Maybe I am wrong in my use of the word "anti-inflammatory," but to my mind drugs like ACTH and prednisolone are anti-inflammatory. I have seen no comparable effect following the use of B.663 at a dose of 100 mgm. daily.

(8) As to the use of higher doses, I must emphasize that when a trial is started there is no place for variation in dosage. The authorities that Browne cites did not, to the best of my remembrance, even attempt a controlled study. So I do not understand how Browne can "state with assurance" that higher dosage would be successful. I am frightened by this reliance on uncontrolled investigations and can only reiterate that I hope my paper will stimulate a more scientific approach to a disease in which, in Browne's own words, patients suffer from "recurring crops" of lesions and where there is a tendency to "subside spontaneously."

(7) On the problem of ENL and sulphones, I will say no more until experienced leprologists read the paper by Waters and myself (7), which bases our conclusions on a larger and more representative series.

As to the rest of Browne's letter, I am afraid that I do not always follow the reasoning. Earlier he stated that he would not expect a majority of patients to improve on "inadequate amounts," i.e., 100 mgm. of B.663, but later he stated that in cases comparable to those in my paper, 100 mgm. was sufficient in some patients. I look forward to hearing more of the hitherto unpublished work of Dr. Imkamp and particularly I will be interested to learn of his methods of control.

I must perhaps make it clear that in my experience, using the dosage described, B.663 does not work convincingly. In my reply to Dr. Fowler's letter I asked for evidence of the anti-inflammatory effect of B.663 in other diseases. In a personal communication Dr. Fowler stated that he has tried to get other people interested in this project without much success. Perhaps this implies that others, like myself, are not impressed by the claims that B.663 has an anti-inflammatory effect.

Browne fears that a potentially valuable drug may fail to be investigated because of my paper. I feel that he may be safely reassured on this matter; I have personally written papers claiming success for B.663 in low doses against lepromatous leprosy (4), against sulphone-resistant M. leprae infections (4), and against M. ulcerans infections (5). I do not believe that my work will cause the drug to fall into disrepute.

J. H. S. PETTIT

REFERENCES

ENL in Borderline Leprosy

To the Editor:
We have carefully read the comments from Dr. Kwittken and Dr. Harter regarding our paper.
With reference to Dr. Kwittken's remarks on our paper we have two comments. First, our paper was submitted for publication on 15 August 1966, his on 5 October 1966. Second, in his paper he gives a clinical description and diagnosis of ENL but fur-
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nishes no histologic confirmation of this diagnosis. Such confirmation is provided neither by Dr. Trautman nor by Dr. Schulz, whose papers Dr. Kwittken has quoted. They merely state that ENL occurs in borderline leprosy.

We submit that in borderline leprosy during exacerbated phases of the disease, tender erythematous and nodular lesions of borderline leprosy itself appear in different parts of the body, which are difficult to differentiate from ENL purely on clinical grounds. In our experience these lesions need to be examined histologically before they can be labelled as ENL. Besides, whereas the patient we have described had BB lesions in multiple skin biopsies and had a positive Mitsuda lepromin reaction that showed a mixed granuloma histologically (refer to photomicrographs in the original paper), it is vital to note that both Dr. Kwittken and Dr. Harter are referring to patients with borderline leprosy toward leproma (BL according to Ridley’s classification) with negative lepromin tests.

As for Dr. Kwittken’s contention that in his patient there was no need to withdraw sulfones during the exacerbated phase, and his statement that the patient “improved” with “good results,” one or two comments seem pertinent. From the description given of the clinical course it is obvious that the patient continued to develop episodes of exacerbation while on sulfones, and that these exacerbations were attributed to attacks of “asthma” and were promptly suppressed by adequate steroid therapy. It is well known that steroids can suppress or modify the reactive phase of all varieties of leprosy, and therefore it is not surprising that this happened in Dr. Kwittken’s case. What we are not able to assess is what Dr. Kwittken considered as “improvement.” We would have liked to know the criteria used to judge “improvement,” since we do not find any reference to clearance of bacilli (fall in BI), nor to precise change in neurologic status, e.g., in muscle power, sensory loss, etc.

Our comments regarding the lack of histologic confirmation in the reported cases of ENL in borderline leprosy (BL) apply to Dr. Harter’s references as well. However, we are in agreement with Dr. Harter’s statement that erythema nodosum leprosum is a complication that may be seen in all bacilliferous forms of leprosy rather than only in lepromatous leprosy.

Finally, the view of Dr. Kwittken regarding the use of the words “form,” “type” and “group” with reference to the various points in the spectrum of leprosy is an interesting point of semantics, but it is doubtful if any useful comments can be made on them at this stage.

—A. B. A. Karat
—C. K. Joh
—S. Karat

Schieffelin Leprosy Research Sanatorium
Karigiri, via Katpadi
N. Arcot District, South India
3 July 1967