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Letters to the Editor

From The Director, Lepra, Fairfax House, Causton Road, Colchester CO1 1PU

The Medical Advisory Board of Lepra, at its meeting held on the 10 June, 1976, expressed its deep concern for the action taken by the President of the American Leprosy Mission in using the recent claim that the leprosy bacillus has been cultivated *in vitro*—as a basis for a special fund raising drive. Members of the Board consider that any attempt, by organizations dedicated to the control of leprosy and the welfare of the leprosy patients, to exploit a discovery of this magnitude before it has been documented and proved, can only harm the work of anti-leprosy organizations generally and more especially so far as their fund raising activities are concerned. The public will soon lose confidence and become disillusioned if claims of this nature are exploited without adequate explanation and before the claim can be substantiated.

Members of the Lepra Medical Advisory Board strongly condemn such publicity and trust that those in responsible positions in anti-leprosy organizations throughout the world, will exercise great care to ensure that their fund raising literature and publicity is not in any way misleading.

G. F. HARRIS

From Dr G. Warren, The Leprosy Mission, c/o Christian Hospital, Manoram, Thailand

The Editorial of *Leprosy Review* of March 1976 (Vol. 47, No. 1) on the complications of treatment with clofazimine provides interesting reading for those who are familiar with this drug. But it has caused real concern amongst the less clinically experienced, some of whom have become fearful that its use will often produce a high incidence of undesirable toxic effects and may even cause death.

All who have worked for any length of time with clofazimine realize that only a small proportion of patients on the drug do develop gastro-intestinal symptoms (the figures to support this are quoted in the Editorial), and that in an even smaller proportion are the symptoms severe enough to warrant a change in drug therapy. Well supervised trials with clofazimine were carried out at widely separated centres and as in any drug trial, deaths did occur which were usually well investigated. In most cases some other cause of death was easily found, but the possibility of the use of clofazimine, or any other drug being given at or immediately before death, contributing to the cause of death would be difficult to rule out.

These drug trials showed clofazimine to be an effective means of controlling E.N.L. and that its dangers are so much less than those of prednisolone that it has

become the drug of choice in many centres for treating patients with E.N.L. Although the usual dosage is 100 mg daily this may need to be increased to 200, 300 or even more per day adequately to control the E.N.L. in severe cases. Once the E.N.L. is controlled we do try to reduce the dosage of clofazimine to the lowest level that will control the E.N.L., but many patients do need 900-2100 mg/week for long periods of time. Even on these higher doses the incidence of gastro-intestinal symptoms is low.

In many clinics the control of E.N.L. must be achieved by clofazimine or prednisolone, and especially where close monitoring is not possible clofazimine is the drug of choice. It is hoped that an Article such as this one does not lead to discarding the use of clofazimine and so to less efficient E.N.L. control and irregular anti-leprotic drug therapy.

The two cases are interesting and it would be informative to read the full case histories including the results of the tests done and to know the final medical state of the second patient. From the details given it is obvious that clofazimine cannot be assumed as the undoubted cause of the gastro-intestinal symptoms in either case—especially as in the second patient the first symptoms reported occurred more than 3½ years after cessation of treatment with clofazimine. Such cases should of course be properly investigated and reported to the Dunlop Committee, but until definite proof is obtained incriminating a specific drug, it is hoped that any printed reports, while informing workers of the possible complications will not be such that will deter the field workers from using the drug in adequate dosage to achieve the desired therapeutic effect.

Personally I have treated many more than 100 patients with clofazimine—some on 300 and 400 mg daily for many months, and can only remember 2 patients with significant gastro-intestinal symptoms. In one of these the symptoms settled when we realized he had been taking the drug on an empty stomach, and he took it with his food thereafter. In the other a very detailed investigation was carried out—including laparotomy which revealed heavily pigmented lymph nodes in the mesentery—and revealed no obvious cause of the symptoms. Once he was reassured the symptoms subsided without stopping the clofazimine which was only being given at 100 mg at the time, and had been at that dose for many months. Fortunately, when these complications occur they rarely if ever cause an acute crisis, so that awareness of their possibility should help the field worker in the management of patients. In the areas where clofazimine is frequently used there are also many other causes of gastro-intestinal symptoms; these must be ruled out before clofazimine is definitely incriminated. If we do a proper work-up and treat the patient for any intercurrent diseases we should not be caught blaming a drug which has solved far more problems than it has caused.

GRACE WARREN

COMMENTS ON DR WARREN'S LETTER BY DR W. H. JOPLING

The purpose of the Editorial in question was not to condemn the use of clofazimine in the treatment of leprosy but to alert doctors using the drug to be aware of possible gastro-intestinal side effects. Like Dr Warren I have used the drug extensively and have had no cause to change my policy; in fact the only side effect seriously mitigating against its use is skin discoloration in light-skinned patients. Regarding the last case recorded in the Editorial, the chief interest lies in the finding of dense deposits of clofazimine crystals in mesenteric glands 4 years after stopping the drug.