

Editorial: Importance of early diagnosis in ocular leprosy

The revival of interest in ocular leprosy will prove timely, since the disease now ranks as one of the major contributors to world blindness. Leprosy patients, with their loss of mobility due to the crippling deformities of the disease, should qualify for special consideration in these statistics. Sadly the numbers with severe visual impairment and blindness are unlikely to diminish in the short term despite the widespread introduction of multidrug therapy in the management of the disease.

The ocular complications of leprosy can often be avoided, not only by early diagnosis and rigorous control of the disease and the encouragement of compliance among patients notoriously indifferent to their condition, but also by changing social attitudes to leprosy, which for too long have prevented patients from presenting early in the course of the disease because of the age-old stigma.

Multidrug therapy may well prove effective in reducing the incidence of ocular involvement, provided patients are treated energetically under supervision. Those that slip through the net may develop eye complications which can still be prevented from causing blindness by the constant attention of leprologists, paramedical workers, and the patients themselves.

A word of caution needs to be added at this stage. Evidence is accumulating that some eyes in lepromatous leprosy may harbour living organisms or antigen long after the skin is bacteriologically negative, and ocular disease may recur after patients are released from control. This implies that some form of ocular supervision may need to be continued indefinitely in multibacillary disease irrespective of current practice. The problem of early diagnosis and detection of ocular involvement therefore becomes more

pertinent, and it is here that solid information is lacking at present.

It is known that in early lepromatous leprosy the organisms enter the eye through the blood stream and lodge mainly in the tissues of the ciliary body and iris. Reactions to antigen may occur, giving rise to acute iridocyclitis and obvious clinical symptoms and signs. Frequently, however, the progression of the ocular disease may not be punctuated by acute episodes of inflammation and a slow, relentless, and usually asymptomatic condition occurs, culminating eventually after many years in iris atrophy, miosis, and ciliary body failure. Certain signs may become evident, but they are subtle in the early stages, and by the time they become obvious the eye is irreversibly affected. These signs indicate impairment of autonomic function and include diminished pupil reactions, particularly those involving the dilator muscle, reduced accommodation, and lowered intraocular pressure. None of these can be used singly as a pathognomonic sign of ocular disease, and yet it is of paramount importance to identify those cases with early involvement of the anterior uveal tract, since these may be the patients who run into problems later on.

The paper by Lewallen, Courtright, and Lee in this issue shows that comparisons of intraocular pressure measurements in the upright and supine positions can give useful information about disturbances of autonomic function in the ciliary body with the implication that the tissue has been invaded by *Mycobacterium leprae*. It is to be hoped that this and allied tests can be used to identify those patients at risk, so that they can be singled out for long-term ophthalmic follow-up even after they have been classified as 'cured' by multidrug therapy and released from supervision.

TIMOTHY FFYTCHIE