REPLY: THE ROLE OF NERVE BIOPSIES IN THE DIAGNOSIS AND MANAGEMENT OF LEPROSY

Sir,

The paper by Dr R Nilsen *et al.* (*Lepr Rev*, 1989; **60**: 28–32) provides further evidence to substantiate the differences in bacterial load and histological response between nerve and skin lesions and the risk of relapse commencing in nerve. It is an important fact that skin lesions may not be representative of nerves, especially in paucibacillary patients. However, the authors go on to state, incorrectly, that their findings are at variance with our previous conclusion¹ that skin rather than nerve demonstrates the general tissue response to *Mycobacterium leprae*. Since they may not be alone in this view we should like to try to elucidate the position.

It is now 14 years since Pearson & $Ross^2$ and later Stoner³ interpreted the predilection of M. *leprae* for nerve by reference to the immunological protection afforded to the bacillus by the Schwann cell basement membrane, the multilayering of the perineurium, the absence of lymphocyte recirculation within the fascicles and the blood-nerve barrier. This hypothesis, as far as we know, has not been contested and our own results^{1,4} are strong evidence in support. Yet more often than not, attempts are made to interpret neural leprosy as the consequence of some uniqueness in the neural-bacillary relationship. It really does not make sense to say that 'an immunologically nonresponsive form of leprosy can reside in nerve lesions and a responsive form in skin lesions'. A leprosy infection, like the patient, is one, even though the bacterial load and the response to it vary between sites some of which are immunologically protected and others exposed. For two reasons the general tissue response is the one seen in unprotected (or relatively unprotected) sites such as skin: 1, the majority of sites in the body are unprotected, the minority protected; and 2, it is only after exposure of antigen that the immunological response to it can be evaluated. Similarly the general bacterial level is the one which has developed at open sites, not behind immunological barriers. This in no way diminishes the importance of the vital events that do take place behind the barriers such as nerve. But to suggest that the classification of leprosy ought to be based on the status of nerve rather than skin lesions is not right. The events in a protected site are variable and unpredictable, and the findings in a nerve biopsy are valid only for that particular site.

The widely held view that there is an affinity between Schwann cell and leprosy bacillus is of course not incorrect, but the uniqueness of the affinity pertains to the bacillus, not the Schwann cell. There are a number of other human tissues besides nerve in which, perhaps uniquely, *M. leprae* is remarkably well tolerated. There are also a number of other protected sites, though none in which the bacilli multiply so freely as in nerve. In the context of leprosy, peripheral nerve is not unique, but it is pre-eminent among protected sites as the one with the best growth potential.⁵

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