been suggested to prevent borderline leprosy reaction ([1]), one may question if the better treatment response from 1974–1978 was not in part due to the increase of the DDS dosage. Hence the provocative heading of this letter, which only illustrates that a controlled trial is needed to prove the value of long term steroid treatment in reversal reaction.

—J. Van der Meulen, M.D.

All Africa Leprosy & Rehabilitation Training Centre (ALERT)
P.O. Box 165
Addis Ababa
Ethiopia

REFERENCES

1. Barnetson, R. St. C. and Pearson, J. M. H. Evidence for the prevention of borderline leprosy reactions by dapsone. Lancet **2** (1978) 1171.
2. Hill, A. B. Past events as a standard of comparison. *In: Statistical Methods in Clinical and Preventive Medicine*. London: E. S. Livingstone Ltd., 1962, p. 5.
3. Naafs, B., Pearson, J. M. H. and Wheate, H. W. Reversal reaction: The prevention of permanent nerve damage. Comparison of short and long-term steroid treatment. Int. J. Lepr. **47** (1979) 7–12.

Reply to Dr. Van der Meulen's Letter to the Editor

To the Editor:

Dr. Van der Meulen's comments on our article, "Reversal Reaction: The prevention of permanent nerve damage" are, in our opinion, incorrect.

Only when Dr. Van der Meulen states that comparison of results from a prospective study can easily lead to biasing past observations is he right. However, when we analyzed our results, we were perfectly aware of such a pitfall. We did our best to avoid it, and we think we succeeded.

When he claims that the mild neuritis group in the 1969–1973 period is different from the group in the 1974–1978 period, based on a difference of 1½ points, he has to redo his statistics; such a difference is hardly significant.

We performed regularly careful assessments during the treatment of our neuritis cases, not only using the voluntary muscle testing (VMT) but also the motor nerve conduction velocity measurements and the sensory testing. Therefore, we were able to detect deterioration early. A patient, as mentioned by Dr. Van der Meulen, will certainly deteriorate after the discontinuation of steriod treatment. However, in this patient further VMTs were not done—as he claims—so deterioration was not noticed. It may be advisable to compare the original VMT-deficits of such patients with the present ones (their records are still available). We are afraid that, when these patients are included, the difference between the 1969–1973 and the 1974–1978 period will be even more striking and will not prove his point.

When he compares the treatment results in points and states that the measured differences do not justify a longer period of steroid treatment, he should realize where these points stand in terms of disability.

In the 1969–1973 period, after 3 years of treatment of 25 patients, only 6 were without disabilities and 12 (50%) had at least 1 ulnar or median palsy. In the 1974–1978 period 20 out of 23 did not have any nerve damage while only 2 (10%) had more than an ulnar or median palsy. In our opinion these differences do matter, and we will seriously plead for longer periods of steroid treatment.

Recently, we were able to compare our results with a study done in the Masanga Leprosy Hospital (Sierra Leone). Their results agree perfectly with ours although they had slightly more complications (Dr. Kazen, personal communication; Lepr. Rev., in press).

We agree that a high dosage of DDS may prevent reactions and may diminish the dosage of steroids needed. We were able to monitor the improvement of the patients in

Dr. Barnetson's and Dr. Pearson's study. There was no difference in response to the steroids of patients on 5 mg DDS daily compared with those on 50 mg daily. Therefore we do not think that a difference in DDS dosage explains the differences in treatment results.

When Dr. Van der Meulen plans a prospective trial, he is welcome to do so since our findings are too important not to be confirmed. At ALERT all the equipment needed for careful nerve assessments is available.

—Ben Naafs, M.D.

Department of Dermatology
Binnengasthuis
University of Amsterdam
The Netherlands

The Fingers in Non-Lepromatous Leprosy

TO THE EDITOR:

Four recent publications ([1,2,4,5]) have drawn attention to the unexpected significance of the fingers in leprosy with particular regard to slit-skin smears, and the subject has been fully reviewed by Jopling ([3]). These interesting and original observations have so far related entirely to patients with lepromatous leprosy and are to some extent understandable since apparently uninvolved skin in this form of leprosy quite regularly contains bacilli. Clearly this new knowledge should now be applied to much larger numbers of lepromatous patients, before and after treatment, and including those who are dapsone resistant.

The purpose of this letter, however, is to suggest that such examination should be extended to include patients with non-lepromatous leprosy in whom apparently uninvolved skin is considered not to contain leprosy bacilli. This suggestion is prompted by the observations of Pearson (Pearson, J. M. H. personal communication, 1979), who took skin biopsies from patients with BT leprosy in reversal (upgrading) reaction. These patients showed active discrete lesions of the trunk and limbs, but there were no evident lesions of their warm swollen hands and feet. Biopsies were taken from a skin lesion and from the dorsum of a finger; they showed epithelioid granulomata at both sites though no acid-fast bacilli were seen.

It is generally believed that reversal reactions are the clinical manifestations of temporarily enhanced cell mediated immune responses to antigens of *M. leprae*. Pearson's findings suggest that the hand may, even in non-lepromatous leprosy, be a site of predilection for leprosy bacilli. Skin smears and biopsies could be taken from the fingers and hands in patients with active untreated non-lepromatous leprosy; such studies might provide new information on the localization of *M. leprae* and could also throw light on the pathogenesis of reversal reactions. Dr. Jopling has reminded me that the possible presence of "hidden foci" of *M. leprae* in dermal nerves in the hands and fingers would be of particular interest and importance.

—A. Colin McDougall, M.D., F.R.C.P.

The Slade Hospital
Headington
Oxford OX3 7JH
England

REFERENCES

1. HAIDAR, A., BELEHU, A., STONER, G., TOUW, J. and ATLAW, T. Selection of sites for slit-skin smears. Lepr. Rev. **50** (1979) 283–287.
2. HIRAMALINI, S., JOSEPH, N. A. and CHACKO, C. J. G. Concentration and persistence of bacilli in the fingers and toes of patients with lepromatous leprosy. Lepr. Rev. **49** (1978) 223–229.
3. JOPLING, W. H. The saga of the skin smear. Editorial. Lepr. Rev. **50** (1979) 272–273.
4. JOPLING, W. H., REES, R. J. W., RIDLEY, D. S., RIDLEY, M. J. and SAMUEL, N. M. The fingers as sites of leprosy bacilli in pre-relapse patients. Lepr. Rev. **50** (1979) 289–292.
5. RIDLEY, M. J., JOPLING, W. H. and RIDLEY, D. S. Acid-fast bacilli in the fingers of long-treated lepromatous patients. Lepr. Rev. **47** (1976) 93–96.