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ELECTROMYOGRAPHIC DIAGNOSIS OF LEPROSY

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SUMMARY — Eighty untreated patients suspected to have leprosy were submitted to neurophysiological examination and later compared with the clinical diagnosis. Among the patients who had leprosy confirmed, 98% had EMG abnormalities. Motor and sensory amplitude reduction was the earliest and the most frequent abnormality. Low conduction velocity of the ulnar nerve across the elbow was present in over 55% of the patients. A «mosaic» peripheral polyneuropathy was the most characteristic finding, and seems to be helpful to the diagnosis of leprosy. All of the clinical forms showed EMG abnormalities, and even some asymptomatic contacts, however the abnormalities increase from the indetermined and tuberculous to the borderline and Virchow's forms.

Diagnóstico eletromiográfico da lepra.

RESUMO — Hanseníase tem sido considerada doença dermatológica, embora sempre provoque lesões de nervos periféricos e nem sempre provoque lesões dermatológicas. O diagnóstico de hanseníase jamais seria considerado sem alterações sensitivas. Partindo desse princípio, 80 pacientes com suspeita de hanseníase, não tratados anteriormente, foram submetidos a cuidadoso exame eletrofisiológico na busca de lesões de nervos periféricos. Cerca de 98% dos pacientes em que hanseníase foi confirmada pelos métodos tradicionais apresentaram alterações eletromiográficas. O achado mais comum e precoce foi redução da amplitude das respostas motoras e sensitivas, usualmente duas vezes mais freqüente que redução nas velocidades de condução nervosa no mesmo nervo. O nervo mais freqüentemente alterado foi o ulnar, em que a amplitude sensitiva estava reduzida em 62% dos pacientes e a velocidade de condução nervosa sensitiva em apenas 29%. A velocidade de condução nervosa motora do nervo ulnar no canal cubital estava reduzida em 55% dos pacientes (síndrome do túnel cubital). Síndrome do túnel carpiano foi observada em 16% dos pacientes. Alterações eletromiográficas foram registradas em todas as formas clínicas, discretas na forma indeterminada, moderadas na forma tuberculóide e severas nas formas dimorfa e virchowiana. Surpreendentemente, alguns contatos assintomáticos apresentaram alterações eletromiográficas, mais freqüentemente a síndrome do túnel cubital, o que sugere a possibilidade de uma «cicatriz» neurofisiológica: o «complexo primário» da hanseníase. Alguns desses contatos poderiam ainda pertencer à forma I, incipiente. Juntamente com a síndrome do túnel cubital e a redução da amplitude sensitiva no território do nervo ulnar, distribuição em «mosaico» das lesões nervosas foi o achado mais característico da hanseníase (polineuropatia em mosaico). Nossos achados evidenciam a utilidade da eletromiografia no diagnóstico da hanseníase, em qualquer estágio ou forma, particularmente nos estágios iniciais, quando os testes atualmente em uso são ineficientes.

Although leprosy always produces peripheral nerve damage, it remains the concept of dermatological disease. A systematic and prospective neurophysiological study of peripheral nerve involvement in leprosy has not been performed yet. Several authors have demonstrated sensory and motor nerve conduction velocity abnormalities,

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besides reduction of sensory and motor action potential amplitudes [4,6,7,10,14,18,19]. Histopathological abnormalities of peripheral nerve are well known, and demyelination has been described by many authors [5,14,16,19]. There are evidences that the earliest nerve lesion in leprosy occurs in the non myelinated and small diameter myelinated fibres, and axonal neuropathy has been suspected [1,4,11,15,16,19].

Since the diagnosis of leprosy necessarily implies in sensory disturbance and very often in motor dysfunction, we decided to study the disease as peripheral neuropathy.

MATERIALS AND METHODS

Eighty untreated patients, referred to the Department of Infectious Diseases of the Secretary of Health of Goias' State, suspected of suffering leprosy, underwent neurophysiological studies. All of them went later through routine procedures for Hansen's disease, which included physical examination, skin biopsy, bacilloscopy, Mitsuda test and, occasionally, nerve biopsy.

Orthodromic bilateral sensory study was performed at all of the digital branches of the median nerve (thumb, fore, middle and ring fingers), at the two branches of the ulnar nerve (ring and little fingers) and at the radial nerve (thumb). Antidromic study was done at the sural nerve. Recording electrodes consisted of silver discs, each disc being 4 mm in diameter, separated from each other by 28 mm, placed over the median, ulnar and radial nerves, proximal to the flexor skin crease, and just posterior to the lateral maleolus over the sural nerve. These electrodes have acute border to mark the skin, in order to permit more accurate measurements. The stimulating electrodes were a pair of clips. The cathode was placed at the distal phalanx of the thumb and at the middle phalanx of the fore, middle, ring and little fingers. The anode was placed at the proximal phalanx, as close as possible of the palm, not touching it. The sural nerve was stimulated with the disc electrodes at the calf, 10 to 12 cm above the recording electrodes.

Evoked muscle responses were recorded with silver strips 45 mm long and 4 mm wide placed over the end plate zone of the abductor policis brevis muscle (APB) for the median nerve, abductor digiti minimi muscle (ADM) for the ulnar nerve, extensor digitorum muscle (EDB) for the peroneal nerve and plantar muscles for the posterior tibial nerve [9]. Stimulating electrodes were of the same type used for recording sensory responses.

The median nerve was stimulated at the wrist and at the lower extremity of the arm, just besides the internal border of the biceps brachial tendon. The motor conduction velocity of the median nerve was measured along the forearm. The ulnar nerve was stimulated at the wrist, at the elbow, just below the cubital tunnel, and 10 to 12 cm above the elbow. The motor conduction velocity of the ulnar nerve was measured from the elbow to wrist and across the elbow. The peroneal nerve was stimulated at the ankle and just below the head of the fibula. The motor conduction velocity of the peroneal nerve was measured along the leg. The tibial nerve was stimulated at the ankle. Instead of the conduction velocity, the H-reflex was used to evaluate the function of the peroneal nerve.

Measurements were made directly from the EMG machine (Polimed PL 1002) which has storage oscilloscope with maximal amplification of 5 UV/cm and latency measurer varying with 0.1 ms. Electrical stimuli for motor and sensory fibres consisted of rectangular voltage pulses, 50 to 100 us in duration. Amplitude was measured peak to peak both for sensory and motor responses. Latency was measured from the onset of the stimulus artifact to the initial negative peak, always with a high gain, to increase the accuracy. The responses were registered from the oscilloscope by an Apple computer (Unitron AP II). Hands temperature was kept between 30 and 34 degrees centigrades. When necessary they were heated with infrared lamp. A control group of 20 nerves from 12 healthy person was done.

RESULTS

The motor and sensory amplitude and conduction velocity of the control group (Table 1) is within the average of the literature [2,3,8,10,13,17]. Since we were interested in the abnormal findings, we have used the lower limit of normal. Verghese et al. [18] have admitted in their study on leprosy: «for all practical purpose 50 m/s and above can be considered as normal for the upper arm segment». The following values, and above, have been considered normals. Motor and sensory conduction velocities: 50 m/s for the upper limbs (except for the thumb which was considered normal above 44 m/s); 40 m/s for the lower limbs. Motor amplitudes:

Nerve		Thumb		Fore	Middle	Ring	Little
Median	SA	68 24		50 12	66 18	38 12	
	SV	52 05		59 04	59 03	59 04	
	MA	(thenar)	14 02				
	MV		61 04				
Ulnar	SA					29 06	27 07
	SV					60 04	59 05
	MA	(hipothenar)	14 02				
	MV		61 05				
	EV		58 05				
Radial	SA	31 07					
	SV	52 04					
Peroneal	MA		07 01				
	MV		47 04				
Tibial	MA		09 02				
	HR		42 03				
Sural	SA		31 12				
	SV		50 03				

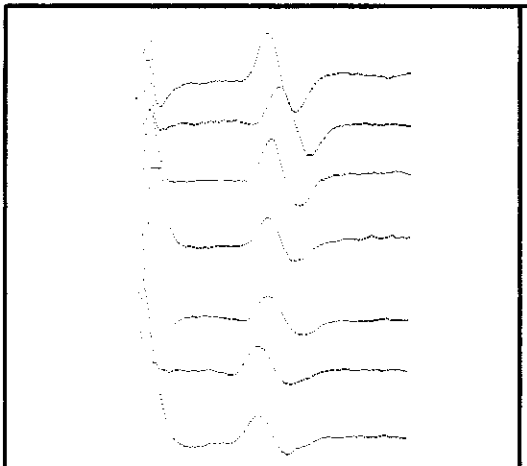
Table 1 — Controls. SA, sensory amplitude; SV, sensory velocity; MA, motor amplitude; MV, motor velocity; EV, elbow velocity; HR, H-reflex. All responses are the mean plus or minus standard deviation.

APB muscle (median nerve) 9.0 mV, ADM muscle (ulnar nerve) 10.0 mV,EDB muscle (peroneal nerve) 6.0 mV, Plantar muscle (tibial nerve) 7.0 mV. Sensory amplitudes: Thumb (median nerve) 20 uV, Fore (median nerve) 30 uV, Middle (median nerve) 30 uV, Ring (median nerve) 15 uV, Ring (ulnar nerve) 15 uV, Little (ulnar nerve) 15 uV, Thumb (radial nerve) 15 uV.

The routine examination excluded 9 patients considered either normal or with another pathology but leprosy. Fifteen were contacts (three of them had leprosy confirmed later) and 59 had leprosy confirmed (including the three contacts). The patients were classified according to the international classification in: 1. indetermined leprosy (I form), 13; 2. tuberculoid leprosy (T form), 18; 3. borderline leprosy (D form), 23; 4. lepromatous leprosy (V form), 5.

From the 59 patients with the clinical and laboratorial diagnosis of leprosy 56 (95%) had some EMG abnormality. The three who had complete normal EMG belonged to the indetermined form, and one of them had only paresthesia on the face. The other admitted that she was on treatment for a long time and the last one actually had sensory conduction velocity slightly reduced; however, because we were not able to keep her skin temperature warm, we decided to disregard this abnormality. All of the contacts had subjective complains of paresthesias (numbness and tingling) and spontaneously looked for healthy care; most of them had EMG abnormalities (Table 2). Contrary to many authors [1,7,10,18], who have found reduced conduction velocity as the most frequent EMG abnormality, we have low amplitude as the most common abnormal finding, both in the pool and in each clinical form itself (Fig. 1).

Ulnar sensory amplitude (pool) was either reduced or not detectable in 62% of patient nerves, while ulnar sensory conduction velocity was reduced only in 29% of them. The same relationship was observed for all of the nerves but the sural, where the amplitude was reduced in 33% of the patients and the conduction velocity was reduced in 45% of them (Table 2). The same behaviour was observed at the motor nerves. The ratio between the percentage of reduction of the motor amplitude and the percentage of reduction of motor conduction velocity was (pool): Posterior tibial nerve, 58/16; Median nerve, 40/6; Peroneal nerve, 44/6; Ulnar nerve (forearm), 29/6. The exception, and one of the most frequent finding was the motor conduction velocity of the ulnar nerve across the elbow (in the



NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
median	thumb	20 uV	1.9 ms	10.1 cm	53 m/s
median	fore	60 uV	2.0 ms	12.4 cm	62 m/s
median	middle	65 uV	2.0 ms	12.2 cm	61 m/s
median	ring	40 uV	1.9 ms	11.8 cm	62 m/s
ulnar	ring	30 uV	1.9 ms	12.2 cm	64 m/s
ulnar	little	30 uV	1.7 ms	11.2 cm	66 m/s
radial	thumb	30 uV	1.7 ms	10.6 cm	62 m/s

FIG-1A

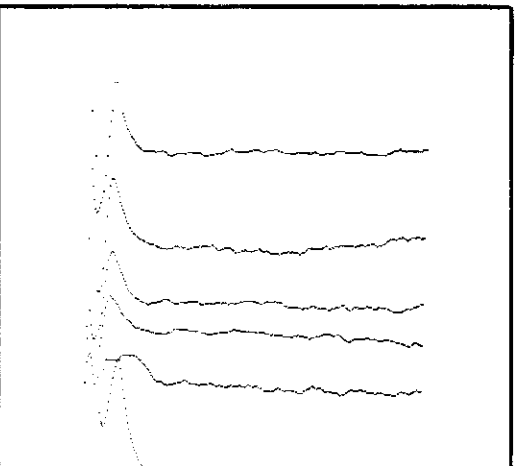
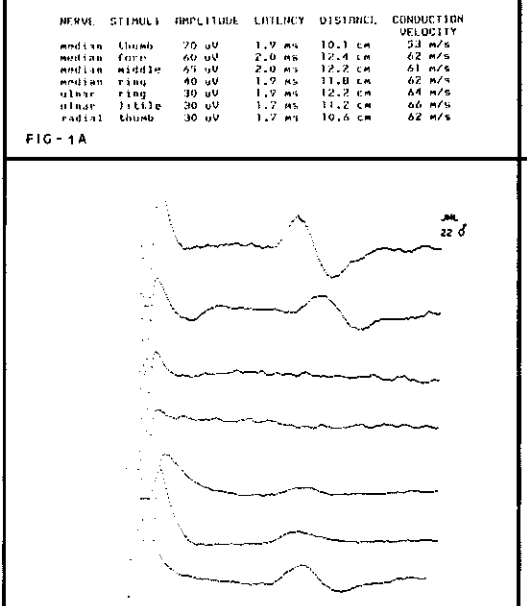
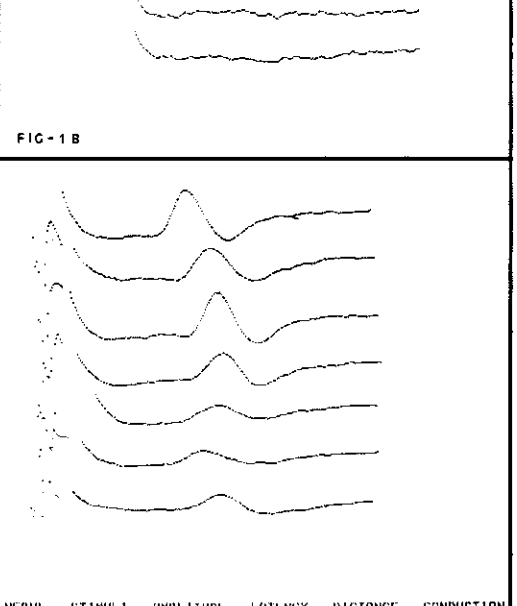


FIG-1B



NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
median	thumb	40 uV	2.3 ms	9.4 cm	41 m/s
median	fore	25 uV	2.6 ms	12.5 cm	48 m/s
median	middle	no	detectable	response	
median	ring	no	detectable	response	
ulnar	ring	4 uV	2.3 ms	12.0 cm	52 m/s
ulnar	little	0 uV	2.2 ms	10.6 cm	48 m/s
radial	thumb	19 uV	2.5 ms	9.8 cm	39 m/s

FIG-1C



NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
median	thumb	40 uV	1.9 ms	9.7 cm	51 m/s
median	fore	25 uV	2.0 ms	12.0 cm	60 m/s
median	middle	40 uV	2.2 ms	12.0 cm	55 m/s
median	ring	25 uV	2.2 ms	12.0 cm	55 m/s
ulnar	ring	10 uV	2.1 ms	11.9 cm	56 m/s
ulnar	little	10 uV	1.9 ms	10.5 cm	55 m/s
radial	thumb	19 uV	2.2 ms	11.0 cm	50 m/s

FIG-1D

Fig. 1 — Sensory responses on the digital branches of median, ulnar and radial nerves. A, normal control. B, lepromatous form: no detectable responses. C, tuberculoid form: "mosaic" distribution of nerve damage. D, contact: ulnar sensory loss.

Nerve		Total	R	%R	%RI	%RT	%RD	%RV	%RC	%RO
Median	SA	102	47	46	32	37	60	50	7	33
	SV	101	37	37	27	31	46	37	7	37
	MA	98	39	40	31	35	47	37	38	50
	MV	98	6	6	5	3	7	12	0	17
Ulnar	SA	102	63	62	32	56	90	50	42	55
	SV	101	29	29	0	22	49	37	0	22
	MA	103	30	29	9	12	50	44	17	22
	MV	103	6	6	0	3	10	11	0	0
	EV	103	57	55	29	51	67	78	37	11
Radial	SA	98	37	38	19	29	54	43	12	55
	SV	97	27	28	5	16	47	43	0	22
Peroneal	MA	70	31	44	37	30	58	50	21	33
	MV	70	4	6	0	0	9	50	0	33
Tibial	MA	52	30	58	50	54	67	0	52	83
	HR	49	8	16	0	9	29	0	4	40
Sural	SA	84	28	33	0	29	46	50	12	33
	SV	84	38	45	0	29	71	67	12	50

Table 2 — Patients. Patients number, 80; HD confirmed, 59; excluded leprosy, 9; contacts, 15 (3 had HD confirmed). SA, sensory amplitude; SV, sensory conduction velocity; MA, motor amplitude; MV, motor conduction velocity; EV, velocity at the elbow; HR, H-reflex; R, reduced; I, indetermined; D, borderline; T, tuberculoid; V, lepromatous; C, contacts.

cubital tunnel) which was reduced in 55% of the nerves, while the motor conduction velocity along the forearm was normal in 94% of these nerves (Table 2) (Fig. 3). The H-reflex was reduced in 16% of the plantar muscles (tibial nerve), while motor responses were reduced in 58% of the muscles.

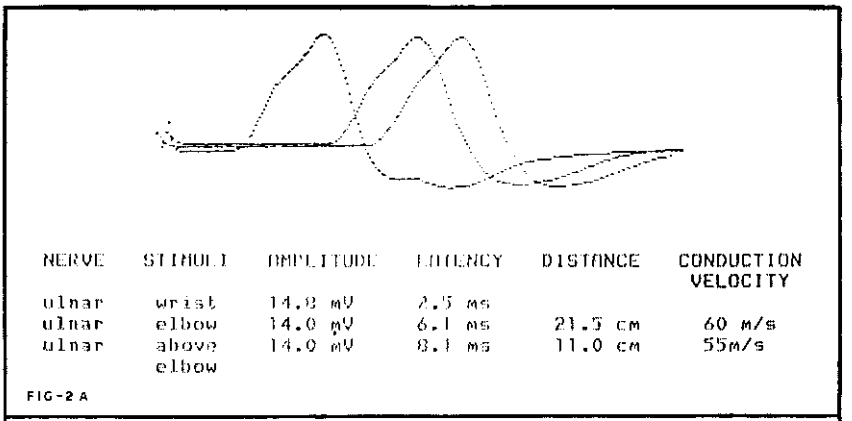
If we look at the patients through the different clinical forms, there is an obvious predominance of motor and sensory denervation among the borderline (D) and lepromatous (V) forms over the tuberculoid (T) and indetermined (I) forms. As it was expected, the denervation declines from the T form to the I form, being less intense among the contacts.

Actually the contacts revealed a surprisingly high incidence of denervation, rising up to 42% for the ulnar sensory amplitude (Table 2) (Figs. 1 and 2). However, the ulnar conduction velocity was normal in 100% of the ulnar contact nerves and in 92% of their median nerves. The ulnar conduction velocity across the elbow was reduced in 37% of the contacts (including those who had the disease confirmed later).

Few patients belonging to the I and T forms who had only hypoesthesia at the elbow, and with EMG findings either normal or slightly abnormal at that arm, showed quite abnormal cerebral evoked responses when stimuli were applied at the hypoesthesic area, and compared with the response of the normal side (Fig. 3), suggesting the possibility of lesion at the skin receptors, preceding the nervous trunk damage in these cases.

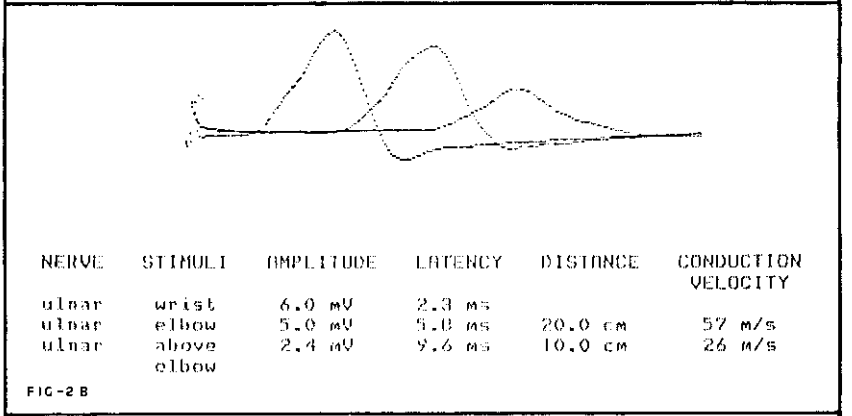
Carpal tunnel syndrome was observed in 46% among the patients (pool) and in 8% among the contacts (Table 3), most of them superposed the cubital tunnel syndrome. However, 26% of the carpal tunnel happened without ulnar nerve involvement. Contrary to what one observes among the population, there was no higher incidence of carpal tunnel syndrome in the females, suggesting its association with the leprosy.

Asymmetry of nerve involvement was found in almost all of the patients. A randomic distribution of the nerve lesions, sometimes affecting digital branches of one nerve and



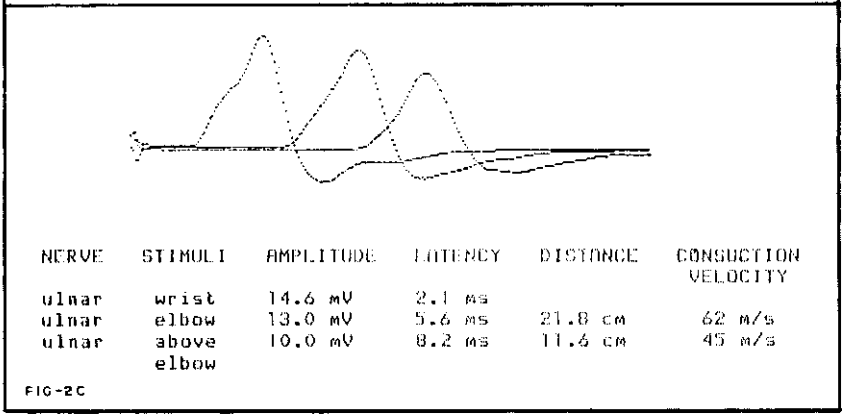
NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
ulnar	wrist	14.8 mV	2.5 ms		
ulnar	elbow	14.0 mV	6.1 ms	21.5 cm	60 m/s
ulnar	above elbow	14.0 mV	8.1 ms	11.0 cm	55m/s

FIG-2 A



NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
ulnar	wrist	6.0 mV	2.3 ms		
ulnar	elbow	5.0 mV	5.8 ms	20.0 cm	57 m/s
ulnar	above elbow	2.4 mV	9.6 ms	10.0 cm	26 m/s

FIG-2 B



NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
ulnar	wrist	14.6 mV	2.1 ms		
ulnar	elbow	13.0 mV	5.6 ms	21.8 cm	62 m/s
ulnar	above elbow	10.0 mV	8.2 ms	11.4 cm	45 m/s

FIG-2C

Fig. 2 — Motor responses of the ulnar nerve (ADM muscle). A, normal amplitude and conduction velocity. B, lepromatous form: reduced amplitude and reduced conduction velocity across the elbow. C, contact: normal amplitude and reduced conduction velocity across the elbow.

sparing other digital branches of the same nerve (Fig. 1C) was the most striking finding. The figure of this particular distribution of the denervation reminds a mosaic, so we have called it «mosaic» polyneuropathy.

Pool	38 %
Contacts	8 %
Indetermined (I)	38 %
Tuberculoid (T)	44 %
Borderline (D)	48 %
Virchow's (V)	40 %
I + T + D + V	46 %
Male	52 %
Female	48 %
Carpal tunnel syndrome without cubital tunnel syndrome	26 %

Table 3 — Incidence of carpal tunnel syndrome among the different forms of leprosy.

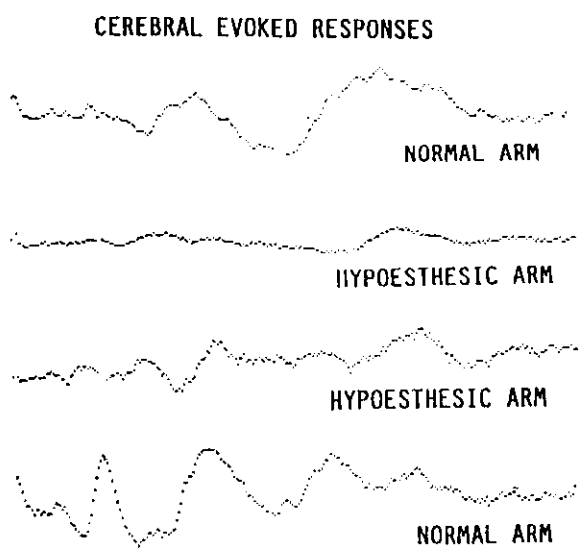


Fig. 3 — Severely reduced amplitude and prolonged latency of the cerebral evoked responses in a patient from the tuberculoid form.

COMMENTS

Full and precise EMG examination seems to be extremely helpful to the diagnosis of leprosy, since we can demonstrate neurophysiological abnormalities in at least 95% of the patients, including those of the indetermined form, considered not having nerve damage. Actually abnormal nerve conduction velocity has been shown by Donde et al.[4] in a third of clinically normal patients nerve, and by Karat et al.[7] and Verghese et al.[18] in nerves of leprosy patients and some contacts. The discrepancy between the EMG abnormalities and the clinical findings is obviously due to the higher sensibility of the neurophysiological methods, which are able to detect subclinical nerve dysfunctions.

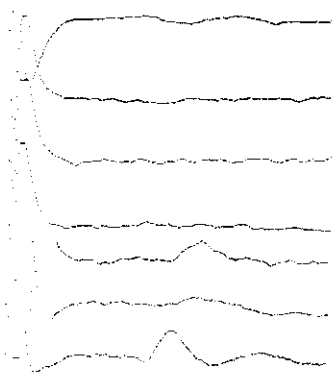
In fact we found reduced amplitude responses as the most frequent abnormal finding, usually twice as much frequent as the reduced conduction velocity in the same nerve (except for the sural nerve) (Table 2). Since root lesion may produce peripheral denervation, particularly motor amplitude reduction in the APB muscle, in the EDB muscle and in the plantar muscle, we must consider that some of our patients may have root lesion besides leprosy. However the great improvement of amplitude responses with specific treatment is an evidence that the method must be worthwile (Fig. 4). Apparently the drop of amplitude precedes the conduction velocity reduction, as we can see by the difference of incidence of the two abnormalities (Table 2), and comparing the ratio between these abnormalities in the different forms of the disease. The percentage of reduction of the ulnar sensory responses (pool) over the percentage of reduction of ulnar sensory conduction velocity was: indetermined form, 32/0; tuberculoid form, 56/22; borderline form, 90/49. We are considering that the patients must pass through the indetermined form before reaching either the tuberculoid or the borderline forms.

These findings are suggestive of axonal neuropathy which may occur without the presence of bacilli, as was described by Shetty et al.[16], possibly due to antibodies against nerve components. Very often the motor and sensory denervation were found both in asymptomatic patient nerves, as has been reported[4,6,12], or asymptomatic contacts.

Among the patients who have already returned to control, after 4 months under Dapsone therapy, someone have shown nearly complete recovery of nerves which had no sensory and low motor amplitude responses (Fig. 4). This finding permits the supposition that a functional blocking plays an important role at the initial nerve lesion in leprosy, which may precede the axonal and the demyelinating lesions. Towards this supposition is the fact that in the same patient, in the same limb while the median nerve has recovered, the ulnar sensory responses remain unaltered (Fig. 4). Again we are assuming that the ulnar sensory amplitude is the earliest neurophysiological lesion to happen. Some other patients, even with clear dermatological improvement, exhibit nerve deterioration suggesting the possibility of either immunological or inflammatory process provoked by the treatment (Fig. 5).

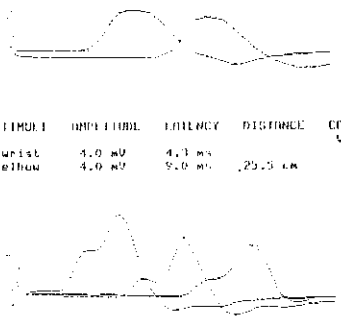
Some contacts, untreated, remain with abnormal findings, particularly low ulnar conduction velocity across the elbow and reduced ulnar amplitude sensory responses, after one year of follow up, suggesting either a very mild and stationary peripheral neuropathy or, more likely, the neurophysiological scar of the primary infection (leprosy primary complex). It seems that the high incidence of EMG abnormalities among the contacts is caused by the difficulties in making the diagnosis of leprosy in the early stages. Surely many of the patients regarded as contact in this study actually belong to an initial stage of leprosy, as we could see when 3 of 15 (20%) had leprosy clinically confirmed later.

Conclusions — (1) Leprosy is a «mosaic» primary peripheral polyneuropathy, involving motor and sensory fibres. (2) The earliest nerve lesion seems to be a functional blocking, followed by asymmetric axonal neuropathy, represented by reduced motor and sensory amplitudes with normal conduction velocities. (3) Diffuse demyelination (reduced conduction velocities) apparently is a late stage of the nerve lesion, affecting more severely and frequently the ulnar nerve in the cubital tunnel and the median nerve in the carpal tunnel. (4) The neuropathy involves all of the clinical forms, including some contacts; the borderline and lepromatous forms are the most



NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
median	thumb	3 uV	2.9 ms	8.9 cm	31 m/s
median	fore	no detectable		responses	
median	middle	no detectable		responses	
median	ring	no detectable		responses	
ulnar	ring	3 uV	2.3 ms	11.2 cm	51 m/s
ulnar	little	1 uV	2.1 uV	10.1 cm	48 m/s
radial	thumb	10 uV	1.9 uV	9.3 cm	48 m/s

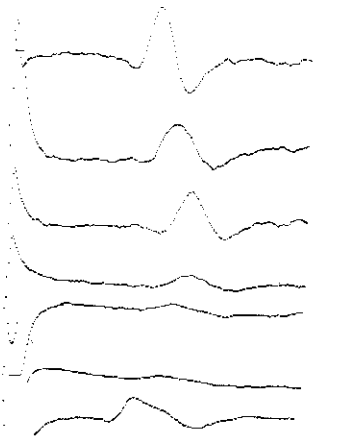
FIG-4A



NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
median	wrist	4.0 mV	4.3 ms		
median	elbow	4.0 mV	9.0 ms	25.5 cm	54 m/s

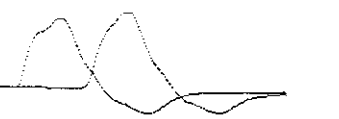
NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
ulnar	wrist	7.5 mV	2.7 ms		
ulnar	elbow	7.7 mV	6.5 ms	20.8 cm	55 m/s
ulnar	above	4.3 mV	10.3 ms	10.0 cm	26 m/s

FIG-4A1

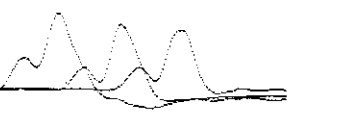


NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
median	thumb	70 uV	2.0 ms	9.0 cm	45 m/s
median	fore	35 uV	2.3 ms	11.8 cm	51 m/s
median	middle	40 uV	2.6 ms	11.9 cm	46 m/s
median	ring	8 uV	2.6 ms	11.3 cm	43 m/s
ulnar	ring	4 uV	2.3 ms	11.6 cm	50 m/s
ulnar	little	1 uV	2.2 ms	10.0 cm	45 m/s
radial	thumb	22 uV	1.9 ms	9.7 cm	51 m/s

FIG-4B



NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
median	wrist	7.0 mV	3.1 ms		
median	elbow	8.2 mV	7.3 ms	25.9 cm	62 m/s



NERVE	STIMULI	AMPLITUDE	LATENCY	DISTANCE	CONDUCTION VELOCITY
ulnar	wrist	7.0 mV	2.1 ms		
ulnar	elbow	6.0 mV	5.8 ms	21.9 cm	59 m/s
ulnar	above	1.0 mV	9.0 ms	10.5 cm	33 m/s

FIG-4B1

Fig. 4 — Borderline leprosy. A, sensory responses before treatment. A1, motor responses before treatment. B, sensory responses after 4 months under Dapsone therapy. B1, motor responses after therapy.

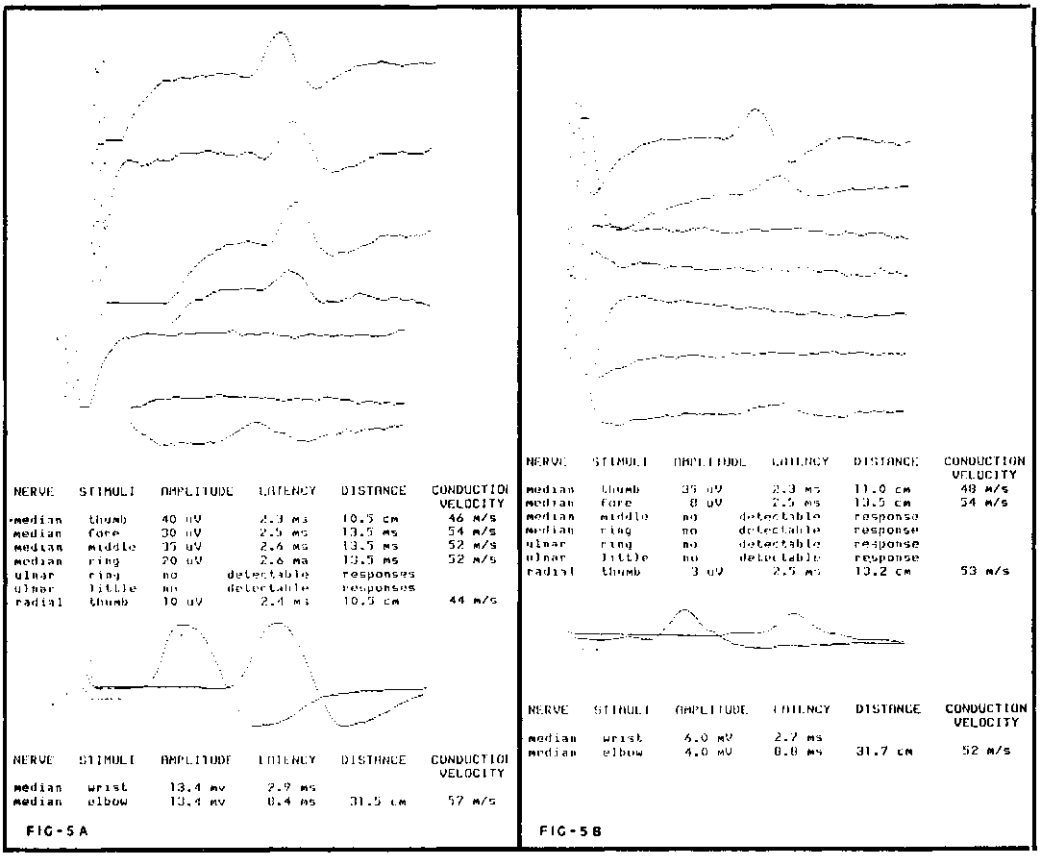


Fig. 5 — Bordeline leprosy. A, motor and sensory responses before therapy. B, motor and sensory responses after 4 months under Dapsone therapy.

widespread and severely affected. (5) The initial neuropathy promptly recovers with specific (Dapsone) therapy, however the old lesions are irreversible. (6) EMG seems to be a precise sensitive and precocious method for leprosy diagnosis and also helpful to evaluate the efficacy of therapy and to make the prognosis of nerve recovery.

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