AN ELECTRODIAGNOSTIC STUDY OF THE LOWER MOTOR UNIT IN LEPROSY [1, 2]

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The concept of rehabilitation in leprosy is one that has developed only during the past 20 years. Before then efforts had usually been directed at effecting a cure, and little thought had been given to the eventual reintegration of the patient suffering from leprosy into society. The acceptance of the idea of rehabilitation has been due largely to the ideas and work of Cochrane ([12]), Brand ([8, 9]) and Antia ([2]), though many others, principally in Asia and South America, have played an important part ([11, 16, 17, 18, 22, 25, 38–43, 46, 48]).

Mycobacterium leprae may damage not only skin, but also other organs, especially peripheral nerves, bone and the eye. Sensory loss and muscular weakness, with subsequent trophic changes and deformity, all common sequelae of leprosy, are accepted as the result of peripheral nerve damage. The lesion of the peripheral nerve may be either "constrictive" or "infiltrative," depending on the type or form of the disease.

An extensive review of the literature of the past 15 years failed to reveal any exact information about the state of muscle in leprosy from the histologic point of view. In such studies as had been performed, the changes found were considered to be secondary to nerve damage.

Electrodiagnosis has been used occasionally, in recent years, in evaluation of the motor unit in leprosy. However, the electrodiagnostic methods used have consisted mainly of the faradic-galvanic test and chronaximetry. These tests are based on the direct response (i.e., from the nerve point) or the indirect response (i.e., from the muscle motor point) to external electric stimulus and provide almost no information about the muscle itself. Gross pathologic changes in the peripheral nerve are required before abnormal reactions are obtained through these technics ([29]).

The purpose of the present study was to investigate the condition

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of the motor unit in leprosy through use of the electrodiagnostic equipment now available. We have tried to compare the accuracy and reliability of the various tests and to evaluate the importance of various clinical and laboratory data in relation to the nerve and muscle, as these structures are, from the medical point of view, of extreme significance for the final rehabilitation of the patient.

MATERIAL AND METHODS

The investigations were carried out in 80 leprosy patients, of whom 58 suffered from the lepromatous type, 8 from the tuberculoid type, 12 from the indeterminate form and 2 from the borderline form of the disease. These 80 patients comprise about half of the known leprosy cases in Israel. The remaining patients could not be included in the present study mainly because of lack of cooperation. Forty-four of the patients were male and 36 female; the age classification may be seen in Table 1 and an analysis of geo-ethnic origin in Table 2. Clinical evaluation in this study included general, dermatologic, neurologic, and ophthalmologic examinations (on the latter we will not elaborate). In the neurologic examination, emphasis was placed on muscle power, the presence of atrophy, superficial sensation (pain, touch, and temperature), periosteal and tendon reflexes, the presence of a Tinel sign, and of thickened nerves.

One of the major problems that arose at the beginning of the study was the need for definite criteria and standardization of the

TABLE 1.—*Classification of patients by age groups.*

Age group	Type of leprosy				
	Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
10–19 years	2	—	—	—	2
20–29 "	12	1	5	—	18
30–39 "	9	1	—	1	11
40–49 "	8	1	1	—	10
50–59 "	14	1	4	1	20
60 and over	13	4	2	—	19

TABLE 2.—*Classification of patients by ethnic regions of origin.*

Region	Type of leprosy				
	Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Middle East (including Israel)	29	3	8	1	41
Asia	18	4	4	1	27
North Africa	9	1	—	—	10
South America	1	—	—	—	1
Europe	1	—	—	—	1

clinical neurologic signs, since, in most of the cases, the degree of damage varied greatly from one site to another, from one nerve to another and even from one branch to another of the same nerve. Standardization was required in order to enable us to perform electrical tests on the same structures without having to choose a different site in each patient. Such a choice would, in our opinion, have prejudiced the objectivity of the results. In order to circumvent this difficulty, we chose a group of 4 peripheral nerves and 4 muscles, which were examined in each patient. These were the ulnar and median nerve on each side and the opponens pollicis and abductor digiti quinti muscles in each hand. Our choice was based on the following facts, which had been found in a pilot study:

1. These particular peripheral nerves and muscles had been found to be damaged in the majority of our patients.
2. Whenever loss of motor power was clinically evident in more than 6 muscles in the upper limbs, 2 of the weak muscles belonged to the selected group.
3. These peripheral nerves and muscles were usually among the first damaged, if any neurologic damage was present at all.
4. The selected nerves and muscles are of extreme functional importance in the hand, and, as such, their condition is of great significance in the social, economic, and psychologic state of the patient.
5. Successful rehabilitation of the patient usually presupposes a functional hand. Reconstructive surgery in the hand is often related to at least one of these structures, which are of basic functional importance.
6. Examination of the power of these particular muscles is technically easy and accurate and requires a minimum of understanding and cooperation from the patient.
7. Local atrophy of these muscles and thickening of these nerves, if present, are easy to ascertain and evaluate.
8. The technical approach for the electrical tests is comfortable, for the patient and physician alike.

The results obtained in the 80 patients will be presented therefore as related to the muscle power of 320 muscles (opponens pollicis and abductor digiti quinti, bilaterally), 320 possible sites of atrophy (thenar and hypothenar eminences, bilaterally), 320 possible thickened nerves (ulnar and median, bilaterally), and 320 possible Tinel signs (ulnar and median nerves, bilaterally). The Tinel sign was considered positive when pressure or percussion of the nerve point triggered a sensation of electric current or pain along the nerve, distal to the point of pressure ([14]). The sign points to a lesion of the nerve (regeneration most often) ([14, 28]), but it is not related to the degree of nerve damage or resultant muscle weakness. The results of examination of tendon and periosteal reflexes are not included, since

changes in or absence of reflexes occurred in a very low percentage of our patients, and, moreover, the condition of the reflex may be related to any part of the reflex arc, without being directly relevant to the condition of the motor unit. The same observations are pertinent to the results of the sensory testing, which bears no relation to the motor condition, except in extreme cases.

The laboratory evaluation of the patient consisted of investigations of the bacteriologic status in histologic preparations and smears, the immunologic status (lepromin test) and serologic status (WAR, Kahn test), sheep hemagglutination test (SHAT), and latex fixation test (LFT).[5] All the tests were performed on admission except the SHAT and LFT, which were not in regular use at the time of admission of many of the patients) and again at the time of the present study, just before the electric testing. Henceforth, we refer to the laboratory data as "at time of admission" and "at present." The average period between admission and "at present" was 4 years (one to 9 years).

While the present study was under way, 67 patients were receiving diaminodiphenylsulfones; 6, thiosemicarbazone; 3, diphenylthiourea; and 4 combined therapy

According to the clinical and laboratory data at the time of the present study, 22 of the 80 cases were active and 58 inactive.

The electrodiagnostic investigation of the motor unit included:

A. *Faradic-galvanic test (henceforth referred to as* FGT) *was performed on a Fischer apparatus.* Each examination included direct and indirect (i.e., from the nerve point and from the muscle motor point) testing of the ulnar and median nerves. For the ulnar nerve, the nerve point is located in the groove between the medial border of the olecranon and the medial epicondyle of the humerus; the median motor point is located either on the anterior aspect of the elbow next to the biceps tendon or on the anterior surface of the wrist next to the palmaris tendon. For the indirect test, the opponens pollicis motor point was located in the medial proximal third of the thenar eminence, and the abductor digiti quinti motor point in the proximal palmar aspect of the hypothenar eminence. Each motor point was tested first by faradic current (400 cycles per second) and then by galvanic current using a manual interrupter. The galvanic test included measurement of the rheobase (the minimal intensity that elicits a minimal muscle contraction without any time limit) and of the chronaxy (the shortest time in which a current with an intensity double that of the rheobase will elicit a minimal muscle contraction). The results of the FGT could be divided into three main groups:

1. Normal: the faradic stimulus caused a sustained tetanic

[5] For convenience in reference all abbreviations used are listed at the end of the text of this paper. See page 859.

reaction; galvanic stimulus caused a brisk muscular contraction, on both direct and indirect testing; the rheobase was less than 5 milliamperes (ma) and the chronaxy faster than 1 millisecond (ms).

2. Partial degeneration reaction (henceforth referred to as "partial DR"): faradic stimulus did not elicit any muscular contraction; galvanic stimulus triggered a brisk or sluggish muscle contraction; the rheobase was higher than 5 ma and the chronaxy above 1 ms.

3. Total degeneration reaction (total DR): no response to faradic or galvanic stimulus, either on direct or indirect testing (for purposes of convenience; the complete and absolute DR are included in this type of reaction).

The normal FGT is, in general, accepted as indicating a healthy nerve, though it cannot diagnose or evaluate fine nerve lesions. The partial DR indicates nerve damage of various degrees; it should be stressed that when the nerve lesion is severe the galvanic current actually stimulates the muscle fibers directly, as evidenced by the sluggish contraction and deviation of the point of lowest intensity of current from the motor point to the point of highest muscle fiber concentration. A total DR obviously implies that the number of functioning nerve or muscle fibers is so reduced that it cannot respond to any stimulus ([29]).

B. *Time-intensity curve* (TIC). For this test we used a Medelec electromyograph. The curve was obtained by plotting the intensities of galvanic current (expressed in milliamperes (ma)) required to elicit a minimal muscle contraction at various fixed stimulus durations (in milliseconds (ms)). This method is widely used throughout the world ([37, 49]).

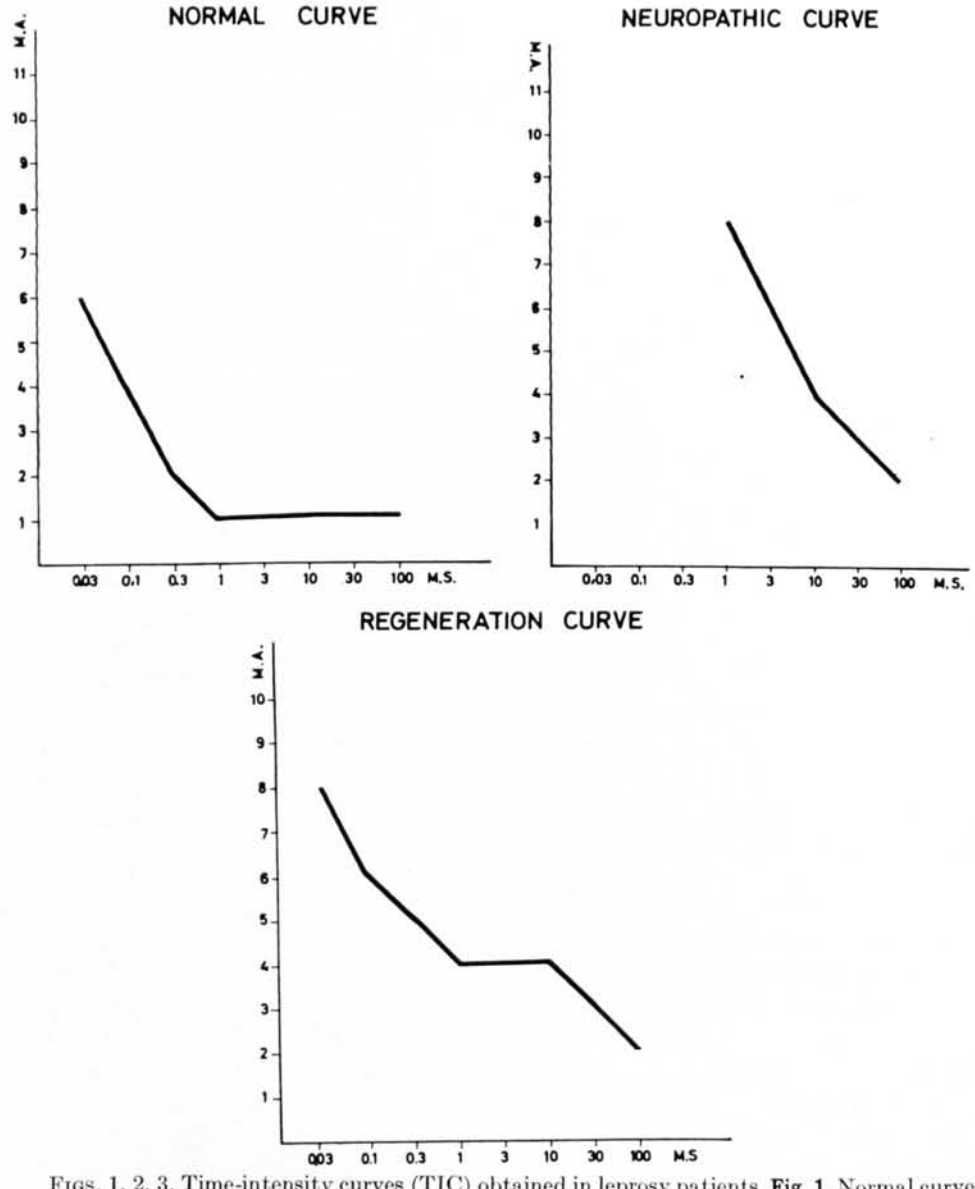
The fixed stimulus durations were 100 ms (approximating the actual rheobase), 30 ms, 10 ms, 3 ms, 1 ms, 0.3 ms, 0.1 ms. and 0.03 ms. The interpretation of the test is based on the slope, shift and form of the curve. In addition, we evaluated the ratio of intensities at 100 ms and 1 ms durations; this method, introduced by Bauwens ([5]) is much faster and double-checks the TIC. If the intensity required to give a minimal contraction at 1 ms is less than double the intensity at 100 ms duration, the nerve is probably healthy, while if the ratio is higher it points to a nerve lesion. In the great majority of examinations we have found a complete correlation between the two methods. In the few cases in which the interpretation was doubtful we repeated the examination. The results of TIC evaluation were classified in 3 groups:

1. Normal (Fig. 1): flat curve with a sharp rise at the short durations, without kinks; intensity at 1 ms less than double the intensity at 100 ms.

2. Pathologic curve: in this group we have included all the TIC with signs of nerve damage, without differentiating between the curves indicating complete (Fig. 2) or partial denervation (Fig. 3). The reason for this was that in the present study we were interested

only in the diagnostic value of the test. The prognostic importance of TIC will be discussed in a following study. The diagnosis of a pathologic curve was based on the presence of at least 2 of the following requirements: a shift of the curve to the right, a slanted curve, kinking and loss of contraction at an intensity of 50 ma; the required intensity at 1 ms duration more than double the intensity at 100 ms.

3. Unobtainable: when an intensity up to the pain or burn level (usually above 40 ma) at 100 ms duration failed to trigger any



FIGS. 1, 2, 3. Time-intensity curves (TIC) obtained in leprosy patients. **Fig. 1.** Normal curve. **Fig. 2.** Neuropathic curve. **Fig. 3.** Regeneration curve.

muscular contraction. Usually this lack of response paralleled a total DR in the FGT. The few discrepancies to be seen in the results may be explained by the reluctance of the investigator to use in one or another of the tests a high intensity, because of the danger of local burn or of mass contraction of other surrounding muscles, either of which may have induced in the patient an antagonism toward the use of electric testing.

The TIC is considered the most sensitive technic available in the diagnosis of nerve lesions ([33]). Almost any lesion of the nerve should manifest itself by the appearance of a pathologic curve. The inability to obtain any response would mean, as mentioned for the total DR of the FGT, that no nerve fiber is left to respond to the galvanic stimulus.

C. *Electromyography* (EMG).—The examinations were performed on a Medelec two-channel apparatus provided with a synchronized mnemoscopic unit, loud-speaker and filming camera. For technical reasons, we were unable to use a frequency analyzer. The electrodes used were of the coaxial needle type. The insulated examining room was kept at an agreeable temperature. The patient was seated comfortably, the hands and forearms were placed on a padded table, and the test and technic were explained to him in order to insure his cooperation. The patient was taught, before the actual examination, what movements were expected and how to relax completely the opponens pollicis and abductor digiti quinti muscles. Each EMG testing included recordings at the time of insertion of the needle, with the muscle in complete relaxation, and in minimal and maximal volitional contraction. This pattern was repeated at least twice and then the needle was rotated and redirected both crosswise and in depth. A few patients received 25–50 mg. meperidine hydrochloride (Pethidine) before the examination in order to relieve their apprehension; this did not affect the EMG in any way. Seven types of EMG pattern were observed among the 320 examinations:

1. Normal: complete electric silence with the muscle in relaxation. With the muscle in minimal volitional contraction, di- or triphasic spikes, with an average amplitude of 900–3,000 μV, duration of 4–10 ms and a few polyphasic units. In maximal volitional contraction, a summation of motor units forming a uniform interference pattern (Fig. 4). This pattern caused a loud, uninterrupted noise on the synchronized loudspeaker.

2. A normal pattern from all points of view with the exception of a relatively high number (above 4–10% of the recording) ([15, 30, 33, 47]) of polyphasic potentials of the same amplitude, but with a slightly longer duration; no other pathologic potential was observed. This finding was not constant in all the electrode positions within the same muscle, and as the rest of the recording was normal from all points of view we have included this type of pattern in the normal group.

3. Neuropathic pattern: spontaneous EMG fibrillations at rest,

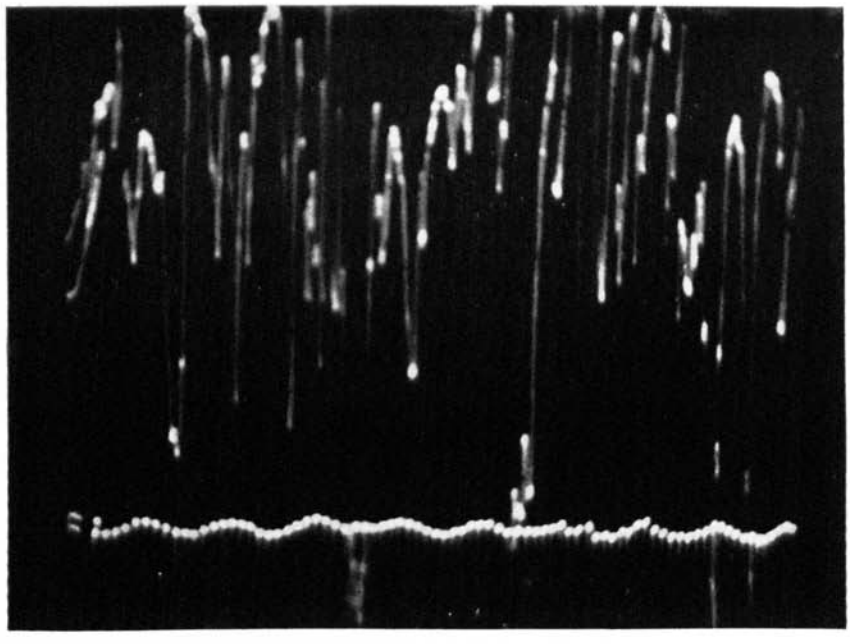


FIG. 4. Normal EMG recording. Interference pattern obtained in maximal volitional contraction. Calibration 1200μV and 1 ms.

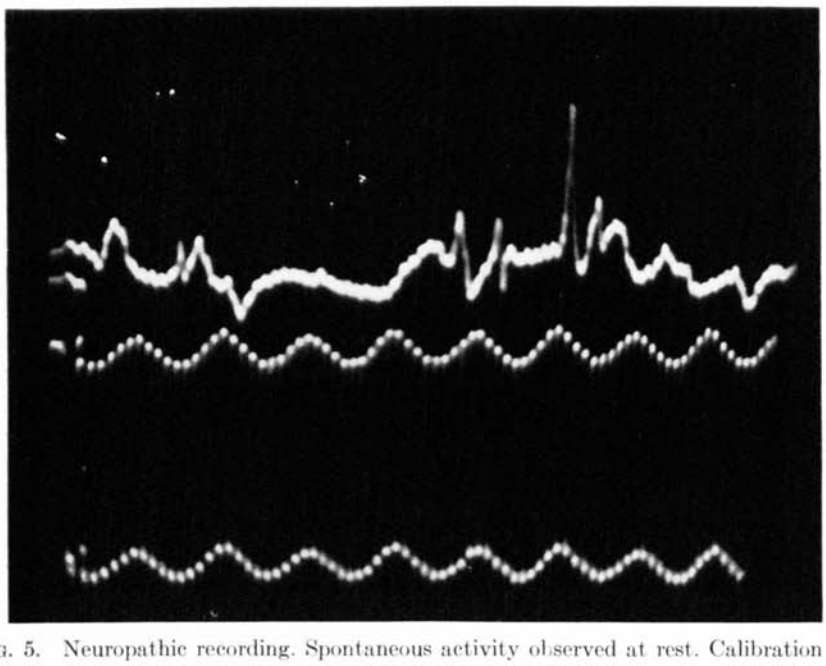


FIG. 5. Neuropathic recording. Spontaneous activity observed at rest. Calibration 300 μV and 1 ms.

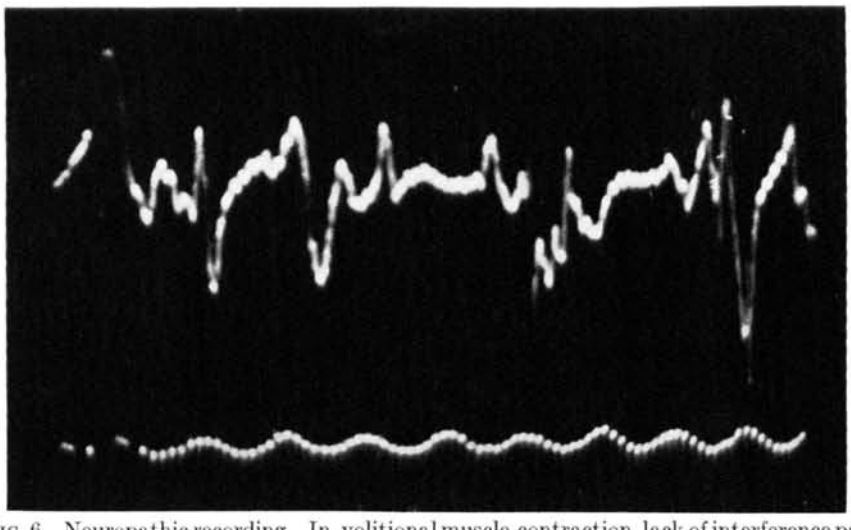


FIG. 6. Neuropathic recording. In volitional muscle contraction, lack of interference pattern, with loss of motor units. Calibration 600 μV and 1 ms.

sometimes accompanied by positive sharp waves (Fig. 5). In volitional minimal contraction, a high percentage of mono- and diphasic potentials, with a decreased amplitude and prolonged duration. In maximal volition, absence of an interference pattern with gaping, implying a loss of motor units (Fig. 6) ([10, 31, 34]).

4. The same type of neuropathic pattern with or without spontaneous activity, with a high percentage of polyphasic potentials with a markedly longer duration (12–24 ms) and a proportionately high incidence of giant spikes with an amplitude up to 9,000 μV (Fig. 7). These potentials might indicate a certain degree of nerve regeneration ([27, 50]), but as this distinction is not pertinent to the present study, and as the rest of the EMG evidence pointed to a nerve lesion, we have included these recordings also in the "neuropathic pattern."

5. A special group of EMG recordings in which there was, in general, no activity with the muscle in relaxation, though occasionally, a few small inconstant fibrillation potentials of an amplitude not higher than 150 μV could be observed ([7]). In minimal volition the recording appeared normal with the exception that the amplitude was markedly lower than normal (not higher than 600 μV) and the duration appeared to be shorter than normal, 2–4 ms (though no accurate measurement could be made by frequency analysis); occasional polyphasic potentials of the same amplitude as the general recording were observed. In maximal volition an interference pattern was always obtained, though often the clinical muscle power appeared to be markedly reduced. A typical feature found in these interference patterns was that though there was no "gaping," there was often a marked "thinning," with a general low amplitude, no matter how much the needle electrode was moved (Fig. 8). All these characteristics point to pure muscle damage (myopathy) without involvement of the axon ([7, 19, 23, 32, 33, 37]). In using this term we admit to much

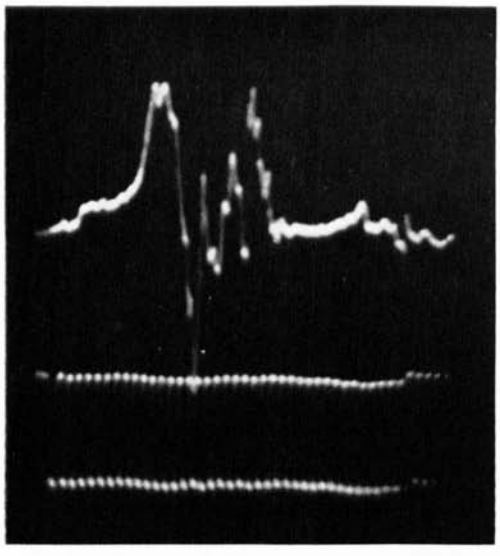


FIG. 7. Neuropathic recording with probable signs of regeneration; polyphasic potential with high duration. Calibration 600 μV and 1 ms.

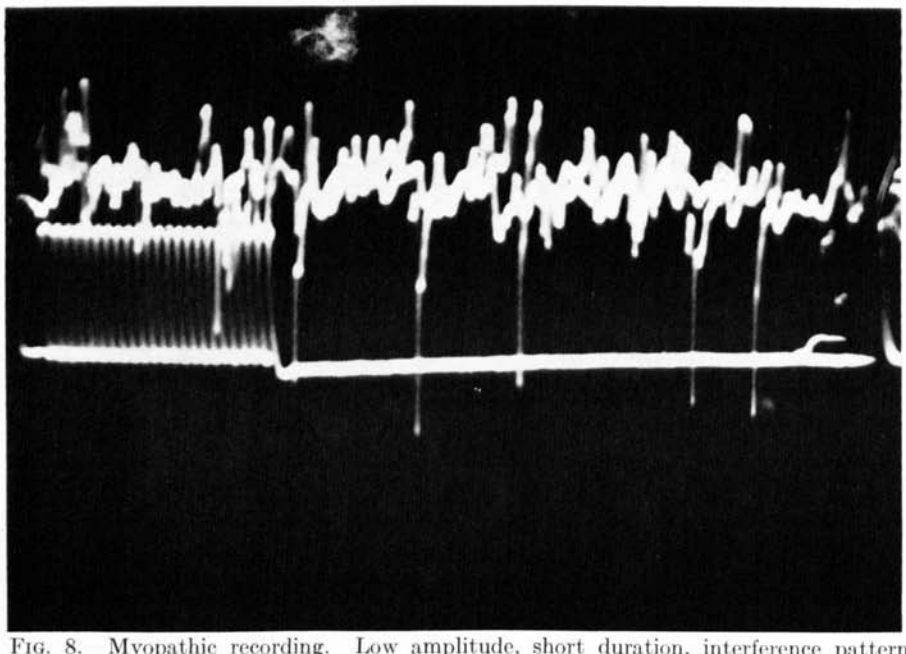


FIG. 8. Myopathic recording. Low amplitude, short duration, interference pattern in maximal volitional contraction. Calibration 600 μV and 1 ms.

hesitation, since we have no controlled proof, frequency analysis, or accurate duration measurements. However, the decision to use this term was made easier by a comparison with recordings taken from other myopathies.

6. The same type of recording as above, with the addition of long duration polyphasic units and high insertional potentials. This type of recording is consistent in our experience, and that of others [19, 36], with a polymyositis. As we were uncertain of the measurement of the

polyphasic potentials, and because we did not have any additional histologic supportive data, we have included these recordings in the "myopathic pattern" group.

7. Electric silence: complete lack of any potential, both in muscle relaxation and in volitional contraction, indicating complete atrophy and replacement of the nerve and muscle.

To summarize, the EMG recordings are classified as normal, neuropathic pattern, myopathic pattern, and electric silence.

In addition to the FGT, TIC and EMG, we examined, in a limited number of patients, the motor impulse velocity conduction. As the data are at present insufficient to provide any useful comparative values, the results of this test have not been included in the present report.

Evaluation of muscle power was carried out by two separate investigators at different times, to limit error. Muscle power was graded from 0–5, but the results of grading are not introduced here since such detail would only complicate the presentation of our results without adding useful information. The electrodiagnostic tests were always performed by the same two investigators, regardless of the dermatologic or neurologic condition of the patient. The evaluation and comparison of clinical, laboratory and electrodiagnostic data were carried out only after the completion of all the investigations.

RESULTS AND INTERPRETATION

This investigation has been carried out in 80 leprosy patients of whom 58 suffered from the lepromatous type and 8 from the tuberculoid type. Twelve were of indeterminate and 2 of borderline form. The bacteriologic status of the patients is presented in Table 3 (histologic preparations) and Table 4 (smears). From these two tables, it can be seen that the therapy afforded good results. Fifty-eight of the patients are bacteriologically negative at present. Among the remaining 22, there is a group of 6 new patients and also a number who did not receive or accept therapy under regular conditions. These remained histologically positive. The slight discrepancy in the number of positives in histologic preparations (22) and smears (18) is of no statistical significance.

The lepromin test (Table 5) was negative in the great majority of cases (68) before therapy was instituted.

The results of serologic examinations are presented in Table 6. In comparing the false WAR and Kahn tests before and during therapy, it is worthwhile mentioning that almost half the patients had a positive Kahn test on admission and of these about half became negative under therapy. The WAR showed similar results with an even greater proportion showing reversion under therapy. The SHAT and LFT are probably an expression of an aberrant immunologic reaction. With these tests, a positive result depends mainly on the presence of the "rheumatoid factor." This substance has been

proven by immunoelectrophoresis to be a β_2M globulin ([21]). In the general population 0.7—2 per cent SHAT and up to 14 per cent LFT ([3, 4, 20, 45]) examinations may show a positive reaction. There may be false positive responses in a variety of lesions, especially disseminated lupus erythematosus. We were interested in the response to these two tests in leprosy because the WAR and Kahn tests are accepted as giving a high percentage of false positives. As we have already mentioned, the SHAT and LFT were not in routine use at the time of admission and, therefore, we present only the "at present" results, without possibility of comparison. A positive SHAT was found in only two patients, an incidence approximating that of the general population in Israel. The LFT was found positive in 10 cases, a slightly higher incidence than that found by us in examinations of a random Israeli population sample ([1]). This LFT incidence is approximately equal to the number of false positive WAR tests. As the LFT is very sensitive, it seems worthwhile to investigate its value

TABLE 3.—*Bacteriologic findings (in histologic preparations).*

Bacteriologic findings		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
On admission	Positive	55	8	11	2	76
	Negative	3	—	1	—	4
At present	Positive	17	2	2	1	22
	Negative	41	6	10	1	58

TABLE 4.—*Bacteriologic findings (in smears).*

Bacteriologic findings		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
On admission	Positive	50	3	9	—	62
	Negative	8	5	3	2	18
At present	Positive	17	—	1	—	18
	Negative	41	8	11	2	62

TABLE 5.—*Lepromin test.*

Lepromin test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
On admission	Positive	3	4	3	2	12
	Negative	55	4	9	—	68
At present	Positive	1	2	5	—	8
	Negative	57	6	7	2	72

TABLE 6.—*Serologic status.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
On admission						
Kahn	Positive	29	3	3	—	35
	Negative	29	5	9	2	45
WAR	Positive	29	2	2	—	33
	Negative	29	6	10	2	47
At present						
Kahn	Positive	17	1	—	—	18
	Negative	41	7	12	2	62
WAR	Positive	10	2	—	—	12
	Negative	48	6	12	2	68
SHAT[a]	Positive	2	—	—	—	2
	Negative	56	8	12	2	78
LFT[b]	Positive	9	—	1	—	10
	Negative	49	8	11	2	70

[a] SHAT = Sheep hemagglutination test.

[b] LFT = Latex fixation test.

These two tests were not in regular use at the time of admission of most of the examined patients. Therefore they were not included in the present table at the time of admission.

TABLE 7.—*Neurologic signs.*

Neurologic sign	Type of leprosy				
	Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Muscle power loss[a]	74	14	26	2	116
Atrophy[b]	82	14	16	2	114
Positive Tinel sign[c]	41	6	14	1	62
Thickened nerve[d]	37	8	12	1	58
Sensory loss[e]	30	5	11	2	48

[a] Muscle power loss—opponens pollicis and abductor digiti quinti bilaterally—of a possible total of 320.

[b] Atrophy—thenar and hypothenar eminence bilaterally—of a possible total of 320.

[c] Positive Tinel sign—ulnar and median nerve bilaterally—of a possible total of 320.

[d] Thickened nerve—ulnar and median nerve bilaterally—of a possible total of 320.

[e] Sensory loss—of ulnar and median area—of a possible total of 80.

in fresh cases of leprosy before the institution of therapy, and to elucidate the mechanism of a positive response.

The neurologic signs are analyzed in Table 7. It is obvious from the results that the most common signs were loss of motor power and atrophy. From the respective numbers (116 and 114) it might be expected that these two neurologic signs appeared almost always together. In the following tables, however, it will be seen that this is not correct; in a number of instances there was clinical motor weakness without atrophy, while in others atrophy was present

without loss of muscle power. The same observation, to a lesser degree, applies to the presence of a positive Tinel sign and thickened nerves, which were also found often dissociated. A discrepancy between muscle power loss and atrophy seems to exist in the lepromatous group, in which the atrophy was more common, and the indeterminate group in which muscle power loss predominates. Sensory loss has been analyzed only in relation to the total number of patients, since the lesions were often partial, irregular, patchy, and scattered, and not related to any individual major branch of a nerve. Furthermore, we found that sensory loss was in no way related to motor neuron damage or any other neurologic sign, except in advanced lesions. In view of this, no further analysis of sensory loss will be added.

Table 8 indicates the relationship between muscle weakness and the other neurologic signs. It should be noted that about a third of the lepromatous cases with muscle weakness (23 of 74) did not show any atrophy, and in only 25 was a thickened nerve palpable. In the indeterminate group, half of the weak muscles showed no associated atrophy and only in 10 instances was the respective nerve thickened. In the tuberculoid and borderline groups, there were no significant differences between muscular weakness and atrophy. In all the 10 cases in the indeterminate group in which a weak muscle was associated with a corresponding thickened nerve, the Tinel sign was positive.

TABLE 8.—*Relationship between muscles with power loss and other neurologic signs.*

Neurologic sign	Type of leprosy				
	Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Muscle power loss	74	14	26	2	116
Atrophy	51	12	13	2	78
Positive Tinel sign	30	6	10	1	47
Thickened nerve	25	5	10	1	41

The relationship between the presence of atrophy and the other neurologic signs (Table 9) showed the same marked dissociation, especially in the lepromatous group: only 50 of the 82 cases showing atrophy evinced muscle power loss, and a thickened nerve was palpated in only about a third of the cases (26 of 82). Of the 16 atrophies in the indeterminate groups, only 4 had a corresponding thickened nerve, and in the tuberculoid group only 5 out of 14. These findings will be discussed later.

The comparison of a positive Tinel sign with the other signs (Table 10) indicates that more than one-third of lepromatous cases with a positive Tinel sign showed no other sign of nerve involvement. A similar proportion was seen in the indeterminate group. Whether

TABLE 9.—*Relationship between atrophy and other neurologic signs.*

Neurologic sign	Type of leprosy				
	Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Atrophy	82	14	16	2	114
Muscle power loss	50	12	13	2	77
Positive Tinel sign	29	5	9	1	44
Thickened nerve	26	5	4	1	36

TABLE 10.—*Relationship between positive Tinel sign and other neurologic signs.*

Neurologic sign	Type of leprosy				
	Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Positive Tinel sign	41	6	14	1	62
Muscle power loss	25	5	9	1	40
Atrophy	28	5	9	1	43
Thickened nerve	17	4	7	1	29

in these patients the nerve lesion was in the incipient stage, with the positive Tinel sign as the only manifestation, or whether the nerve damage was very slight and arrested by the treatment, cannot be decided. Furthermore, the meaning of this sign in the 40 instances of loss of muscle power requires clarification.

Similar results were found in relation to thickened nerves (Table 11). One conclusion that can be drawn from these figures could be that a thickened nerve appeared in about a third of the cases before any other neurologic sign (excluding sensory damage). However, we are still somewhat doubtful whether this is actually so. We feel that more examinations over a longer period of time are required before final conclusions can be made.

TABLE 11.—*Relationship between thickened nerve and other neurologic signs.*

Neurologic sign	Type of leprosy				
	Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Thickened nerve	37	8	12	1	58
Muscle power loss	24	5	9	1	39
Atrophy	25	5	4	1	35
Positive Tinel sign	16	4	7	1	28

The overall results of the electric tests, according to the type and form of the disease, appear in Table 12. Further comparisons will be made later, but, at this stage, attention should be drawn to three

important points. First is the marked discrepancy in the numbers of "normals" among the three tests. It is obvious that the FGT does not reflect finer degrees of lesion. However, whether the "normal" of TIC or of EMG is the correct one, or whether the true result lies somewhere in between, is difficult to ascertain. Second, the relatively high number of "total DR" and of "unobtainable" TIC (79 and 69 respectively), as compared with only 24 instances of EMG "electric silence," is noteworthy. One possible explanation for this phenomenon may be that the total number of nerve and muscle fibers was insufficient to show an objective response to extraneous stimuli, though their presence could be ascertained by EMG. Third, the presence of 45 EMG "myopathic patterns" is of great importance. This finding, if confirmed by additional diagnostic investigations, can be detected only by EMG, as the other electric tests (including motor impulse velocity transmission) provide information only about the condition of the axon, and not about the muscle proper.

TABLE 12.—*Results of the faradic-galvanic test (FGT), time-intensity curve (TIC) and electromyography (EMG).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
FGT	Normal	173	19	26	7	225
	Partial DR [a]	11	—	5	—	16
	Total DR	48	13	17	1	79
TIC	Normal	80	3	15	3	101
	Pathologic curve	108	19	19	4	150
	Unobtainable	44	10	14	1	69
EMG	Normal	116	14	23	7	160
	Neuropathic pattern	69	13	9	—	91
	Myopathic pattern	37	2	6	—	45
	Electric silence	10	3	10	1	24

[a] DR = Degeneration reaction.

In Table 13 we relate normal FGTs to muscle power and atrophy. It is of consequence that in 28 muscles with various degrees of power loss the FGT was normal. Theoretically, this can only imply that the weakness is related to the pathology of the muscle proper. We shall return to this point later. Furthermore, in 51 instances there was local atrophy, but, as we have already noted, this sign does not necessarily imply motor damage.

The relationship of FGT with partial DR to muscle power loss and atrophy (Table 14) shows, again, 5 muscles with normal power, in spite of a DR. As this finding was suspicious, we repeated the examination, both clinically and electrically, with the same results. We can only speculate that the axon was damaged at some earlier

time, when we might have found loss of power too, and that in the meantime an improvement occurred as a result of compensatory hypertrophy of the muscle.

TABLE 13.—*Relationship between normal faradic-galvanic test and the degree of muscle power and atrophy.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Normal FGT		173	19	26	7	225
Muscle power	Normal	153	18	20	6	197
	Loss	20	1	6	1	28
Atrophy	Absent	132	16	20	6	174
	Present	41	3	6	1	51

TABLE 14.—*Relationship between faradic-galvanic test, partial degeneration reaction and the degree of muscle power and atrophy.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Partial DR		11	—	5	—	16
Muscle power	Normal	3	—	2	—	5
	Loss	8	—	3	—	11
Atrophy	Absent	10	—	3	—	13
	Present	1	—	2	—	3

The FGT total DR (Table 15) shows the expected correlation with muscle power and atrophy, with the exception of the 2 muscles with normal power in spite of total DR. The only explanation that can be offered for this contradiction is that a mistake in evaluation has occurred. This will be evidenced in the succeeding tables. It is also of interest that 20 muscles did not reveal any clinical atrophy, although the FGT pointed to complete atrophy of the whole nerve-muscle unit.

Table 16 compares the normal FGT with the other two electric tests. The 5 instances in which the TIC was unobtainable are probably related to the investigator's reluctance to use a higher intensity. It is remarkable, however, that in 124 cases, the TIC pointed to neurologic damage, while in only 55 instances did the EMG show a neuropathic pattern. We look upon this fact as evidence that in at least a quarter of the cases the FGT is inaccurate and does not reflect the true condition of the nerve, and secondly, as will be seen in the following results, that the TIC is a much more sensitive,

though less accurate parameter, than EMG. It is also of significance that 18 of the EMG recordings showed evidence of a myopathic pattern.

TABLE 15.—*Relationship between faradic-galvanic test, total degeneration reaction and the degree of muscle power and atrophy.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Total DR		48	13	17	1	79
Muscle power	Normal	2	—	—	—	2
	Loss	46	13	17	1	77
Atrophy	Absent	9	2	9	—	20
	Present	39	11	8	1	59

TABLE 16.—*Relationship between normal faradic-galvanic test and the time-intensity curve (TIC) and electromyography (EMG).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Normal FGT		173	19	26	7	225
TIC	Normal	79	2	12	3	96
	Pathologic curve	90	17	13	4	124
	Unobtainable	4	—	1	—	5
EMG	Normal	113	14	18	7	152
	Neuropathic pattern	45	5	5	—	55
	Myopathic pattern	15	—	3	—	18
	Electric silence	—	—	—	—	—

Table 17 compares the partial DR with the TIC and EMG. The striking fact emerging from this table is that in 4 instances the EMG was normal, while in 7 instances a myopathic pattern was found. These findings may be explained if we accept that our needle electrode happened to be in 6 places in the muscle (this is the number of repositionings in each muscle) in which the local motor units were healthy or in which only the local terminal nerve fibers were involved. In support of this supposition, it may be noted that most of these 7 myopathatic recordings were of the polymyositic type. We intend to repeat the electric tests in the 4 muscles with normal EMG and confirm their condition also with histologic studies.

The total DR relation to TIC (Table 18) shows 4 instances of a normal curve; as the TIC is substantiated by the EMG, we can blame for the total DR reaction of FGT only the reluctance of the investiga-

tor to cause pain. We should stress at this point that the patients with intact sensation tended to be less tolerant of pain during faradic stimulation. As the faradic current is utilized before the galvanic stimulus (to exclude the presence of a tetanic contraction, which, if existent, would preclude the galvanic test), many patients evinced a marked dislike for and lack of cooperation during FGT. This further accentuates the inaccuracy of the test. Confirmation of this inaccuracy was obtained in the 13 instances in which a TIC was obtained at a later time. Furthermore, it is of interest that of 62 muscles with total DR confirmed by an unobtainable TIC, only 27 showed electric silence on EMG, a fact that stresses the greater and more discriminating reliability of EMG as a test for the presence of nerve or muscle fibers.

In Table 19 we relate normal EMG recordings to muscle power and atrophy. The 12 muscles with loss of power in spite of a normal

TABLE 17.—*Relationship between faradic-galvanic test, partial degeneration reaction and the time-intensity curve (TIC) and electromyography (EMG).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Partial DR		11	—	5	—	16
TIC	Normal	—	—	1	—	1
	Pathologic curve	10	—	3	—	13
	Unobtainable	1	—	1	—	2
EMG	Normal	1	—	3	—	4
	Neuropathic pattern	4	—	1	—	5
	Myopathic pattern	6	—	1	—	7
	Electric silence	—	—	—	—	—

TABLE 18.—*Relationship between faradic-galvanic test, total degeneration reaction and the time-intensity curve (TIC) and electromyography (EMG).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Total DR		48	13	17	1	79
TIC	Normal	1	1	2	—	4
	Pathologic curve	8	2	3	—	13
	Unobtainable	39	10	12	1	62
EMG	Normal	2	—	2	—	4
	Neuropathic pattern	20	8	3	—	31
	Myopathic pattern	16	—	1	—	17
	Electric silence	10	5	11	1	27

EMG show that the EMG may not be sufficient as a test either. It is obvious that in many instances the EMG should be recorded from a greater number of sites in the muscle, as our choice of the site for the needle insertion is entirely arbitrary. As the nerve or muscle damage in leprosy is in most instances scattered, the EMG examination becomes a matter of conjecture. Nineteen normal EMG were in sites that were obviously atrophied. It is of further interest that of the 12 discrepancies between EMG and clinical muscle power, 8 were in the indeterminate form. We think, however, that this is a fortuitous contingency of no practical significance.

TABLE 19.—*Relationship between normal electromygraphic activity and the degree of muscle power and atrophy.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Normal EMG		116	14	23	7	160
Muscle power	Normal	113	14	15	6	148
	Loss	3	—	8	1	12
Atrophy	Absent	103	12	20	6	141
	Present	13	2	3	1	19

In Table 20 we may observe that 38 muscles were normal in power but showed a neuropathic pattern on EMG. We ascribe this to fortuituous placing of the electrode in abnormal tissue. Thirty-two of these muscles did not show any atrophy, a finding on which we will elaborate further; suffice it to say at present that the absence of atrophy did not in general correspond with normal muscle power. We are now following the patients with normal muscle power and an EMG neuropathic pattern in order to observe their further development.

TABLE 20.—*Relationship between electromyographic neuropathic pattern and the degree of muscle power and atrophy.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
EMG neuropathic pattern		69	13	9	—	91
Muscle power	Normal	29	4	5	—	38
	Loss	40	9	4	—	53
Atrophy	Absent	24	6	2	—	32
	Present	45	7	7	—	59

The analysis of muscle power and atrophy in those patients showing EMG myopathic pattern (Table 21) does not allow us to draw any conclusion, as it is clear that the EMG does not reflect any quantitative changes and, therefore, does not necessarily relate to the clinical state of the muscle.

EMG complete electric silence (Table 22) proves the reliability of EMG in severe cases. All the 24 muscles without electric activity were clinically completely paralyzed. We think this state proves beyond doubt that clinically obvious atrophy does not necessarily entail neurologic motor damage. There were 10 instances in which there was no clinical atrophy though the muscle was completely paralyzed and the EMG indicated complete disappearance of the entire motor unit.

TABLE 21.—*Relationship between electromyographic myopathic pattern and the degree of muscle power and atrophy.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
EMG myopathic pattern		37	2	6	—	45
Muscle power	Normal	16	—	2	—	18
	Loss	21	2	4	—	27
Atrophy	Absent	19	—	4	—	23
	Present	18	2	2	—	22

TABLE 22.—*Relationship between electromyographic electric silence and the degree of muscle power and atrophy.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
EMG electric silence		10	3	10	1	24
Muscle power	Normal	—	—	—	—	—
	Loss	10	3	10	1	24
Atrophy	Absent	4	—	6	—	10
	Present	6	3	4	1	14

The relationship between normal EMG recordings and the two other electric tests (Table 23) shows that out of 160 cases with a normal EMG, 152 FGTs were normal too. A possible explanation for the 4 total DR and one unobtainable TIC was provided previously. It should be stressed, however, that 75 TIC were found pathologic. The interpretation of the curves was according to that accepted throughout the world and, thus, we can only presume, as we have

already suggested, that the TIC is able to indicate fine lesions of the nerve when electromyographic exploration is not adequate. It is also possible that TIC, depending as it does on an extraneous stimulus, and being influenced by many factors, is prone to error in a higher percentage of cases than EMG. Again, it may indicate states of nerve pathology that are actually transient and scattered. By further follow-up on these cases by repeated TIC and EMG, we may eventually be able to decide which of these hypotheses is correct.

The analysis of the neuropathic EMG (Table 24) stresses the inadequacy of the FGT (56 normals), which requires a gross nerve lesion before it shows a disturbance, and the relatively much higher sensitivity of the TIC. The 10 instances of normal TIC point to the fact that an elaborate EMG exploration of the muscle may show very minute local changes which even a sensitive test depending on external stimulus cannot demonstrate.

Table 25 compares the EMG myopathic pattern with the FGT and TIC. It seems to us that both the FGT and TIC adduce

TABLE 23.—*Relationship between normal electromyographic activity and the faradic-galvanic test (FGT) and time-intensity curve (TIC).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Normal EMG		116	14	23	7	160
FGT	Normal	113	14	18	7	152
	Partial DR	1	—	3	—	4
	Total DR	2	—	2	—	4
TIC	Normal	70	1	10	3	84
	Pathologic curve	45	13	13	4	75
	Unobtainable	1	—	—	—	1

TABLE 24.—*Relationship between electromyographic neuropathic pattern and the faradic-galvanic test (FGT) and time-intensity curve (TIC).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
EMG neuropathic pattern		69	13	9	—	91
FGT	Normal	43	6	5	—	56
	Partial DR	4	—	1	—	5
	Total DR	20	7	3	—	30
TIC	Normal	7	2	1	—	10
	Pathologic curve	45	6	6	—	57
	Unobtainable	17	5	2	—	24

corroborative evidence that our interpretation of the EMG recordings may be correct. The combination of normal and total DR of FGT and normal and unobtainable TIC are findings consistent with any myopathy. Furthermore, the 7 instances of partial DR and 18 pathologic TIC could be explained by the presence of a widespread polymyositic process. In general it is accepted that a weak or paralyzed muscle with a normal TIC or a normal FGT indicates the presence of myopathy ([6, 33]). Here, we have in addition EMG recordings which also point to a myopathic lesion. Moreover, another clinical fact is of importance: in most of these cases, the sensory loss was minimal or nonexistent. Though the sensation was found an unreliable indicator of nerve lesion, still, in this case, the lack of sensory loss was constant and compatible with lack of nerve lesion.

The EMG electric silence (Table 26) was accompanied by a parallel total DR and unobtainable TIC. The table requires no further explanation.

TABLE 25.—*Relationship between electromyographic myopathic pattern and the faradic-galvanic test (FGT) and time-intensity curve (TIC).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
EMG myopathic pattern		37	2	6	—	45
FGT	Normal	15	—	3	—	18
	Partial DR	6	—	1	—	7
	Total DR	16	2	2	—	20
TIC	Normal	3	—	4	—	7
	Pathologic curve	18	—	—	—	18
	Unobtainable	16	2	2	—	20

TABLE 26.—*Relationship between electromyographic electric silence and the faradic-galvanic test (FGT) and time-intensity curve (TIC).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
EMG electric silence		10	3	10	1	24
FGT	Normal	—	—	—	—	—
	Partial DR	—	—	—	—	—
	Total DR	10	3	10	1	24
TIC	Normal	—	—	—	—	—
	Pathologic curve	—	—	—	—	—
	Unobtainable	10	3	10	1	24

In the next four tables (Tables 27–30) we have taken the most sensitive and accurate laboratory test (the bacteriologic status) and compared its positive results in histologic preparations and in smears with muscle power, atrophy and electrodiagnostic tests, respectively. It is evident from the data presented that no relationship exists between a positive bacteriologic finding and the neuromuscular or electrodiagnostic results.

TABLE 27.—*Relationship between the positive bacteriologic findings in histologic preparations and the neurologic signs.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Positive test[a]		17	2	2	1	22
Muscle power	Normal	49	4	4	3	60
	Loss	19	4	4	1	28
Atrophy	Absent	59	6	4	3	72
	Present	9	2	4	1	16
Tinel sign	Negative	55	6	5	4	70
	Positive	13	2	3	—	18
Thickened nerve	Absent	59	4	4	4	71
	Present	9	4	4	—	17

[a] For each positive bacteriologic test, 4 possibilities are considered for every neurologic sign.

TABLE 28.—*Relationship between the positive bacteriologic findings in histologic preparations and the electric tests.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Positive test[a]		17	2	2	1	22
FGT	Normal	54	4	4	4	66
	Partial DR	7	—	1	—	8
	Total DR	7	4	3	—	14
TIC	Normal	32	2	2	—	36
	Pathologic curve	28	2	5	4	39
	Unobtainable	8	4	1	—	13
EMG	Normal	42	2	1	4	49
	Neuropathic pattern	16	6	4	—	26
	Myopathic pattern	10	—	3	—	13
	Electric silence	—	—	—	—	—

[a] For each positive bacteriologic test, 4 possibilities are considered for every electric test.

TABLE 29.—*Relationship between the positive bacteriologic findings in smears and the neurologic signs.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Positive test[a]		17	—	1	—	18
Muscle power	Normal	50	—	2	—	52
	Loss	18	—	2	—	20
Atrophy	Absent	48	—	—	—	48
	Present	20	—	4	—	24
Tinel sign	Negative	60	—	2	—	62
	Positive	8	—	2	—	10
Thickened nerve	Absent	64	—	4	—	68
	Present	4	—	—	—	4

[a] For each positive bacteriologic test, 4 possibilities are considered for every neurologic sign.

TABLE 30.—*Relationship between the positive bacteriologic findings in smears and the electric tests.*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Positive test[a]		17	—	1	—	18
FGT	Normal	61	—	2	—	63
	Partial DR	3	—	1	—	4
	Total DR	4	—	1	—	5
TIC	Normal	31	—	1	—	32
	Pathologic curve	27	—	1	—	28
	Unobtainable	10	—	2	—	12
EMG	Normal	40	—	4	—	44
	Neuropathic pattern	20	—	—	—	20
	Myopathic pattern	8	—	—	—	8
	Electric silence	—	—	—	—	—

[a] For each positive bacteriologic test, 4 possibilities are considered for every electric test.

As atrophy proved to be a sign of vacillating importance, we have compared all the instances of clinical atrophy with the electrodiagnostic tests (Table 31). Apart from the fact that the presence of atrophy does not imply a neuromuscular lesion, no direct relationship could be shown.

In Table 32 we evaluate the electrodiagnostic tests in muscles with power loss. Again, it is evident here, that the TIC is probably the most sensitive test for the evaluation of the axon, while the EMG

is the only test that can give information about the possible presence of a myopathy; in the latter case, as we have suggested above, the TIC and, to a lesser degree, the FGT may confirm the myopathy by inference. It is also evident in this table that the electrodiagnostic tests of all the neurologic and motor signs are most directly related to the muscle power.

TABLE 31.—*Relationship between atrophy and the faradic-galvanic test (FGT), time-intensity curve (TIC) and electromyography (EMG).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Atrophy		82	14	16	2	114
FGT	Normal	42	3	6	1	52
	Partial DR	1	—	2	—	3
	Total DR	39	11	8	1	59
TIC	Normal	10	1	3	—	14
	Pathologic curve	35	5	6	1	47
	Unobtainable	37	8	7	1	53
EMG	Normal	13	2	3	1	19
	Neuropathic pattern	45	7	7	—	59
	Myopathic pattern	18	2	2	—	22
	Electric silence	6	3	4	1	14

TABLE 32.—*Relationship between muscles with power loss and the faradic-galvanic test (FGT), time-intensity curve (TIC) and electromyography (EMG).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Muscle power loss		74	14	26	2	116
FGT	Normal	20	1	6	1	28
	Partial DR	8	—	3	—	11
	Total DR	46	13	17	1	77
TIC	Normal	2	1	5	—	8
	Pathologic curve	31	3	8	1	43
	Unobtainable	41	10	13	1	65
EMG	Normal	3	—	8	1	12
	Neuropathic pattern	40	9	4	—	53
	Myopathic pattern	21	2	4	—	27
	Electric silence	10	3	10	1	24

Finally, the last table (Table 33) analyzes muscles found healthy from the clinical point of view with respect to the electric tests. Out

TABLE 33.—*Relationship between muscles with normal power and the faradic-galvanic test (FGT), time-intensity curve (TIC) and electromyography (EMG).*

Test		Type of leprosy				
		Lepromatous	Tuberculoid	Indeterminate	Borderline	Total
Normal muscle power		158	18	22	6	204
FGT	Normal	153	18	20	6	197
	Partial DR	3	—	2	—	5
	Total DR	2	—	—	—	2
TIC	Normal	78	2	10	3	93
	Pathologic curve	78	16	11	3	108
	Unobtainable	2	—	1	—	3
EMG	Normal	113	14	15	6	148
	Neuropathic pattern	29	4	5	—	38
	Myopathic pattern	16	—	2	—	18
	Electric silence	—	—	—	—	—

of the 204 FGTs, 197 were normal, while 5 showed lack of response to faradic stimulus. The TIC shows a surprisingly high number of pathologic curves (108), while in 3 instances we could not obtain a response to galvanic stimulus. Although some pathologic curves were borderline ones, and the investigator (who did not know the clinical status) might have considered them doubtful, a remarkable number of nerves (38) have shown clear electric signs of lesion, in spite of the lack of any actual clinical loss of muscle power. Of the 3 instances in which TIC was considered unobtainable, 2 examinations were performed while the patients were in reaction and suffering from acute pain, while the third was suffering from an acute causalgia of the examined area. As the patient with causalgia and one of those with an acute reaction were the same as those included in the total DRs, they should not be taken into consideration. EMG showed 148 muscles as normal, 38 with a neuropathic pattern, and 18 with a myopathic pattern. More than a quarter of the clinically healthy muscles, therefore, have shown EMG signs of damage. The EMG neurogenic findings paralleled the TI pathologic curves, while in most of the EMG myopathic patterns the TIC was normal.

DISCUSSION AND CONCLUSIONS

It is obvious that the motor state of the lower neuron does not depend in any way on the type or form of the disease, though it may be said that the numbers of cases of tuberculoid type and borderline form are too small to permit firm conclusions. Moreover, the activity or inactivity of the disease does not play any role, once the lesion of the nerve has occurred. Furthermore, the dermatologic state and the various laboratory tests bear little relationship to the neurologic

status, nor do they play any prognostic part in relation to the motor condition. Sex, age and ethnic origin are of no importance in the neurologic picture.

Of the neurologic signs, by far the most important is the clinical muscle power. This examination depends to a large extent on the degree of cooperation and understanding of the patient, on one hand, and the technic, experience, and patience of the investigator on the other hand. Nevertheless, it gives the best information about motor function. Atrophy, on the other hand, proved to be an unreliable sign. In our experience with the clinical aspects of many other lower motor neuron and muscle diseases, we have been accustomed to rely on atrophy as a fair, clinically objective sign of nerve damage. To our surprise, this did not prove to be so in leprosy. The frequency (38 cases) of muscle power loss without atrophy and of atrophy without muscle power loss (37 cases) suggests that lost muscle substance may be replaced by some other material, while, on the other hand, we may probably have atrophy of the subcutaneous tissue without clinically evident loss of muscle function. One additional point requires clarification here: at the beginning of our study we examined muscle power by grading 0–5 and atrophy by grades mild to severe. We have found, however, that this unnecessarily complicated the presentation of the material and that in any case the actual quantitative grading was not significant. It suffices to say that only the qualitative test was pertinent and that the loss of muscle power and atrophy were signs present almost beyond doubt.

The physiologic reflexes of the upper extremity were found completely unreliable as indicators of the motor condition, except in extreme lesions in which they were not needed for diagnosis anyway. Sensory change, when present, was not relatable to actual motor loss, again excluding the severe nerve trunk lesions. In some cases we noted a few small patches (1–2 cm. in diameter) of loss of superficial sensation, often dissociated, with an unproportionate loss of power, while in other cases the loss of sensation was extensive and muscle power was preserved. On the other hand, the preservation of sensation was an important corroborative finding when the decrease in muscle power was due, electromyographically, to a "myopathy." It seems clear that the clinical picture was caused by selective scattered lesions in isolated bundles of muscle fibers.

The Tinel sign proved to be a fair indicator of nerve trunk lesion. At present, however, we cannot evaluate its prognostic importance. It is a sign that the nerve is both damaged and functional. It might be possible by long follow-up to ascertain whether its presence is a sign of improvement (nerve regeneration), or of an active lesion and whether its disappearance has any prognostic meaning.

The thickened nerve is similarly a fair sign of nerve lesion. It is a clear palpable sign that the nerve is infiltrated, although about a third of such cases did not show any motor lesion (Table 11). Without doubt, these two signs show a distinct nerve lesion, a fact proven

also by the EMG recordings (not presented in tables) of neuropathic patterns to the complete exclusion of any EMG sign of myopathy. An interesting observation is that the lesions of the ulnar nerve outnumbered those of the median nerve in a 3:1 proportion, but as a careful comparison did not reveal any difference between the lesions of the two nerves we have presented the material without further analysis. In order to prevent possible criticism or objection to our method of arbitrarily choosing the opponens pollicis and abductor digiti quinti muscles as representatives of those muscles innervated by the median and ulnar nerves, we examined neurologically and graded all the muscles innervated by the same nerves. In only a few instances did we find other muscles damaged to the exclusion of one of our choice. (When such a situation did exist, it was usually found that the interossei were involved.) Muscles in the lower limb mainly innervated by the peroneus communis nerve were damaged in a similar manner. The electric tests of these muscles (interossei, peroneus longus and tibialis anterior) did not reveal any difference warranting their inclusion. Furthermore, it seemed to us that the clinical and electric evaluation would be more liable to error.

From the data we have presented, we feel that it can be accepted that clinical neurologic examination is not sufficient in certain aspects of leprosy. This is especially true when the neurologic lesion appears small or when the muscle is slightly weakened. It is of paramount importance when surgery of the hand is contemplated to have an evaluation of the qualitative changes of the motor function of any healthy muscle to be transferred. When this need arises, the information that may be supplied by electrodiagnostic methods is of great value and may provide information on the true condition. The problem that arises is that of choosing the electrodiagnostic test to be used. We have no doubt now that the faradic-galvanic test is an unreliable, inaccurate test that can demonstrate only gross nerve lesions, which are clinically apparent anyway. Furthermore, the test relies on an external stimulus that is often painful. The reaction, moreover, may be affected by extraneous factors such as room temperature, humidity, technic, or congenital anomalies in the location of the motor points. This test does not, in fact, investigate the state of the muscle substance. The time-intensity curve appears to be the most sensitive tool for the detection of a nerve lesion, though, of course, it is subject to at least some of the limitations mentioned above. Its importance is not only diagnostic but also prognostic. We are convinced that repeated examinations at intervals of 2 to 3 weeks will not only reveal errors in technic or interpretation, but also indicate the trend of the lesion.

The most serious limitation of this test, as with the FGT, is that the TIC does not evaluate the muscle proper. However, information may be obtained indirectly. For example, a clinically weak muscle with a normal TIC would indicate muscle damage without nerve

involvement. We will not dwell at the present time upon the importance of the motor impulse transmission, although it should be clear that this test is applicable only to the long peripheral nerves of the extremities. Its main use is in investigating the exact localization of the lesion along the nerve.

Electromyography has proven to be a reliable technic for the examination of the nerve and the muscle. Although it is probably less sensitive than the TIC, because of the physical limitations imposed by the use of needle electrodes, a careful search will show the actual pathology in most cases.

We do not believe that skin electrodes would be beneficial in spite of the bigger surface covered and relative painlessness; their main drawback is that wherever two or more muscles are superimposed, differentiation is difficult. Moreover, the number of motor units thus examined is too high to allow for precise analysis. From the EMG results, two findings emerge as most important. First, the suggestion of myopathic patterns in leprosy is new. In the past, all the muscle changes have been ascribed to the nerve lesion, while the muscle substance, it has been thought, is never damaged primarily. In our 80 leprosy patients, 45 of the 320 muscles examined have shown on EMG that the pathology lies within the muscle itself. Although we still have some doubts about this finding (since the diagnosis of an EMG myopathic pattern is so difficult and because at present we have no corroboration by frequency-duration analysis or histologic studies), we have in the present material two hints that the findings might be true: one, that most of these muscles appeared to be either healthy from the clinical point of view (Table 33), or weak but unaccompanied by the other neurologic signs commonly seen in a nerve lesion. The second hint is that in most of the EMG myopathic patterns the TIC was normal or unobtainable (Tables 25 and 33). In most of the instances in which the TIC has shown a neuropathic lesion parallel with an EMG myopathic pattern, the EMG picture fitted the diagnosis of "polymyositis" (a picture that could combine both nerve and muscle pathology). It is evident that no final conclusion can be drawn before additional data are collected.

The second important EMG finding is of 56 pathologic recordings (over one quarter) in the 204 clinically healthy muscles. If this finding is proven correct by additional tests, it will prove of cardinal importance in the field of reconstructive surgery. It is a well accepted fact that in other neurologic conditions in which muscle transfers and translocations are performed (for example poliomyelitis), the muscle to be transferred should be healthy in order to give a good functional outcome. The manipulation and transfer of the muscle at the time of the intervention may cause a loss of its power [13, 24, 41] even in the most experienced hands. Furthermore, if the muscle is already affected by leprosy, the chances are that even under good therapeutic control it will be further damaged in the next leprosy

reaction. This could make the surgery performed futile and unwarranted.

In conclusion, it is our impression from the results presented that all leprosy patients, especially candidates for reconstructive surgery and those in whom a motor lesion is suspected, should undergo repeated EMG examinations for evaluation of the nerve and muscle

At present we are conducting further EMG studies by frequency analysis, and amplitude and duration histograms of mean averages. Histologic studies are also being carried out. These should give us better and more accurate information on these problems. We are studying also the possibility of localizing the exact site of nerve lesion (when compression or strangulation of the nerve trunk is suggested by the clinical picture) by motor impulse velocity transmission.

ABBREVIATIONS USED

DR	Degeneration reaction	ma	milliamperes
EMG	Electromyography	ms	milliseconds
FGT	Faradic-galvanic test	SHAT	Sheep hemagglutination test
LFT	Latex fixation test	TIC	Time-intensity curve
		WAR	Wassermann reaction

SUMMARY

1. Faradic-galvanic tests, time-intensity curve estimations, and electromyography were used in the investigation and evaluation of the motor unit in 80 patients suffering from leprosy (58 lepromatous type, 8 tuberculoid type, 12 indeterminate form, and 2 borderline form).

2. Dermatologic, neurologic, bacteriologic, immunologic, and serologic evaluations were carried out in each patient.

3. The following factors were specifically evaluated: muscle power, atrophy, Tinel sign, and nerve thickening.

4. For reasons given, neurologic and electrodiagnostic examinations were confined to the ulnar and median nerves and the opponens pollicis and abductor digiti quinti muscles, bilaterally.

5. Comparisons of the laboratory data, neurologic signs, and electrodiagnostic tests revealed the following:

(a) Lack of direct relationship between the activity of the disease and the condition of the motor unit, once the motor damage had occurred.

(b) Absence of correlation between the type or form of the disease and the neurologic signs and electrodiagnostic findings.

(c) Absence of relationship between the dermatologic condition and laboratory data, on one hand, and the neurologic and electrodiagnostic findings on the other.

(d) Proof that muscle power loss was the most reliable neurologic sign.

(e) A conspicuous discrepancy between muscle power loss and atrophy.

(f) Demonstration that the Tinel sign and thickening of the nerve were good indicators of the nerve lesion, though not in relation to motor power.

(g) Demonstration that the faradic-galvanic test is unreliable and inaccurate whenever a minor lesion is present.

(h) Demonstration that the time-intensity curve is the most sensitive test for the overall evaluation of any particular muscle in relation to its nerve of supply. It is dependent, however, on an extraneous stimulus and does not evaluate the muscle proper, nor can it identify disseminated fine lesions.

(i) Demonstration that electromyography is the most exact test, provided exploration is sufficiently broad.

(j) Indication, from a number of electromyographic recordings, of the possibility of direct muscle damage. If this finding is confirmed, it is of importance in the basic concept of motor damage in leprosy.

(k) Demonstration by electromyography, in a significant number of clinically healthy muscles, of signs of damage. If these findings are proven correct, they are of major importance in the planning of surgical rehabilitation.

6. At present we are investigating the significance of the time-intensity curve in the prognosis of the nerve lesion. We are also conducting histologic studies and frequency analyses of the electromyographic recordings in order to ascertain the possibility of primary muscle damage and occult nerve lesions. The relationship between clinical muscle power loss and atrophy is being further investigated.

RESUMEN

1. En la investigación y evaluación de la unidad motora en 80 pacientes sufrientes de lepra (58 tipo lepromatoso, 8 tipo tuberculoide, 12 formas indeterminadas y 2 formas limites-borderline) fueron usados las pruebas farádico-galvánicas, la estimación de las curvas de tiempo-intensidad y la electromiografia.

2. En cada paciente fueron realizadas las evaluaciones dermatológicas, neurológicas, bacteriológicas, inmunológicas y serológicas.

3. Fueron especificamente evaluados los siguientes factores: poder muscular, atrofia, signo de Tinel y espesamiento de los nervios.

4. Por razones dadas, los exámenes neurológicos y electrodiagnósticos, fueron confinados bilateralmente a los nervios cubital y mediano, a los musculos flexor del pulgar y abductor del quinto dedo.

5. Las comparaciones de los datos de laboratorio, de los signos neurológicos y pruebas electrodiagnósticas, revelaron lo siguiente:

(a) Perdida de la relación directa entre la actividad de la enfermedad y la condición de la unidad motora, una vez que se produjo el daño motor.

(b) Ausencia de correlación entre el tipo o forma de la enfermedad y los signos neurológicos y hallazgos electrodiagnósticos.

(c) Ausencia de relación entre la condición dermatológica y los datos de laboratorio por una parte, y los hallazgos neurológicos y electrodiagnósticos por otra.

(d) Pruebas de que la perdida de poder muscular fué el signo neurológico de mas confianza.

(e) Una conspicua discrepancia entre la perdida del poder muscular y la atrofia.

(f) La demostración de que el signo de Tinel y el espesamiento del nervio fueron buenos indicadores de la lesión nerviosa, aunque no en relación con el poder motor.

(g) Demostración que la prueba farádica-galvánica es insegura e inadecuada cuando una lesión menor está presente.

(h) Demostración que la curva de tiempo-intensidad es la prueba mas sensitiva para la evaluación total de un músculo particular en relación con el nervio correspondiente. De cualquier manera, es asimismo dependiente de un estimulo extraño y no evalua al musculo propiamente dicho, ni puede identificar finas lesiones diseminadas.

(i) Demostración que la electromiografia es la prueba mas exacta, provisto que la exploración sea suficientemente amplia.

(j) Indicación, con una cantidad de electromiografias registradas, de la posibilidad de un daño muscular directo. Si estos hallazgos son confirmados son de importancia para el concepto basico del daño motor en la lepra.

(k) Demostración por electromiografia, de signos de daño en un significativo numero de músculos sanos clinicamente. Se estos hallazgos prueban ser correctos, son de mayor importancia en el planeamiento de la rehabilitación quirurgica.

6. En el presente, estamos investigando el significado de la curva tiempo-intensidad en el pronóstico de la lesión nerviosa. Tambien estamos efectuando estudios histológicos y análisis de frecuencia de los registros electromiográficos, en orden de aseverar la posibilidad del daño primario muscular y las lesiones nerviosas ocultas. Tambien es investigada la relación entre la perdida clínica del poder muscular y la atrofia.

RÉSUMÉ

1. En vue d'étudier et d'évaluer l'unité motrice chez 80 malades atteints de lèpre (58 du type lépromateux, 8 du type tuberculoide, 12 atteints de la forme indeterminée, et 2 dimorphes), on a eu recours á des épreuves galvano-faradiques, à des estimations de la courbe d'intensité en fonction du temps. et à l'electromyographie.

2. Des examens dermatologiques, neurologiques, bactériologiques, immunologiques et sérologiques ont été pratiqués chez chaque malade.

3. La force musculaire, l'atrophie, le signe de Tinel et l'épaississement nerveux ont été spécialement étudiés.

4. Pour des raisons qui sont expliquées, les examens neurologiques et l'electrodiagnostic ont été limités aux nerfs cubital et médian, ainsi qu'à l'opposant du pouce et à l'abducteur du cinquiéme doigt, et ce de chaque côté.

5. La comparison des données fournies par le laboratoire, des signes neurologiques et de l'electrodiagnostic ont révelé les faits suivants:

(a) Il n'existe pas de relation directe entre l'activité de la maladie et la condition de l'unité motrice, une fois qu'est apparue la lésion motrice;

(b) Aucune corrélation n'a été notée entre le type, ou la forme, de la maladie, et les signes neurologiques ou les rèsultats de l'électrodiagnostic;

(c) Il n'y a pas de relation entre la condition dermatologique et les données de laboratoire, d'une part, et entre les observations neurologiques et electriques d'autre part;

(d) Il a été établi que la perte du pouvoir musculaire est le signe neurologique le plus fidèle;

(e) L'absence de concordance entre la perte du pouvoir musculaire et l'atrophie est frappante;

(f) Il a été démontré que le signe de Tinel et l'épaississement nerveux constituent des signes présomptifs valables d'une lésion nerveuse, quoique ces manifestations ne soient pas liées à la capacité motrice;

(g) L'épreuve galvano-faradique, ainsi qu'on a pu le démontrer, n'est pas une épreuve fidèle ou valable lorsqu'une lésion mineure est présente;

(h) La courbe d'intensité en fonction du temps constitue l'épreuve la plus sensible pour l'évaluation globale de n'importe quel muscle en particulier en relation avec le nerf qui l'innerve. Cette courbe, toutefois, est dépendante de l'excitation extérieure et ne permet pas l'évaluation du muscle liu-même, pas plus qu'elle ne permet d'identifier des lésions disséminées;

(i) L'electromyographie est l'épreuve la plus précise, pour autant que l'exploration soit suffisament étendue;

(j) Les résultats d'enregistrements electromyographiques multiples indiquent qu'il pourrait y avoir des atteintes directes du muscle. Cette observation, si elle était confirmée, revêtirait une grande importance dans la conception de base du dommage moteur au cours de la lèpre;

(k) L'electromyographie a permis de mettre en évidence un dommage au niveau d'un nombre significatif de muscles qui paraissaient cliniquement sains. S'il apparait que cette observation est correcte, ceci serait d'importance primordiale pour la réhabilitation chirurgicale.

6. Pour le moment, les auteurs étudient la signification de la courbe d'intensité en fonction du temps pour le pronostic de la lésion nerveuse. Ils ménent aussi des études histologiques ainsi que des analyses de fréquence des enregistrements electromyographiques, en vue de mettre en évidence, le cas échéant, une lésion musculaire primaire ou des lésions occultes du nerf. La relation entre la perte de la capacité musculaire évaluée cliniquement et l'atrophie fait l'objet d'études supplémentaires.

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