A Study of the Conduction Velocity of the Motor Fibers of Ulnar and Median Nerves in Leprosy[1,2]

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Every leprosy patient has peripheral nerve involvement of one sort or another and with over 10 million patients in the world, this becomes the largest single peripheral nerve disorder in the world. Peripheral neuropathies may be classified in three different ways—by pathology, impaired function or etiology. Pathologic classification is based on the site of primary involvement: axon, Schwann cell or the interstitium ([4, 10, 14]). In arsenic toxicity, alcohol toxicity, beriberi and carcinomatous neuropathy the primary attack is on the axon or the anterior horn cell. Diseases affecting the Schwann cells such as diphtheritic and lead neuropathies result in segmental demyelination. When the interstitial tissues are primarily affected by vascular disorders or infection, the nerve fibers may be affected by direct involvement or pressure or ischemia. In neuropathies where myelin or interstitial tissue is the primary site of involvement, the changes in the nerves produce interruption of axonal function; only later does actual degeneration occur. Such axons are capable of recovery. Electrical studies show the following characteristics: (a) when the nerve is stimulated distal to the site of lesion, the muscles supplied by it contract (b) electromyography studies reveal electrical silence at rest or few fibrillation potentials if some nerve fibers are degenerated, and (c) conduction velocity studies show slowing or block at the level of lesion.

On the basis of impaired function, neuropathies may be classified as (a) predominantly motor neuropathy, (b) distal sensory neuropathy, (c) distal sensory motor neuropathy, (d) mononeuritis, and (e) mononeuritis multiplex ([15]). The sensory and motor deficit in leprosy can be explained on the two basic types of neuropathies, viz., distal sensory polyneuropathy and mononeuritis or mononeuritis multiplex ([2]). The pattern of mononeuritis multiplex in leprosy has been studied clinically and pathologically and is fairly well established. The following are the common sites of the lesion in the nerve trunks ([1]): (a) the ulnar nerve in the lower half of the upper arm, (b) the ulnar nerve just above the wrist, (c) the median nerve just above the wrist, (d) the common peroneal nerve at the level of knee joint above the neck of the fibula, (e) the anterior tibial nerve just above the ankle joint. (f) the posterior tibial nerve just above the ankle joint, and (g) zygomatic branch of facial nerve in the region of zygoma. Occasionally the radial nerve as it emerges from the triceps muscle, the median nerve in the upper arm and the whole of the facial nerve in the facial canal are damaged. The etiologic classification is based on the association of neuropathy to known disease, toxins, trauma or biochemical disorders. The associated disease does not always explain the cause of the nerve damage ([4]). In leprosy since the lesion is often where the nerve is superficial ([9]), Brand ([1]) has put forward the theory that it is the temperature variation which is the major factor in determining the site of the lesions. Other possible factors are: (a) pressure from thickened sheath and interstitial fibrous tissue, (b) pressure on the inflammed nerve by ligaments such as the carpal tunnel or the fibrous band at the elbow, (c) immunologic and (d) vascular causing ischemia of nerve.

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MATERIALS AND METHODS

Since the sensory deficit in many cases is due to the distal sensory neuropathy and is not always due to the lesion in the nerve trunks, this study which was aimed at understanding the electrophysiologic changes in the nerve trunks was restricted to motor fibers ([13]). Motor conduction velocities of 156 ulnar nerves of 96 leprosy patients were recorded. Of the 156 nerves, 71 were without any clinical evidence of enlargement or tenderness or associated muscle weakness. Of these 71, seven had some sensory loss in the hands and the other 64 had normal sensation. Eighty-five of the total ulnar nerves studied had clinical evidence of nerve involvement either in the form of thickening and/or tenderness and/or muscle weakness. These nerves were classified into three groups: (a) typical tuberculoid, (b) typical lepromatous, and (c) the "intermediate" group. The "intermediate" group of this study included indeterminate as well as borderline (dimorphous) cases. There were 10 lepromatous, 29 tuberculoid and 46 "intermediate" nerves. Motor conduction velocity studies were done on 19 median nerves of 12 leprosy patients. Of these, 10 were tuberculoid and nine belonged to the "intermediate" group. Fourteen had clinical evidence of nerve involvement either in the form of enlargement and/or tenderness and/or muscle weakness. Five were the normal nerves of patients who had clinical evidence of contralateral neuritis.

A clinical examination was done before electrical studies were begun to note whether there was any tenderness or enlargement in the nerve. Detailed sensory and motor assessments were also made. A Medelec four channel electromyography machine was used for electromyography and conduction velocity studies. In the case of ulnar nerves, electromyographic studies of the flexor carpi ulnaris, the abductor digiti minimi and the first dorsal interosseus were done. Motor conduction velocity studies of the ulnar nerves were recorded by inserting a coaxial needle electrode in the abductor digiti minimi and stimulating the nerve at the following sites; (a) at about 3 cm. above the distal wrist crease, (b) 2 cm. below the medial epicondyle which is the lowest end of the superficial segment of the nerve, and (c) at the posterior fold of the axilla. The conduction velocity of the ulnar nerve in the upper arm which is the usual site of lesion in leprosy and the conduction velocity of the nerve in the forearm which is below the site of lesion were thus obtained. Median nerve

TABLE 1. *Conduction velocities of upper arm and forearm segments in relation to classification.*

Conduction velocity in meters per second	Upper arm				Forearm			
	Leprom-atous	Tuber-culoid	Inter-mediate	Total	Leprom-atous	Tuber-culoid	Inter-mediate	Total
0–10	Nil	3	6	9	Nil	Nil	Nil	Nil
11–20	2	2	4	8	Nil	1	1	2
21–30	2	4	8	14	1	1	Nil	2
31–40	3	6	7	16	Nil	2	2	4
41–44	2	6	4	12	Nil	1	2	3
45–49	Nil	2	6	8	2	Nil	8	10
50 & above	1	6	11	18	7	24	33	64
Total	10	29	46	85	10	29	46	85

TABLE 2. *Conduction velocities of the upper arm segments of the ulnar nerves in relation to thickening, muscle grading and presence of fibrillation potentials in the nerves.*

Conduction velocity in meters per second	Total	Thickening			Muscle grading				Presence of fibrillation (definite evidence of axon degeneration)
		Nil or mild	Moderate	Severe	No weakness Gr. 5	Mild weakness Gr. 4	Moderate weakness Gr. 1–3	Clinically paralyzed Gr. 0	
0–10	9	Nil	6	3	1	Nil	7	1	8
11–20	8	Nil	6	2	1	1	5	1	4
21–30	14	2	9	3	4	2	8	Nil	7
31–40	16	3	12	1	5	3	7	1	9
41–44	12	1	9	2	3	1	8	Nil	5
45–49	8	1	6	1	2	3	2	1	1
50 & above	18	7	11	0	12	2	4	Nil	3
Total	85	14	59	12	28	12	41	4	38

studies were done by inserting a coaxial needle electrode in the abductor pollicis brevis and stimulating the nerve at the distal wrist crease and at the proximal end of the superficial segment of the median nerve in the forearm which is five to ten centimeters proximal to the distal wrist crease. The conduction time from the distal wrist crease to the muscle action potential and the conduction velocity of the nerve between the two stimulating points were recorded.

Results. Of the 71 ulnar nerves without clinical evidence of nerve involvement, 69 had conduction velocities above 49 M/sec. (meters/second) in both the upper and forearm segments, the range being 50–79 M/sec. The other two had 43 M/sec. and 48 M/sec. respectively in the upper arm segments and 50 M/sec. and 65 M/sec. respectively in the forearm segments. The results of clinical examination, electromyography and conduction velocity studies of the 85 ulnar nerves with clinical evidence of neuropathy are tabulated in Tables 1 and 2.

Results of median nerve studies. The conduction delay from the wrist to muscle action potential was less than four seconds for 16 of the median nerves tested, the range being 2.3 to 3.6 M/sec. This value can be considered as normal. For two nerves the values were 4.6 M/sec. and 5.2 M/sec. respectively. These two nerves had marked slowing of the conduction velocity in the lower third of the forearm, the values being 4 M/sec. and 12.2 M/sec. respectively. One median nerve with clinical signs of nerve involvement could not be excited at the wrist or in the forearm because of the high threshold of excitability of the nerve. When this nerve was stimulated above the elbow at a distance of 38 cm. from the needle electrode, a conduction delay of 55 M/sec. was obtained indicating marked reduction in the conduction of the nerve.

DISCUSSION

Thomas and Lambert analyzed the records of 188 healthy adults and found that the conduction velocity of the forearm segment of the ulnar nerve ranged from 47 to

73 M/sec. with a mean of 60 M/sec. Thomas, Sears and Gilliat studied 46 subjects using coaxial needle electrodes and for their subjects, the conduction velocity ranged from 49–65.6 M/sec. Subsequent reports by others also give similar figures [3, 5, 6, 8]. Conduction velocity of the upper arm segment of the ulnar nerve is usually slightly higher than that of the forearm segment. For all practical purposes 50 M/sec. and above can be considered as normal for the upper arm segment and values below 45 M/sec. as slowing. In our preliminary studies on 12 normal subjects (not recorded here) the values without exception, were above 50 M/sec. Of the 71 leprosy patients without any clinical evidence of nerve involvement all but two had values above 49 M/sec. Analysis of the values for the 85 ulnar nerves with clinical evidence of nerve involvement indicates a marked slowing of the conduction velocity in the upper arm segment in a large percentage of these nerves. However 18 nerves had conduction velocities above 50 M/sec. Evidently some of the fast conducting fibers of these 18 nerves were not involved in the pathologic process (Table 3). Marked slowing of conduction velocity in the upper arm segments with normal conduction velocities in the forearm segments was a significant finding in 38 nerves. Eleven nerves had conduction velocities below 45 M/sec. (range being 12 M/sec. to 44 M/sec.) in both upper and forearm segments. In some of the nerves this may be due to neuritis in the segment of the nerve above the wrist but in certain nerves it could be due to changes in the myelin distal to the site of lesion in the upper arm which is seen in other localized lesions of peripheral nerves such as pressure syndromes. Marked slowing of the conduction velocity was seen in lepromatous, tuberculoid and "intermediate" nerves without significant variations. Though marked slowing was seen in more of the moderately enlarged nerves, the degree of slowing was not always in proportion to the thickening of the nerves. Six nerves without thickening had definite slowing of the conduction velocities. For 11 of the moderately thickened nerves conduction velocities were above 50 M/sec. Slowing of the conduction velocity (below 45 M/sec.) in 14 ulnar nerves without any muscle weakness was another significant observation. For six of these, values were less than 30 M/sec. and for two they were

TABLE 3. *Conduction velocities of 18 median nerves in the lower third of forearm in relation to clinical signs.*

Conduction velocity in meters per second	Muscle Gr. 5 No enlargement or tenderness in the nerves	Muscle Gr. 5 Enlargement and/or tenderness present	Muscle Gr. 4	Muscle Gr. 1–3
0–10	Nil	—	—	2
11–20	Nil	—	—	2
21–30	Nil	1	—	—
31–40	1	2	—	1
41–44	Nil	—	1	—
45–49	Nil	—	—	—
50 & above	4	3	—	1
Total	5	6	1	6

less than 20 M/sec. On stimulation of some of the ulnar nerves at the wrist (distal to the presumed site of lesion) good response was observed in the clinically paralyzed or very weak muscles indicating that the paralysis was due to axonal interruption without actual degeneration. These findings suggest that Schwann cell and/or the interstitial tissue is the primary target of the disease with later affection of the axon. Although the cause remains uncertain, diminished conduction in peripheral nerves is considered to be due to disorder of the myelin sheath ([7, 12]) and the cause may be pressure, ischemia, or metabolic or immunologic injuries. If the invasion of Schwann cells by leprosy bacilli is the cause of neuropathy in leprosy, it may be that infection interferes with the metabolism of Schwann cells resulting in demyelination or it may be that immunologic response (as in Guillain Barre syndrome or experimental allergic neuritis) is the major factor in causing demyelination ([10]). Inflammatory reaction in the interstitial tissue may cause damage to myelin sheaths and later to axons by direct pressure or by ischemia ([4]). There probably are multiple factors contributing to the pathogenesis of neuropathy in leprosy.

An interesting finding in the electrical study of median nerves was the marked slowing of the conduction velocity in the lower third of the forearm with normal conduction times from the wrist to the muscle potential. In carpal tunnel syndromes where pressure from the carpel tunnel is the cause of damage to the nerve, conduction time from wrist to muscle potential is increased without any slowing in the conduction velocity proximal to the pressure point. Since the conduction velocity of the median nerves in the lower third of the forearm is calculated for a short distance (5 cm. to 10 cm.) small errors in measuring the length of the nerve percutaneously and minor errors in the conduction time measurements can produce significant error in the conduction velocity values. Leaving an allowance for this experimental error, values below 30 M/sec. can be considered as definite slowing and values below 40 M/sec. as probable slowing.

Conclusion. Electrical studies such as (a) percutaneous stimulation of nerve distal to the site of lesion, (b) electromyography, and (c) conduction velocity studies are useful tools when used together with proper clinical examination, for evaluating the nature and severity of nerve damage in leprosy.

SUMMARY

Motor conduction velocity studies of 186 ulnar nerves and 19 median nerves were recorded. Seventy-one of the ulnar nerves and five of the median nerves were without clinical evidence of nerve involvement either in the form of enlargement or tenderness or associated muscle weakness. Eighty-five ulnar nerves and 13 median nerves had enlargement and/or tenderness and/or muscle weakness. Conduction velocities of the upper arm segments (a usual site of lesion in leprosy) and of the forearm segments were recorded separately. Marked slowing of the conduction velocity in the upper arm segment with or without slowing of the conduction velocity in the forearm segment was observed in a large percentage of involvement either in the form of thickening and/or tenderness and/or muscle weakness. This was seen in all types of leprosy without significant variation. Marked slowing of the conduction velocity in the upper arm segment was seen in many ulnar nerves without muscle weakness. Some of the clinically paralyzed muscles responded when the nerves were stimulated distal to the site of the lesion. In the case of median nerves, marked slowing of the conduction velocity was noticed in the lower third of forearm without slowing under the carpel tunnel or distally.

RESUMEN

Se registraron estudios de conducción motora de 186 nervios ulnares y 19 nervios medianos. Setenta y uno de los nervios ulnares y cinco de los nervios medianos no presentaban evidencia clínica de compromiso nervioso, ya sea en forma de engrosamiento, sensibilidad o debilidad muscular asociada. Ochenta y cinco nervios ulnares y 13 nervios medianos mostraban engrosamiento y/o sensibilidad y/o debilidad muscular.

La velocidad de conducción de los segmentos del brazo (una localización corriente de lesiones de lepra) y de los segmentos del antebrazo se registraron separadamente. Se observó una marcada reducción de la velocidad de conducción en el segmento del brazo, con o sin disminución de la velocidad de conducción del segmento del antebrazo, en un alto porcentaje de los nervios ulnares con evidencia clínica de compromiso, ya sea en forma de engrosamiento y/o sensibilidad y/o debilidad muscular. Esto se encontró en todos los tipos de lepra sin variaciones significativas. En muchos nervios ulnares se observó una marcada reducción de la velocidad de conducción en el segmento del brazo, sin debilidad muscular. Algunos de los músculos paralizados clínicamente respondieron cuando los nervios se estimularon distalmente al sitio de la lesión. En el caso de los nervios medianos, se notó una marcada disminución de la velocidad de conducción en el tercio inferior del antebrazo, sin disminución del tunel del carpo o distal.

RÉSUMÉ

On a enregistré le résultat d'études de la vitesse de conduction motrice au niveau de 186 nerfs cubitaux et de 19 nerfs médians. Parmi les nerfs cubitaux, 71, et parmi les nerfs médians, 5, ne présentaient pas de signes cliniques d'atteinte nerveuse, soit sous la forme d'épaississement ou de sensibilité à la palpation, ou encore de faiblesse musculaire associée. Quatre-vingt cinq nerfs cubitaux, et treize nerfs médians, présentaient soit un épaississement, soit de la douleur à la palpation ou de la faiblesse musculaire, ou encore une association de deux de ces symptômes ou ces trois symptômes à la fois. Les vitesses de conduction au niveau du segment supérieur du bras, qui constitue le site habituel de lésions dans la lèpre, ainsi qu'au niveau de divers segments de l'avant-bras, ont été enregistrées séparément. Dans un large pourcentage des nerfs cubitaux, présentant des signes cliniques d'une atteinte d'épaississement, de douleur à la palpation ou de faiblesse musculaire, ou d'association de ces symptômes tels que décrits ci-dessus, on a observé un ralentissement marqué de la vitesse de conduction au niveau du segment supérieur du bras, avec ou sans ralentissement de la vitesse de conduction au niveau de l'avant-bras. Ces résultats ont été relevés dans tous les types de lèpre, sans variation significative. Un ralentissement marqué de la vitesse de conduction au niveau du segment supérieur du bras a été observé dans beaucoup de nerfs médians sans que l'on relève en même temps de faiblesse musculaire. Certains des muscles cliniquement paralysés ont rèpondu lorsque les nerfs étaient stimulés à un niveau distal par rapport à l'endroit de la lésion. Dans le cas des nerfs médians, un ralentissement marqué de la vitesse de conduction a été noté au niveau du tiers inférieur de l'avant-bras, sans que ceci soit accompagné par un ralentissement au niveau du tunnel du carpe ou plus distalement.

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