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Letters to the Editor

I should like to place on record two interesting clinical observations that may be relevant in other of the more sophisticated societies.

Miss L. K. B., aged 28 years, was admitted to our care in 1965. On admission she denied previous anti-leprosy treatment, but it was later discovered that she had been treated for 6 years in China and had discontinued treatment for 3 years before coming to us. Her leprosy was BL-LL in type, appearing to be more LL, with a Bacterial Index (B.I.) of 4.7 and a Morphological Index (M.I.) of 5%. Progress was satisfactory until 1967, when she began to develop neuritis of both ulnar nerves at the elbow. She experienced crops of *erythema nodosum leprosum* (ENL), and was being treated with dapsone at the time, but this was changed to Vadrine and later to thiambutosine. However, the neuritis had certain atypical features, and responded to none of the usual medications nor to rest.

In November 1968, her clinical state was very bad, her face being more infiltrated and showing many ENL lesions. The numbness and pain in her arms were increasing. It was then discovered that she was treating herself with vitamin A and D tablets which, she had been told, would do her good and increase her energy. She was taking 120,000 units of vitamin A daily, but stopped taking this after being given an explanation of its side-effects. The arm pain gradually diminished and did not return. When she discharged herself at the end of 1971, there was no obvious neural deficit of her hands except slight anaesthesia of left ulnar distribution.

At the time, two articles had appeared in the *Medical Journal of Australia* (Cleland and Southcott, 1969*a*, *b*), indicating that hypervitaminosis-A might in certain circumstances induce peripheral neuropathy. Reading these articles led to the suspicion that a grossly high intake of vitamin A by this patient might be playing a part in the production of the signs of peripheral neuropathy.

The possibility is also mentioned in a standard textbook (Cecil and Loeb, 1971) as causing bone and joint pain and neurological signs, especially in children, as well as other signs that were not present in our patient. However, in a patient who already has inflammation in the nerve, an excess of vitamin A might produce definite pain and discomfort.

Little more attention was paid to this possibility until a second similar case occurred. Mr S. Y. was aged 24 years on first admission in 1962, with BL-type leprosy and a B.I. of 4.0. He discharged himself against advice in 1965, and had no treatment for 2 years. On his return in April 1967, the B.I. in his skin smear had risen to 2.8, the M.I. being 0%. In February 1968, he complained of auricular nerve pain, and had slight ENL while receiving thiambutosine by injection. The neuritis and ENL continued on and off through the year. He had left ulnar neuritis in January 1969; at that time he was found to be taking vitamin A, 10,000 units daily (possibly more), in addition to the prescribed dose of multivitamins (vitamin A x 5000 units). He was advised to stop his self-medication; within a month the pain had disappeared, and he had no further

nerve pain for the next 6 months. He did, however, continue to have ENL and occasional bouts of mild nerve pain for the next 18 months till he again discharged himself against advice.

Since there was no other real change in the medication of either of these patients, it would appear that the extra dosage of vitamin A may well have been the relevant factor in the production of the neuritis. In countries where drugs are freely available and widely advertised, the problem of self-medication must always be considered in the presence of bizarre symptoms. I wonder if any of your readers have met similar situations or are interested in investigating the problem further?

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How many medical workers interested in the physical treatment of leprosy would agree that the inclusion of electrical stimulation, either galvanic or faradic, is not of primary importance in the leprosy clinic or physiotherapy department?

Firstly, for diagnosis of the extent of a motor nerve lesion, it is not needed, since the selectivity of the *Myco. leprae* in its place of attack presents the same recurring patterns that tell their own story.

Secondly, for treatment. The premise is that electrical stimulation of a muscle, either directly or through its motor nerve, can only maintain muscle tone that is not already lost, and cannot increase muscle power. Only active contractions of a muscle will lead to an increase in muscle strength. Therefore, the electrical muscle stimulator is discarded when even weak active contractions are present. Among those patients who attend leprosy clinics regularly, the proportion of patients who would be helped by electrical stimulation is low—either they are still at home hoping for self-healing, or they come to a clinic only once or twice monthly to obtain their anti-leprosy medicine.

Thirdly, for post-operative re-education following tendon transfer. Is it not better to work through the motor cortex, so that the cortex registers the changed action of a transferred muscle (by working through the "thought pattern" of the muscle movement prior to its transfer) than to use a by-pass in an attempt to stimulate the muscle itself?

Another point—wax baths versus water soaking. Why spend time and money on wax baths, when cool water—available in most homes, and in field work—is more efficient in softening dry skin? Leprosy patients whose sweat glands in hands or feet have been destroyed need rehydration.

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After reading Miss Gardiner's provocative letters *Leprosy Review* asked her to amplify her remarks. This she has done in her articles entitled "Wax Baths in Leprosy" and "Querying the Absolute Need for either Faradic or Galvanic Stimulation in the Physical Treatment of Leprosy" in this issue (pages 215 and 213).—*Ed.*

Leprosy in the Republic of South Africa

Our previous publication (Schultz and Pentz, 1970) dealt with statistics up to the end of 1968, when the incidence of new cases was 2.0 per 100,000.

The number of new cases diagnosed since then is shown in Table 1.

TABLE 1

Year	Bantu	Coloured	Asiatic	White	Total
1969	373	5	2	1	381
1970	301	2	0	1	304
1971	312	6	1	3	322
1972	323	3	0	1	327

It is of interest that one of the three "white" patients diagnosed in 1971 was then a recent immigrant from Spain.

The incidence of new cases of leprosy per 100,000 of the population for the years 1969, 1970, 1971 and 1972 was 1.9, 1.5, 1.4 and 1.4 respectively.

In a recent report on Leprosy in the Republic of South Africa (*Leprosy Review*, News and Notes, 1972), it was stated that 749 notifications of leprosy were received during 1970. We would draw attention to the fact that this figure does not represent new patients only, but includes old patients admitted to the Institution for various treatments.

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