EDITORIAL

The relationship of leprosy to diet and especially to the vitamins is one of importance. Dr. Blueth's paper in this issue shows relief of leprosy neuritis by treatment with synthetic vitamin B_1. Chowhan and Chopra, writing on *Cobra Venom in Nerve Leprosy* in the last December number of the Indian Medical Gazette, state that they have used mixed injections of Vitamin B_1 and cobra venom with marked success in a few cases of leprosy neuritis which had not yielded to cobra venom alone, believing that most of these resistant cases were due to vitamin B_1 deficiency. How these drugs act is still a matter of conjecture, but anything which relieves this most painful of all the conditions in leprosy should be welcomed. It is the general opinion that it is impossible permanently to relieve the pains of leprosy without first improving the general health of the patient. In the strong healthy leprous patient severe nerve pain seldom, if ever, occurs. The use of vitamin B_1 seems to be on the right lines, as vitamin B_1 deficiency so frequently stands in the way of improved general health. Curiously, however, this product seems to require to be given by injection. Badger and Patrick, in the article referred to in Blueth's paper (p.109), first tried dried brewer's yeast and synthetic B_1 concentrate by mouth without results, though their intramuscular injections were most effective.

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We would also call our readers' attention to three more forms of treatment recommended in articles appearing in or reviewed in this issue. Sir C. Sprawson (p.106) reports on the treatment of nerve conditions with novocaine used in Russia. Dr. de Carvalho (p.133) describes the use of intravenous injections of hypertonic glucose in 74 cases, with complete recession in 51 per cent. Dr. Boenjamin (p.140) obtained favourable results in ten out of twelve cases of lepra reaction with intramuscular injections of omnadin.

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Dr. Rotberg's article, an abstract of which we print (p.130), opens up interesting speculations. If his hypothesis is confirmed, that the majority of people are born with a definite " N factor " which renders them immune to leprosy, or at least to its lepromatous and infectious form, then methods of prophylaxis will call for revision. The most effective control of leprosy would then be through finding out and concentrating on the minority in endemic areas who are proved by the lepromin test to be susceptible to

leprosy. To prove or disprove Rotberg's hypothesis considerable further investigation would have to be carried out, both in endemic and non-endemic countries, the lepromin test forming the basis of the investigation.

In this connection Dr. Burnet's success in inoculating Syrian hamsters with human leprosy (see p.132) seems to uphold the hypothesis of the susceptibility of a minority of individuals in a community. Only one of his six hamsters developed the disease, and this one developed it in a very definite form. This suggests that hamsters, with the exception of a minority, are resistant to leprosy. If this is proved to be so then this rodent, and possibly other species of the same order, may yet be of immense value in the investigation of the unknown factor which underlies susceptibility and resistance to leprosy.

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The abstract from the Burma Report appearing below (p.142) is certainly of a startling nature. If among the thirteen million inhabitants of Burma there are 200,000 lepers, a percentage of more than one and a half, then the situation is a serious one. In an article appearing in *Leprosy in India* (Oct. 1938) Dr. Lowe discussing the question concludes : " Thus it appears that the lack of resistance to leprosy of Burmans is racial and hereditary." Seventy-five per cent. of Burmans in the Rangoon Leper Asylum were found to be of the lepromatous type, while only thirty-nine per cent. of the Indians were of this severe type.

Dr. Oberdorffer's paper (p.112) puts forward the suggestion that this difference in racial susceptibility is dietary and that sapotoxins from aroideal food (plants like colocasia) damage the adrenals, thus proving the deciding factor in racial susceptibility. This suggestion is certainly one that deserves consideration and further investigation by leprologists and others throughout the world. In connection with racial immunity Dr. Flandin's speech quoted from the *Lancet* (p.135), and the short paragraph about leprosy among white peoples in Australia, show that leprosy is not yet an obsolete problem even in Europe and among Europeans.

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The treatment of trophic ulcers is always a difficult problem. Various methods have been suggested at times, and we publish two in this issue. Dr. Maynard (p.118), after cleaning up with antiseptic dressings, pours on a mixture of beef suet, ghee and beeswax. Dr. Mehta injects locally a solution of rivanol (p.140). There is no doubt that subcutaneous injection in the region of the ulcers of irritant fluids such as hydnocarpus oil and esters, dettol

and many other drugs, has a marked effect in causing healing of sluggish sores. The thick skin of the sole will peel off within a few days and the surface of the ulcer become vascular and healthy. Whether rivanol has any properties which make it preferable to these other stimulating injections awaits confirmation. It is possible that a triple method of treatment may prove best: first, the removal of dead bone and gross sepsis; second, stimulation by local injections; third, some such method as that of Dr. Maynard. The depth, area and chronicity of the ulcer, the condition of the leg and of the patient obviously call for variations in the method used; but the triple principle is a useful one to bear in mind—cleanse, stimulate, protect.