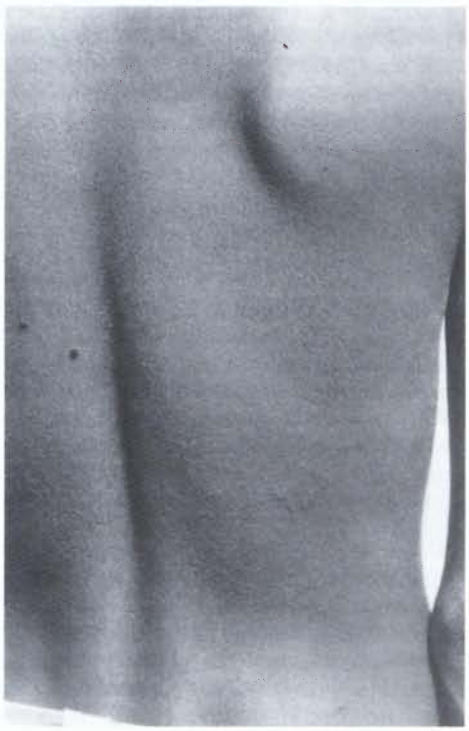
"A Natural Sweat Test" in Leprosy

TO THE EDITOR:

Leprosy is a chronic infectious disease with varied presentations. Nerve involvement is undoubtedly of fundamental importance in the pathogenesis of leprosy. Apart from motor paralysis and sensory loss, autonomic neuropathy is reported in varying degrees as indicated by sweat function tests, histamine triple response and tests for cardiovascular and respiratory system involvement in leprosy ([4]). It has been observed that on the anesthetic areas of leprosy the lesions of psoriasis either do not occur or disappear because degenerated dermal nerve fibers cannot produce neuropeptides like substance P, vasoactive intestinal peptide (VIP) and calcitonin G related peptide (CGRP) which are essential for neurogenic inflammation in the pathogenesis of psoriasis ([5]). The lack of sweating in lesions of leprosy which is well-known, also confirms the destruction of nerves in the infiltrates of leprosy ([4]). Recently we noticed an interesting finding of miliaria rubra sparing lesions of leprosy, which again can be explained on the basis of neural involvement in leprosy.



THE FIGURE. Extensive miliaria rubra over back sparing leprosy patches.

A 20-year-old male patient, an established case of borderline leprosy (BL) under treatment, complained of extremely pruritic lesions over his trunk which on examination were found to be miliaria rubra. During the examination the most noticeable finding was complete sparing of leprosy patches by the lesions of the miliaria rubra (The Figure). The multiple anhidrotic leprosy lesions were dramatically highlighted by a 'natural sweat test' which resulted in the production of the lesions of miliaria rubra. There was no other abnormality of any nature detected on systemic or cutaneous examination.

Miliaria rubra is a condition in which free flow of eccrine sweat to the skin is impeded. It is commonly seen in hot humid environments which result in injury to the epidermal cells lining the eccrine duct producing keratinous plugging. It presents as small, discrete, erythematous papulovesicles with a predilection for the clothed areas of the body ([1]). Leprosy lesions are known to be anhidrotic due to the destruction of sweat glands by local infiltrate around them and perineural granuloma resulting in autonomic nerve damage ([2]). A useful adjunct to the early diagnosis of a leprosy lesion is a sweat test, which is usually done using pilocarpine nitrate injected intradermally. The leprosy lesions become obvious being hypohidrotic or anhidrotic ([3]). The absence of miliaria rubra over leprosy lesions in our patient can be attributed to the reduced or absent functioning of sweat glands, which plays a central role in the pathogenesis of miliaria. This phenomenon is perhaps common in the hot humid climate of the Indian subcontinent, but has probably gone unnoticed in the past. Its explanation lies in the basic pathological changes which occur in and around nerves and appendages. To the best of our knowledge this observation has not been reported in the past.

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Single-Dose ROM Treatment for Multilesion Paucibacillary Leprosy—Further Observations

To the Editor:

A single dose treatment with ROM in the single skin lesion-paucibacillary (SSL-PB) leprosy group has been well received by the leprosy control programs. A small proportion of relapses and other clinical problems have been reported ([2, 3]). ROM single dose (ROM-1) treatment in paucibacillary patients with two to five lesions (PB 2–5) is currently under trial and the initial observations including relapses have been reported ([2, 3]). It is necessary to record longterm follow-up of such cases before this regimen can be considered for leprosy control programs.

A treatment period cohort analysis of ROM-1-treated 335 PB (2–5) leprosy patients followed up for a period ranging from 6 months to 70 months is reported. The mean period of follow up was 2.8 years. All the clinical problems other than reactions were recorded. The reactions were noted separately. The clinical problems were mainly in the nature of new lesions, persistence of lesions and an increase in the size of the patches. All these problem cases were given steroids before they were labeled as relapses. The rate of relapse observed in this study is compared with already published relapse rates after PB-MDT. The confidence interval (CI) is calculated for all the values at 95%. Table 1 shows that 10.4% (95% CI 7.03, 13.78) of the patients presented with various clinical problems other than reactions. The relapse rate is 1.4% (95% CI 012, 2.68) or 5.3 relapses (95% CI 0.26, 1.32) per 1000 person years of follow up. The annual rate is 0.5%. Two of them were bacteriologically positive when they relapsed. One of these bacteriologically-positive patients relapsed after 26 months and the other one after 70 months. All these 5 relapses have been retreated.

Table 1. *Relapse rate after ROM-1 in PB (2–5) leprosy.*

Description of events	No.
Patients followed up	335
Person years of follow-up	940
Patients with clinical problems	35 (10.4%)
Patients relapsed	5 (1.4%)
Relapse rate/1000 person years (py)	5.3