Letters to the Editor

Dear Sir,

I read with a great interest the stimulating article of Dr. R. E. Pfaltzgraff on 'Classification of Leprosy' in the January issue of the Review, and would like to offer the following comments:

Regarding the implication of the inevitability of deformity in leprosy which still remains, may I draw Dr. Pfaltzgraff's attention to paragraph 4 of my Conclusions to the effect that: 'deformity is *not* an inescapable feature of leprosy work. It is preventable by proper measures. All those experienced in leprology are agreed that most of the disabilities in leprosy can be prevented and that those which cannot be can be corrected by reconstructive surgery. *The philosophical acceptance of the inevitable is an attitude which can hardly be tolerated in this day and age*, when the present study shows that there is ample time for therapy to be carried out.'

Dr. Pfaltzgraff states that I have failed to show that the above is not the case, primarily because I do not fully acknowledge the direct relationship of disability to the classification of leprosy. I would point out from the start that disabilities are a *fait accompli* to the tune of several millions throughout the world today, a massive and alarmingly enough evidence in itself which prompted my study whilst adopting, as a working basis and for practical purposes, the Madrid classification recommended by the WHO. Whereas I do subscribe to Dr. Pfaltzgraff's views for the need of relating disability to the classification of the disease, yet I do feel that, at this juncture, it is primarily a question of securing the *consensus omnium* as to a common acceptance of terms in their entirety, hence the remark in the Introduction of my study to the effect that the classification of leprosy is *disputed* still.

Dr. Pfaltzgraff goes on to say that Indeterminate Leprosy, if correctly defined, is a group in whom deformity *never* occurs unless transformed into one of the definitive types of leprosy. I beg to differ here: the Indeterminate group is indeed notably unstable, most of the patients belonging to it changing eventually to either polar type of the disease, but there is still a number of these patients who carry on as such, whether one considers their persistently achromic macules residua or scars indicating healed lesions. There is ample evidence purporting to this fact in Northern Burma. On the other hand, in Pogiri, India—the Danish Centre where I was the WHO Team Leader in 1962-64, now coping with some 35,000 patients—I was able to follow but a dozen Indeterminate patients which changed mostly to the Tuberculoid form within a 6-9 months' period. In effect, out of the 1,568 patients I examined, 186, i.e. 11.2%, were Indeterminate, 25 among whom, i.e., 13.4%, with disabilities; allowing for marginal errors in classification since I worked mostly under field conditions, there still remains a small group of these patients significantly disabled, yet with the least potential.

I do agree with Dr. Pfaltzgraff that it would have been preferable to have divided the Tuberculoid group into its varieties, noting specifically the ones where deformity occurred, but I wish to stress that the majority of the patients I studied were longstanding patients and that it would have been, therefore, hazardous to have done so at their stage of the disease. Furthermore, I cannot concur with Dr. Pfaltzgraff's statement that deformity *never* occurs in Minor Tuberculoid nor that it may occur in one limb alone and seldom in all four in Major Tuberculoid when (i) 432 out of the 700 patients, i.e., 61.7%, showed a combined deformity of hand and foot, (ii) 262, i.e., 60.2%, among them had quadrilateral involvement, and (iii) their comparative frequency between clinical forms, as it would be expected, was identical as in the case of the hand and foot alone, 48.5% and 34.01% concerning the Tuberculoid group alone !

Finally, as regards my not carefully relating the *incidence* of deformity to the spectrum of disease accurately conceived, may I point out that the primary objective of my study was to

establish the *onset* of deformity *per se* and to sort out its pattern within the context of clinical observations, other workers having already shown to what an extent disabilities occur in the various forms of leprosy.

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2nd February, 1967.

Dear Sir,

In *Leprosy Review* of January, 1967, Rodrigo Gutiérrez published a Preliminary Report on the Effects of L-Triiodotyronine, Radioactive Iodine[131] and Methimazole on Experimental Murine Leprosy. The conclusion of this work is as follows: 'Our preliminary data seems to point to some "protective action" of Iodine[131] and of L-Triiodotyronine on experimental infection with the Stefansky bacillus.' On the other hand, Rodrigo Gutiérrez comments that Lurie *et al.* have found that the administration of thyroid hormones can increase the level of native resistance to TB in rabbits and that thyroidectomy and anti-thyroid drugs have the opposite effects. Concerning the conclusions of Lurie and Gutiérrez I must express the following opinions. Gutiérrez notes that all the animals were fed Purina Lab Chow. I do not know what nourishment was used by Dr. Lurie in his experiments with rabbits. It is possible that Dr. Lurie had used a nourishment for the rabbits which was analogous to Purina Lab Chow. Purina Lab Chow contains a quantity of iodine equivalent to 600 microgrammes per kilo. All these nutriments for animals have a content of minerals fixed by the National Research Council, Washington, D.C., U.S.A. I know the facts about nutrient 301 as revised January, 1954, by the Committee on Animal Nutrition.

In previous publications (Rojas) I have suggested the appropriateness of associating a hypo-iodic diet with the treatment of human leprosy per the medium of anti-thyroid substances such as Methimazole. I think that experiments with Methimazole in laboratory animals and other anti-thyroid substances should use a hypo-iodic diet so as to obtain adequate supporting evidence of the action of the drug.

It is my opinion that tactical compatibility between the anti-iodic substances such as Methimazole and the nutriment used (Purina Lab Chow) does not exist and I may mention that military terms are very suitable to the attack against the bacterium of leprosy. I wish to suggest a new experiment for the future, namely to use the nutriment with the lowest content of iodine at least in 50% of any new group of rabbits who should get Methimazole or Triiodotyronine.

The hypo-iodic diet well known at present for experiments on laboratory animals is a Remington diet. It contains 15 microgrammes of iodine per kilo. Though difficult it should be possible to prepare hypo-iodic nutriments for human use analogous to the Remington diet and usable in several pathological conditions. Thus we can initiate a therapeutic procedure wherein iodine will operate not by its presence but by its absence. References about the Remington diet are as follows:—

Levine, H., Remington, R. E. and von Kolnitz, H. *J. Nutr.*, 1933, **6**, 325.
Levine, H., Remington, R. E. and von Kolnitz, H. *J. Nutr.*, 1933, **6**, 347.
Chapman, A. *Endocrinology*, 1941, **29**, 680.
Axelrad, A. A., Leblond, C. P. and Isler, H. *Endocrinology*, 1955, **56**, 387.

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