Paralysis of Nerves in Leprosy

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 cou...
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 Weckdell, Foes and Palmer gave some interesting insight into the microscopic relationships of the bacilli and ciliary 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 interstitial 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 disabilty and deformity. —Paul W. Brand

BCG Vaccination Against Leprosy

The importance and value of a protective vaccine against leprosy are obvious. Unfortunately a specific vaccine prepared from killed or attenuated bacteria is not available for testing because the causative organism of leprosy, M. leprae, has not been cultured in vitro. However, the original observation of Fernández in 1939 (1) that conversion to lepromin positivity occurred in a large proportion of lepromin-negative children following BCG vaccination, an observation subsequently confirmed by many workers, led him to suggest that BCG vaccination might confer some protection against leprosy. The later work and writings of Fernández (8) and Chauvinand (3) drew attention particularly to the possible similarities between tuberculin and lepromin sensitivity as a measure of protective immunity against tuberculosis and leprosy respectively. Because BCG vaccination could induce positivity to both skin tests in negative subjects, and because there was already evidence that BCG vaccination gave protection against experimental tuberculosis, there was a strong case for using BCG vaccination against leprosy in man. Their views dominated the field of leprosy, and several small trials of BCG vaccination against leprosy followed, in particular those by de Souza Campos (9), Fernández (8) and Conloit (1). Although there was some suggestion of protection in the vaccinated group, especially against the development of lepromatous type leprosy, none of the trials was carried out on a large enough scale or with adequate control or special care to establish the vaccinated and unvaccinated groups by random allocation, so as to withstand critical analysis.

This was no particular criticism of the leprosy workers, because, in fact, exactly the same criticism and uncertainty existed at that same time regarding the value of BCG vaccination against tuberculosis. In fact, it was not until 1956, with the first publication of the definitive trials undertaken by the British Medical Research Council (1) that the value of BCG against tuberculosis in man was finally established to the satisfaction of all the critics. For several years recommendations had been made to establish large-scale trials of BCG vaccination against leprosy which would yield scientifically acceptable results, and these had been urged by successive WHO Expert Committees on Leprosy (14, 19) and International Congresses of Leprology. Therefore the first progress report by Kinnear Brown and Stone on a large-scale trial of BCG vaccination of children against leprosy (15) is a most important contribution to this difficult and controversial subject. The report is of an investigation into the prophylactic effect of BCG vaccine planned by the Uganda Government with continuing scientific and technical guidance by the Leprosy Committee of the British Medical Research Council. The controlled trial was initiated in September 1960 in the Teso District of Eastern Uganda, and, by September 1962, 19,079 children, more than 80 per cent of whom were aged under 10 years, had been included. All were relatives or contacts of known leprosy patients. All the children were examined and those with leprosy or with suspected leprosy lesions were recorded; all were tuberculin-tested also, by the Heuf multiple puncture method, but the majority of the trial children were not lepromin-tested. The children with negative reactions (Grade 0) or with weak positive reac-