DAMAGED EAR CARTILAGE AND TYPE OF THE DISEASE

In the correspondence section of this issue are replies to a question about the cause of damage of the margin of the cartilage of the ear in leprosy. The question was, what would be the retrospective diagnosis of the type of the disease responsible for the condition when it is seen in a case in which active manifestations have cleared up—in effect, whether loss of substance of the cartilage could be due to tuberculoid leprosy or should be regarded as pathognomonic of the processes of lepromatous leprosy.

The question arose at Culion when, in a supposedly lepromatous case of long duration which for some time was supposed to have been cleared up, there suddenly blossomed forth an extensive eruption of lesions which clinically and histologically were of straightforward tuberculoid character. Because of the nature of this reaction the problem arose whether or not it could be proved that the case had ever been really lepromatous, and that led to the question whether the most conspicuous of the residuae of the previous condition, a "nibbled" or serrated condition of the ear cartilage, could be regarded as pathognomonic of that type of the disease. As has been the case in other inquiries of the kind, some of the replies received indicate how the question could have been made more precise.

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[3] ARGUELLO PITT, L., CONSIGLI, C. A., DEGOY, A. and PEÑA, J. M. Experiencia acerca de las relaciones inmunológicas entre lepra y tuberculosis. (Premunición con B. C. G.; su valor en la profilaxis de la lepra.) Mem. VI Congr. Internac. Leprol., 1953; Madrid, 1954, pp. 643-656.

[4] HANKS, J. H. and FERNANDEZ, J. M. M. Enhancement of resistance to murine leprosy by BCG plus specific antigen. *Internat. J. Leprosy* **24** (1956) 65-73.

For one thing, it was evidently not made clear that the question was not how to make a presumptive type diagnosis in retrospect of a *case* as a whole, with all its features, but of the one particular condition. (See Figs. 1 and 5 of the plate accompanying the symposium.)

It was also asked whether or not the question, admittedly a minor one, merited a symposium. A few of the twenty-one persons who replied thought not, but a much larger number felt otherwise; one remarked that "everything in leprosy is interesting." Many wrote at some length; three (Lara, Tolentino and Contreras) surveyed available cases before replying; and three (Wolcott, Chung-Hoon and Basombrio) supplied illustrative photographs.

A large majority of the contributors have seen the condition, definitely or probably. Three (Muir, de Souza Lima, Basombrio) have not observed it and venture no opinions, while one (Cochrane) recalls a single case seen twenty years ago. Four do not indicate whether they have or not, and their replies seem distinctly speculative.

As for the form of leprosy in which the condition arises, the vote is overwhelmingly for lepromatous, if in some instances doubtful and in others with exceptions. Eight are definitely for it, and certain of them are as definitely against tuberculoid. Two other (Yokota and Nojima, the latter conveying the result of a conference discussion) are apparently for it. One (Contreras) is for it—the neural variety of lepromatous—mostly, but thinks that neural tuberculoid leprosy may also leave such stigma; another (Rodriguez) is for it but speculates that the condition may occur in reactional tuberculoid cases; and another (Chaussinand) says lepromatous is usual but tuberculoid—lazarine only—may sometimes be involved. Only one writer (Ross Innes) voted for tuberculoid, and that may have been entirely on speculative grounds.

Especially significant because they were based on the investigation of actual cases and their histories, although not on observation of the process itself, is the conclusion of Lara and of Tolentino, working completely independently in different institutions, that it is the borderline form or phase of leprosy in which the deformity occurs. Furthermore, Fiol believes that it is either borderline or lepromatous; and the single case recalled by Cochrane was of that kind.

As for the actual process, it seems generally agreed that breakdown and ulceration of lesions is involved, several contributors invoking secondary infections or/and local disturbance of blood supply; one (Davey) speaks of general debilitation; and one (Contreras) of a neurotrophic element. Five writers mention complicating conditions which may have similar effects on the ear cartilage: trauma, frost-bite, etc. (Yokota); yaws (Ross Innes); leishmaniasis (Convit); "erythematous lupus" (Basombrio); and tophi of gout (Fiol).

It remains to be seen if this inquiry will arouse sufficient curiosity about the matter to bring out reports of definitive observations of the process of ear-cartilage damage. It may be, as suggested by Lara and Wolcott, that present-day treatment no longer permits its occurrence. Be that as it may, the notes of the two contributors who examined cases and their records before replying, with certain others more or less in accord, direct attention to the important but much-neglected borderline form of leprosy.

—H. W. W.