

THE NASAL EXCRETION OF LEPROSY BACILLI

On the following pages, Dr T. F. Davey describes the nasal excretion of leprosy bacilli from one untreated lepromatous patient, during the course of one day. As he ruefully remarks in the opening paragraphs, it is a matter of continuing concern that so little *practical* notice has been taken of the enormous numbers of bacilli now proven to issue from the nose and upper respiratory tract of patients with this type of leprosy. Bearing in mind the many millions of leprosy patients currently estimated by WHO to be still undiagnosed, and that in some countries 50% or more of these may be lepromatous, one wonders if future generations will look back on this era of leprosy control as one in which a tap was running full tilt in an upstairs bathroom, causing water to pour down the stairs, perpetually flooding the lower floors, while the occupants moved from one room to another with rather small mops. Dr Davey has graphically described the dissemination of leprosy bacilli from only one patient, emphasizing the paramount importance of finding and treating the maximum number of lepromatous patients in all control schemes. Whilst admitting that most patients are not lepromatous, and that there have been some interesting observations on the apparent infectivity of non-lepromatous patients in maintaining endemics in some areas, there is surely no longer any doubt about the overwhelming importance of the nose in the pathogenesis and spread of this disease, and it may here be worth considering some of the implications of this for the patient, the community, and for the survival of the leprosy bacillus itself.

A high percentage of all untreated lepromatous patients have nasal symptoms, sometimes for many years before diagnosis, and these may include blockage, chronic discharge and epistaxis. Destruction of the nose and cartilaginous septum are of course common events and they may be difficult to correct surgically, even if the facilities are available. From a personal point of view, the nose is a central point in an area which may already be affected by skin nodulation, loss of eyebrows, eye disease, defective teeth and impairment of the voice. The additional stigma of permanent nasal deformity may have an almost irreversible effect in undermining the patient's confidence and return to normal life.

As regards the community and the health risks to susceptible people of nasal shedding, it is relevant to consider what happens during sneezing from virus infections such as the common cold, particularly as leprosy is being

increasingly recognized as a disease of crowded, urban communities. The facts (Mims, 1977) are as follows—

“In a sneeze, up to 20,000 droplets are produced... the largest droplets (1 mm in diameter) fall to the ground after travelling 15 feet or so, and the smaller ones evaporate rapidly, depending on their velocity, water content, and on the relative humidity. Many have disappeared within a few feet and the rest, including those containing microorganisms, then settle according to size. The smallest... in fact stay suspended indefinitely, because air is never quite still. Particles of this size are likely to pass the turbinate baffles and reach the lower respiratory tract... shedding from the nasal cavity is much more effective when fluid is produced, and among the viruses that are shed from this site, evolution has favoured those that induce a good nasal discharge.” And again “(In a sneeze), most of the droplets in fact originate from the mouth, but larger masses of material (“streamers”), as well as droplets, are expelled from the nose when there is excess of nasal secretion....” Much of this, though in lesser detail, was well appreciated many years ago by those carrying out work on the transmission of tuberculosis, in which the importance of inhalation is beyond doubt, and the pros and cons of an analogy between the mode of spread in these two diseases have been discussed by Dungals (1961), Rees and Meade (1974) and more recently (1977) by Leiker. Dr Davey draws attention to experimental work in the mouse which indicates that inhalation may also be a mode of entry of the leprosy bacillus, but in this context it should be kept in mind that in most forms of leprosy, nasal lesions are not described and that the lung is not a target organ in any form of the disease. What matters in comparing leprosy and tuberculosis in the present context is that there is in both instances a well-established “open” positive case; an excretor of bacilli on a massive scale. The danger of this type of patient in tuberculosis has been recognized for a very long time; in leprosy, it is difficult to escape the comment that measures for dealing with the open lepromatous case, which are at the same time humanitarian and medically effective, are as yet poorly conceived.

Finally, in looking at the thing which is of most interest to the bacillus itself, namely survival, the nose, as opposed to the intact skin or the peripheral nerves, may have peculiar advantages. The induction of a good nasal discharge may clearly be of benefit, but an even more important and subtle advantage has been suggested by Shepard (1965): “the low optimum temperature of *M. leprae* might have come about through natural selection, because it is mainly those bacilli living in the cool nasal passages that cause contagion.” Coupled with this, it may be relevant to recall that there is a continuous bacteraemia in lepromatous leprosy which is associated in a high percentage of cases with the finding of bacilli, many of them solid-staining and presumably viable, in the endothelial lining cells of blood vessels. These include a wide range of vessels in the nasal mucous membrane, a tissue which is delicate, easily shed, and subject to secondary infection. Having presented such a wealth of data on the nasal route of excretion in the transmission of leprosy, Dr Davey does not elaborate on the possible role of biting insects, wisely commenting that its importance has yet to be more fully established.

Some important data from this area of research have already been published in *Leprosy Review*, and it is certainly one that is worth pursuing. In view of the known importance of the vascular endothelium as a site of replication and shedding of viruses and rickettsiae that are transmitted by blood-sucking arthropods (Mims, 1977), we await with interest further research which might point to yet one more subtle device by the leprosy bacillus—a link between a continuous (and totally asymptomatic) bacteraemia, loading of endothelial lining cells by bacilli, and biting arthropods.

Taking an objective look at the “increasing complexity of leprosy control” (Lechat, 1978), particularly in the treatment of new lepromatous patients, dapsone-resistant patients, and of adverse reactions, together with the unavoidably slow pace of developments in research which are likely to have a fundamental effect on the prospects for leprosy control in the foreseeable future, it may be that we should try, yet again, and even harder, to find as many lepromatous patients as possible, and to stop their nasal excretion of bacilli, as a matter of priority. The interesting question which then arises is: Where should such an activity come in the list of priorities? Should it be at the top?

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References

- Dungals, N. (1961). Is Leprosy Transmitted by Arthropods? *Lepr. Rev.* **32**, 28.
- Lechat, M. F. (1978). Sulfone Resistance and Leprosy Control. *Int. J. Lepr.* **46**, 65.
- Leiker, D. L. (1977). On the mode of transmission of *Mycobacterium leprae*. *Lepr. Rev.* **48**, 9.
- Mims, C. A. (1977). *The Pathogenesis of Infectious Disease*. Academic Press, London.
- Rees, R. J. W. & Meade, T. W. (1974). Comparison of the modes of spread and the incidence of tuberculosis and leprosy. *Lancet*, January 12, 47.
- Shephard, C. C. (1965). Temperature optimum of *Mycobacterium leprae* in mice. *J. Bact.* **90**, 1271.