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EDITORIALS

Editorials are written by members of the Editorial Board, and occasionally by guest editorial writers at the invitation of the Editor, and opinions expressed are those of the writers.

Paralysis of Nerves in Leprosy[1]

This number carries two very interesting articles on a study of facial nerves in leprosy patients. The authors are to be congratulated on a bold and careful investigation of a subject that has not had enough attention. We hope that others will follow up with investigations of a different type, aimed at prevention of the paralysis of facial muscles which has caused so much distress and blindness among those who have suffered from leprosy.

The authors have not attempted to mask the difficulties of investigating nerve lesions in leprosy, nor have they minimized the fact that it is not without danger to the patient. Such extensive dissections of the nervous system were justifiable only because the investigator was a skilled plastic surgeon who could use a standard exposure, and could use the same operation to give the patient a much needed face lift, and an essential correction of eyelid paralysis. Even so, the high incidence of some degree of postoperative facial weakness must be a caution to any surgeon who wishes to undertake surgical dissection of nerves that are partially paralyzed by leprosy. The surgeons at Vellore, India, had a similar experience when they undertook extensive stripping of the sheath of the ulnar nerve for decompression. Although the patients all experienced dramatic relief of their pain, careful sensory and muscle testing sometimes showed that the paralysis had increased.

These studies have demonstrated that careful electromyographic investigations before operation were confirmed by electric stimulation at operation and by the biopsy specimens removed. The gross appearance of the nerve, however, gave very little help in diagnosis, and would have been an unreliable basis for defining the level or the extent of paralysis. Fibrosis has again been identified as a probable precursor of paralysis, and much of the fibrosis is within the nerve. Even though some fibrous tunnels were observed around some branches, the operative decompression of these constrictions does not seem to have been followed by clinical improvement in nerve function.

These observations tend rightly to discourage those who advocate routine surgical intervention in cases of early neuritis and to encourage those who seek for con-

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servative measures for the prevention of paralysis. They should also encourage the use of the electromyograph as a tool of clinical investigation, to give a measure of the changing response of the nerves to various forms of treatment. We should be stimulated by the careful and objective way in which those studies have been carried out.

These papers focus some attention on all three of the main areas of interest concerning nerves in leprosy. Since all three areas need a great deal more study, I will try to define them and point out why we need to keep them separate from each other in our thinking.

1. The relationship of nerve elements to the *Mycobacterium leprae*.
2. The investigation of the causes of paralysis.
3. The management of the patient who is paralyzed.

1. In their careful study of the biopsies, the team in Bombay has again drawn attention to the Schwann cells, and to their relationship to the bacilli. Last year the studies of Weddell, Rees and Palmer gave some interesting insight into the microscopic relationships of the bacilli and bacillary debris to various neural elements and to the Schwann cells. If it proves true that the mycobacteria are in some sort of protective environment in the Schwann cell, and if this may prove to be a reservoir of infection in patients whose other tissues are negative, then this opens up a very important field of research. This is a study that may help us to understand and to control leprosy, but it is not so likely to have a direct effect on the course of paralysis, because *M. leprae* is known to exist and to multiply in nerves that show no signs of paralysis. Conversely, an intense reaction to a very few bacilli may result in paralysis.

2. The paralysis of a major nerve seems to require not only the presence of bacilli, but also an inflammatory reaction or fibrosis or both. This localized edema and inflammation occurs in association with some other factors that have never been well defined. Antia *et al*, point out that in the facial nerve "one anatomic feature that seems peculiar to the damaged nerves was the background constituted by the bony prominence of the zygoma underlying them." Others have noted that nerves become paralyzed near joints, or near the surface of the body, or at sites where they are liable to trauma, or at sites where the nerve is subject to variations of temperature, or at situations where they may suffer compression or entrapment, and many workers have noted the relationship between paralysis and certain types of reactive states. A number of surgeons have practiced and recommended various ingenious operations designed to change the situation of a nerve and to protect it from paralyzing factors. So far not one of these methods has been supported by a convincing controlled study.

Now, with the careful use of the electromyograph, it should be possible to pinpoint with greater accuracy not only the common sites of paralysis but also every variation in the progress of the damage. Thus the surgeon or physician who holds a theory about the precipitating factor in nerve paralysis may use surgical or medical measures to modify the environment of a nerve that is becoming paralyzed and may then monitor its progress day by day, for comparison with control cases.

In the absence of such careful follow-up studies, it is our personal opinion that surgical intervention in leprosy neuritis should be limited to exposure of the nerve at the site of inflammation, and to longitudinal incision of the sheath, or other constrictive structure, without disturbing the blood supply of the nerve coming from its deeper surface. An inflamed edematous nerve may have lost its normal longitudinal blood supply, and depend largely on accessory blood supply from its surrounding tissues.

Routine treatment of leprosy neuritis at the present time must depend upon medical measures to reduce the inflammation in the nerve, and conservative measures to keep the inflamed nerve at rest and warm, for example by means of a padded splint.

3. Dr. Antia has just touched on the third aspect of the problems of paralysis, and he reminds us that we need to do something for the patient in whom we

have failed to prevent paralysis. The temporalis muscle transfer for lagophthalmos is just one of many operations that need to be made available to those who may be regarded as failures of medical treatment, but who can still be saved from gross disability and deformity.

—Paul W. Brand