

EDITORIAL

In this issue we welcome the first fruits of two new projects of the British Empire Leprosy Relief Association.

In Dr. Cochrane's "Practical Textbook of Leprosy" the following sentence occurs:—"The number of hospitals or sanatoria dealing with leprosy which can boast of a department devoted to work of this kind (physiotherapy) is comparatively small, despite the prevalence of neural involvement in leprosy".

Dr. Cochrane would be the first to agree that this statement is a conservative one. His short account of physiotherapy is the only one in any English textbook on leprosy. The prevention and restoration of trophic changes in leprosy should be an integral part of treatment in every leprosy institution. The insistence on physiotherapeutic treatment in every case of nerve leprosy meets all too frequently with apathy and lack of enthusiasm on the part of leprosy workers and patients alike. We are therefore particularly glad to introduce Mr. S. Alderson's able and practical observations in this field.

The British Empire Leprosy Relief Association has also sponsored a Leprosy Research Unit in Nigeria, with Dr. John Lowe as its director. Dr. Lowe's preliminary views on sulphone research are given in this issue. This represents the first of a series of reports on modern research from an authoritative source consisting of a highly qualified team of workers.

The important and difficult question of type mutability in leprosy is discussed by a number of authors in this issue. The subject is of primary importance. Among Chinese in Malaya cases of leprosy normally start clinically and histologically as pure tuberculoid. Again in the normal course of events they degenerate into lepromatous cases. This is not a matter of opinion but of proven fact. In other parts of the world a careful study of the early history of lepromatous cases reveals presumptive evidence of a tuberculoid onset. Again, in many lepromatous cases physical evidence can be found suggestive of a previous tuberculoid condition. There can be few leprologists who have not heard the statement from a lepromatous case "At first the doctor said it was ringworm"—again presumptive evidence of a primary tuberculoid phase.

There is thus both factual and presumptive proof of the mutability of tuberculoid into lepromatous leprosy. We are immediately faced with the paradox that able and experienced workers like Lowe, Cochrane and Fernandez have not in their

vast experience observed such a change to be common or even possible. Differences in the interpretation of the histology or clinical appearance of tuberculoid leprosy do not account for this apparent contradiction.

Let us assume, therefore, that there are significant type variations both in neural tuberculoid and lepromatous leprosy. We should then have in neural tuberculoid leprosy (1) the non-anaesthetic depigmented macule peculiar to Nigeria; (2) the immutable tuberculoid of Cochrane and Lowe; (3) the Malayan tuberculoid, with its progress to lepromatous change; (4) the type where both lepromatous and tuberculoid lesions appear at the same time on separate parts of the body; (5) the mixed type with a combination of tuberculoid and lepromatous leprosy. Equal differences could be made in lepromatous leprosy. There is, for instance, a marked clinical and prognostic difference between the mild and indolent lepromatous leprosy seen in Nigeria and certain parts of India; as compared with the virulent and eruptive form of the disease seen in Malaya. Further development along these lines can only lead to hopeless confusion in classification.

Aristotle has said that the only insoluble problem is the problem in which the premises are incorrect. If so, the symposium in this issue would seem to call for a basic reorientation of our ideas on the classification of leprosy.