were performed on a group of sixteen normal subjects and twenty-five patients with lepromatous leprosy. The normal subjects were employees of the USPHS Hospital, Carville. None had a history of disease involving peripheral nerves. Their age ranges were 22-51 years, with a mean age of 36 years.

The patients were consecutive lepromatous patients admitted or re-admitted to the USPHS Hospital, Carville, Louisiana, and were otherwise unselected. Their age ranges were 20-73 years, with a mean age of 41 years.

The bulk and strength of the following muscles were assessed: extensor digitorum brevis, extensor hallucis longus, extensor digitorum longus, anterior tibialis, peronei, gastrocnemius-soleus, tibialis posterior, and intrinsic muscles of the foot. For certain of these muscles, strength cannot be directly tested against resistance and judgments were made based on the size and hardness of the contracting muscle (extensor digitorum brevis), or ability to perform an act such as making a cup of the ball of the foot (intrinsic muscles of the foot) or inverting the foot (tibialis posterior). Ankle jerks were noted to be present or absent. Attempts were made to palpate the peroneal nerve in the popliteal fossa, lateral to the head of the fibula, and at the ankle, and the tibial nerve in the popliteal fossa and behind the medial malleolus. Sensation was not assessed, for topical sensory loss occurs in lepromatous leprosy due to destruction of cutaneous nerve networks which may be unrelated to nerve trunk damage [24].

Motor nerve conduction velocities and latencies were determined according to the method of Hodes *et al* [10], using a Teca T4 Electromyograph (Teca Corp., White Plains, New York). Surface electrodes were used. The potentials were displayed on an oscilloscope and simultaneously recorded on photo-sensitive paper. In all cases supramaximal stimuli were used.

In order to assess segmental changes, conduction velocity was calculated for two peroneal nerve segments: from popliteal fossa to the proximal head of the fibula (P_1) and from the head of the fibula to the ankle (P_2). Latencies to two peroneal-innervated muscles also were recorded: from the ankle to the extensor digitorum brevis (P_3) and from the head of the fibula to the tibialis anterior (P_4). Tibial nerve conduction was measured from the popliteal fossa (popliteal skin crease) to 2 cm posterior and 1 cm proximal to the point of the medial malleous (T_1). Latencies to three tibial-innervated muscles also were recorded: from the ankle to the flexor digiti quinti (T_2), from the ankle to the abductor hallucis brevis (T_3), and from the popliteal crease to the lateral head of gastrocnemius (T_4) (see Fig. 1).

In order to obtain accurate latency values it is necessary to measure to the initial negative deflection of the compound motor unit action potential. This is best done by placing the external recording electrode directly over the motor point of the muscle. This point may be found without difficulty for the extensor digitorum brevis and anterior tibialis, but often only after much searching for the abductor hallucis, lateral gastrocnemius and flexor digiti quinti. In general the optimum location for the abductor hallucis conformed to that found by Mavor and Atcheson [21] (Fig. 1). The motor point of the anterior tibialis was usually found one-third the distance (average 120 mm) on a line between the head of the fibula and the medial malleolus; that of the lateral gastrocnemius was one-third the distance (average 120 mm) on a line between the center of the popliteal crease and the lateral malleolus.

Statistical analysis was performed for each of the eight values to compare control subjects with patients, and to compare patients grouped according to symptoms or findings.

RESULTS

Normal subjects. Clinical examination was normal in all subjects. Mean nerve conduction, latencies, ranges, and standard deviations were as shown in Table 1. These conform to previously published series [1-3, 10, 12, 14, 17, 21, 33]. Measurement of conduction in the segment P_1 (peroneal nerve, popliteal fossa to head of the fibula)

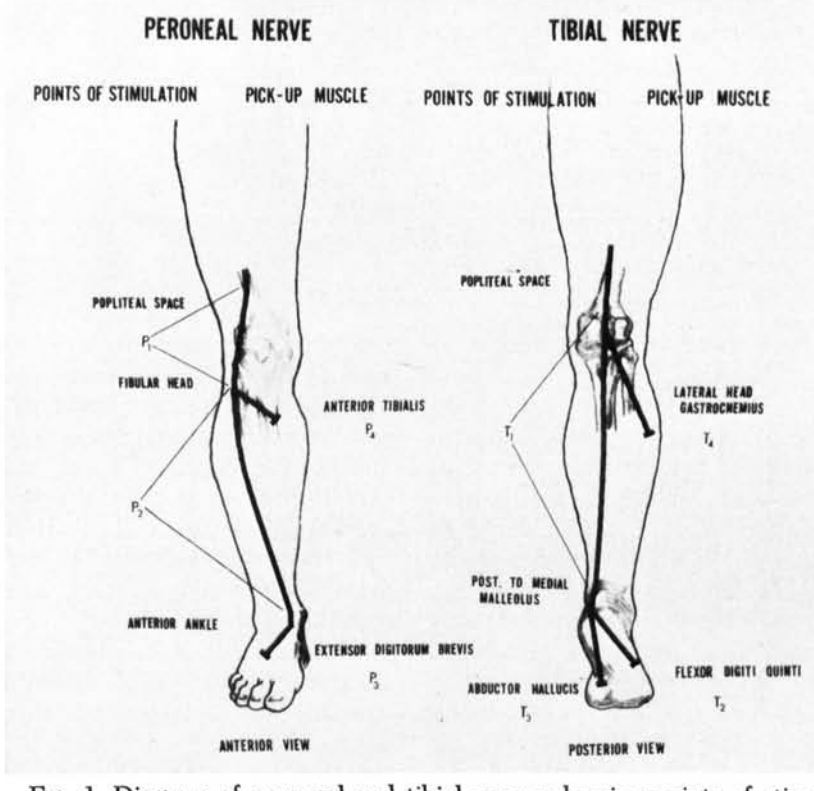

FIG. 1. Diagram of peroneal and tibial nerves showing points of stimulation, muscle pick-ups, and segments over which conduction was measured.

has not previously been reported, but appears not to be significantly different from P_2 (peroneal nerve, lateral fibula head to ankle). It is of interest that of the 256 observations made on the sixteen control subjects, only seven could be considered abnormal. All of these were latencies, rather than conduction velocities, and are possibly explainable on the basis of the sizes of the subjects involved. Six of the seven prolonged latencies occurred in a single individual male, six feet five inches in height, who has a size 12 foot. The only other abnormal latency occurred in another male subject, six feet four inches in height with a size 13 foot. The latencies in nor-

TABLE 1. *Nerve conduction and latency values.*

	Patients 50[a]			Normals 32[a]		
Segment	Mean	Range	Standard deviation	Mean	Range	Standard deviation
P_1 meters/sec.	45.3*[b]	24.6-70.9	9.5	51.9	40.9-66.9	6.8
P_2 meters/sec	47.7	39.4-59.6	5.3	49.5	43.5-56.0	4.2
P_3 msec	4.0*	8.2-2.3	1.0	3.6	5.4-2.6	0.7
P_4 msec	3.3	4.5-2.0	0.7	3.1	4.4-2.3	0.5
T_1 meters/sec.	44.9*	25.0-69.8	8.3	48.3	39.3-60.9	5.3
T_2 msec	5.6	9.0-3.7	1.1	5.5	6.7-4.2	0.7
T_3 msec	5.2	7.6-2.8	1.3	4.5	8.4-3.3	1.0
T_4 msec	4.3*	8.3-2.3	1.3	3.8	6.9-2.8	0.9

[a] Indicates number of legs on which figures are based.
[b] Asterisks indicate values significantly slowed compared to normal (two-tail T test $P = <0.05$).

TABLE 2. *Minimal normal values of nerve conduction velocities and latencies.*

Peroneal	P_1	P_2	P_3	P_4
	$\geq$ 38.3 Meters/second	$\geq$ 41.1 Meters/second	$\leq$ 5.0 msec	$\leq$ 4.1 msec
Tibial	T_1	T_2	T_3	T_4
	$\geq$ 37.7 Meters/second	$\leq$ 6.9 msec	$\leq$ 6.5 msec	$\leq$ 5.6 msec

mal subjects showed a wide range P_3 5.4-2.6 msec, P_4 4.4-2.3 msec, T_2 6.7-4.2 msec, T_3 8.4-3.3 msec, T_4 6.9-2.8 msec. As described, these ranges are partly explainable on the basis of height. However nerve conduction velocity, which is not height-dependent, also shows a wide range of normal values: P_1 40.9-66.9 meters/second, P_2 43.5-56.0 meters/second, T_1 39.3-60.9 meters/second. This wide range probably represents individual variation in normal subjects. For the purposes of this study, normal values for each of the eight measurements were arbitrarily defined as being within two standard deviations of the mean. When calculated in this way, minimum normal values are as listed in Table 2. One control subject had an anomalous branch of the peroneal nerve as described by Infante and Kennedy ([11]).

Clinical studies. Weakness and atrophy were present in muscles in the peroneal distribution in ten patients (18 nerves) and in the tibial distribution in seven patients (13 nerves). For almost all muscles this was bilateral. Involvement of the extensor digitorum brevis muscle was the most common, being present in 15 of 50 legs. Other peroneal-innervated muscles were involved as follows: extensor hallucis longus, 10 of 50 legs; extensor digitorum longus, 10 of 50 legs; tibialis anterior, 10 of 50 legs; peronei, 8 of 50 legs. Involvement of these muscles suggests peroneal nerve involvement at or above the head of the fibula. The greater involvement of the extensor digitorum brevis suggests additional involvement of the peroneal nerve at the ankle, since this is the only muscle tested in the peroneal distribution below the ankle. Atrophy and weakness of the tibial-supplied muscles were present in seven patients (13 nerves). The intrinsic muscles of the foot were the most often involved, weakness and atrophy having been found in 12 of 50 legs. Tibialis posterior was involved in 9 of 50 and gastrocnemius-soleus in 6 of 50 legs. In almost all instances these muscles were paretic but not paralyzed. The greater involvement of the intrinsic muscles of the foot than the calf muscles suggests additional involvement of the tibial nerve in the distal third of the leg.

Loss of ankle jerks occurred in only five patients, in all of whom it was bilateral. In four of these five patients there was evidence of weakness and atrophy of the calf muscles.

Nerve enlargement was quite common for the peroneal nerve, occurring in 26 of 50 legs, and was always bilateral. Enlargement was most often noted at the head of the fibula. In fewer instances the nerve could be palpated at the ankle and in the popliteal fossa. Tibial nerve enlargement occurred in 7 of 50 legs and was bilateral in two patients. Enlargement was noted most often behind the medial malleolus and less often in the popliteal fossa. Most of the enlarged peroneal and tibial nerves were firm and tender to palpation, and referred paresthesiae could be elicited on manipulation.

Documented instances of episodes of acute neuritis (acute nerve pain and sudden increase in neurologic deficits) occurred in 18 peroneal nerves. It was always bilateral. Tibial neuritis occurred in three nerves, and was bilateral in one patient.

Electrical studies. Electrical studies revealed abnormalities in 23 of 50 peroneal nerves and 19 of 50 tibial nerves. The mean values, ranges, and standard deviations for all 16 observations on each patient are listed in Table 1. Those values followed by an asterisk are significantly different from

TABLE 3. *Patients with selective slowing of P_1 segment.*

Patient no.	P_1 N ≥ 38.3	P_2 N ≥ 41.1	P_3 N ≤ 5.0	P_4 N ≤ 4.1
6 right	40.0	48.6	5.0	4.4*
6 left	33.3*[a]	42.2	4.0	3.9
8 right	25.2*	45.9	3.9	3.1
8 left	24.6*	51.1	4.3	2.6
9 right	30.6*	48.4	3.9	3.4
9 left	36.4*	51.0	4.7	3.7
11 right	39.0	50.0	3.4	3.4
11 left	48.9	50.8	4.1	3.2
17 right	28.6*	48.4	3.1	2.9
17 left	52.6	59.1	5.3*	4.0
22 right	54.4	42.5	3.0	2.7
22 left	36.0*	43.5	4.0	3.7
23 right	37.2*	51.8	3.1	2.4
23 left	50.6	47.1	3.5	2.8

[a] Asterisks indicate values significantly slowed compared to normal (two-tail T test P = <0.05).

normal. As may be seen, P_1 and T_1 conduction velocities and P_3 and T_4 latencies are significantly different from normal (two-tail T test, P = <0.05). Those patient nerves showing no response to electrical stimulation (P_1, 2 nerves; P_2, 2 nerves; P_3, 1 nerve; P_4, 1 nerve) or which could not be stimulated due to obesity (P_1, 1 nerve), are not included in the statistical analysis. The greater variability in patients than control subjects is shown by the standard deviation which is higher for every determination in the patient group. The slowing of P_1 segment was the most common of the nerve segments tested, occurring in 13 of 49 legs (in one leg no response could be obtained because of obesity). Slowing occurred in P_3 in 8 of 50, T_1 in 8 of 50, and T_4 in 7 of 50 legs, with lesser instances of slowing of the other segments.

In several patients this segmental slowing was quite impressive. Results are given in Table 3 for seven patients that demonstrate selective slowing in the P_1 segment with normal conduction further distally. An important point to be made on this table is that if peroneal nerve conduction studies had been performed in the traditional way only, i.e. from the head of the fibula to the ankle, the marked slowing of peroneal conduction of 8 nerves in P_1 segment would not have been found, and all 14 nerves shown in the table would have been considered normal.

Prolonged latency from the ankle to the extensor digitorum brevis was present statistically in the patient group (Table 1), but relatively few values were abnormal. Selective involvement of this segment is suggested by an analysis of four patients with prolonged P_3 latencies (Table 4). In this table, six of the eight P_3 values are prolonged (≥ 5.0 msec.) In all but one of these nerves there is normal conduction

TABLE 4. *Patients with selective slowing of P_3 segment.*

Patient no.	P_1 N ≥ 38.3	P_2 N ≥ 41.1	P_3 N ≤ 5.0	P_4 N ≤ 4.1
4 right	48.6	49.3	6.5*[a]	2.4
4 left	52.9	57.8	5.1*	2.8
5 right	40.0	56.8	5.1*	3.4
5 left	62.5	48.7	3.8	3.2
17 right	28.6*	48.4	3.1	2.9
17 left	52.6	59.1	5.3*	4.0
24 right	43.5	47.8	8.2*	4.7*
24 left	45.0	55.5	5.2*	3.8

[a] Asterisks indicate values significantly slowed compared to normal (two-tail T test P = <0.05).

in all other areas of the nerve. In patient 17 there was selective and severe slowing of the right P_1 segment and the left P_3 segment but not in any other portions of the nerve.

Although mean values of P_2 and P_4 for patients were both different from normal, these differences are not statistically significant.

There was a significant difference in conduction velocity of the tibial nerve from the popliteal fossa to the ankle between control subjects and patients. That this slowing was due to involvement of the tibial nerve in the popliteal fossa and/or in the upper calf is suggested by the associated prolongation of the T_4 latency. This segment represents the latency to the lateral gastrocnemius, and this nerve leaves the tibial nerve in the popliteal fossa. Somewhat surprisingly, the distal latencies to the intrinsic muscles of the foot, T_2 and T_3, were normal, again suggesting that tibial nerve involvement was occurring further proximally. However, additional involvement of the tibial nerve above the ankle in the distal portion of the leg cannot be ruled out on the basis of this study.

An attempt was made to correlate 1) the presence of nerve enlargement with NCV slowing, 2) the presence of weakness with NCV slowing, and 3) the presence of nerve pain with NCV slowing. Of the three correlations, only the last showed a significant difference in NCV between patients without nerve pain and those with nerve pain. In Figure 2, P_1 and P_2 velocities are plotted for control subjects, patients without weakness, and patients with weakness. The large dot is the mean for each group, and one standard deviation is given. As may be seen, the patients with weakness

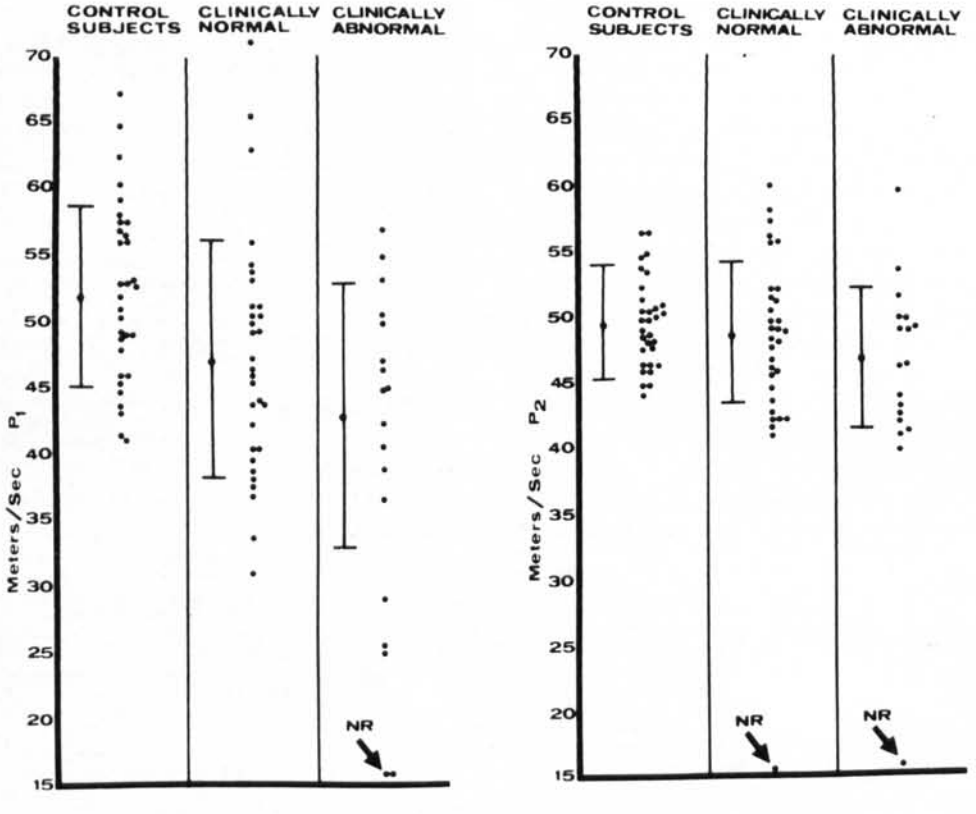

FIG. 2. Graph showing individual conduction velocities of P_1 and P_2 segments for control subjects, patients without weakness, and patients with weakness. Mean conduction velocity and one standard deviation above and below the mean are given for each group. (NR = no response to stimulation)

have a lower mean P_1 and P_2 conduction velocity. According to the method of calculation, this difference while possibly showing a trend, is not statistically significant (two-tailed T test, $P = < 0.05$). However, as can be seen, in two legs in both P_1 and P_2, no values could be obtained for patients with weakness and if these observations are included, the differences are significant. For nerve pain, however, there is a significant correlation in the P_2 segment, Figure 3. A history of nerve pain, therefore, appears to have a more important correlation with slowing of nerve conduction velocity than either nerve enlargement or weakness.

Since tibial nerve pain occurred in only two patients and tibial nerve enlargement in only five, meaningful correlations could not be drawn between these parameters and slowing of conduction velocity. Although tibial nerve conduction was slower for patients with weakness than for those without weakness of tibial supplied muscles, the differences were not statistically significant.

DISCUSSION

This study shows that in a large group of unselected patients with lepromatous leprosy, clinical evidence of peroneal and tibial nerve involvement is quite common. Fifty-two percent of peroneal nerves were enlarged to palpation, 36% had a history of nerve pain at some time, and 36% had weakness in the muscles they supplied. Fourteen percent of tibial nerves were enlarged, 6% had a history of nerve pain at some time, and 26% had weakness in the muscles they supplied. Nerve conduction velocity studies support the clinical findings. Nerve conductions and latency values were abnormal in 46% of peroneal and 38% of tibial nerves.

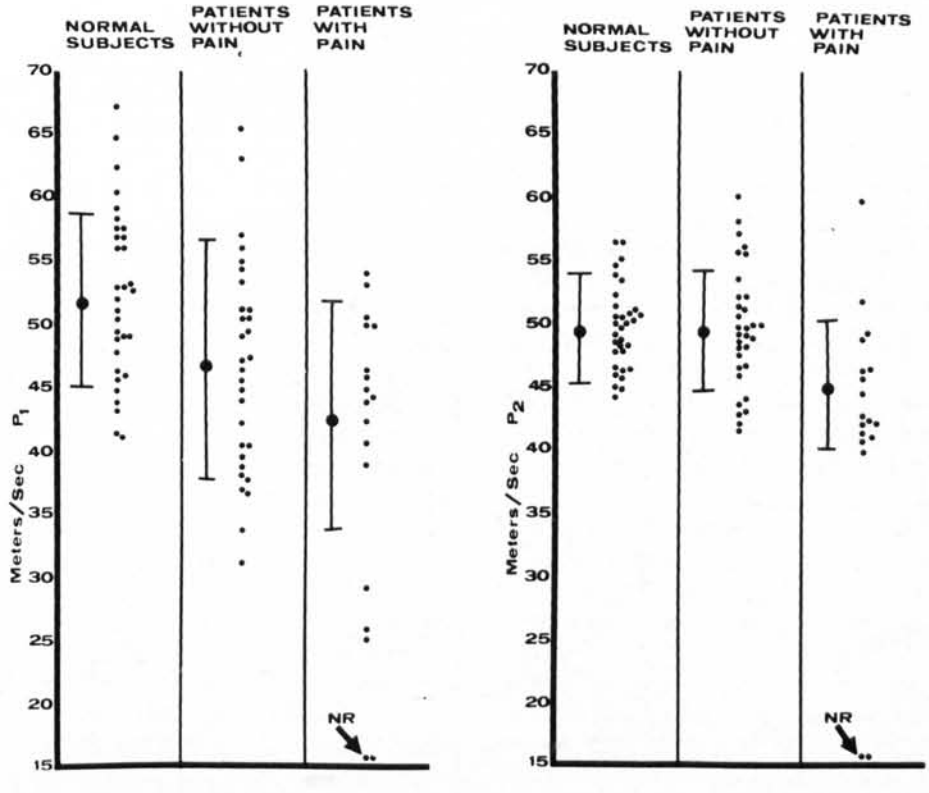

FIG. 3. Graph showing individual conduction velocities of P_1 and P_2 segments for control subjects, patients without peroneal nerve pain, and patients with peroneal nerve pain. Mean conduction velocity and one standard deviation above and below the mean are given for each group. (NR = no response to stimulation)

As a group, patients with lepromatous leprosy have slowed conductions of the peroneal nerve from the popliteal fossa to the head of the fibula and at the ankle. Tibial nerve conduction is slowed from the knee to the ankle and in the branch to the gastrocnemius. This study points up the importance of including the P_1 segment in peroneal conductions in leprosy, despite the fact that this segment is relatively short and the margin for error is correspondingly greater. This segment was chosen for study because, as originally proposed by Brand [2] and confirmed by later work [24], nerve involvement in lepromatous leprosy occurs in areas of low temperature, presumably due to a temperature preference for growth of *M. leprae* [26]. If the peroneal conduction velocity is measured only for the deep peroneal nerve, i.e. between the head of the fibula and the ankle, most of the cool areas of the peroneal nerve will be missed and conduction will be normal. This may account for the lower incidence of abnormal conduction velocities in the peroneal nerve reported by Magora *et al* [19]. Likewise the P_3 segment, also in a cool area, is significantly prolonged in the patient group. Temperatures of the peroneal nerve bed at the knee, deep in the anterior tibial compartment and at the ankle recently have been investigated using thermistor-tipped needle probes by Sabin, Hackett and Brand [25]. The temperatures at the knee and ankle are lower by 2.4° and 3.5°C, respectively, than in the anterior tibial compartment. A similar relationship to temperature has also been determined for the ulnar and median nerves [25].

The value of performing peroneal and tibial nerve conduction velocity studies as described is apparent from the fact that greater involvement was found on electrical testing of nerves than on clinical examination of muscles these nerves supply. Prominent slowing of conduction with no evidence of muscle atrophy, such as occurred in 15 peroneal and 14 tibial nerves in this series, is consistent with segmental demyelination of nerve with preservation of axons. In addition, the slowing of the proximal segment of the peroneal nerve with normal conduction further distally, similar to slowing in the elbow segment of the ulnar nerve, suggests a process of segmental demyelination. This is supported by recent studies which have revealed histologic evidence of segmental demyelination in leprous nerve [5, 6, 32].

In nine peroneal nerves and nine tibial nerves in this series, weakness and/or atrophy was present with normal conduction velocities, suggesting axonal degeneration. Loss of nerve fibers in the face of normal nerve conduction velocity is also possible where maximal damage is occurring to certain fascicles with relatively little involvement of others. Since nerve conduction velocity is calculated on the basis of the fastest conducting fibers only, this might still be normal. Another process that would explain weakness and atrophy in the presence of normal nerve conduction is myopathy. Histologic evidence of leprous myositis has been found [13, 20, 22], and a few studies have reported electromyographic evidence of myopathy [16, 18, 20]. However, this has not been found in other histologic and electrical studies [4, 23, 30], and our experience with electromyography in a large group of leprosy patients has failed to uncover myopathic changes.

SUMMARY

Clinical examination and segmental nerve conduction velocity studies of peroneal and tibial nerves were carried out on 25 patients with lepromatous leprosy and on 16 control subjects. Muscle atrophy and weakness occurred most often in the extensor digitorum brevis muscle (15 of 50 legs) and intrinsic foot muscles (12 of 50 legs), with lesser instances of weakness in other muscles. Nerve enlargement and nerve pain were common for the peroneal nerve and less common for the tibial nerve. Nerve conductions and latencies revealed significant slowing in the patients in the segment of the peroneal nerve from the popliteal fossa to the head of the fibula and in the latency from the ankle to the extensor digitorum brevis muscle. Tibial slowing occurred in the segment from the popliteal fossa to the ankle and in the latency from the popliteal fossa to the lateral head of the

gastrocnemius. This study shows that clinical and electrical evidence of segmental involvement of both nerves is common in lepromatous leprosy, and points out the importance of performing nerve conduction velocity studies on the segment of the peroneal nerve between the popliteal fossa and the head of the fibula.

RESUMEN

Se realizaron estudios clínicos de velocidad de conducción nerviosa segmentaria en los nervios peroneales y tibiales en 25 pacientes con lepra lepromatosa y en 16 sujetos controles. La atrofia muscular y la debilidad se observaron con mayorf frecuencia en el músculo extensor digitorum brevis (15 de 50 piernas) y músculos intrínsecos del pié (12 de 50 piernas), con una menor frecuencia de debilidad en los otros músculos. Se encontró que el engrosamiento y dolor nervioso se observaban con frecuencia en el nervio peroneal y con menos frecuencia en el nervio tibial. La conducción nerviosa y la latencia se mostraron significativamente disminuídas en los pacientes en el segmento del nervio peroneal que va desde la fosa poplítea hasta la cabeza de la fíbula y en la latencia desde el tobillo hasta el músculo extensor digitorum brevis. El retardamiento tibial se observó en el segmento que va desde la fosa poplítea hasta la cabeza lateral del grastrocnemius. Este estudio muestra evidencia clínica y eléctrica de que el compromiso segmentario de ambos nervios es común en lepra lepromatosa, e indica la importancia de llevar a cabo estudios de velocidad de conducción nerviosa en el segmento del nervio peroneal que va de la fosa poplítea hasta la cabeza de la fíbula.

RÉSUMÉ

Chez 25 malades atteints de lèpre lépromateuse et chez 16 sujets témoins, on a procédé à des examens cliniques et à des études de la vitesse de conduction nerveuse segmentaire des nerfs péroniers et tibiaux. Une atrophie musculaire, de même qu'une faiblesse des muscles, a été constatée plus souvent dans le court extenseur des orteils (15 jambes sur 50), et dans les muscles intrinsèques du pied (12 jambes sur 50), alors que les autres muscles présentaient des signes de faiblesse en moins grand nombre. Un élargissement des nerfs, de même que des douleurs nerveuses, étaient communes au niveau de nerf péronier, mais moins fréquente au niveau du nerf tibial. Les études de conduction nerveuse et des temps de latence, ont révélé un ralentissement significatif chez les malades, au niveau du segment du nerf péronier allant de la fosse poplitée à la tête du péronier. Un ralentissement du temps de latence a été constaté entre la cheville jusqu'au niveau du court extenseur des orteils. Un ralentissement tibial était présent dans le segment allant de la fosse poplitée à la cheville; un ralentissement du temps de latence était constaté entre la fosse poplitée jusqu'à l'insertion latérale du couturier. Cette étude montre dès lors qu'il existe des signes cliniques électriques d'une atteinte segmentaire de ces dux nerfs; cette atteinte est commune dans la lèpre lépromateuse. Ces résultats montrent en outre l'importance qu'il y a à procéder à des études de la vitesse de conduction nerveuse au niveau du segment du nerf péronier entre la fosse poplitée et la tête du péronier.

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