ercises for early lagophthalmos. It also finds application during the relearning period after the temporalis transfer operation. Both groups of patients need the constant supervision of a physiotherapist to accomplish the task of activating the eyelids at a specified rate. The device presented here is an unambiguous substitute for the pacing normally provided by a physiotherapist.

The device fulfills the following design criteria: It should a) be portable: It measures 5 cm × 5 cm × 3 cm and weighs 96 g with a 9-volt battery pack; b) consume minimal current to ensure economic operation: The circuit is based on CMOS NAND gates. The current consumption in the quiescent state is 7 μa, and during the brief audible state, it is 500 μa. The 9-volt battery should last for more than 1 year of continuous use; and c) be unobtrusive: An optional earphone socket may be included to cut off the piezo-ceramic buzzer and to deliver the sound through an earphone to the ear.

The circuit is based on a single Quad CMOS-dual input NAND gate integrated circuit of type number 4011B (The Figure). N_1 and N_2 form the delay circuit along with R_1 and C_1. D_1,D_2 and the preset potentiometer P_1 determine the duration of the sound. N_3 and N_4, with the associated components R_3, C_2 and R_4, form a square wave audio oscillator, the output of which is delivered directly to a piezo-ceramic buzzer. A further gain stage must be added to this oscillator if an earphone is to be driven since the output impedence is 400 ohms. However, the addition of an earphone will place a greater current demand on the power source.

The cost of the device is approximately Rs.60/- (Indian rupees) and all components are readily available in the local electronics market. There may, of course, be possible variations in the performance of the device due to component variability and tolerance.

This device has had an initial period of testing with two patients, and the response of the participating physiotherapists and patients has been encouraging.

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REFERENCES

1. Anderson, J. G. Surgical treatment of lagophthalmos in leprosy by the Gillies' temporalis transfer. Br. J. Plast. Surg. **14** (1961) 339–345.
2. Antia, N. H. Reconstruction of the face in leprosy. Ann. R. Coll. Surg. Engl. **32** (1963) 71–98.
3. Johnson, H. A. A modofication of the Gillies' temporalis transfer for the surgical treatment of lagophthalmos of leprosy. Plast. Reconstr. Surg. **30** (1962) 378–382.
4. Lennox, W. M. Management of lagophthalmos in leprosy. Lepr. Rev. **37** (1966) 151–157.

Leprous Neuritis, Classification of Leprosy, and Multidrug Therapy

To the Editor:

My letter is intended to initiate discussion and obtain clarification concerning several issues of importance in leprology and regarding leprous neuritis.

I have often asserted in textbooks and publications that neural lesions in leprosy are classified into tuberculoid and lepromatous lesions. The tuberculoid lesion is usually described as infiltrated by giant and

epithelioid cells, and the lepromatous lesion by numerous lepra cells. In my histopathological report of leprous neuritis I do not use this classification. Indeed, I have found it illusory to classify neural lesions in leprosy based on the type of inflammatory infiltrate as is done for dermal leprous lesions. In my experience, a typical tuberculoid granuloma is seen essentially in cases where reversal reaction has been clinically diagnosed, and I rarely see in nerve biopsy a typical lepromatous infiltrate as observed in dermal lesions. Histologically, what we see generally in leprous neuritis is a lymphohistiocytic infiltrate of a certain density associated with the existence of varying bacillary load. According to the bacillary load, I report paucibacillary [bacterial index (BI) $\leq$ 2] or multibacillary (BI >2) leprous neuritis. In multibacillary leprous neuritis the bacilli are, principally, found in Schwann cells and also in some macrophages. Some authors consider a multibacillary neural lesion with an inflammatory infiltrate of a mild density as an immunological landmark of lepromatous leprosy, while others prefer not to take into account for the classification of the disease these multibacillary neural lesions. (Is field-work condition the whole idea behind this latter assertion?)

These notions may be important in cases where a discrepancy exists between the bacterial index found in the dermal and that found in the neural lesions. There are cases where tuberculoid leprosy has been diagnosed clinically and histopathologically on a skin lesion while nerve biopsy reveals a multibacillary lesion. May these cases be considered as dimorphic leprosy? Does the multibacillary neural lesion reflect the true immunological lepromatous profile of the patient? It would not be surprising if some leprologists may in the future propose nerve biopsy as a routine procedure for proper evaluation and classification of patients with paucibacillary dermal lesions.

All of these considerations become important when we consider the multidrug therapy (MDT) regimen to be given appropriately for each patient: for instance, at least 2 years of MDT for patients with BT dermal lesions and multibacillary neural lesions. On the other hand, if the neural feature is not taken into account, one can consider only 6 months of MDT for these patients with BT dermal leprosy and multibacillary neural leprosy.

I think this will need further investigation and evaluation in order to distinguish true BT patients from "BT" patients harboring potentially multibacillary leprosy.

In conclusion, I would like to raise some questions:

1. Is it always possible to classify histologically neural lesions in leprosy according to the histopathological criteria used for the dermal lesions?
2. Does multibacillary neural leprosy reflect the immunological lepromatous profile of the patient?
3. Should one take into account the neural finding before initiating the appropriate MDT regimen?
4. Since in 15–25% of cases a discrepancy exists between the bacillary load found in the skin and the nerve, should one consider routine nerve biopsy as a procedure for proper evaluation of patients with paucibacillary dermal leprosy?

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