

- epidemiology of leprosy. Proc. VI Pacific Science Cong., 5 (1939) 939-945.
11. ROTBERG, A. The influence of allergic factors in the pathogenesis of leprosy. Proc. VI Pacific Science Cong. 5 (1939) 977-982.
 12. ROTBERG, A. "N-factor" of resistance to leprosy and its relationship to reactivity to lepromin and tuberculin. Doubtful value of BCG in anti-leprosy immunization. Rev. Bras. Leprol. 25 (1957) 85-106.
 13. ROTBERG, A. Uma visão panorâmica da leprologia moderna. In: *Medicina Tropical*. Lisbon, 1966.
 14. ROTBERG, A. The specific defect of immunity to hanseniasis ("anergic margin"), a 40-year-old Brazilian theory. (Editorial) Hansen. Int. 2 (1977) 12-14.
 15. The Cairo Congress number; immunology and serology. (Editorial) Int. J. Lepr. 6 (1938) 374.
 16. The lepromin test. (Editorial notes). Lepr. India 12 (1940) 115-116.

Simultaneous Type I and Type II Reactions

TO THE EDITOR:

Dr. Pfaltzgraff⁽¹⁾ raises the question of Type I and Type II reactions occurring together. I believe that this is not too rare a combination in BL patients, and in three cases which readily come to mind, it would appear to have been curative.

The first was a teenage girl seen more than ten years ago with numerous, bewilderingly dissimilar skin lesions, before we had considered the possibility of simultaneous Type I and II reactions.

She had been on low-dose dapsone, and while this was raised she was given high-dose steroids. Within a few months there was not a mark on her, and she took herself off treatment. Despite much encouragement to continue with dapsone, she refused. When she became pregnant, we waited for her to relapse but she passed this penultimate test unscathed, and remains well five years later.

Two men have been seen with a similar clinical picture, both with the addition of severe and widespread paralysis. One man

suffered ulceration of numerous nodules and also of most of the patch on one arm which had been his first (presumably BT) lesion years ago. Both made unusually good recoveries on prolonged steroids and clofazimine and, in the last case, with the addition of thalidomide. Of course, one has scars, but their otherwise normal appearance and neurological status is quite remarkable. They both continue to take dapsone, but I have often wondered whether severe and simultaneous Type I and Type II reactions has cured them, as it apparently did the girl.

—J. K. A. Clezy, F.R.C.S., F.R.A.C.S.

5 Rugby Street
College Park
South Australia 5069

REFERENCE

1. PFALTZGRAFF, R. Short-term, multi-drug chemotherapy of leprosy and Type I reaction. (Letter to the Editor). Int. J. Lepr. 50 (1982) 365-366.

Risks of Treating Leprosy in a General Hospital

TO THE EDITOR:

The paper of Mathai, Rao and Job, "Risks of Treating Leprosy in a General Hospital,"⁽⁶⁾ contains very valuable data for persons who must design health care and leprosy control programs in highly endemic areas,

such as India. It would seem to be very desirable that the medical care of leprosy patients should be incorporated into the same system of general medical care available to patients with other diseases. Whereas prolonged isolation of patients with mul-

tibacillary infectious leprosy may have been a rational and, at times, useful strategy in the pre-chemotherapy era, such isolated care is no longer required because of the availability of effective chemotherapy. With prompt use of appropriate drug treatment, prolonged isolation probably adds little if anything toward limiting the spread of leprosy. All too often prolonged physical isolation of leprosy patients from society has isolated them also from the benefits of the best medical care. While considerable circumstantial evidence has led many workers to the hypotheses that isolation may not be necessary, scant data are available which attempt to directly quantitate the risk of infection among health care workers having frequent contact with infectious leprosy cases in hospitals and clinics.

This careful study of a large number of health care workers who, during their hospital employment, had frequent contact with leprosy patients is most welcome. It is very encouraging to learn that the rate of new leprosy cases among hospital staff in this study was lower than that in the general population served by the hospital. However, with respect to the critical question of whether there is an increased relative risk among hospital workers who are caring for infectious leprosy cases when no isolation procedures are used, further consideration should be given to the appropriate data to be used for comparison. Defining an appropriate control or comparison group to measure the relative risks of a given exposure is a common problem in epidemiological research.

We question whether the most relevant comparison to estimate the risk in this situation is between hospital workers and either the incidence or prevalence of leprosy in the general population served by the hospital. Health care workers and students in this study population differed from the general population in many important ways, as was noted by the authors of this study. Health care workers were screened for illness on employment and admission to their training. They almost certainly differed significantly in socio-economic status from the general population. The age distribution and age at the time of exposure to infectious patients differed between hospital workers

and the general population. Health care workers were followed medically much more carefully than the general population; they were examined annually in this study.

One might estimate the risks to hospital workers more reliably by comparing the incidence rates of leprosy, when the disease appeared before the end of a (hypothetical) incubation period after employment in the hospital, to the rates occurring during an equal later interval in this population. One can then compare the attack rates of leprosy occurring before with those measured after the end of the incubation period for nosocomial exposure in the same population of hospital workers. If we assume that any increased risk in hospital workers from nosocomial exposure would be apparent only after the incubation period is passed, one might avoid the numerous confounding variables inherent in comparing rates of leprosy in different populations. Underestimation of the relative risk associated with a given exposure is a frequent problem when chronic diseases are studied and the incubation, induction, or latent periods are not taken into account when estimating the risk (⁷). However, in leprosy this problem can be dealt with, to some extent at least, since data are available on the probable length of the incubation period (^{1, 3, 4, 9}).

If one assumes that none of the leprosy cases detected among hospital staff during the first few years of hospital work could be due to exposure in the hospital, one could compare these early rates to those that occurred later after a reasonable incubation period of leprosy from nosocomial exposure after beginning employment. Among those 1614 persons who had been working at the Vellore Hospital for 0–5 years, there were six cases of leprosy, an attack rate of 4/1000. Of the 824 persons who had worked in the hospital for 6–10 years, 11 developed leprosy, an attack rate of 13/1000. Therefore, there was a relative risk of 3.25 for hospital workers at Vellore in their second five years of employment when compared to that experienced during their initial five years of work in the hospital. This attack rate of leprosy is significantly greater ($Z = 2.697$, $p < 0.003$) in the second five-year period.

It is not absolutely clear from the data given whether or not all of the 11 cases which

occurred in persons who had been employed for 6–10 years had the onset of their leprosy during the second five years of their employment. We have interpreted the data as though this was the case. However, even if some of the cases of leprosy in hospital workers who had worked at the hospital for 6–10 years had the onset of their disease during the initial five years of employment, the incidence rate is probably higher in this group of workers than in those who had worked five years or less. One could estimate the number of cases which could be expected by assuming that the rate of 4 cases per 1000 persons, that was observed in the first five years of employment, was the endemic rate in this selected population. During the ten-year period in the 824 persons who were employed for 6–10 years, one could expect to find 6.59 cases. Since 11 cases occurred in this group of employees, the relative risk would be 1.67 using these calculations.

Based upon our analysis of the data in this valuable paper, it would appear that while there may be a small increased risk of developing clinical leprosy in persons caring for leprosy patients in a general hospital setting when no isolation practices are utilized, the increased risk, if present, is fairly small. It is quite possible that minimal isolation procedures directed at infectious cases early in their hospitalization would obviate this risk. In addition, a policy of instituting supervised antileprosy chemotherapy on outpatients with an effective regimen, such as that recently recommended by WHO (²), prior to their admission to the hospital would be very likely to render even multibacillary patients non-infectious after a short period of such therapy (⁸). It was very encouraging to note that most of the infections which were detected in hospital staff were readily treatable, since all except two cases were of indeterminate or tuberculoid type. It is noteworthy that the Centers for Disease Control, Atlanta, Georgia, U.S.A., at present makes no recommendation concerning isolation techniques to be used for the hospital care of patients with leprosy (⁵). This emphasizes the lack of good data on this issue.

We feel that the treatment of leprosy patients in a general hospital is medically sound and will go a long way toward ensuring better care for such patients. Whether brief periods of isolation when chemotherapy is being instituted, or an initial brief period of chemotherapy of newly diagnosed cases on an outpatient basis, would further reduce the small risks associated with caring for infectious patients is not clear at present. Additional data, such as that provided by Mathai, *et al.* (⁶), would be very useful in answering this question.

—Kenrad E. Nelson, M.D.

Professor, Preventive Medicine

—Victoria Schauf, M.D.

Professor, Pediatrics

College of Medicine

University of Illinois

835 South Wolcott Avenue

Chicago, Illinois 60612, U.S.A.

REFERENCES

1. BADGER, L. F. Epidemiology. In: *Leprosy in Theory and Practice*. Cochrane, R. C. and Davey, T. F., eds. Bristol: John Wright and Sons, 1964, 2nd ed. pp. 69–97.
2. Chemotherapy of leprosy for control programmes. WHO Tech. Rep. Ser. No. 675, 1982.
3. DOULL, J. A., GUINTO, R. S., RODRIGUEZ, J. N. and BANCROFT, H. Risk of attack in leprosy on relation to age of exposure. *Am. J. Trop. Med.* **25** (1945) 423–429.
4. FELDMAN, R. A. and STURDIVANT, M. Leprosy in the United States, 1950–1969: an epidemiologic review. *South. Med. J.* **69** (1976) 970–979.
5. *Isolation Techniques for Use in Hospitals*. Centers for Disease Control, USPHS. Washington, D.C.: U.S. Government Printing Office, 5th ed. 1974.
6. MATHAI, R., RAO, P. S. S. and JOB, C. K. Risks of treating leprosy in a general hospital. (Letter to the Editor) *Int. J. Lepr.* **48** (1980) 298–302.
7. ROTHMAN, K. J. Induction and latent periods. *Am. J. Epidemiol.* **114** (1981) 253–259.
8. SHEPARD, C. C., LEVY, L. and FASAL, P. Rapid bacteriocidal effect of rifampin on *Mycobacterium leprae*. *Am. J. Trop. Med. Hyg.* **21** (1972) 446–449.
9. WORTH, R. M. and WONG, K. O. Further notes on the incidence of leprosy in Hong Kong. Children living with a lepromatous parent. *Int. J. Lepr.* **39** (1971) 745–749.