Lepr. Rev. (1972) **42**, 208-218

Nerve Enlargement in Relation to Classification of Leprosy*

M. A. FURNESS and D. A. RANNEY

Schieffelin Leprosy Research Sanatorium, S.L.R. Sanatorium P.O., via Katpadi, N.A. District, S. India

While certain details of the pathogenesis of nerve involvement in leprosy are rather obscure, it is well known that peripheral nerves are damaged at certain sites of predilection. The nerves become enlarged and tender and exhibit a varying consistency on palpation. Although the changes are due essentially to the presence of *Myco. leprae* in the nerve, the consistence of the enlarged nerve depends on the amount of oedema and cellular infiltration within the nerve and of fibrosis in the sheath or in the nerve itself (Browne, 1963). Superficiality, repeated trauma, constricting bands, lower temperature, and a greater temperature variation (Brand, 1959) have all been suggested to explain the apparently complex pathogenesis of nerve involvement in leprosy. Not only truncal nerves but also cutaneous nerves, which are normally difficult or impossible to palpate, may become so enlarged as to be easily palpable and sometimes visible. Occasionally nerve abscesses may be encountered.

From the detailed studies of peripheral-nerve enlargement reported by Chatterji (1933, 1936) at the Calcutta clinic, it was obvious that he was dealing mainly with the tuberculoid type of leprosy. Murdock (1949), following an intensive study, documented the frequency and distribution, without reference to classification, of peripheral-nerve enlargement in 117 leprosy patients and compared his results with those of Chatterji. Browne (1965), in an interesting paper, reported some less common neurological findings made over a period of 28 years and covering a large number of patients.

The present prospective study was undertaken to obtain more precise information with regard to the pattern of clinical manifestations, and of the frequency and distribution of enlargement of the cutaneous and truncal nerve in the various types of leprosy.

Patients and Methods

A total of 540 leprosy patients were chosen by stratified random selection from 41 peripheral clinics of the leprosy control programme undertaken by the Schieffelin Leprosy Research Sanatorium, Karigiri. Of these, 130 patients were classified as having indeterminate leprosy, 130 tuberculoid, 130 lepromatous, and 150 borderline leprosy. The classification of the type of leprosy was determined by the usual clinico-bacteriological criteria employed for this purpose; both male

* Approved for publication 3 September, 1971.

and female patients were included in the study. All the patients were receiving antileprosy treatment at the time of assessment. Patients in acute lepra reaction and those with quiescent leprosy were excluded.

The study was conducted by means of repeated visits to all peripheral clinics over a period of 6 months and was based on the patients who were attending the clinic at the time of the visit. Special forms listing all the cutaneous and truncal nerves in the head and neck, trunk and upper and lower limbs were used in the course of the clinical examination, which consisted in careful inspection and palpation of the nerves. Palpation in each was done across the course of the nerve and compared with that on the opposite side. It was sometimes necessary to distinguish an enlarged nerve from an enlarged lymphatic gland or leprotic nodule. Skin lesions and the areas surrounding them were palpated to detect nerve enlargement. In addition, cutaneous and truncal nerves were routinely palpated at the usual levels where they are subcutaneously placed, and any pain or tenderness was recorded. Particular attention was paid to the consistency of enlarged nerves, which were classified as hard, soft, and beaded or node-like. In a few cases where a nerve abscess was suspected, surgical exploration was undertaken by one of the authors (D.A.R.). Fibrosis of the nerve and paralysis when present were noted. In all cases the patient's age, sex, duration of the disease, and treatment received were recorded (Table 1).

Results

Table 1 shows the number of male and female patients studied in each group. It also records the group's average age and the duration of disease and treatment according to classification.

In Table 2, the nerves that were found enlarged on palpation in 130 patients with indeterminate leprosy are recorded. The consistency of the nerve and its association with any skin lesions, pain and tenderness, fibrosis, or paralysis are shown where present. Table 3 shows the results in the 130 patients with tuberculoid leprosy. The frequency of unilateral and bilateral involvement of nerves and the consistency of the nerve is noted. The relationship of nerves to skin lesions, pain, tenderness and paralysis is also recorded.

Table 4 records the findings on examination made of the cutaneous and truncal nerves of 130 patients with lepromatous leprosy. The consistency of the nerve

TABLE 1

Age, sex, duration of disease and treatment according to classification

Classification	Male	Female	Total	Average duration of disease (years)	Average duration of treatment (years)	Average age of patients (years)
Indeterminate	76	54	130	2.8	1.7	14.4
Tuberculoid	76	54	130	3.3	2.3	22.3
Lepromatous	85	45	130	7.3	3.0	31.4
Borderline	96	34	130	5.1	2 9	28.0

No significant difference in nerve involvement was noted between male and female patients in any of the groups studied.

TABLE 2

Indeterminate leprosy

Nerves	Unilateral		Bilateral		Hard		Enlargement Beaded		Soft	
	No.	%	No.	%	No.	%	No.	%	No.	%
Head and neck										
Greater auricular	4	3.1	1	0.8	2	1.5	–	–	4	3.1
Upper limb										
Lat. antebrach. cut.	3	2.3	4	3.1	7	5.4	–	–	4	3.1
Ulnar	23	17.7	15	11.5	20	15.4	–	–	33	25.4
Radial	1	0.8	–	–			–	–	1	0.8
Ulnar cutaneous	6	4.6	–	–	1	0.8	–	–	5	3.8
Radial cutaneous	12	9.2	3	2.3	3	2.3	–	–	15	11.5
Lower limb										
Lateral popliteal	15	11.5	14	10.8	20	15.4	–	–	23	17.7
Musculo-cutaneous	10	7.7	–	–	5	3.8	–	–	6	4.6
Posterior tibial	5	3.8	4	3.1	12	9.2	–	–	2	1.5
Totals	79	–	41	–	70	–			93	–

TABLE 3

Tuberculoid leprosy

Nerves	Unilateral		Bilateral		Enlargement: Hard		Enlargement: Beaded		Enlargement: Soft		Skin lesion		Tenderness		Pain		Fibrosis		Paralysis	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No	%	No.	%
Head and neck																				
Supratrochlear	2	1.5	1	0.8	1	0.8			3	2.3	4	3.1								
Supraorbital	1	0.8			1	0.8					1	0.8								
Infraorbital	6	4.6	1	0.8	2	1.5			6	4.6	8	6.2								
Upper division (F)	3	2.3	1	0.8	3	2.3.	1	0.8	1	0.8	5	3.8								
Greater auricular	16	12.3	13	10.0	27	10.8	2	1.5	13	10.0	41	31.5								
Smaller occipital	1	0.8	1	0.8	1	0.8			2	1.5	3	2.3								
Cervical cutaneous	1	0.8	–	–	–	–			1	0.8	1	0.8								
Upper limb																				
Dorsal antebrach. cut.	4	3.1	2	1.5	8	6.2					6	4.6								
Med. antebrach. cut.	4	3.1			3	2.3			1	0.8	4	3.1								
Lat. antebrach. cut.	34	26.2	20	15.4	56	43.1			18	13.8	53	40.8	1	0.8						
Ulnar	39	30.0	40	30.8	65	50.1	2	1.5	29	22.3	72	55.4	2	1.5	2	1.5			5	3.8
Median	10	7.7	2	1.5	12	9.2			3	2.3	11	8.5							1	0.8
Radial	5	3.8	2	1.5	8	6.2			3	2.3	7	5.4	1	0.8						
Ulnar cutaneous	21	16.2	6	4.6	23	17.7			9	6.9	28	21.5	1	0.8						
Radial cutaneous	34	26.2	27	20.8	54	41.5	2	1.5	28	21.5	73	56.2	2	1.5						
Lower limb																				
Lateral popliteal	29	22.3	33	25.4	67	51.5			25	19.2	92	70.8					1	0.8	1	0.8
Musculo-cutaneous	20	15.4	12	9.2	32	24.6			12	9.2	44	33.8								
Sural	1	0.8	1	0.8					3	2.3	3	2.3								
Posterior tibial	6	4.6	9	6.9	20	15.4	1	0.8	3	2.3	22	16.9								
Totals	237	–	171	–	383	–	8	–	160	–	478	–	7	–	2	–	1	–	7	–

(1) Ulnar nerve abscess

TABLE 4

Lepromatous leprosy

Nerves	Unilateral		Bilateral		Enlargement: Hard		Enlargement: Beaded		Enlargement: Soft		Skin lesion		Tenderness		Pain		Fibrosis		Paralysis	
	No.	%	No.	%	No.	%	No.	%	No.	%	No	%	No.	%	No.	%	No.	%	No.	%
Head and neck																				
Supratrochlear	3	2.3	3	2.3	2	1.5			7	5.4										
Supraorbital	3	2.3	9	6.9	1	0.8			20	15.4										
Lacrimal	3	2.3							3	2.3										
Infraorbital	7	5.4	6	4.6	2	1.5			18	13.8										
Upper division (F)	1	0.8							1	0.8										
Greater auricular	24	18.5	43	33.1	26	20.0	1	0.8	80	61.5										
Smaller occipital	1	0.8							1	0.8										
Greater occipital			1	0.8					2	1.5										
Supraclavicular	7	5.4	2	1.5					11	8.5										
Upper limb																				
Dorsal antebrach. cut.	6	4.6	2	1.5	6	4.6			4	3.1										
Med. antebrach. cut.	6	4.6	2	1.5	6	4.6			4	3.1										
Lat. antebrach. cut.	31	23.8	34	26.2	49	37.7	1	0.8	33	25.4			3	2.3	2	1.5				
Ulnar	16	12.3	95	73.1	84	64.6	3	2.3	110	84.6			4	3.1	2	1.5	1	0.8	1	0.8
Median	3	2.3	15	11.5	2	1.5			29	22.3			2	1.5	2	1.5	2	1.5	2	1.5
Radial	11	8.5	4	3.1	10	7.7			9	6.9										
Ulnar cutaneous	28	21.5	30	23.1	26	20.0			56	43.1										
Radial cutaneous	21	16.2	84	64.6	69	53.1	1	0.8	103	79.2			3	2.3	2	1.5				
Lower limb																				
Lateral popliteal	23	17.7	89	68.5	88	67.7			110	84.6			2	1.5	2	1.5				
Musculo-cutaneous	34	26.2	30	23.1	36	27.7			58	44.6										
Sural	4	3.1			2	1.5			2	1.5										
Posterior tibial	27	20.8	41	31.5	43	33.1			66	50.8			2	1.5						
Totals	259	–	490	–	452	–	6	–	727	–			16	–	10	–	3	–	3	–

TABLE 5

Borderline leprosy

Nerves	Unilateral		Bilateral		Enlargement: Hard		Enlargement: Beaded		Enlargement: Soft		Skin lesion		Tenderness		Pain		Fibrosis		Paralysis	
	No.	%	No.	%	No	%	No.	%	No.	%	No.	%	No.	%	No.	%	No	%	No.	%
Head and neck																				
Supratrochlear	3	2.0	2	1.3					7	4.7	4	2.7								
Supraorbital	6	4.0	6	4.0	1	0.7			17	11.3	7	4.7								
Lacrimal	4	2.7	4	2.7					12	8.0	6	4.0								
Infraorbital	10	6.7	6	4.0	2	1.3			20	13.3	10	6.7								
Mental	2	1.3							2	1.3	2	1.3								
Upper division (F)	5	3.3	1	0.7	2	1.3			5	3.3	6	4.0			1	0.7			3	2.0
Lower division (F)	3	2.0							3	2.0	1	0.7								
Greater auricular	25	16.7	41	27.3	31	20.7	6	4.0	70	46.7	16	10.7			2	1.3				
Smaller occipital	3	2.0			2	1.3			1	0.7	3	2.0								
Curvical cutaneous	2	1.3							2	1.3	1	0.7								
Supracalvicular	3	2.0	3	2.0	2	1.3			7	4.7	2	1.3								
Upper limb																				
Dorsal antebrach. cut.	5	3.3	5	3.3	10	6.7			5	3.3	11	7.3								
Med. antebrach. cut.	7	4.7	2	1.3	5	3.3			6	4.0	8	5.3								
Lat. antebrach. cut.	43	28.7	34	22.7	62	41.3			47	31.3	22	14.7								
Ulnar	26	17.3	96	64.0	73	48.7	1	0.7	134	89.3	35	23.3	6	4.0	2	1.3	8	5.3	8	5.3
Median	10	6.7	17	11.3	7	4.7			35	23.3	11	7.3	4	2.7	1	0.7	2	1.3	3	2.0
Radial	10	6.7	6	4.0	6	4.0			17	11.3	11	7.3	3	2.0						
Ulnar cutaneous	39	26.0	28	18.7	28	18.7	2	1.3	65	43.3	24	16.0	2	1.3	1	0.7				
Radial cutaneous	42	28.0	73	48.7	57	38.0	3	2.0	128	85.3	36	24.0	2	1.3	1	0.7				
Lower limb																				
Lateral popliteal	29	19.3	94	62.7	59	39.3			134	89.3	25	16.7	4	2.7	2	1.3	3	2.0	3	2.0
Musculo-cutaneous	36	24.0	41	27.3	25	16.7			88	58.7	24	16.0		2.0	2	1.3				
Sural	3	2.0	3	2.0					9	6.0	8	5.3								
Posterior tibial	30	20.0	38	25.3	33	22.0			71	47.3	14	9.3	4	2.7	2	1.3				
Totals	346	–	500	–	405	–	12	–	885	–	287	–	28	–	14	–	13	–	17	–

Rt. ulnar abscess (1) Beaded

Hard nerves, unilateral involvement and association of enlarged nerves with skin lesions were abscessed in the borderline-tuberculoid group. Soft nerves and bilateral involvement were seen in the borderline-lepromatous group.

and the frequency of unilateral and bilateral involvement of these nerves are shown. Pain, tenderness, fibrosis, paralysis, and the association of enlarged nerves with skin lesions are also recorded. Lastly, Table 5 shows the results in the 150 patients with borderline leprosy. Patients with borderline-tuberculoid, borderline-borderline, and borderline-lepromatous were included in this group. The number of nerves thickened unilaterally and bilaterally, the consistency of the nerves, and the association of the nerves with skin lesions, pain and tenderness, as well as fibrosis and paralysis are recorded.

Discussion

Since the earliest lesions of leprosy usually constitute the indeterminate group, and also because many of the patients were children, the average age was distinctly lower and the duration of the disease and of treatment shorter in this group than in the other three groups (Table 1). There was also a relative infrequence of nerve enlargement in the indeterminate group, only one or a few nerves being involved in each patient. Although 34 nerves were routinely examined in each case enlargement was recorded only in the 9 nerves listed in Table 2. The nerve enlargement was discrete, was just perceptible on palpation, with the consistency varying from hard to soft; pain and tenderness were not elicited in any of the nerves examined. This confirms the accepted view that thickening of peripheral nerves is usually not detected in the early indeterminate group of leprosy, although later some of the nerves may be found enlarged. It may be assumed that even though acid-fast bacilli may be found within the nerves, the bulk of the nerve parenchyma remains unaffected at this stage of the disease (Iyer, 1965). Another interesting finding was that cutaneous nerves supplying the area in which skin lesions are situated were not found to be enlarged. It seems appropriate to comment on the particular involvement of the ulnar nerve and the lateral popliteal nerve even at the stage of indeterminate leprosy. In addition to such factors as superficiality, temperature, constricting bands and trauma already mentioned, it was observed by Sunderland (1953*a*, 1953*b*) that the ulnar nerve behind the medial epicondyle of the humerus and the lateral popliteal nerve behind the neck of the fibula are composed of few and larger funiculi with little connective tissue between them. On the basis of the variation in the relative number of funiculi and amount of connective tissue in different nerves, and also along the course of a particular nerve, it is apparent that nerves or segments of nerves which are composed of only a few, but large, funiculi will have a much larger proportion of the nerve affected than those with the same number of funiculi destroyed but whose funiculi are smaller and more numerous. Furthermore, the thinness of the interfunicular connective tissue would render the nerve more vulnerable to extraneural trauma, as well as allowing the transmission from one funiculus to another of pressure and possibly even bacteria. Also, since nerves which have few funiculi tend to have a greater proportion of their blood supply more superficially placed, such nerves are more vulnerable to trauma. These factors may combine to allow the adverse influences of trauma and infection to operate and so make the nerves particularly vulnerable to damage.

The characteristic feature in the group of 130 patients with tuberculoid leprosy (Table 3) was the enlargement of cutaneous and truncal nerves supplying the area in which skin lesions were situated. This feature was marked in most of the nerves examined in the head and neck and the upper and lower limbs. Enlargement of

branches of the facial and trigeminal nerves was in all cases associated with skin lesions on the face. One or more cutaneous branches and one or more truncal nerves were observed to be associated with a single skin lesion. Sometimes a nerve supplying a small lesion was grossly hypertrophic while that supplying an extensive lesion was found to be only slightly thickened. Other features were that only a few nerves were involved in each patient and that the involvement was more frequently unilateral than bilateral and depended to a large extent on the distribution and number of skin lesions, the neurological patern being determined by the asymmetrical distribution of a few skin lesions. As may be expected with tuberculoid leprosy, where the cellular reaction to paucibacillary infection in nerves is vigorous, the consistence of the nerves was mostly hard. Tuberculoid leprosy is a form of disease in which the body defences are adequate and in which attempts are made to localize the disease. Since the bacilli are also located in the nerve, the reaction takes place within the nerve. This is probably an antigen-antibody disorder confined to nerve tissue (Iyer and Desikan, 1968), the *Myco. leprae* being the antigen which provides the trigger enabling an immune response to be adjuvated and to take place rapidly (Weddell *et al.,* 1963). Therefore nerve involvement in tuberculoid leprosy may be considered a delayed hypersensitivity reaction, with *Myco. leprae* merely acting as an adjuvant. Other agents, traumatic or toxic, acting on nerve tissue may also be precipitating factors in determining this nerve damage (Browne, 1965).

In the softer nerves the absence of tenderness may be due, to some extent, to the effect of therapy. Some beaded enlargement was encountered. One beaded ulnar nerve was surgically explored and found to contain an area of softening. In this group of patients with tuberculoid leprosy there was little relation between nerve thickening and paralysis.

In all the patients with lepromatous leprosy many nerves were found to be enlarged, and more nerves were involved bilaterally than unilaterally (Table 4). The consistence was recorded as soft in 727 nerves, compared with 452 nerves which were felt to be hard. A few beaded nerves were encountered, but none were surgically explored. Although pain and tenderness were present in some of the nerves examined in both upper and lower limbs, paralysis was noted in only one ulnar and two median nerves. The average age, duration of the disease and of treatment were also proportionately greater in this group. It has been demonstrated by Job and Desikan (1968) that bacilli are found throughout the course of the nerve but are particularly abundant at those sites where the nerves are placed subcutaneously. As cellular infiltration of the nerves is generally minimal the clinical evidence of damage is generally late, that is, from the third year onwards (Browne, 1965). An epidemiological study by Srinivasan (1965) showed that deformities were generally more frequent in lepromatous than in non-lepromatous leprosy. It was suggested that in a systemic disease like lepromatous leprosy, where there is a haematogenous spread of the infection, more nerves are likely to be involved with, in consequence, a greater likelihood of nerve damage. In addition to non-paralytic deformities that are likely to occur during lepra reaction (Furness *et al.,* 1968) a significant increase in neurological deficit was also recorded (Karat *et al.,* 1969) in a group of patients with recurrent and/or chronic erythema nodosum leprosum (ENL) as compared with a control group without reactions. The unrestrained multiplication of *Myco. leprae* within the reticulo-endothelial cells in the anergic type of leprosy seems to suffer no limitations except those imposed by some little understood need for proximity to

the surface of the body. There is a polyneuritis and nerves are enlarged at sites where they are superficial, suggesting that their involvement may be temperature-dependent.

In the borderline group of 150 patients, the characteristics of nerve enlargement represented an admixture of those seen in tuberculoid and lepromatous leprosy. In these cases 23 different nerves out of a total of 34 examined were found to be enlarged in the head and neck, trunk, and upper and lower limbs (Table 5). Whereas 500 nerves were enlarged bilaterally, only 346 showed unilateral enlargement. A soft consistence was recorded in 885 nerves, 405 nerves felt hard, and 12 beaded on palpation. Although 287 enlarged cutaneous and truncal nerves were found to be associated with skin lesions, this was less than the 478 nerves found to be associated with skin lesions in tuberculoid leprosy (Table 3). Unilateral involvement, hard nerves and some relation of nerve involvement to skin lesions were observed mainly in the borderline-tuberculoid group in this study, while bilateral enlargements and soft nerves were noted in patients with borderline-lepromatous. An abscess was found during surgical exploration of a right ulnar nerve in a patient with borderline-tuberculoid leprosy. Pain and tenderness were present in several nerves in the upper and lower limbs and face. Paralysis was more frequent in borderline leprosy than in any of the other groups studied.

In none of the groups studied did there appear to be any relationship between nerve enlargement and paralysis. Sherron (1945) has stated that about one-third of a nerve can be divided without producing demonstrable motor or sensory deficit. Sunderland (1945), following studies made by intraneural tomography, observed that owing to reassortment of fibres in progress in the proximal portion of the nerve and to the fibre composition of funiculi at high levels, the injury may involve those bundles which contain only a few fibres of some or all of the branches, so that the resultant loss of function may not be detected clinically. These reasons explain in part the normal function of infected, enlarged nerves in leprosy. Although local tenderness has been known to persist in some enlarged nerves after clinical and bacteriological arrest of the disease, its marked infrequency in the groups studied can be attributed only to effective therapy.

Summary

A group of 540 leprosy patients, of whom 130 had indeterminate leprosy, 130 tuberculoid, 130 lepromatous, and 150 borderline disease, were examined by inspection and palpation to study the pattern of clinical manifestations and frequency and distribution of cutaneous and truncal nerve enlargement according to classification.

A relative infrequency of nerve enlargement was recorded in the group with indeterminate disease, only one or a few nerves being involved in each patient. The nerve thickening was discrete, just perceptible on palpation, with a consistence varying from hard to soft. The particular predilection for involvement of the ulnar and lateral popliteal nerves, even at the indeterminate stage, is discussed.

The characteristic feature of the tuberculoid group was the enlargement of cutaneous and truncal nerves supplying the area in which the skin lesions were situated, the neurological pattern being determined by the asymmetrical distribution of a few skin lesions. The nerves were felt to be mostly hard. The

opinion is expressed that the nerve enlargement is probably a delayed hypersensitivity reaction, with *Myco. leprae* merely acting as an adjuvant.

Many nerves were involved in lepromatous leprosy. The involvement was predominantly bilateral rather than unilateral, while the consistence of the nerve was felt to be soft. The unrestrained multiplication of *Myco. leprae* at sites of predilection where such sites are superficial suggests that nerve involvement in lepromatous leprosy is temperature-dependent.

In the borderline group, the characteristics of nerve enlargement represented an admixture of those seen in tuberculoid and in lepromatous leprosy. Hard nerve enlargements, unilateral involvement, and association of nerves with skin lesions were noted in the borderline-tuberculoid group, while bilaterally involved soft nerves were seen in the borderline-lepromatous group. Nerve enlargement was most frequent in this latter group of patients.

No significant difference in nerve involvement was noted between male and female patients in any of the groups studied.

In none of the groups studied did there appear to be any relationship between nerve enlargement and paralysis. Some reasons for this discrepancy are suggested. The marked absence of pain and tenderness in the nerves was probably due to the effect of treatment.

The duration of disease and of treatment was longest in the group of patients with lepromatous leprosy, and shortest in the patients with indeterminate leprosy.

Acknowledgements

Our thanks are due to Mr. D. Ramachandra Rao, physiotherapy technician, for his invaluable assistance in the collection of data for the study, and to Mrs. L. Furness for secretarial help in preparing the manuscript. Financial assistance was given by the Social and Rehabilitation Service of the United States Department of Health, Education and Welfare (SRS-IND-32), for which we are also grateful.

References

Brand, P. W. (1959). Temperature variation and leprosy deformity. *Int. J. Lepr.* **27**, 1.

Browne, S. G. (1963). Some less common neurologic findings in leprosy. Reprinted in *Int. J. Lepr.* (1968) **31**, 881.

Browne, S. G. (1965). Some clinical problems awaiting solution by research. *Int. J. Lepr.* **33**, 759.

Chatterji, S. N. (1933). Thickened nerves in leprosy in relation to skin lesions. *Int. J. Lepr.* **1**, 283.

Chatterji, S. N. (1936). Neural affections in leprosy and their diagnosis, pathology and treatment. *Indian Med. J.* **30**, 414.

Furness, M. A., Karat, A. B. A. and Karat, S. (1968). Deformity in the reactive phases of leprosy. Aetiology and physiotherapeutic management. *Lepr. Rev.* **39**, 135.

Iyer, C. G. S. (1965). Predilection of *M. leprae* for nerves. Neuro-histopathologic observations. *Int. J. Lepr.* **33**, 634.

Iyer, C. G. S. and Desikan, K. V. (1968). Nerve involvement in leprosy. Pathogenesis and significance. *Neurology (India)* **16**, 89.

Job, C. K. and Desikan, K. V. (1968). Pathologic changes and their distribution in peripheral nerves in lepromatous leprosy. *Int. J. Lepr.* **36**, 257.

Karat, A. B. A., Furness, M. A., Karat, S. and Rao, P. S. S. (1969). Patterns of neurological involvement in relation to chronic and/or recurrent erythema nodosum leprosum. *Lepr. Rev.* **40**, 49.

Murdock, J. R. (1949). Thickening of superficial nerves as a diagnostic sign in leprosy. *Int. J. Lepr.* **17**, 1.

Sherron, J. (1945). *Injuries of Nerves and their Treatment.* New York, William Wood & Co. 1907. Quoted by Sunderland, S. (1945). The intraneural tomography of the radial, median and ulnar nerves. *Brain,* **68**, 243.

Srinivasan, H. (1965). Personal communication. Quoted by Iyer, C. G. S. (1965) q.v.

Sunderland, S. (1945). The intraneural tomography of the radial, median and ulnar nerves. *Brain,* **68**, 243.

Sunderland, S. (1953*a*). Funicular suture and funicular exclusion in the repair of severed nerves. *Br. J. Surg.* **40**, 581.

Sunderland, S. (1953*b*). The relative susceptibility to injury of the medial and lateral popliteal divisions of the sciatic nerve. *Br. J. Surg.* **41**, 300.

Weddell, G., Palmer, E., Rees, R. J. W. and Jamison, D. G. (1963). *Experimental Observations Related to the Histopathology of Leprosy,* pp. 3-15. CIBA Foundation Study Group No. 15. London, J. & A. Churchill.