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# LEPROSY REVIEW

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# Leprosy Review

## A journal contributing to the better understanding of leprosy and its control

## British Leprosy Relief Association
## LEPRA





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*Leprosy Review* is published by the British Leprosy Relief Association (LEPRA) with the main objective of contributing towards the better understanding of leprosy and its control. Original papers on all aspects of leprosy, including research, are welcomed. In addition, *Leprosy Review* seeks to publish information of educational value which is of direct benefit to the control of leprosy under field conditions, and hence to the individual patient. The Journal aims to interpret what is being done in other disciplines, particularly for field workers.

From time to time the Editorial Board invites special articles or editorials from experts in various parts of the world, and gives consideration to the production of a supplement or special number devoted to a particular subject or theme of major importance.


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# Editorial

## EVALUATION OF A MULTIDRUG THERAPY PROGRAMME OF LEPROSY CONTROL

### Introduction

Multidrug therapy (MDT) as recommended by the World Health Organisation (WHO)[1] has now been in use for 12 years. However, the availability of MDT alone does not constitute an adequate leprosy control programme. Other essential components are early case-finding and the diagnosis of relapse; equally important are the detection of reactional states and adequate activities for the prevention of disability (POD). Much credit has been given to MDT for the dramatic reduction in the prevalence of leprosy worldwide,[2] but this is mainly due to the shortening of the duration of treatment and the 'register-cleaning' that has accompanied its introduction. It is changes in incidence rather than prevalence that provide evidence that the transmission of the disease is being interrupted.[3]

Evaluation is defined in a variety of ways when applied to programme management. It may be interpreted only as the comparison of the achievements of a programme with pre-set objectives;[4] here we will use it to include 'situation analysis', and for any activity that assesses the effectiveness of a leprosy programme. At the outset of a discussion on evaluation, it is therefore important to define the two basic objectives of MDT programmes of leprosy control:

to control leprosy in populations by interrupting transmission; and
to control leprosy in individual patients by arresting the disease process and preventing disabilities.

Programmes, and components of programmes, should be evaluated on the basis of both these objectives. Effective evaluation of the different parts of the programme will require different approaches. Epidemiological indicators of transmission are needed, but may react slowly to changes and be difficult to measure. Operational indicators are important, and those that are useful now are very different from those advised in the pre-MDT era.[5] Indicators of quality may be difficult to standardize when the diversity of programme contexts is considered; the establishment of 'benchmarks' to encourage self-evaluation may be a better aim. Indicators should ideally be complete, to allow comprehensive evaluation; relevant to the matter to be evaluated; repeatable between

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different observers and different conditions; readily available, so that the results are promptly available for action; simple, neither too complicated nor too costly; and in general use, so there is no controversy about the value or definition of the indicator.[6]

Two levels of evaluation can be considered: first the review of routinely produced programme statistics; and then the detailed evaluation undertaken by independent review, which includes going to see the programme in action. The first is inexpensive and should be done on a continuous basis by the programme manager and by the programme purchaser (whether government or a nongovernmental organization). This is possible, for example, by review of the ILEP 'B' returns over a number of years. Review of a single years' data can be very misleading: trends in indicators are much more useful. Such evaluation of data should be done annually, whereas site visits need only be done periodically (e.g. 3–5 yearly) or when indicated by trends in routine statistics. Pragmatic evaluation, by a site visit and an interview, is also valid and useful for the review of operational and administrative aspects of programmes; though it is more time-consuming and expensive, and needs some structure to gain maximum value, it may reveal much about a programme that is not obvious simply from statistics.

## Evaluation of the control of leprosy in populations

The evaluation of leprosy programmes using epidemiological indicators has been comprehensively surveyed.[7] Lechat *et al.* mention the use of 25 different markers. Since then more limited lists have been proposed.[8–10] However, almost all current indicators have drawbacks as indicators of outcome because they all largely reflect programme activities.

*Prevalence.* This indicator of the number of cases of the disease at a given time has become increasingly invalid in recent years. Defined as the incidence multiplied by the duration of the disease, the indicator reflects the treatment duration rather than giving specific information about disease transmission. Prevalence depends on the case definition in use: at present this is a patient needing chemotherapy.[11] Prevalence therefore also depends on the duration of treatment. Already, many PB patients never appear in prevalence figures, especially if they complete treatment in only 6 months, as they are treated and released from control before the year-end report is drawn up. The situation will become more complex in future as shorter and shorter regimes are introduced. The ultimate state would be reached if there were ever a single-dose treatment, administered on diagnosis—the prevalence of the disease would then be zero, even though there was a continuing incidence. However, the WHO goal of elimination is based on prevalence, and this indicator will continue to be used.

*Incidence.* This is a difficult indicator in leprosy but is the most useful in evaluating the effect of MDT on transmission. It is defined as the number of new cases occurring in a set period (usually 1 year). However, it has long been recognized that it is very difficult to measure as it would require annual population surveys, which are expensive, as well as complete accuracy in the diagnosis of early leprosy, which needs great skill.

As a proxy for incidence, case detection rate is often used. However, this has a major disadvantage when used for programme evaluation—it is highly programme-dependent as well as disease-dependent. Examination of the records of many programmes shows that case detection varies considerably from year to year. Enquiry will often reveal a

relationship between these variations and changes in the programme: case detection goes down when a crucial staff member is on leave, or goes up when a new detection strategy or health education programme is initiated. The situation can sometimes be made clearer by the consolidation of the results from different programmes in the same area. Local influences are then cancelled out, though national or regional policy changes may still be reflected. Case detection methods and diagnostic practices must remain constant for case detection to be a good reflection of incidence. Case detection rate, therefore, needs careful interpretation, as do two other programme-dependent indicators, child rate and mode of detection.

*Child rate.* The proportion of children aged 0–14 years among newly detected patients is highly dependent on the programme's strategy for dealing with indeterminate (especially single macular) lesions, and on the use of school surveys—a change in the priority or periodicity of school surveys can cause wild fluctuations in the trend of child rate.

*Mode of detection.* This is also often recorded, and high rates of self-reporting (voluntary reporting) are regarded as evidence of good community knowledge about leprosy.[12] The point has to be made, however, that any case detected through self-reporting might have been detected at an earlier stage by some active case-finding method, and this indicator is not a sufficient marker by itself of programme efficiency. Cessation of all active methods of case detection leads to high proportions of new cases detected by self-reporting and a drop in the absolute numbers of new cases.

Other epidemiological markers often reported are the gender ratio, and the lepromatous rate.

*Gender ratio.* This varies in new cases between programmes and countries and this variation may be biological or cultural. In many leprosy-endemic countries, women are less able to obtain health care, and/or are less likely to accept adequate examination by a male health worker. Recruiting of female health staff to redress this imbalance is now frequently undertaken. The imbalance remains, however, in situations widely differing in terms of health care access and the acceptability of examination, and also between those where women stay mainly at home (protected from exposure to *Mycobacterium leprae*), and those where they take a full part in community life. It seems likely that the difference is multifactorial, but this matter is becoming more difficult to research, as many programmes do not now report cases by gender.

*Lepromatous rate.* In new cases, intrinsically variable by race, lepromatous rate is also thought to change as leprosy transmission stops.[13] This is assumed to be because the incubation period of lepromatous leprosy is longer than that of tuberculoid, and cases therefore continue to appear for some time after interruption of the transmission of the disease and the reduction in the number of tuberculoid cases.

*Disability rate at detection.* This is another indicator which represents a useful indicator for evaluation. This index, normally taken as the percentage of new cases with WHO Grade 2 disability, gives a double insight into the effectiveness of control activities. It monitors both the promptness and the completeness of case detection—promptness as early case detection should occur before disabilities have had the chance to occur, and completeness on the assumption that all patients with leprosy disabilities will eventually come to the notice of the programme. Disability rate is a useful marker, both by itself, and in association with others mentioned above. For example, if the mode of detection proportions suggest that the programme is mostly relying on self-reporting,

the rate of disability at detection may indicate delay between the onset of disease and case detection. Equally, if the case detection rate is falling, but disability at detection is rising, it suggests incompleteness of case detection rather than a genuine fall in incidence. Disability rate may prove to be the most useful outcome indicator of case detection activities.

## Evaluation of the control of leprosy in individuals

It is assumed that MDT is effective in killing *M. leprae* in humans and in stopping the disease process. Similarly it is assumed that prevention of disability activities, such as treatment of reactions[14] and use of appropriate footwear,[15] are effective. Evaluation of an MDT programme is therefore assessing effectiveness in treating patients using MDT, and in preventing disabilities.

### *Effectiveness in treating patients with MDT*

The two most useful measures of the programme's effectiveness in treating patients with MDT are the MDT coverage and MDT completion rates.

*MDT coverage.* Among new patients and among registered patients, this is commonly used. However, coverage alone is an inadequate indicator, as a single dose of MDT followed by default achieves coverage, even though it is clearly unsatisfactory management. Coverage is usually presented as a percentage which is dependent on the denominator, and patients may be inappropriately deregistered in order to achieve a higher MDT coverage statistic.

*MDT completion rates.* As used by ILEP in their 'B' form, these give a better indicator of genuine achievement in MDT programmes, and are calculated on a cohort of patients who began treatment during the year up to 9 months before the reporting date (PB) and during the year up to 3 years before the reporting date (MB). These groups are the most recent which can be assessed within the current WHO completion criteria (6 doses in 9 months or 24 doses in 36 months), but in fast-developing programmes, this information may already be out of date, and judgments of 'quality' based on them may therefore also be anachronous. MB completion rates will reflect previous performance rather than current work. Completion rates, even though more complex to calculate, should be the most reliable indices of the effectiveness of programmes in treating patients adequately with MDT, though they are a new indicator for some programmes, and record systems that permit their calculation may take time to develop.

### *Effectiveness in preventing disabilities*

The difficulty of monitoring POD activities associated with MDT programmes is shown by the numerous methods proposed to assess nerve function. While controversy about filaments and ball-pens rages among the experts, most patients do not have access to even the most basic neurological supervision. Monitoring nerve function using WHO grading is a very coarse tool, as much deterioration can happen within grade 2, and therefore not be recorded as change: a slight clawing at detection places the patient in that grade, and even the grossest deterioration thereafter is not recorded as change.

However, there is no other system that could easily replace WHO grading, and the worsening from no disability (Gr. 0) to anaesthesia (Gr. 1) or to visible deformity (Gr. 2) is certainly significant. WHO disability grading is therefore useful in establishing the level of disability but is of less use in assessing change. WHO grading is often done at diagnosis, but less frequently at the completion of treatment to monitor change during the course. More complex methods of assessment are not suitable for use under field conditions but are useful for research purposes. Careful monitoring of sensory and motor nerve function is possible in some centres; it is more difficult to train staff to take action based on any changes recorded. The reproducibility of the tests used may not be high, and variations between assessments may lead to overdiagnosis of 'silent neuritis' and overuse of steroids. Scoring of disability records to get a measure of the situation within a programme has been recommended as a management tool.[16]

Sole wounds (ulcers and cracks) are the most characteristic deformity of leprosy, and the 'doctrine of the first ulcer'[17] has long been a strategy in dealing with patients. Trends in wound counts within the population covered by a leprosy control programme may be a useful and highly reproducible method for evaluating the programme's effectiveness in treating and preventing plantar ulcers, both by self-care and footwear.[18] It may also reflect the effectiveness of other POD activities.

## Evaluation of leprosy control programmes by site visit

Site visits are essential for a full evaluation of a programme, whether to develop an overview of the work ('situation analysis'), or to undertake formal evaluation against preset targets. In addition to physical checking of the registers to confirm data previously presented (and to see the quality of the registers and records) site visits allow particular information to be gathered on programme context, methods, and management; and on staff attitudes to their work, to each other, and to their patients. Programmes and organizations often have their own checklists of items to be covered in such visits, but the following areas should be included.

It is essential to know the context of the leprosy control programme for any evaluation to be valid. Programmes may be 'vertical', joint (with TB or dermatology, for example), fully integrated into primary health care (PHC), or integrated with 'vertical' supervision. Additionally they may be running within a Government health service or independently (NGO or private). Programme managers may be defensive of their own position—the more so as the falling prevalence of leprosy is seen to threaten jobs—but should understand the different models, and be open to any modifications that may be appropriate. The programme methods—case finding, case holding, surveillance, prevention of deformity, rehabilitation—may be set by national or organizational policy, and be outside the control of the local manager. However, managers who have an understanding of the benefits and limitations of the strategies used will be better able to maximize their effectiveness. A further essential aspect of management to be assessed is the ability of managers to interpret information that is received (from staff, or by feedback from their own superiors), and to take appropriate action based on it. Managers will also be able to indicate problems within the programme in areas such as the reliability of drug supply, the quality of the staff, the regularity of financial remittances, the availability of transport, and the level of community support.

From the staff it is important to determine their own feelings on matters such as the adequacy of their training (was it appropriate for the job they are doing?), and whether they feel confident and interested in their work. A general enquiry, such as asking about anything that would help them give a better service to the patients, will give an insight into their own attitudes to the work, as well as identifying programme constraints that may not have seemed relevant to the management.

The supervision of the programme is an important area for organizational evaluation. Do the supervisors have a regular timetable, or do they make spot checks? Do they have a supervision checklist? Do they encourage their junior staff or only criticize them? Perhaps the most important point, especially in integrated programmes with specialist supervision, is to determine whether the supervisors really supervise, or whether they simply take over the leprosy clinics that they attend. The supervisory hierarchy must also be determined, to see that supervisors are themselves responsible to someone else; and it should be established whether the supervisors have had specific training in supervision/management, or have simply achieved seniority through long service in leprosy work.

Operational aspects of the programme also need to be examined. In particular, details of the current case-finding strategy, and the confidence and competence of the staff in diagnosing leprosy are both important. In looking at the treatment component, an overlooked problem may be the dropping-out of patients between detection and treatment. Particular note must be taken of any 'noncompliance' which is actually the fault of the programme: due to failures of manpower, transport, or drug supplies. Case notes may be reviewed for specific operational indicators (such as the regularity of disability grading records, or the correct implementation of release-from-treatment criteria) or for completeness of treatment delivery records.

The regularity of attendance can be checked from registers, and the acceptability of this compared with local criteria. By talking to staff and patients, it may be possible to identify the constraints—lack of motivation, lack of opportunity or lack of understanding—that lead to poor compliance. If possible, new and old patients should be reviewed during clinic or drug delivery sessions.

Prevention of deformity activities are particularly appropriate for site visit evaluation. The commitment of the staff to the whole concept of POD can best be elicited person to person. Technical questions about the content of health education, the use of prednisolone and the advice given to patients can be asked, and an assessment of the clinical skills of the staff can also be made.

Attitudes and relationships are important in any programme. On-site evaluation allows the complex relationships among the staff, between staff and patients, and between the programme and the local community, to be assessed. Staff who have a good relationship with patients will be well motivated to give a high quality service, and will also be better able to communicate with their patients.

## Summary

MDT programmes for leprosy control have two objectives, controlling leprosy in populations and controlling leprosy in individuals. Evaluation of such programmes needs to address both objectives and this can be done by a review of the trends in key indicators and by site visits. Site visits are more expensive and should be done less

frequently, but they can reveal issues not apparent in routinely produced statistics. Evaluation on an annual basis is the responsibility of programme managers and programme funders. Evaluation by programme staff themselves should be encouraged and supported.

Evaluation of an MDT programme's effectiveness in controlling leprosy in a population should be by analysis of case detection as a proxy for incidence. Prevalence rates will continue to be monitored because of the WHO elimination goal, but these do not reflect disease transmission. Case detection is a proxy measure of incidence and depends on consistency in case detection activities. Case detection data by age, gender, mode of detection, disability ratio and lepromatous (MB) rate need to be analysed over at least 5 years and preferably 10 years to give an indication of trends in incidence. Caution is needed, however, as the pattern seen when case detection deteriorates may resemble the pattern expected when transmission is reduced. The site visit is important in this situation in allowing examination of the case detection activities, as well as in looking for new, undetected cases in the population.

Evaluation of the MDT programme's effectiveness in controlling leprosy in patients should be by analysis of both the effectiveness of MDT delivery and the changes in disability. For drug delivery, MDT coverage of new and registered patients is used, but this only reflects the basic minimum of treatment, that each patient has started MDT. MDT completion rates are the best indicators, with PB rates reflecting the situation in the previous year, and MB rates the longer-term position. In monitoring disability, WHO gradings are of limited use in assessing change, and are not always recorded. If they were available for the start and end of each patient's treatment it would give a crude indicator of the programme's effectiveness in preventing disabilities. Better methods have not yet been proved to be either adequately reproducible or simple enough for PHC-based programmes. More research is urgently needed in this field. It may be that simple counts of sole wounds will prove to be the most suitable indicator of effectiveness in the prevention and treatment of disabilities. A site visit will help to reveal what is actually going on in the area of prevention of disabilities as well as in treatment delivery. Remember that it is always worthwhile speaking to the patients and not only to the staff!

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# Editorial

## WILL THERE BE A NEED FOR LEPROSY CONTROL SERVICES IN THE 21ST CENTURY?*

Since the introduction of MDT in the early 1980s the global number of leprosy patients registered for chemotherapy has been reduced by more than 60%. What will be the consequences of this changing leprosy situation for the implementation of leprosy control? Will there still be a need for leprosy control services in the next century? Before we can answer this question, it must be made clear what actually is the leprosy problem and the change we are talking about.

What do we mean by 'the leprosy problem' and how do we express this problem? The suffering due to leprosy is caused by irreversible damage to the peripheral nerves, which leads to sensory loss, paralysis and loss of function of hands, feet and eyes. The resulting deformities are the main cause of the stigma attached to the disease. This stigma especially leads to the serious psychological, social and economical consequences for leprosy patients as well as for their families. The *raison d'être* of leprosy control is that leprosy is a disabling disease. For the public leprosy is synonymous with deformity. Basically leprosy control means prevention of leprosy related deformity and disability. With the current definition of a case of leprosy (a patient in need of antileprosy chemotherapy), there is a danger that we neglect the actual leprosy problem as it is perceived by patients and communities: the physical, social, economical and psychological impact of the disease on the affected individuals, their families and their communities. Mere data on the number of patients in need of MDT (prevalance) insufficiently reflect the leprosy problem. However, as we do not have adequate methods to measure the magnitude of the suffering caused by leprosy, the prevalence of registered leprosy cases, the incidence (as reflected by case detection figures) and the number of persons with leprosy associated disability remain the best possible indicators for the leprosy problem. But we must be aware of the limited value of these figures.

At present we do have the knowledge and tools to control leprosy. These tools are early diagnosis, MDT and early identification and appropriate treatment of nerve function impairment. Yet many leprosy control programmes have not been very successful in this respect, because most health services have proved incapable of adequately delivering these tools. A wide gap exists between the number of estimated cases and the number actually diagnosed and less than 50% of the cases registered are on MDT and a smaller proportion completes full course MDT treatment. Moreover, even

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among registered cases impairment of nerve function, resulting in new disabilities and worsening of existing disabilities, occurs with distressing frequency.

In April 1993 the total number of patients was estimated to be 3·1 million in 90 endemic countries. There were 2·3 million patients registered for treatment ('known prevalence') of which only 1·1 million cases were on MDT (47·8%). The number of persons with leprosy related disability probably lies between 5 and 7 million. The estimated number of new cases (incidence) is 900,000 per year (WHO 1993). While the annual number of new cases is slowly decreasing in some countries (especially those showing socio-economic development and having well functioning leprosy control programmes based on dapsone monotherapy already existing a long time before the introduction of MDT), on a global scale case detection increased from 575,000 in 1990 to 590,000 in 1991 and 650,000 in 1992. Although this increase of case detection figures may partly be attributed to improved self-reporting and intensified active case finding, and to the lack of precision inherent to global data based on routine field reports, it definitely does not reflect a worldwide declining incidence.

Let me now come back to my introductory question: what are the consequences regarding the need for leprosy control services in view of the reduced prevalence since the introduction of MDT? The declining prevalence is largely caused by the shortening of treatment duration with MDT as compared with dapsone monotherapy and the clearing of the registers of patients not requiring chemotherapy any more; each account approximately for 50% of the observed reduction in prevalence. While the introduction of MDT had a tremendous impact on the prevalence, the incidence figures (as reflected by case detection) so far show little change in many countries where MDT is implemented. We have not controlled the disease until the incidence is contained. Unless incidence is reduced all problems regarding case finding, diagnosis, treatment and disability prevention remain basically unchanged. In summary: reduced prevalence does not necessarily reflect a decline in incidence, and we do not yet have convincing evidence that MDT has an additional impact on the incidence of leprosy.

In view of the long incubation period of leprosy and the fact that wide MDT coverage has only been established in some countries during recent years, it is, however, too early to already expect clear evidence that the implementation of MDT has an impact on the incidence. I am convinced that leprosy is a disappearing problem and that in the long run even complete eradication of leprosy is feasible as a result of a combination of various factors, including socioeconomic development, BCG vaccination and early diagnosis and MDT. This will, however, be a slow process. Despite a slowly declining trend the incidence of leprosy will remain a significant problem till far beyond the turn of the century.

All new cases have to be detected at an early stage of the disease and be submitted to regular and complete treatment with MDT. Moreover, a significant proportion of new cases will already show disability at diagnosis and many patients will develop disability after diagnosis. In addition, all current patients with nerve function impairment are at risk of developing additional disabilities. In conclusion: despite the strongly declining prevalence leprosy and leprosy related disabilities will, for decades to come, continue to exist as an important problem, not only for the patients and their relatives, but also for the health services and social services. Leprosy services will be needed in the 21st century.

The 44th World Health Assembly (1991) adopted in a resolution the goal of attaining global elimination of leprosy as a public health problem by the year 2000. 'Elimination'

is defined as reaching a level of prevalence below 1 case per 10,000 population. This important initiative has, at least temporarily, promoted the commitment of endemic countries to leprosy control. However, the elimination goal should not give the false impression that the leprosy problem will have been solved by the year 2000. After a WHO press release on the elimination goal in July 1993 the Dutch press brought the news under headings such as: 'leprosy will have disappeared by the year 2000'. In creating overexpectation we lay the foundation for disappointment and thus future demotivation of the health workers, administrators, politicians and the public, including the contributors to the donor agencies. We must be extremely careful in the way we inform the public. Overoptimistic information may easily become counterproductive as the funds required for the treatment of all cases with MDT will not be available any more.

With the reducing number of patients the cost per patient cured will increase. WHO has roughly estimated the direct cost for the health services to diagnose and cure a leprosy patient under diferent prevalence situations. With a prevalence of more than 10 per 10,000 the average costs for a PB patient are US$30 and for an MB patient $150; at a prevalence rate of about 5 per 10,000 the costs are $70 and $280 respectively and at a prevalence below 1 per 10,000 $100 for a PB patient and $400 for an MB patient. It may be expected that with the declining incidence and prevalence of leprosy and thus the decreasing relative importance of leprosy as a public health problem, governments in the endemic countries will make less funds available for leprosy control. In order to achieve eradication there will be a continuing need during the next decades for technical and financial resources from international donor agencies. Here we face the danger that the recent success of leprosy control may have negative effects on fund raising by the NGOs. It is usually not a problem to sell a success story, but this may become the case in leprosy control. Leprosy control should not become a victim of its own success, just as we are getting close to our goal to eradicate the disease. Therefore, whenever the elimination goal is presented it should be made clear that even when this goal is attained, there will continue to be significant numbers of (new) cases of leprosy and people with severe psychological, economical and social problems caused by leprosy who need assistance. Leprosy will not be under control when the 'elimination' goal has been achieved.

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# The role of IgM antiphenolic glycolipid-1 antibodies in assessing household contacts of leprosy patients in a low endemic area

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*Summary* This study was carried out to assess the role PGL-1 antibodies may have to play in assisting with early diagnosis in close contacts of leprosy patients. Blood samples were collected from patients and contacts. It was found that 6·9% of index cases and 1% of healthy contacts were positive for PGL-1 antibody. None of the healthy contacts developed clinical leprosy and all had become seronegative at follow-up. We conclude that screening for PGL-1 antibodies has a limited role in the screening of healthy contacts and may not be of use in low endemic areas.

## Introduction

The effective implementation of multidrug therapy (MDT) for the treatment of leprosy in a geographically defined region results in the reduction in the number of leprosy patients and the case load for the leprosy control programme. Optimal strategies for active case detection in such low endemic areas where MDT has been implemented have yet to be defined. The relative risk for development of clinical leprosy is greater in the household contacts of leprosy patients than in the general community.[1] There are, however, substantial differences in the rates of leprosy in close contacts of patients between different regions, with higher attack rates being associated with communities with a higher overall prevalence of leprosy. A number of cross-sectional serological studies have demonstrated an increased proportion of household contacts of leprosy patients with IgM antiphenolic glycolipid-1 (PGL) antibodies, than in the general community (reviewed by Smith[2]). In one large prospective study of the contacts of leprosy patients in a high endemic area, there was a strong association between the antiPGL-1 antibody levels and the risk of leprosy.[3] Therefore, screening for antiPGL-1 antibodies in household contacts may help in detecting those at increased risk of disease.

Active leprosy control measures have been undertaken in the Lalitpur District in the Central Region of Nepal, close to Kathmandu, since 1962 when the Anandaban Leprosy Hospital was established. This was intensified in 1986 when a mass survey of the district was commenced. Active case-finding and the implementation of the World Health

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Organisation (WHO) recommended MDT regimens[4] was conducted until 1990. During the period of the survey, 234 new cases were registered from a district population of 210,538, a case-detection rate of 2·2/10,000 per year. For the purposes of this study, during 1991–92 all registered patients in the district were revisited and their household contacts examined. In order to assess the additional benefit of testing for antiPGL-1 antibodies in this low endemic area, blood samples were collected from both patients and contacts and tested for antiPGL-1 antibodies.

## Materials and methods

During 1991–92 every patient ever registered in the Lalitpur District was visited at home by paramedical workers. The southern area of this district has poor access and therefore leprosy control staff only visit there occasionally. The current status and residence of patients were updated. Many patients had moved away from the district or died. All previous patients and those patients currently receiving MDT who were home at the time of the initial visit or one follow-up visit were examined physically with detailed voluntary muscle testing and sensory examination. Skin smears were performed on these patients and a finger prick blood sample was collected for antiPGL-1 antibodies. The healthy household contacts of these patients were also examined and where possible a capillary blood sample was also collected. Capillary blood samples were collected onto chromatography paper (Whatman, UK) which was stored with desiccant at −20°C until tested. A 6-mm circle was punched out of the blood spot and eluted overnight in 500 $\mu$l of 0·9 M phosphate-buffered saline, ph 7·2, with 0·05% Tween 20. The eluant, which was approximately a 1:50 dilution of serum, was then used in IgM antiPGL-1 enzyme linked immunosorbent assay (ELISA).

This utilized the glycoconjugate, disaccharide-bovine serum albumin (dBSA), provided through the Immunology of Leprosy Programme of WHO, in a solid-phase ELISA as previously described.[5,6] Samples with an absorbent >0·199, the mean +3 standard deviations of serum samples from 91 healthy Nepali subjects, were considered positive. Previously a strong correlation between simultaneous venous and capillary blood samples for antiPGL-1 antibodies was demonstrated in a group of 62 leprosy patients $r_8 = 0{\cdot}81$, $P < 0{\cdot}001$) (data not shown).

**Table 1.** The classification of index cases and their contacts and the number in each group who were IgM antiPGL antibody positive

| Classification* | TT | BT | BB | BL | LL | IN | PN |
|---|---|---|---|---|---|---|---|
| No. of index cases | 23 | 76 | 6 | 23 | 16 | 3 | 12 |
| Total no. of contacts | 61 | 194 | 12 | 64 | 38 | 3 | 31 |
| PGL-1 Ab + ve contacts | 0 | 2 | 1 | 0 | 0 | 0 | 1 |
| PGL-1 Ab − ve contacts | 61 | 192 | 11 | 64 | 38 | 3 | 30 |

* Classification: tuberculoid (TT), borderline–tuberculoid (BT), midborderline (BB), borderline lepromatous (BL), lepromatous (LL), indeterminant (IN) and primary neuritic (PN).

**Table 2.** Year of release from treatment for the different types of index cases

| Classification | TT | BT | BB | BL | LL | IN | PN |
|---|---|---|---|---|---|---|---|
| RFT year | | | | | | | |
| 1970–79 | 1 | 2 | 1 | 1 | 0 | 0 | 0 |
| 1980–84 | 11 | 37 | 3 | 5 | 7 | 1 | 5 |
| 1985–89 | 8 | 22 | 0 | 5 | 5 | 2 | 4 |
| 1990–92 | 2 | 11 | 1 | 8 | 1 | 0 | 0 |
| On treatment | 1 | 4 | 1 | 4 | 3 | 0 | 3 |

* Classification as in Table 1.

## Results

A total of 159 leprosy patients (113 male, 46 female) were re-examined. The classifications of these index cases, all of whom were adults, are shown in Table 1. The duration since release from chemotherapy varied from 1 to 22 years with a mean duration of 5 years (Table 2). In all, 16 of the index cases were still receiving chemotherapy. Of the index cases, 55 had been treated with multibacillary (MB) MDT, 93 with paucibacillary (PB) MDT and 11 had received dapsone monotherapy alone.

During this follow-up survey, 403 household contacts of 159 index cases were examined. Of these contacts, 178 were male, 225 female and 93 were aged <15 years. No new cases of leprosy were detected among the 403 contacts.

Of the index cases, 11 (6·9%) had IgM antiPGL-1 antibodies. The proportion of index cases who were seropositive among those still receiving chemotherapy (4/16, 25%) or who had ceased chemotherapy in the preceding 2 years (4/23, 17·4%) were higher (Table 3); 4 of the contacts (1·0%) were positive for IgM antiPGL antibodies (Table 4). The index cases of the antibody positive contacts (2BT, 1BB, 1PN) were antibody negative at the time of testing (Table 1) and had completed therapy for variable periods of time (Table 3). None of the 4 antibody positive contacts had clinical features of leprosy. All 4 subjects were re-examined 6 months later when there were also no features of leprosy. On repeat testing, the antiPGL-1 antibodies had become negative at the 6-month follow-up.

**Table 3.** IgM antiPGL-1 antibodies in index cases and contacts analysed by year of release from treatment

| | Index case | | Contact | |
|---|---|---|---|---|
| | Ab + ve | Ab – ve | Ab + ve | Ab – ve |
| RFT year | | | | |
| 1970–79 | 0 | 5 | 0 | 4 |
| 1980–84 | 3 | 66 | 2 | 181 |
| 1985–89 | 0 | 46 | 1 | 108 |
| 1990–92 | 4 | 19 | 1 | 57 |
| On treatment | 4 | 12 | 0 | 38 |

**Table 4.** Actual absorbance values for antiPGL-1 antibody in the 4 healthy contacts (normal <0·200)

| Contact | | |
|---|---|---|
| 1 | 35-year-old female | 0·276 |
| 2 | 8-year-old male | 0·226 |
| 3 | 35-year-old female | 0·959 |
| 4 | 21-year-old female | 0·355 |

## Discussion

The role of PGL-1 antibodies in the serodiagnosis of leprosy has been examined in a number of cross-sectional household studies.[2] The seropositivity rate in household contacts ranged from 7 to 43% with higher rates being observed in contacts of MB cases. In contrast only 1·0% of the contact group in this study had demonstrable IgM antiPGL-1 antibodies. This may reflect the fact that 89% of the index cases had completed chemotherapy, in many cases up to 10 years beforehand. Further, the overall prevalence of registered leprosy case load had fallen dramatically in the district from 70/10,000 to 2·2/10,000 over a 20-year period (unpublished observations). Therefore the exposure to leprosy in the household and community was also substantially reduced.

As there were no new cases among this group of contacts examined no test can be used to define a high risk group. However, in a low endemic situation there are few new cases and those that occur do not seem to be related to close exposure to an index case. This is typical, however, for many low endemic districts following the successful implementation of leprosy control programmes.

The small number of seropositive subjects had no evidence of leprosy and rapidly became seronegative. Although there is an increased risk of leprosy in seropositive household contacts the majority of such individuals in follow-up studies in the Philippines (92%),[7] Tahiti (99%)[8] and Papua New Guinea (99%),[9] did not develop disease over 2–5 year follow-ups. Rather than being a marker of clinical leprosy, IgM antiPGL-1 antibodies may be evidence of subclinical infection, particularly in children and adolescents[9,10] which revert to negative spontaneously. Further, in longitudinal studies both in household contacts[3] and in the general population,[9] the majority of new leprosy cases develop in seronegative rather than seropositive subjects. Therefore screening for IgM and antiPGL-1 antibodies has a limited role in the screening of contacts of leprosy patients in a low endemic region.

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# Field evaluation of WHO–MDT of fixed duration at ALERT, Ethiopia: the AMFES project—I. MDT course completion, case-holding and another score for disability grading

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*Summary* We report on 286 new leprosy patients (128 PB, 158 MB) enrolled in the AMFES project, a field study in which patients are monitored during WHO–MDT and during 5 years thereafter, by active surveillance. This first paper describes the purposes, organization and methods of the study, patient enrolment and preliminary results of MDT completion and case-holding.

Of 128 PB patients 102 (79·7%) completed MDT and of 91 on surveillance for more than 1 year, coverage with reviews had been good or very good for 31, fair or poor for 36 and very poor or nil for 21 PB patients. Of 158 MB patients 64 had completed MDT, and 26/128 (20·3%) PB and 18/158 (11·4%) MB patients were lost to follow-up during treatment, with 76 MB patients still on treatment.

At first diagnosis, 159/286 (55·6%) had nerve function impairment, with no significant differences in disability grade by gender or between PB and MB patients. The proportion of disability grade 0 amongst new cases decreased very significantly with age, from 28/41 (68·3%) for age 0–14 years to 13/57 (22·8%) for 50 years and above. In view of the limitations of patient disability grades, a score per patient of the sum of disability grades for the four extremities, named 'HF-impairment score', is shown to be more informative.

Incidence of leprosy reactions and neuritis in these patients, during treatment and during surveillance, is reported upon in Part II (on pp. 320–332 of this issue).

## Introduction

In order to evaluate the effectiveness of the WHO-recommended multidrug therapy (MDT) regimens[1] in the operational conditions of a leprosy control programme, ALERT's MDT Field Evaluation Studies (the AMFES project) started patient intake in 1988. The

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main objectives are to assess: the incidence of relapse and factors associated with the occurrence of relapse; the incidence of leprosy reactions and factors associated with the occurrence of reactions; and the incidence of new or increased nerve dysfunction and its progression to permanent nerve function impairment.

In this long-term prospective study, paucibacillary (PB) and multibacillary (MB) patients are observed for reactions and nerve dysfunction during the course of MDT and for reactions, nerve dysfunction and relapse occurring after the course of MDT during a 5-year period of active surveillance.

Observations are made under routine leprosy control service conditions. Criteria for diagnosis, for allocation to categories PB and MB, for relapse, reaction, nerve dysfunction and also the treatment regimens used, are all similar to those in the routine leprosy control services.[2]

Enrolment started in April 1988. This paper gives findings up to the end of June 1992, for 286 new patients registered in the first 3 years of the project, who had been observed for between 15 and 51 months. Characteristics of patients are given and results to date regarding completion of MDT courses and changes in BI during MDT. For 102 PB patients who completed MDT, we give preliminary results of case holding during up to 3 years of active surveillance.

A second paper (Part II, pp. 320–332) gives preliminary findings on the incidence of leprosy reactions, neuritis and nerve dysfunction in this cohort of patients.

## Methods

In the absence of active case detection, practically all patients enter the control programme as self-reporting new cases at one of the 99 leprosy clinics held in the area. ALERT's leprosy control programme is essentially still a vertical leprosy service run by specialized health personnel. All new, untreated leprosy patients are eligible for enrolment. Thirty-nine patients who had received some dapsone just before the start of their MDT treatment for a short period only (for up to 8 weeks for PB and 16 weeks for MB) have also been regarded as 'new, untreated'. Apart from that small amount of pre-MDT dapsone these patients did not differ from the others.

### DIAGNOSIS AND CLASSIFICATION

Seven leprosy control supervisors (LCSs) carry out the diagnosis and classification of the patients. These LCSs are paramedical health workers with additional special training in leprosy. All have many years of experience in leprosy work in peripheral clinics. Their work was supervised by two medical officers (MOs) (AdR and SG), who confirmed diagnosis, classification and eligibility for enrolment on the basis of the patient record cards which the LCSs presented to the MOs within 2–4 weeks of the first examination.

Paucibacillary (PB) patients are those clinically classified as tuberculoid (TT), borderline tuberculoid (BT) or indeterminate (I) and who have or had a negative bacterial index (BI) at all sites. Until July 1989, BT patients whose highest BI = 1 were included in the PB group. Multibacillary (MB) patients are those clinically classified as borderline–tuberculoid (BT) with positive skin smears, borderline–lepromatous (BL) or lepromatous (LL) and all those who have, or had at any time, a positive skin smear at one or more sites.

ALERT's field programme, and thus also this study, uses the simplified classification for fieldworkers recommended by Jopling which adds the rarely occurring BB cases to the BL group.[3] The term neural leprosy (NL) is used for leprosy patients with nerve involvement only, lacking skin lesions and whose skin smears are (repeatedly) negative, there is either clear thickening of more than one peripheral nerve with or without functional loss, or at least one clearly thickened nerve with some associated loss of function. For these NL patients, assignment of treatment regimen (PB or MB) was based on the extent of nerve involvement or on the finding of acid-fast bacilli in a nerve biopsy.

For the majority of patients clinical diagnosis and classification posed no problems to the LCSs. Skin smears were taken, for all patients, from both earlobes and from at least two skin lesions. In case of doubt on a paucibacillary condition (advanced BT or BL?), skin smears were repeated after 4 or 8 weeks. If, for patients clinically classified as BL or LL, the first smear results were negative, skin smears were to be repeated and, if the BI was again found to be zero, the patient was examined by one of the MOs who also performed a skin biopsy. In case of doubt in classification between BT and BL, the LCSs were instructed to start treatment with the MDT regimen for PB patients while awaiting the skin-smear results and, if necessary, the assessment by one of the MOs. The following combinations of findings made a re-examination by an MO for diagnosis or classification necessary:

patients clinically BL or LL, for whom the highest BI $< 2$ in two consecutive sets of skin smear examinations;

patients clinically BT, with any skin smear positivity;

patients clinically classified as TT or indeterminate (I);

patients clinically assessed as BT, with no definite sensory loss for light touch in skin lesions; and

patients with negative skin smears and in whom no leprosy-like skin lesions have been seen and who thus should have either neural leprosy or no (active) leprosy.

The MO, when re-examining the patient, usually takes a skin biopsy. Skin biopsies are also taken in other recently registered patients when seen by an MO during a routine supervisory clinic visit. Where the histopathologist's assessment of the classification differs from the classification given on clinical assessment, this does not usually lead to a change of the classification or of the treatment regimen. This is because we aim at an evaluation of the MDT programme under field conditions. In ALERT's field services no biopsies are usually taken for the sake of classification. Therefore, it is only in cases where the MO cannot, on clinical examination including repeated skin smears, draw a conclusion, that the histopathologist's classification is taken into account for the final decision.

## TREATMENT

For PB patients, treatment consists of self-administered dapsone 100 mg daily (50 mg for patients under 15 years) for 27 days, plus that dapsone dose and rifampicin 600 mg (300 mg for patients under 15 years) given under supervision once every 4 weeks, at the time of drug collection. The treatment course is completed when 6 doses of rifampicin have been taken within a period of $(9 \times 4 =)$ 36 weeks.

Treatment for MB patients consists of 100 mg dapsone + 300 mg clofazimine + 600 mg

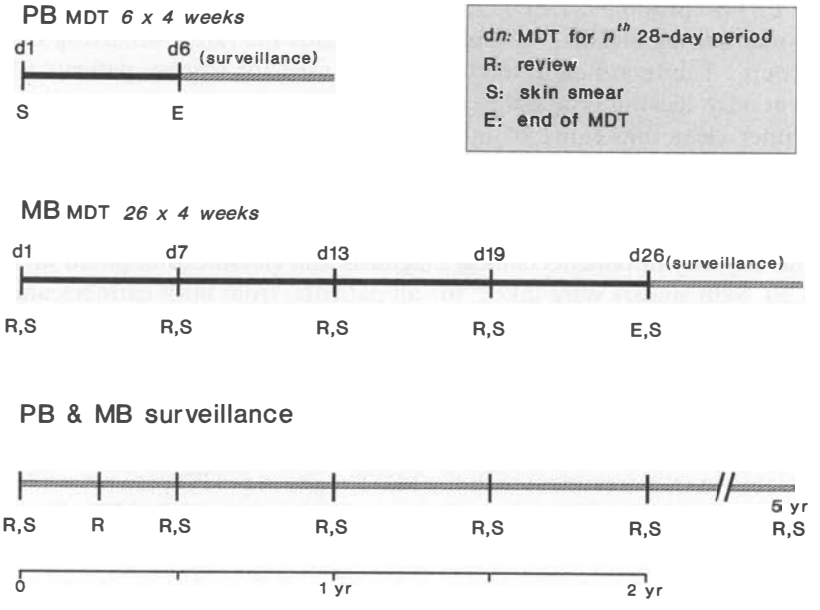


**Figure 1.** Timetable for MDT and surveillance of PB and MB patients, with reviews and skin-smear examinations, in the AMFES project. Surveillance follows the same pattern throughout the 5-year period after completion of MDT. During surveillance skin smears are taken from MB patients at every 6-monthly review and from PB patients only when clinical activity is observed at a review.

rifampicin once in 4 weeks, given under supervision on the day of drug collection, and 50 mg clofazimine + 100 mg dapsone for daily self-administration over 27 days. For children the dosages are adjusted. For MB patients the duration of the MDT course is fixed, limited to 2 years (26×4 weeks of drug supply within a period of up to 3 years), regardless of the BI found in skin smears, while routine leprosy services in Ethiopia follow WHO's 1982 recommendation[1,2] by which the MDT treatment of MB patients is continued until skin smear negativity. Time schedules of MDT for PB and MB patients are summarized in Figure 1.

Since 1990, blister–calendar packs have been in use. In areas where either the patient or the health personnel (or both) cannot reach the treatment point during the rainy season, 4–5 blister packs are given at one time to cover the period of inaccessibility. Thus, for about 30% of the patients, 3–4 of the 13 4-weekly doses per year were given unsupervised.

Drug collections are recorded in treatment registers kept at each clinic. Compliance with drug intake is assessed by urine testing for dapsone at drug collections 1, 3 and 6 for patients on the PB regimen, and at drug collections 1, 3, 7, 13, 19 and 25 for MB patients.

At every 4-weekly drug collection each patient is briefly examined by the clinic's health assistant, who tests for changes in muscle strength of eyelids, hands and feet by a standard set of voluntary muscle tests (VMT) and changes in the sensitivity of eyes, palms and soles by sensory testing (ST).

All patients are also periodically examined by the LCSs. For PB patients at the 6th treatment round, before the treatment course will be completed. MB patients are

reviewed at treatment rounds 7, 13, 19 and 25 or 26. At each of these review examinations (Figure 1), the LCS assesses the skin lesions, takes skin smears from 4 sites, does all routine nerve function tests and records the findings on the patient record card.

### CASE HOLDING

During treatment, case holding is promoted by individual health education on the importance of regular drug collection and faithful drug intake. When a patient is absent, our staff ask other patients about the absentee. If still absent at the next scheduled MDT clinic, action must be taken to get her/him back to treatment. If using other patients as messengers and motivators does not work, the defaulting patient should be visited at home by the health assistant of the clinic. Not all absent patients are visited at home. In some cases the patient's home is inaccessible to the health worker concerned. From the beginning of 1991 until July 1992, the feasibility of home visiting was further reduced, in several areas, due to poor security.

PB patients who have not taken the prescribed 6 doses of supervised treatment within 9 months (36 weeks), and MB patients who have not received 26 supervised doses of their regimen within a period of 3 years, are recorded as 'treatment not completed' (TnC), and excluded from the relapse study. For the incidence of reactions, neuritis or nerve dysfunction, these patients participate in the study for as long as they attend for treatment.

### SURVEILLANCE

A surveillance register is kept and patients are appointed for review at 3 and 6 months after completion of the MDT course and thereafter twice yearly at intervals of 6 months (periods of 6 or 7×4 weeks; Figure 1).

Case-holding action during this active surveillance is basically the same as during the treatment phase. The surveillance period for both MB and PB patients is 5 years.

### QUALITY CONTROL AND DATA MANAGEMENT

The MOs check the cards of all patients once every 4 weeks to supervise timely and accurate data collection, and then write reminder slips for the LCSs, indicating what needs be done next. They also make supervisory visits to clinics according to plans issued every 8 weeks.

Information collected on cards and forms is entered regularly in computerized data files, usually every 4 weeks and at least once in 3 months. From October 1991 onward, all data have been entered twice and compared, to minimize entry errors. The computer is also used for quality control on the LCS's records. Two stages of validation programs identify impossible or unlikely data and inconsistencies, so that recording errors can often be found and corrected before new data are appended to the master file.[4]

Statistical comparisons of numeric quantities have been made by calculating 95% confidence intervals (CIs) of differences between means. Thus a statistical difference exists when the 95% CI excludes zero.

**Table 1.** Enrolment of patients in the first 3 years of AMFES: Classification, sex and age distribution at intake

| Classification: | PB | | MB | | | | | | All | |
|---|---|---|---|---|---|---|---|---|---|---|
| | BT* | | BT | | BL† | | LL | | | |
| Sex: | M | F | M | F | M | F | M | F | M | F |
| Age group (yrs) | | | | | | | | | | |
| 0–14 | 10 | 12 | 1 | 1 | 7 | 4 | 5 | 1 | 13 | 6 |
| 15–29 | 25 | 25 | 1 | 0 | 32 | 6 | 16 | 7 | 9 | 13 |
| 30–49 | 13 | 10 | 1 | 3 | 25 | 13 | 7 | 4 | 33 | 20 |
| ≥50 | 19 | 14 | 2 | 0 | 9† | 6 | 7 | 0 | 18 | 6 |
| All | 67* | 61 | 5 | 4 | 73† | 29 | 35 | 12 | 113 | 45 |
| Total | 128* | | 9 | | 102† | | 47 | | 158 | |

* Including 2 TT (males aged 10 & 67 yrs) and 1 NL (male aged 28 yrs).
† Including 1 NL patient.

## Patients and results

We report on 286 patients, 128 PB and 158 MB, enrolled in the first 3 years of the project. Classification, gender and age distribution at intake, for treatment categories PB and MB, are given in Table 1.

The two most common classifications were BT and BL. Both TT and pure NL were rarely seen, in this series each in 2 patients only. For convenience of presentation, the 2 TT patients and the paucibacillary NL patient have in the tables been included in the BT group, the other NL patient in the BL group. For the PB patients the male:female ratio was 67:61 or 1·1:1. For the MB patients that ratio was 113:45, or 2·5:1. At registration, 41 patients (14·3%) were in the age group 0–14 years. The youngest was 4 years. The overall mean ages of PB and MB patients were not significantly different.

Changes of classification and/or treatment were recorded for 15 patients as shown in Table 2.

**Table 2.** Patients in the AMFES project whose initial classification and/or treatment category were changed

| No. of Patients | Classification | Treatment |
|---|---|---|
| 2 | TT→BL | PB→MB |
| 2 | BT | PB→MB |
| 3 | BT→BL | PB→MB |
| 4 | BL→BT | MB |
| 2 | LL→BL | MB |
| 1 | TT→BT | PB |
| 1 | BL→BT | MB→PB |
| 15 | 13 changes | 8 changes |

**Table 3.** Disability grade for AMFES patients at the start of MDT, by treatment, classification and age group. Percentages for each group are shown in parentheses

| | PB | MB | | | | Age groups | | | | Overall |
|---|---|---|---|---|---|---|---|---|---|---|
| | BT* | BT | BL† | LL | All | 0-14 | 15-29 | 30-49 | ≥50 | Total |
| Disability grade | | | | | | | | | | |
| 0 | 58 (45·3) | 2 (22·2) | 43 (42·2) | 24 (51·1) | 69 (43·7) | 28 (68·3) | 58 (51·8) | 28 (36·8) | 13 (22·8) | 127 (44·4) |
| 1 | 32 (25·0) | 5 (55·5) | 29 (28·4) | 17 (36·2) | 51 (32·3) | 4 (9·8) | 29 (25·9) | 21 (27·6) | 29 (50·9) | 83 (29·0) |
| 2 | 38 (29·7) | 2 (22·2) | 30† (29·4) | 6 (12·8) | 38 (24·1) | 9 (22·0) | 25 (22·3) | 27 (35·5) | 15 (26·3) | 76 (26·6) |
| Total | 128* | 9 | 102† | 47 | 158 | 41 | 112 | 76 | 57 | 286 |

* All BT patients except 2 TT (1 grade 0, 1 grade 1) and 1 NL (grade 0).
† Including one NL patient.

## DISABILITY GRADING

It is common practice in the statistics of both WHO and ILEP to grade a patient's overall disability by the highest grade recorded for anyone of that patient's eyes, hands or feet. Disability grades on this basis, at the start of MDT, are in Table 3. Overall disability grades did not differ significantly between PB and MB patients ($\chi^2 = 2{\cdot}2$, 2df). Although LL patients tended to have lower grades than other MB patients, this difference was also not significant ($\chi^2 = 4{\cdot}7$, 2df).

The differences between the four age groups (Table 3) in the proportions of disability grade 0 are highly significant ($\chi^2 = 24{\cdot}5$, 3df, $p < 0{\cdot}00002$): much larger proportions of disability grade 0 (patients without any functional impairment or deformity) were found in younger patients.

## OTHER SCORES FOR DISABILITY

The WHO disability grading[5] for patients, given in Table 3, though widely used in the patient statistics of leprosy control programmes, has the great disadvantage that it does not distinguish at all between vast differences in conditions of patients. A patient who has lost one small part of one finger is given the same grading 2 as another patient who had gone blind and lost most of both hands and feet.

The AMFES project aims at developing scoring systems and indicators for physical impairment and deformity, which should be more appropriate for monitoring and evaluation of disability prevention in leprosy control programmes. Without yet having made a definite choice for any of various possible scores, we present here the condition of patients at the time of enrolment expressed as the sum of the disability grades of 2 hands and 2 feet. As each hand and each foot can be graded as either 0, 1 or 2, the sum for the 4 extremities ranges from 0 to 8.

In our patient population it appeared to be justifiable to leave the disability grade for eyes out of this indicator. Only 6 of the 128 PB patients and 8 of the 158 MB patients,

**Table 4.** Disability as sum of the grades for four extremities (HF-impairment score) against disability grade per patient (highest of all 6 grades), for 286 AMFES patients

| HF-impairment score | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | Total |
|---|---|---|---|---|---|---|---|---|---|---|
| Disability grade | | | | | | | | | | |
| 0 | 127 | — | — | — | — | — | — | — | — | 127 |
| 1 | — | 18 | 36 | 17 | 12 | — | — | — | — | 83 |
| 2 | 1* | — | 11 | 17 | 12 | 14 | 9 | 6 | 6 | 76 |
| Total | 128 | 18 | 47 | 34 | 24 | 14 | 9 | 6 | 6 | 286 |

* This patient had a disability grade of 2 for 1 eye, with normal hands and feet.

about 5% in all (14/286), had some disability of 1 or 2 eyes (range of score: 1–4; mean = 2·1) and all but 1* of those 14 patients had already reached scores ranging from 2 to 8 (mean = 4·7) for the sum of disability grades for the 4 extremities. Using a scale from 0 to 12, with the eye condition included, would thus appear not to be of much advantage. Only 3 of the 286 patients would reach a score above 8; 2 of the 14 patients with eye disability would have their scores increased from 7 or 8 to 10 and 1 from 8 to 12.

In Table 4 this score for impairment of hands and feet (HF-impairment score) is shown against the conventional patient disability grade for all patients at the start of MDT. The table shows that of 83 patients with disability grade 1, 18 patients (21·7%) had only 1 extremity affected, 36 (43·4%) had sensory loss in 2 extremities, 17 (20·5%) in 3, and 12 patients (14·5%) had all 4 extremities affected. Further, that of 76 patients with disability grade 2, at least 12 (15·8%), with HF-impairment scores 7 or 8, had all 4 extremities affected. In tables for PB and MB patients separately (data not shown) these patterns were very similar. The mean score for patients with disability grade 2 (4·41) was significantly higher than for grade 1 (2·28) (95% CI for difference of means 1·7 to 2·6).

### BACTERIAL INDEX (BI)

As the BI for a patient at start of treatment, we record not the average but the highest BI found in any skin smears taken in the first 3 months of treatment. In the period before July 1989, we prescribed PB treatment regimens for 3 patients with classification BT and BI = 1. All other PB patients always had negative skin smears.

BIs at start for the 158 MB patients are shown in Table 5. The BI at time of diagnosis has an inverse relationship with the HF-impairment score. This is shown in Figure 2. The mean HF-impairment score of 158 MB patients was significantly inversely related to the BI (one-way analysis of variance, 6df, $p = 0{\cdot}0005$). Patients with a high BI at the start of treatment had on average much less physical impairment than those with lower BIs.

### TREATMENT COMPLETION AND LOSS TO FOLLOW UP

Of the 128 PB patients, 102 (79·7%) completed and 26 (20·3%) patients did not complete the MDT course. Among the latter, 12 were from 1 district (Jibat na Mecha) where 42·9% of 28 PB patients did not complete the course. This district was worst affected by

* This patient's eye condition was almost certainly not related to leprosy.

**Table 5.** Bacterial Index (BI) at start of MDT in 158 MB patients in the AMFES project, by classification

| | BT | BL | LL | Total |
|---|---|---|---|---|
| Highest BI | | | | |
| 0 | 5 | 7 | — | 12 |
| 1 | 2 | 6 | — | 8 |
| 2 | 2 | 19 | — | 21 |
| 3 | — | 19 | 1 | 20 |
| 4 | — | 29 | 3 | 32 |
| 5 | — | 15 | 20 | 35 |
| 6 | — | 7 | 23 | 30 |
| | 9 | 102 | 47 | 158 |

the internal war in 1990–91, and in some areas experienced a subsequent period of insecurity, which, we assume negatively influenced the local treatment completion rate.

The mean initial HF-impairment score for PB patients completing treatment was 1·8, compared with 2·1 for those not completing; this difference is not significant.

By the end of June 1992, 64 of the 158 patients on the MB treatment regimen had completed their MDT course, 18 patients had dropped out and 76 were still on treatment. The 18 patients lost were: 1 death, 1 transfer out to another district, 3 patients had left the area without a transfer, 10 patients had been declared out of control, of whom 4 had refused further treatment, and 3 patients had been so irregular that they did not reach an intake of 26 'doses' within 3 years and were recorded as 'TnC'.

Table 6 shows patient attendance rates for the 64 who had completed MDT by the end of June 1992. These figures represent a positive selection of regular patients. For the

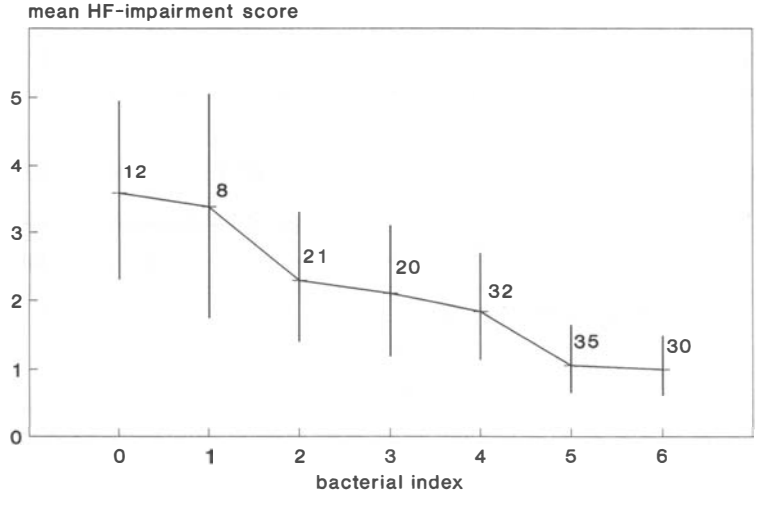


**Figure 2.** Relation between BI at start of MDT and mean initial disability score ('HF-impairment score') for 158 MB patients in the AMFES project.

**Table 6.** Treatment regularity of 64 MB patients in the AMFES project who completed MDT

| Regularity (%) | Attendance rate* at RFT | Patients |
|---|---|---|
| 100 | 26/26 | 33 |
| 81–99 | 26/27–32† | 25 |
| 66–80 | 26/33–35 | 5 |
| < 66 | 26/41‡ | 1 |
| | | 64 |

* Number of 4-weekly MDT collections/number of 4-week periods the MDT course has lasted.
† Including 7 patients who erroneously got 27, 28 or 29 doses.
‡ Exceptionally allowed beyond 39 periods of 4 weeks.

76 patients still on treatment, regularity of drug collection was assessed from recent computer entries of reviews and from data in treatment registers. For 4 there was no up-to-date information when we completed these assessments (31 October 1992). Regular, with an attendance of 80% or more, were 52/72 (72·2%) patients; for 10 the attendance was fair (60–79%), for another 10 poor to very poor.

From this analysis of the 76 patients still on treatment at the end of June 1992, we estimate that 60/76 (78·9%) are likely to complete their MDT course. If we add the other 16 patients to the 18 already reported as dropped out, the loss from the study during the treatment phase would come to 34 (21·5%) of the 158 MB patients. This is similar to the 26/128 (20·3%) for the PB patients.

### BI CHANGE DURING TREATMENT

For the 64 MB patients who completed the MDT course, Table 7 gives the highest BI of skin smears at the end of the MDT course, against the highest BI at start of MDT. Of 59 patients who started treatment with a BI≥2, 29 became negative; and for 57 patients (59–2 unknown), the mean of the highest BIs came down from 4·3 (sd 1·3) at the start to

**Table 7.** Highest BI in skin smears at start and at end of the MDT course, for 64 MB patients in the AMFES project

| BI at end | 0 | 1 | 2 | 3 | 4 | 5 | 6 | (Unk) | Total |
|---|---|---|---|---|---|---|---|---|---|
| BI at start | | | | | | | | | |
| 0 | 4 | – | – | – | – | – | – | – | 4 |
| 1 | 1 | – | – | – | – | – | – | – | 1 |
| 2 | 7 | – | 1 | – | – | – | – | – | 8 |
| 3 | 6 | – | 1 | 1 | – | – | – | (1) | 9 |
| 4 | 8 | 1 | 1 | 1 | 1 | – | – | – | 12 |
| 5 | 8 | 1 | 3 | 2 | 3 | 1 | – | – | 18 |
| 6 | – | – | – | 3 | 3 | 4 | 1 | (1) | 12 |
| | 34 | 2 | 6 | 7 | 7 | 5 | 1 | (2) | 64 |

**Table 8.** Coverage with reviews during surveillance of 91 PB patients in the AMFES project who completed MDT before 30 June 1991

| Coverage | Reviews done as % of required number | Patients |
|---|---|---|
| Nil | None | 14 |
| Very poor | < 30 | 7 |
| Poor | 30–55 | 14 |
| Fair | 56–75 | 25 |
| Good | 76–90 | 11 |
| Very good | ≥91 | 20 |
| Total | | 91 |

1·6 (sd 1·9) at the end of treatment; this difference is highly significant (95% CI 2·1 to 3·3). On average the highest BIs of these patients thus came down 2·7 log points over the notional 2 years of treatment. Of 5 patients with BIs at start ranging from 2 to 6, the highest BI did not come down during treatment.

### SURVEILLANCE

Of the 102 PB patients who completed MDT and were put on surveillance, assessments of compliance for 91 patients who had been on surveillance for 1 year or more are shown in Table 8. For 14 patients (15·4%) we did not get a single review after RFT. These patients are virtually lost although there is a small chance of future contact. Of the 21 (7 + 14) patients whose compliance was scored as very poor or poor, 4 had been seen within 12 months of 30 June 1992, so these patients need not yet be considered as lost.

For the 64 MB patients who completed treatment, the period of follow up had been too short to report on their surveillance.

## Discussion

Three aspects of this study population and our preliminary results deserve to be discussed: patient classification and treatment allocation; disability or grades of physical impairment; and some early observations on the outcome of treatment.

### PATIENT CLASSIFICATION AND TREATMENT ALLOCATION

In this study much attention is given to the distinction between BT and BL and to a proper choice between the PB and MB treatment regimens. However, in a leprosy control service, arbitrary decisions on the classification of patients are unavoidable at times, given the limitations of both the clinical and laboratory (skin smears) assessment.[6] As many of our patients were diagnosed rather late, after several years of active disease, some of the BL patients with negative skin smears might have gone, earlier in their

disease, through a phase* with more BB–BL characteristics and skin-smear positivity. Of the 7 BL patients with BI = 0 reported in Table 5, 1 is a case of neural leprosy; of the 6 others we did not manage to get repeat skin smears or skin biopsies. Of 3 of them one might doubt—in retrospect, on the basis of recorded clinical characteristics—whether these were not BT cases. The other 3 were most probably true BL patients.

As this study is carried out in an existing leprosy control programme, our patient classification has been influenced by the current practice. In ALERT's leprosy control services 673 new BL patients were registered during the same period. Of the 615 patients for whom skin-smear results were known, as many as 161 (26% !) new BL patients had BI = 0. This may reflect shortcomings of the skin-smear service, but amongst these patients are also many with advanced disease of several years' duration, who might have gone through an earlier phase† of BB–BL characteristics, but were at the time of diagnosis more BT like in appearance. Although we do not get positive skin smears from these patients, the sheer extensiveness of their disease, usually with more than 20 skin lesions and many nerve trunks affected, makes them eligible for multibacillary treatment. In ALERT's routine services[3] such smear negative BT/BL? patients who receive MB treatment are, for the sake of convenience, reported as classified BL. In our study, with only 6 smear negative cases amongst 101 BL patients, we thus give evidence of strict attempts at more accurate classification, but the figures also indicate that this problem has not been resolved fully.

Having to change the classification and/or treatment category for some of the patients is also unavoidable when standard programme procedure requires that a clinical diagnosis is made and anti-leprosy treatment started on the day the patient is seen for the first time. In most leprosy control programmes and also in ALERT's routine programme the frequency of such changes is not reported upon. In our study population we had to make such corrections (Table 2) for only 2 (1·6%) of 128 PB patients and for 13 (8·2%) of 158 MB patients. For the purpose of the relapse incidence study it is reassuring that most of the changes in treatment (7 out of 8) were from the PB to the MB regimen. In case of doubt we prefer to prescribe PB treatment, because a change from PB to MB means that the resulting 2-year treatment regimen does not differ much from the standard, whereas if a patient whose definite regimen is PB did get some months of MB treatment, this would make a great difference to the composition of that PB patient's 6-month treatment course. Of the 4 patients who were changed from BL to BT, but kept on MB treatment, 3 had more than 20 skin lesions and all had some characteristics suggestive of BL leprosy and signs of extensive involvement of nerve trunks, but repeated skin-smear examinations had remained negative. For 2 of them skin biopsies had been taken and histologically these were considered BT patients with a BI = 1. All 7 patients shifted from PB to MB treatment had all been found skin-smear positive within the first few months of treatment; 5 were reclassified as BL, 2 were BT patients with positive smears.

In Ethiopia, MB proportions are much higher than elsewhere in Africa.[7,8] In our study population, 55% of the patients were multibacillary. In 2 of the 5 former districts (*awrajas*) where the AMFES project is carried out, we registered less MB than PB patients—an MB proportion of 43·2%—while in the three other districts the MB proportion of patients was 57·3%. These latter 3 districts belong to the 5 with the highest

* We assume that, during several years of disease, before our diagnosis and without specific treatment, these patients' classifications have shifted, through upgrading reversal reactions, from BB (or BL?) to a BT position.

† See previous footnote.

MB proportions of the 12 districts of ALERT's leprosy control programme.[9] We are inclined to consider the observed differences as real, and not to be due to systematic differences in our staff's judgement regarding classification or allocation of treatment category. The possible epidemiological relevance of these findings deserves further investigation.

### GRADING OF DISABILITY OR PHYSICAL IMPAIRMENT

Both in the PB and MB cases—with no significant differences—the disability grades at the time of enrolment (Table 3) were disturbingly high. The fact that 55·6% of our patients either had a disability grade 1 (29·0%) or grade 2 (26·6%) is a very distinct feature of this study population, by which our patients differ greatly from others described in a similar study in Malaŵi[10] and in reports on patients in Asia.[11–13] The paucibacillary patients of the study in Malaŵi, both in the routine LCP services and in the Karonga District with frequent active case-finding surveys, were on average detected in a much earlier stage of the disease. Of their 503 PB patients, 272 (54·1%) had at diagnosis a single skin lesion, against only 2 of our 127 PB patients (1·6%), and 305 (60·6%) of their 503 patients had no palpably enlarged nerves, compared with only 33/128 (25·8%) in our PB patients.

For our study on the incidence of leprosy reaction and neuritis,[14] we thus deal with a study population which at the time of diagnosis and start of MDT obviously had already gone through several spells of such events.

Grading the disability of a patient by giving that patient the highest of the 6 grades of 2 eyes, 2 hands and 2 feet, has been commented upon extensively by several authors[15–17] as far too crude a way of characterizing the condition of a patient. The use of a disability index as proposed in 1971 by Bechelli & Dominguez,[18] would partly solve that problem. The indices they proposed—unfortunately presenting options for three different formulas: DI(1), DI(2) and DI(3), with different levels of complexity—have never been widely used.

Brandsma *et al.*[16] have recently pointed out which important distinctions should be made when speaking about 'disability' and that much of what we term disability should rather be referred to as physical impairment. The WHO-recommended 'disability grade' commonly used for leprosy patients indicates in fact the presence of physical impairment. The more informative sum score of the disability grades of 4 extremities proposed here (Table 4) is, therefore, more correctly termed a 'patient score of physical impairment for hands and feet', shortened to 'HF-impairment score'.

With the relative success of the present antibacterial therapy and less fear of problems with relapse after MDT, prevention of disability is now drawing much more attention than 5–10 years ago.[19] Accurate scoring of physical impairment thus becomes more needed. For a detailed monitoring of a patient's condition over time, e.g. for the evaluation of neuritis treatment, we consider the HF-impairment score, though much more informative than the conventional disability grading, still not sensitive enough.

It is one of the objectives of the AMFES project to develop a more appropriate scoring system of nerve function as indicator of physical impairment and disability, for use in leprosy control programmes. The challenge is to develop a score that is sensitive enough to distinguish relevant improvement or deterioration but simple enough to be used by paramedical field staff.

OUTCOME OF TREATMENT

Regarding the effectiveness of the MDT regimens against *Mycobacterium leprae*, the study on incidence of relapse will need many more years of follow up. So far no relapse has been observed.

The fall in bacterial index of skin smears of MB patients (Table 7)—for this group more than one log point per year of treatment—deserves to be analysed further; also in relation to classification. This will be reported upon when more data have been collected. With 28 (45·2%) of 62 patients still skin-smear positive at the end of the 2-year MDT course, of whom 13 (21·0%) still had a BI of 4 or more, further monitoring of the fall of the BI during surveillance will be an obvious feature of the relapse study. Eventually this should provide data to compare the fall of BI in skin smears for the periods during and after treatment.

At this stage of this long-term study, an important outcome of treatment to reflect upon is loss to follow-up. So far, the loss of patients during the treatment phase has amounted to approximately 20%, with no difference between PB and MB patients. For the short treatment period of the PB patients, the MDT completion rate of 79·7% in our series is disappointingly low. In ALERT's routine programme the average treatment completion rate of 91% for PB patients over the first 6 years (1983–88) of experience with MDT, is much better. However, for the years 1989–91, the routine programme also saw a considerable deterioration, with the average completion rate coming down to 82%.[9] We assume that this deterioration was mainly due to general factors not particularly related to the leprosy control services, i.e. the economic and security situation in the country.

The 20% loss of patients during treatment and the not too good coverage of patients during surveillance so far, does not make this an ideal setting for a longitudinal study on incidence of leprosy reactions, neuritis and relapse. This evaluation project has the disadvantages of a long-term study carried out in the setting of a routine service programme. On top of that we have had to cope with specifically difficult circumstances caused by the recent internal war and its related disturbances. For evidence that, despite such adverse conditions, determined field staff are able to collect interesting data on what happens to patients over several years, the reader is referred to Part II.

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# Field evaluation of WHO–MDT of fixed duration, at ALERT, Ethiopia: the AMFES project—II. Reaction and neuritis during and after MDT in PB and MB leprosy patients

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*Summary* For a cohort of 286 leprosy patients the incidence rates and clinical manifestations of leprosy reactions during treatment and surveillance are described. Currently, individual patients had been observed for up to 4 years. It is intended that surveillance within this project should continue for up to 5 years after treatment. Of 128 PB patients, observed for 267 person-years (mean 2·1) 27 had 35 episodes of reaction, corresponding to an overall incidence rate of 131 events per 1000 person-years-at-risk (pyar).

Of 158 MB patients observed for 402 person years (mean 2·5), 64 had 114 reactions, with an overall incidence of 284 events per 1000 pyar. For both PB and MB patients, incidence rates during treatment and post-MDT surveillance were similar. For PB patients, pre-existing physical impairment at the start of MDT was a significant risk factor for the occurrence of subsequent events, but this was not found in MB patients.

## Introduction

The purpose and design of ALERT's MDT Field Evaluation Studies (the AMFES project) have been described in Part I, giving data on enrolment, completion of treatment and case holding for 286 new leprosy patients observed for 15–51 months, during and after the MDT course. In this second part we give our findings regarding the occurrence of leprosy reactions, neuritis and nerve dysfunction in this cohort of patients during the 4·25 year period from 1 April 1988 to 30 June 1992.

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## Methods

At every 4-weekly drug collection, each patient is briefly examined by the clinic's health assistant (HA), who asks for any complaints, particularly regarding eyes, hands or feet, and inspects hands and feet for wounds, tests for changes in muscle strength of eyelids, hands and feet by a standard set of voluntary muscle tests (VMT) and in the sensory capacity of eyes, palms and soles. Sensory testing (ST) of palms and soles was, until July 1989, done with the tip of a ballpoint pen; thereafter a nylon filament applying a much more standardized pressure stimulus of 10 g was used at a standard series of test points. For the eyes, normal blinking was taken as circumstantial evidence of normal sensation. Only in the absence of normal eye blinking was the sensation of the cornea tested by touching the limbus with a fine wisp of cottonwool.

The HA also palpates 4 peripheral nerve trunks (of the ulnar, median, lateral popliteal and tibialis posterior nerves), on both sides, for tenderness. All findings of this examination are recorded on the individual 'Patient Routine Care Form', which is kept by the health assistant. The procedures of this 'VMT and ST', and its recording, done at every 4-weekly treatment session, are exactly the same in this study as in the routine leprosy control programme. In case of any complaint which may point towards a leprosy reaction, and for each finding of nerve tenderness, or of any deterioration in nerve function, the health assistant has to present the patient to the leprosy control supervisor (LCS) for assesment of a possible 'event' (see below).

During treatment, all patients are also periodically examined by the LCS. For PB patients this is done at the 6th treatment round, before the patient is reminded that next month the treatment course will be completed. MB patients have a full review by the LCS at treatment rounds 7, 13, 19 and 25 or 26. At each of these review examinations, the LCS assesses the skin lesions, takes skin smears from 4 sites, does all routine nerve function assessments and records the findings on the Patient Record Card.

During the 5-year surveillance, periodical reviews are done by the LCS at 3 months, 6 months, and thereafter every 6 months. Patients are, however, also explicitly advised to come at any time when they notice a problem of reaction or nerve function loss. Case-holding procedures during treatment and during surveillance, and results achieved, have been given in Part I, together with methodological details.[1]

### EVENTS

In AMFES, an event is defined as noticeable signs or symptoms associated with leprosy reaction, new nerve dysfunction or a relapse of the disease.

In this field service-based study, we distinguish between only 2 types of leprosy reactions: reversal reaction (RR) or type 1 reaction, occurring mainly in BT and BL patients and associated with cell-mediated hypersensitivity. This category may include reactions which some leprologists may prefer to call 'downgrading reactions', *erythema nodosum leprosum* (ENL), or type 2 reaction, occurring in LL and BL patients and associated with humoral (antibody mediated) responses to antigens of *Mycobacterium leprae*, with immune complexes.

A further distinction is made between mild and severe forms for both RR and ENL reactions. Criteria for assessment of reactions are the same as those used in our routine leprosy control programme, given in ALERT's Manual[2] and summarized in Figure 1.

SIGNS OF REACTION

*Signs of reversal reactions, skin component only, 'RR'*:
Mild skin involvement
—skin lesions become red and raised
—appearance of new lesions, and/or increase in size of pre-existing lesions
—paraesthesia or hypersensitivity in pre-existing skin lesions

Severe skin involvement
—skin lesions become red, raised, very tender and ulcerate
—skin reaction in a patch overlying a major nerve trunk or the eye (here the skin involvement as such may not be severe, but in the instructions for treatment it is grouped with 'severe' because of the high risk of nerve involvement
—marked oedema of hands or feet

*Signs of* erythema nodosum leprosum '*ENL*':
Mild ENL reaction
—appearance in the skin of red and painful nodules which may disappear after a few days, while other similar nodules come up; without ulceration of skin
—nerve involvement: thickening of nerves and/or paraesthesia, without tenderness on palpation or loss of function: mild neuritis
—mild fever and/or malaise

Severe ENL reaction
—appearance of ENL nodules with ulceration

or

—ENL skin lesions with one or more of the following:
- loss of muscle strength and/or loss of sensation in hands, feet or eyes (reduction in VMT and/or ST): severe neuritis;
- painful eyes, with redness around the limbus cornea, increased lacrimation, constriction of the pupil and diminished vision (irido-cyclitis);
- enlarged tender lymphnodes (lymphadenitis)
- painful testicular swelling (orchitis)
- painful swollen joints (arthritis)
- painful swollen fingers (dactylitis); and
- general condition: severe fever and malaise.

**Figure 1.** Signs of reaction (adapted from ALERT's *Manual for Field Treatment of Leprosy Reactions*[2]).

New or increased nerve dysfunction is, for the sake of convenience, hereafter referred to as neuritis, although in some cases, which we may not be able to distinguish, deterioration of function may possibly be due to (post-reaction) scar formation in the nerves. Signs of neuritis and criteria for mild and severe neuritis are summarized in Figure 2.

## CLINICAL MANIFESTATIONS OF REACTION

To facilitate a descriptive monitoring of what is seen in events, we also distinguish between the following elements of reactions:

where apparently no nerve involvement occurred; these are termed either: 'RR' for RR with skin reaction only, or 'ENL' for ENL reaction without neuritis;
conditions where nerve tenderness and/or nerve dysfunction are found without signs of reaction in the skin, termed: 'neuritis' or 'neuritis alone'; and
combinations: 'RR-with-neuritis', 'ENL-with-neuritis' and '(RR + ENL)-with-neuritis'.

The terms 'RR' and 'ENL' are normally used for the whole complex of the reactional process concerned, encompassing the pathology of skin, nerves and other organs. In this

SIGNS OF NEURITIS

New (i.e., in existence for less than 6 months) nerve involvement in a leprosy patient is recorded as a case of:
*Mild neuritis*
if only
— new or increased thickening of nerves
and/or
— paraesthesia, 'tingling'
is found, without pain or tenderness and without loss of function,

and as

*Severe neuritis*
if one or more of the following signs is found:
—*tenderness* of one (or more) nerve(s), with or without loss of nerve function
—*new* (or unquestionable *increase* of) *sensory loss* in the skin of palms and/or soles, i.e.:
2 or more new points of no response out of
4 points tested for the ulnar nerve
6 points tested for the median nerve
10 points tested for the tibial posterior nerve
by a standardised Sensory Test 'ST' carried out with a nylon filament producing a stimulus of 10 g.

— *new* or *increased muscle weakness*: a decrease of muscle strength observed in a simplified 'Voluntary Muscle strength Test' (VMT), which distinguishes three grades only: S (for strong), W (for weak) and P (for paralysed). The VMT is done for the following nerves and functions:
facial nerve—tight closure of eye lids
ulnar nerve—abduction of 5th finger
median nerve—abduction of thumb
peroneal nerve—dorsiflexion of foot
radial nerve—dorsiflexion of hand

**Figure 2.** Signs of neuritis (adapted from ALERT's *Manual for Field Treatment of Leprosy Reactions*[2]).

field study we wish to describe the reactional phenomena in some detail. We therefore categorized the reactions, by clinical criteria (listed in Figures 1 and 2), into those where apparently only the skin was affected, others where only the nerves seemed to be affected and those with both skin and nerve involvement.

When an event is noticed, the LCS does a full review examination, scores the presence or absence of relevant symptoms and signs on the 'Prednisolone Treatment & Report of Event Form' (PTREF), followed by a summary entry, just as for a periodical review, on the Patient Record Card.

At the monthly review of the cards (or, if he happens to be at the clinic, immediately on the spot) one of the MOs categorizes the event and indicates the presence of RR, ENL, or neuritis or combinations of these three categories on the PTREF.

## TREATMENT OF REACTION

In most events prednisolone is prescribed for the patient. This treatment was for study patients the same as for ALERT's routine services.[3] MB patients receive a prednisolone course of 20 weeks. This course starts with 40 mg daily for 2 weeks. The daily dose is then reduced to 30 mg, given for 4 weeks and then tapered down every 4 weeks, via 20 mg, 15 mg, 10 mg, each for 4 weeks, and finally 5 mg daily for the last 2 weeks. For PB patients the dosages of the prednisolone regimen are the same, but the daily dose is reduced every 2 weeks and the course thus lasts 12 weeks only. For pregnant women and for children shorter courses and lower dosages are prescribed.

In case of no response or of a worsening of the reaction or the neuritis, the LCS is to increase the dose. The prednisolone course is then prolonged and may last some 6–12 or more weeks longer. ALERT's Manual[2] details which conditions LCSs are allowed to handle themselves and in which conditions patients have to be referred to the hospital.

At the end of a corticosteroid course another full review is done and findings are recorded both on the PTREF and on the Patient Record Card. The data reported here are obtained from these two records.

### ANALYSIS

Incidence rates for events have been calculated using a person-years-at-risk (pyar) approach. Since the examination at the start of MDT includes detailed enquiries into leprosy-associated findings during the preceding 6-month period, and reaction or neuritis of up to 6-months' duration is still counted and treated as an event, all patients were assumed to have been under 'observation' for this period. There followed a period of MDT, and, after appropriate completion of MDT, a period of surveillance. Patients who had had a 6-monthly review within 6 months of the time limit of this analysis (30 June 1992) were assumed to be on treatment or surveillance up to this date. Patients who had no review after the end of 1991 were assumed to have been observed for 3 more months after their last review.

Statistical comparisons of numeric quantities have been made calculating 95% confidence intervals (CIs) of differences between means. Thus a statistical difference exists when the 95% CI excludes zero.

## Patients

We report on 286 patients, 128 PB and 158 MB, enrolled in the first 3 years of the project, from April 1988 onwards. Procedures and results of patient enrolment, completion of MDT treatment and case holding were described in Part I.[1] Observations up to the end of June 1992 covered 267·0 person-years among PB patients (mean 2·1 years per patient). Similarly for MB patients, observations covered 402·1 person-years (mean 2·5 years).

## Results

### EVENTS IN PB PATIENTS

Of the 128 PB patients, 27 (21·1%) experienced a total of 35 events. All events occurred in patients classified as BT. This corresponds to an incidence rate of 131 per 1000 (pyar). The 35 events were distributed among the 128 patients with 101 patients having no event, 23 patients having 1 event, 2 having 2, 1 having 3 and 1 patient having 5 events. This differed significantly from a random (Poisson) distribution ($\chi^2 = 667$, 5 df). Of the first observed reaction for each patient, 14 out of 27 (51·9%) showed RR with neuritis, 11 (40·7%) neuritis alone, and 2 had RR alone.

The clinical manifestations and the chronology of events in relation to treatment and surveillance are shown in Figure 3. Neuritis was present in 33 of the 35 events. Two events were observed with RR skin reaction alone, i.e. with, by our method of detection,

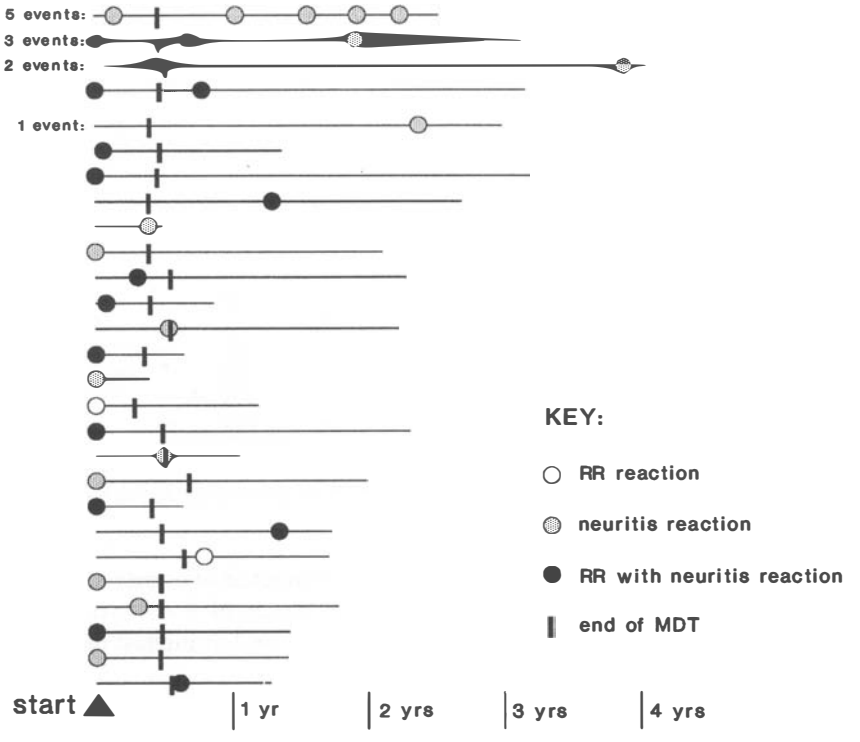


**Figure 3.** Clinical manifestations and chronology of 35 events observed in 27 out of 128 PB patients in the AMFES project, up to the end of June 1992. Patients with an 'end of MDT' mark less than 6 months from the start of MDT underwent an early end-of-treatment assessment, followed by unsupervised completion of MDT, for reasons of inaccessibility during the rainy season.

no evidence of neuritis. In 15 of the 17 events with neuritis alone, the neuritis was 'silent': the new or increased nerve dysfunction occurred not only without accompanying signs of reaction in the skin, but also without any pain or nerve tenderness.[4,5]

The 3 patients with 2 or 3 events had RR-with-neuritis in their first reactions, but 2 had neuritis alone in later events. The woman in whom 5 events were observed had neuritis alone on each occasion. She started with spells of silent neuritis, but by the 5th event she had several tender nerve trunks.

In 13 (48·1%) of the 27 patients who had events, a reaction was found at the time of diagnosis and start of MDT. Another 9 (33·3%) had an event during the treatment course and 5 patients had their first event after MDT. A total of 13 (37·1%) of the 35 events were observed after MDT, over 139·5 pyar during surveillance. Thus the incidence of events during surveillance after MDT was 93 per 1000 pyar. Of these, 4 reactions occurred in the first 6 months after MDT, 3 in the second half year, 3 in the third, 2 in the fourth half year, and 1 in the fourth year after MDT (and this was apparently* not a relapse). Separate incidence rates of events, before and during MDT, and for 2 periods after MDT are shown in Figure 4.

* Only some of the previously existing skin lesions had become red and raised again. On a course of prednisolone, without any antileprosy treatment, these lesions subsided nicely. The patient remains under surveillance.

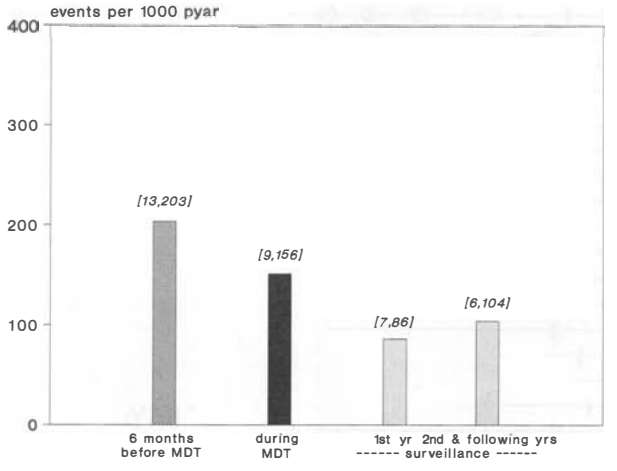


**Figure 4.** Incidence of 35 events in 128 PB patients in the AMFES project before, during and after treatment.

The relation between disability at start and the occurrence of events in PB patients is shown in Table 1. The 58 patients with a disability grade of 0 at start of MDT had significantly less events than the 70 patients with either 1 or 2 as highest disability grade. ($\chi^2 = 6{\cdot}23$ 1 df, $p = 0{\cdot}013$).

Also the mean score of HF-impairment (the sum of the disability grades of a patient's 4 extremities)[1] at the start of MDT among the 16 patients who subsequently experienced events was 2·63 (sd 2·47), compared with the remaining 112 patients for whom that mean score was 1·75 (sd 2·22) (95% CI of difference −0·3 to 2·1, not significant). However, PB patients who already had some impairment at the start of MDT were 2·5 times more likely to experience further events than those without existing impairments (relative risk 2·5, 95% CI 0·9 to 7·3).

For an assessment of changes in the HF-impairment scores of patients over time we have compared scores at start of MDT with latest recorded scores, calculating the change in score for each patient. Of 106 PB patients for whom valid scores were

**Table 1.** Occurrence of events in 27 patients who experienced 1 or more events, out of 128 PB patients in the AMFES project, in relation to their disability grades at the start of MDT

| | All | Patients with event(s) | % |
|---|---|---|---|
| Disability grade | | | |
| 0 | 58 | 6 | 10·3 |
| 1 | 32 | 8 | 25·0 |
| 2 | 38 | 13 | 34·2 |
| | 128 | 27 | 21·1 |

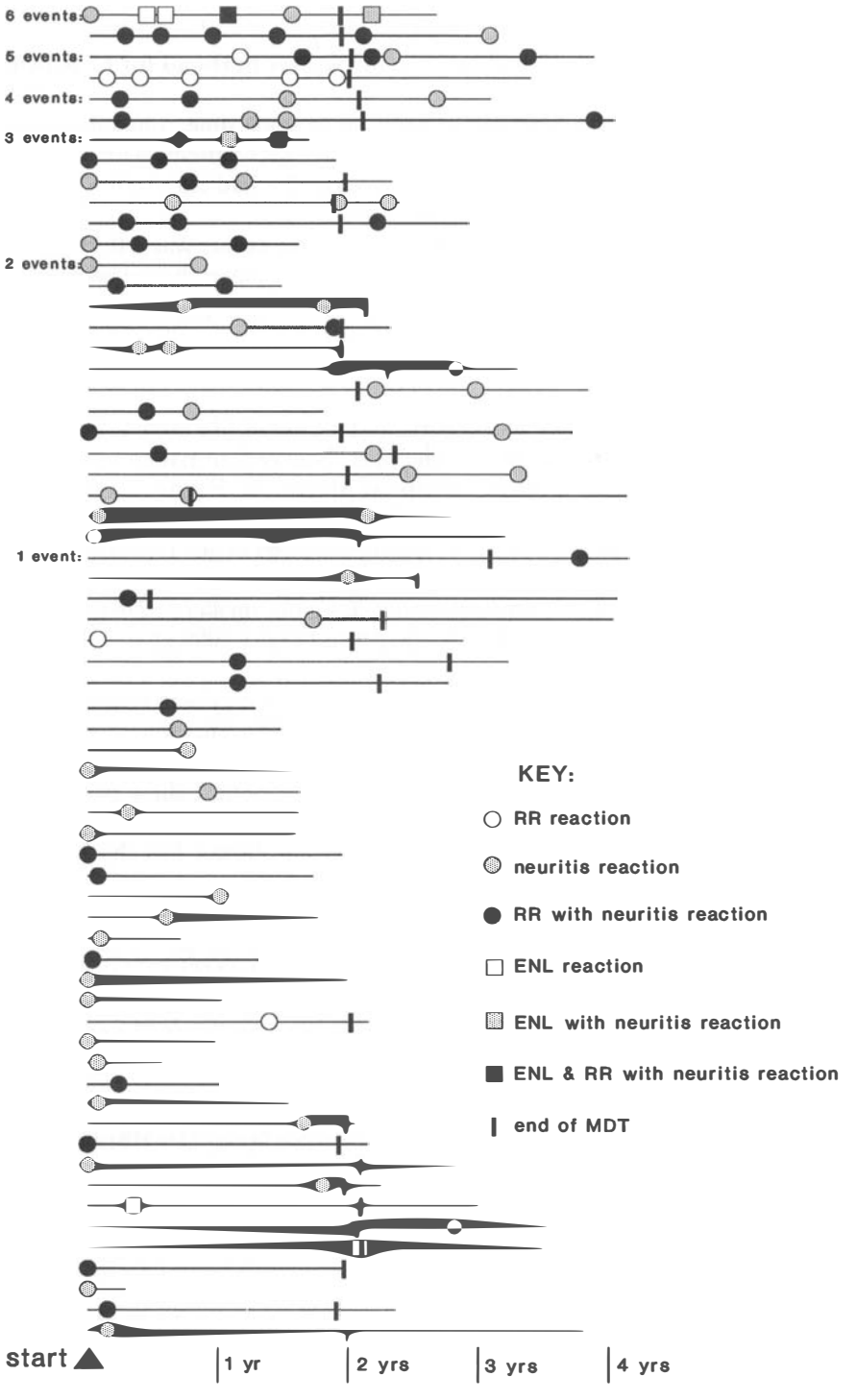


**Figure 5.** Clinical manifestations and chronology of 114 events observed in 64 out of 158 MB patients in the AMFES project, up to the end of June 1992. The 2 cases where 'end of MDT' occurred less than 1 year after the start concerned two skin-smear negative BT patients who were taken off MB-MDT early.

available, 27 had improved, 12 had become worse over time and for 67 patients initial and final scores were the same.

Among 26 patients who experienced an event at some time, 5 had improved, 9 had become worse and 12 ended with no change in score (1/27 patients with an event had no final score). Among 80 patients with both scores who had no event, 22 improved, 3 became worse and 55 ended with the same score.

The mean change in scores for the 26 patients with events was −0·31 (sd 1·12), and for the 80 without events 0·49 (sd 1·11). The difference in mean change between these two groups was 0·80 (95% CI of difference 0·3 to 1·3) which is significant.

### EVENTS IN MB PATIENTS

Of the 158 MB patients, 64 (40·5%) experienced a total of 114 events. This corresponds to an overall incidence rate of 284 per 1000 pyar. Ninety-four patients had no event, 38 had 1 event, and 26 had multiple events as shown in Figure 5. This also differs significantly from a Poisson distribution ($\chi^2$ = 296·7, 6 df).

The majority of events (82/114, 71·9%) were observed in (46/101) BL patients during 248·3 pyar, corresponding to an incidence for BL patients of 330 events per 1000 pyar. Six events occurred in (4/9) BT patients during 26·6 pyar, incidence 226 per 1000 pyar, 24 in (13/47) LL patients during 124·4 pyar, incidence 193 per 1000 pyar, and two events in the single NL patient.

The clinical manifestations and the chronology of all events in relation to treatment and surveillance are shown in Figure 5. Sixteen of the 114 events were present at the time of diagnosis and start of MDT, 79 events occurred during treatment, and so far 19 events have been seen during surveillance following MDT. Amongst the 6 patients with 4 or more events was one (male BL patient) for whom all events consisted of a pronounced RR skin reaction without any evidence of any nerve problems. For the 5 other patients the manifestations of reaction differed over time.

The clinical manifestations of the first events of MB patients are listed in Table 2. Neuritis was also the most common element of reaction in the MB patients. In these first events it was seen in 56 (87·5%) of the 64 patients, and in 33/64 (51·6%) there were not any signs of reaction in the skin. Of the events consisting of neuritis alone, 35/53 (66·0%) were cases of 'silent neuritis' in which there was no tenderness in any of the palpated nerves.

**Table 2.** Clinical manifestations of first reactions among 64 out of 158 MB patients in the AMFES project who experienced 1 or more reactions

| Components of reaction: | RR | Neuritis | ENL | Total events |
|---|---|---|---|---|
| *Reaction type* | | | | |
| RR (skin alone) | 6 | — | — | 6 |
| Neuritis alone | — | 33 | — | 33 |
| ENL (skin alone) | — | — | 2 | 2 |
| RR-with-neuritis | 23 | 23 | — | 23 |
| Total events | 29 | 56 | 2 | 64 |

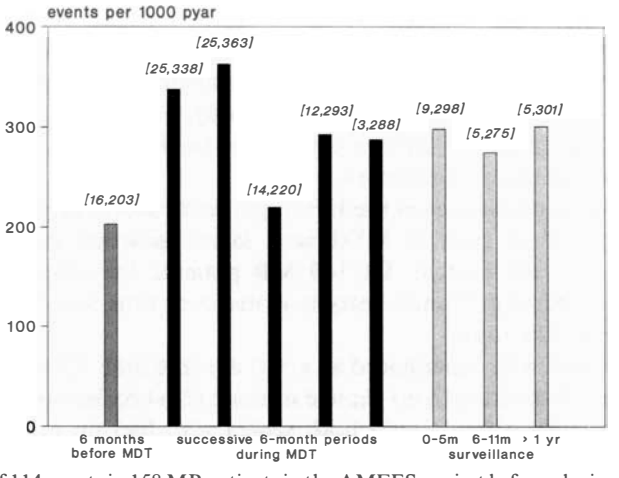


**Figure 6.** Incidence of 114 events in 158 MB patients in the AMFES project before, during and after treatment.

Of the 64 patients with events, 16 (25·0%) already had their first event at the start of treatment, 32 (50·0%) experienced events in the 1st year of treatment, 12 (18·8%) in the 2nd or 3rd year of treatment and 4 patients (6·3%) had their first event during surveillance (Figure 5).

During the first year of treatment, 50 events occurred during 142·9 pyar, giving an incidence of 350 per 1000 pyar. The 29 events recorded during treatment after the first year related to 115·1 pyar, an incidence of 252 per 1000 pyar. The 19 events during surveillance were recorded in the 64 patients who had so far completed MDT, over 65·0 pyar in surveillance. This corresponds to an incidence rate of 292 per 1000 pyar. In Figure 6 incidence rates are given per period of 6 months from before treatment until 1 year after MDT, and thereafter in 1 bar for patients who were on surveillance for more than 1 year.

The relation between disability at start and the occurrence of events in MB patients is shown in Table 3. The 68 patients with a disability grade of 0 at start of MDT had a similar occurrence of events to the 90 patients with either 1 or 2 as highest disability grade.

**Table 3.** Occurrence of events in 64 patients who experienced 1 or more events, out of 158 MB patients in the AMFES project, in relation to their disability grades at the start of MDT

| | All | Patients with event(s) | % |
|---|---|---|---|
| Disability grade | | | |
| 0 | 68 | 26 | 38·2 |
| 1 | 52 | 28 | 53·8 |
| 2 | 38 | 10 | 26·3 |
| | 158 | 64 | 40·5 |

Also the scores of HF-impairment at the start of MDT of the 54 patients who subsequently experienced events (mean of scores 1·50, sd 1·6), were not significantly different from the scores of the remaining 104 patients (mean of scores 1·97, sd 2·2) (95% CI of difference −0·2 to 1·1). MB patients who already had some impairment at the start of MDT were thus equally likely to experience further events as patients starting treatment without existing impairments.

For an assessment of changes in the HF-impairment scores of patients over time, we have compared scores at start of MDT with latest recorded scores, calculating the change in score for each patient. Of 140 MB patients for whom valid scores were available, 35 had improved, 25 had become worse over time and for 80 patients initial and final scores were the same.

Among 62 patients who experienced an event at some time, 15 had improved, 23 had become worse and 24 ended with no change in score (2/64 patients with an event had no final score). Among 78 patients with both scores who had no event, 20 improved, 2 became worse and 56 ended with the same score.

The mean change in scores for the 62 patients with events was −0·15 (sd 1·47), and for the 78 without events 0·47 (sd 0·95). The difference in mean change between these two groups was 0·62 (95% CI of difference 0·2 to 1·0) which is significant.

## Discussion

In Hastings' handbook[4] leprosy reaction is described as 'the appearance of symptoms and signs of acute inflammation in lesions of a patient with leprosy. Clinically there is redness, swelling and sometimes tenderness of skin lesions, and swelling, pain and tenderness of nerves, often accompanied by loss of function. New lesions may appear'. Thus it is not made unequivocally clear that a leprosy reaction may appear as an episode of neuritis alone, without clinical signs of reaction in the skin. Furthermore, the quoted description seems to exclude cases of silent neuritis.[5,6] From a further description in the same chapter, and also elsewhere in the literature[7,8] it is evident that neuritis alone, including silent neuritis, is presently recognized as a possible manifestation of a leprosy reaction. This evident lack of a succinct and comprehensive definition of a leprosy reaction remains a problem.

In this field study project we have recorded and treated new or increased nerve dysfunction as 'neuritis', although we are aware that in some cases the loss of nerve function may be due either to postreaction scar formation[9,10] in a nerve or, particularly in untreated progressive disease, to pathological processes other than those associated with RR or ENL reaction.[7]

There have been very few studies of leprosy reactions on a longitudinal basis. In the only other prospective study in Africa we are aware of, in Malawi,[11] PB patients starting WHO–MDT at a much earlier stage of disease (many of them detected in active case finding) experienced much less reversal reaction. At the start of treatment, 11 of 503 patients (2·2%) had a reaction compared to 13/128 (10·2%) in AMFES. During treatment 7 reactions among 488 Malawi patients gave an incidence rate of 28/1000 pyar, compared to an incidence of 156 events per 1000 pyar in this AMFES group. In the first year of surveillance of PB patients, the Malawi study observed 15 reactions among 314 patients (who had been self-reporting for start of MDT) giving an incidence rate of

48/1000 pyar (AMFES: 88/1000 pyar). In further follow up for 3 more years involving 471 patients, only 2 reactions were seen (no incidence rate given)[12] and among our 102 patients we have observed already 6 reactions (incidence rate 99/1000) in years 2, 3 and 4 of surveillance.

Whilst for all periods the incidence of reaction is higher among the Ethiopian patients, the findings in both studies indicate that for these PB patients the incidence of reaction during the first year of surveillance is not very different from the incidence during the MDT course. This finding, combined with our finding of a higher risk of subsequent reaction in PB patients with an existing nerve function impairment at the start of MDT, may thus be taken as an indication for (more) surveillance attention during that first year, particularly for patients who already have some nerve function impairment. Whether or not this tentative conclusion is supported by further findings in this project, our deduction from these preliminary findings may illustrate the potential of this type of study.

In our study data, both reaction incidence rates and proportions of patients with reactions showed an approximate 2:1 ratio between MB and PB patients. However, we suggest that reaction incidence rates, as also reported by Boerrigter *et al.*[11,12] (and quoted in previous paragraphs), are a more meaningful measure.

Our results are comparable to the findings of a contemporary retrospective study on records of ALERT's field services,[13] which also concluded that in the MB category approximately twice as many patients had reactions as in the PB group. However, our finding of over 10% of patients in reaction at their initial presentation is higher than the 3·5% reported by Becx-Bleumink and Debrezion Behre.[13] As both studies concerned very similar patients, in virtually identical services, this discrepancy may reflect a difference between our prospective study and their analysis of retrospective data.

In both MB and PB groups, reactions were not randomly distributed among patients. Whilst this may in part be due to our definition of a 'new' reaction, it is nevertheless clear that certain patients experienced long periods containing either repeated 'new' reactions or prolonged episodes of on-going reaction activity. Clearly therefore the definition of a reaction also influences incidence rates.

Because of the nonindependence of multiple reactions in individual patients, we have considered clinical manifestations of the first event only for each patient. Over 85% of first reactions included neuritis, of which approximately half did not involve the skin and most of the latter were episodes of silent deterioration of nerves, without any pain or tenderness. Thus, without careful surveillance of nerve function, a large proportion of the occurrence of new or additional nerve dysfunction would not have been detected.

The incidence of events among PB patients was much lower in general than in MB patients, but in both groups similar incidence rates were found during treatment and surveillance periods. Whilst in PB patients pre-existing disability at the start of treatment was a significant risk factor for a subsequent event, this was not found for the MB patients.

Over all patients, the mean change in HF-impairment score between the start of treatment and the latest score represented a deterioration among patients who experienced events, but an improvement among those without events. This difference was statistically significant. Whilst in itself this finding is unremarkable, in view of the common factors in the definition of an event and the HF-impairment score, it supports the validity of the HF-impairment score concept, and of considering mean longitudinal changes in the score when comparing different groups of patients.

## Acknowledgments

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# Field comparison of 10-g and 1-g filaments for the sensory testing of hands in Ethiopian leprosy patients

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*Summary* In ALERT's leprosy control programme sensory testing of hands and feet is done with a nylon filament giving a 10-g stimulus, but doubts arose that early partial sensory loss in hands would not thus be discovered. In order to evaluate the relative performance of 1-g and 10-g filaments for sensory testing on the palms of hands, both filaments were used separately in a series of 1,021 examinations on several consecutive occasions in 159 leprosy patients and 97 nonleprosy controls. The 1-g filament was always felt on normal hands and does not lead to false positive findings of nerve dysfunction. If the 1-g filament were used routinely, almost twice as many instances of 'neuritis' would be discovered and treated, if the criterion for diagnosis and treatment of new nerve dysfunction remained as it is for nerves tested with the 10-g filament.

It appears desirable to distinguish between testing for early sensory loss and for loss of protective sensation. The two tests may each need their own instrument and separate recording of the results.

## Introduction

In leprosy control programmes, sensory testing of the skin of palms of hands and soles of feet was widely introduced in the 1970s. Testing was carried out with the tip of a pencil or a ballpoint pen and the purpose was to identify any lack of protective sensation.[1] This test was commonly carried out a few times a year, in order to identify patients in need of health education and of provision for the protection of insensitive hands and feet (gloves, footwear).

Gradually in the late 1970s and the 1980s more attention was drawn to the role this sensory testing could play in the detection and monitoring[2,3,7,8] of new or additional loss of sensation, particularly when occurring insidiously without other obvious signs of leprosy reaction, in what has been termed 'silent neuritis'.[4]

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Consequently it was suggested that the ballpoint pen should be replaced by one or more nylon monofilaments,[3,5,6] so that standardized and quantifiable stimuli could be used for testing. In ALERT's Physiotherapy Section, such filaments were introduced around 1980[6,9] and in the leprosy control field clinics the ballpoint was replaced by a single nylon filament in 1989. The one filament chosen for use in the field clinics gives a standard stimulus of 10 g. Since then this '10-g filament' has been used for the testing of both palms and soles.

Because of the differences in sensitivity between hands and feet, and of the possible influence of differences in the thickness of skin (particularly among people accustomed to walking barefoot), it would appear desirable that different calibres of filament should be used. On the other hand, it was not considered operationally feasible for field workers to use more than one filament reliably, and so the 10-g filament alone was chosen as a compromise.

Subsequently some patients were found to be complaining of numbness in the palm despite normal responses to testing with the 10-g filament. This suggested that the 10-g filament did not provide a sufficiently sensitive test for early sensory loss in the hand.

This study was therefore initiated to establish whether, for the hand, a finer filament could reliably be used in the field, and to investigate if its use might facilitate the earlier detection of neuritis. This could possibly contribute to the improvement of disability prevention by indicating treatment for neuritis at an earlier stage of disease.

## Methods

In the context of the AMFES project, carried out by ALERT, Ethiopia,[10,11] sensory testing of the hands of leprosy patients was carried out longitudinally using 10-g filaments. For these tests, a nylon monofilament is mounted in the cut-off shaft of a hypodermic needle of suitable size which then, for use, is placed on the nozzle of a disposable 1-ml syringe. When not in use, the syringe (from which the piston has been removed) serves as a protective casing for the filament, giving a small instrument that can easily be carried by field workers (Figure 1). To carry out a test, the end of the filament is pressed perpendicularly against the skin until it buckles, exerting a standard force.

In both hands, 6 points on the skin of the palm area served by the median nerve and 4 points for the ulnar nerve were tested at standard sites on each occasion. After patients had been familiarized with the testing method, response to the 1-g stimulus was usually tested first and thereafter the 10-g filament was applied. Hands feeling the 1 g at all sites did not need to be tested with the 10-g filament. Findings were recorded on a special sheet which was removed from the patient file and stored elsewhere before the next examination 4 weeks later. These examinations were carried out in 38 leprosy clinics of 3 districts. In all, 9 health assistants and 6 supervisors participated. Most of the examinations were carried out by health assistants (HAs). Per patient the sequential tests at 4-weekly intervals were usually done by the same person. The HAs were instructed to ask the visiting leprosy control supervisor to repeat the testing in cases where any finding for the 1-g filament differed from those for the 10-g filament, but the HA was not to show his findings. These repeat tests of the same patient on the same occasion, and also those at periodical supervisor's reviews of patients, were done for interobserver comparison.

In all, 159 leprosy patients were involved. These were ambulatory patients, 41

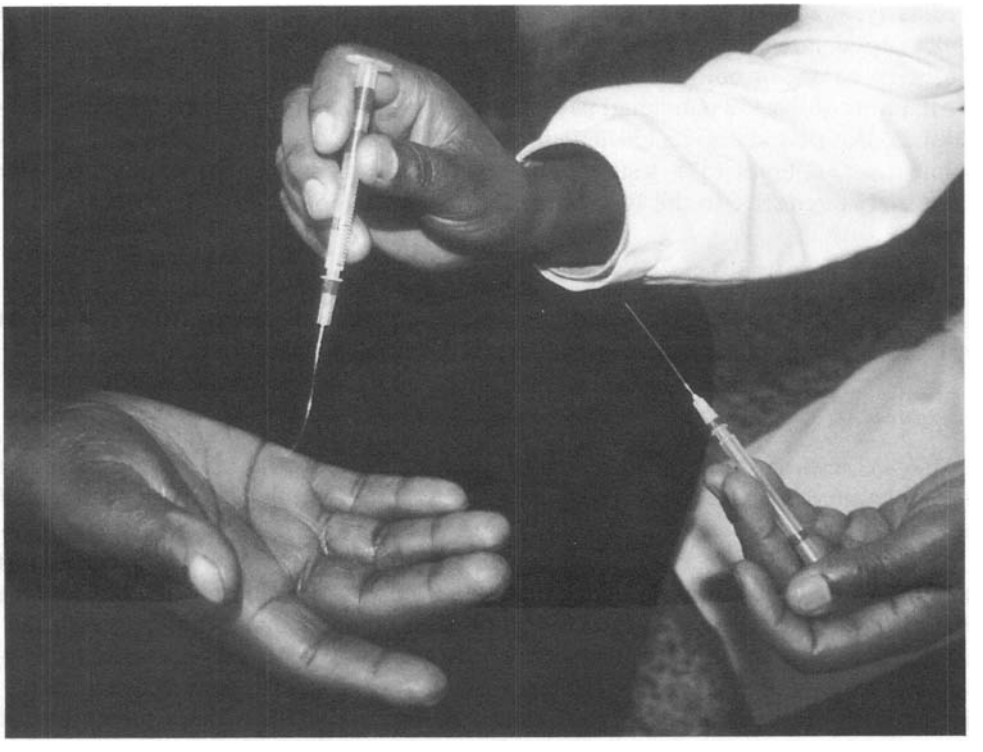

**Figure 1.** Testing sensitivity of the hand using a 10-g filament, mounted in a cut-off hypodermic needle attached to an empty disposable syringe, which acts as a protective cover for the filament when not in use. The alternative 1-g filament is also seen.

paucibacillary and 117 multibacillary, treated with WHO–MDT. The tests were done at the 4-weekly collection of their MDT drugs. (A few patients had also some follow-up tests done during surveillance after completion of the MDT course.)

Findings with the 1-g filament had no consequences for patient management. New sensory loss found with the 10-g filament at 2 or more points of the same nerve area made a patient eligible for an intervention with prednisolone. Tests with the 2 filaments were then continued, often every 2 weeks, to monitor the response to the antireaction treatment.

In addition to these examinations of leprosy patients, a randomly selected group of 97 nonleprosy controls from similar backgrounds were each twice tested in the same way by different examiners, using 10-g and 1-g filaments. These controls consisted of 58 persons who attended a rural health clinic, for other reasons than leprosy, and 39 manual labourers on a building site. This latter group was added to ensure that the control group contained enough men with hands well exposed to manual labour. Age and gender distribution and living conditions of patients and controls were closely similar.

## Results

### LEPROSY PATIENTS

A total of 1,021 examinations were done on 159 patients. In 563/1021 tests (55·1%) total

sensitivity, at all 10 sites of both hands, to the 1-g filament was recorded, and in 20 tests (2·0%) total insensitivity to the 10-g filament was recorded. Thus a partial loss of sensitivity to one or both filaments was found in 438 tests.

In terms of the 159 individual patients, the mean number of tests per patient was 6·4 (sd 4·2). Both hands of 75/159 patients (47·2%) were completely sensitive to the 1-g filament at all times (375 tests in all), and of 5 patients (3·1%) both hands were completely insensitive to the 10-g filament throughout (15 tests).

### NONLEPROSY CONTROLS

Of the 97 nonleprosy controls tested in the same way, the only insensitivity recorded was in 1 point, in 1 person's hand, by 1 observer, for the 1-g filament.

### COMPARISON OF 1G AND 10G RESULTS

Of the 438 tests showing some (but not total) loss of sensitivity, the records of 6 were incomplete and have been excluded from further analysis, leaving 432 tests relating to 79 patients.

The overall results 'per nerve' are summarized in Table 1. Clearly the individual tests (columns 1 and 3) cannot be considered statistically independent of the patients and nerves to whom they relate. Therefore, statistical comparisons of findings with the 2 filaments were made only for frequencies of insensitivity per nerve (columns 2 and 4). Similar patterns of insensitivity for the 1-g and 10-g filaments were observed in all 4 nerves, with the right ulnar nerve showing the highest extent of insensitivity to both filaments. Overall, the tests using the 1-g filament revealed significantly more insensitive nerves than those using the 10-g filament. For the left ulnar nerve the difference was not significant in this series.

The incidence of sensory loss in hands is high in these 79 patients. For the standard 10-g filament, some loss was found in 163 (51·6%) of the 316 (4 × 79) nerves tested and for the 1-g filament this proportion reached 70·9% (224/316). In Table 2, the results of the same tests are presented on the basis of a comparison within each test. In over half of the tests, equal insensitivities were recorded for both filaments. In most of the remaining

**Table 1.** Specification of 432 sensitivity tests in 79 patients in the AMPES project, which showed any loss of sensitivity for either the 1-g or the 10-g filament, or for both. Numbers of tests and nerves with sensory loss are given per nerve and per filament. Comparison (with $\chi$-squares) is shown for the proportions of nerves with sensory loss, as found by the 2 filaments

| | 1-g filament | | 10-filament | | Comparison | |
|---|---|---|---|---|---|---|
| | tests [1] | nerves [2] | tests [3] | nerves [4] | $\chi^2$ | $p$ |
| N | 432 | 79 | 432 | 79 | | |
| Right median | 266 | 58 | 152 | 39 | 8·65 | 0·003 |
| Right ulnar | 321 | 61 | 209 | 43 | 8·13 | 0·004 |
| Left ulnar | 248 | 52 | 160 | 42 | 2·13 | n.s. |
| Left median | 265 | 53 | 155 | 39 | 4·40 | 0·036 |
| (4 nerves) | | (224) | | (163) | (24·00 | < 0·001) |

**Table 2.** Comparisons of 432 sensitivity tests on the hands which showed any loss of sensitivity to either 1-g or 10-g filaments. The results are categorized by differences between the 2 filaments. The tests relate to 79 patients in the AMFES project. Percentages are shown in parentheses

| | 1 g showing higher insensitivity | 1 g and 10 g showing equal insensitivity | 1 g showing lower insensitivity |
|---|---|---|---|
| Right median | 185 (42·8) | 241 (55·8) | 6 (1·4) |
| Right ulnar | 178 (41·2) | 253 (58·6) | 1 (0·2) |
| Left ulnar | 147 (34·0) | 281 (65·0) | 4 (0·9) |
| Left median | 194 (44·9) | 236 (54·6) | 2 (0·5) |

tests, additional insensitivity was revealed using the 1-g filament. Only a very small number of tests showed less insensitivity using the 1-g filament, of which most may be due to procedural errors.

## INTEROBSERVER VARIATION

In Table 3, the proportions of 429 tests showing any degree of sensory loss, by filament and category of observer, are presented. HAs carried out 289 tests and supervisory staff 140. (For 3/432 tests, the examiner was not identified.) For all four nerves, using both filaments, percentages of insensitivity detected by HAs and supervisors were within approximately 5% of the overall proportion. For 3 of the 4 nerve areas tested the supervisors found slightly less sensitivity than the health assistants. For the right ulnar nerve this was the opposite.

In some cases, patients were tested independently on the same day by both categories of examiner. In total, there were 98 paired observations of this kind among the 429 tests. In 45 of these pairs (45·9%), there was complete agreement on sensory loss for all 4

**Table 3.** Results of 429 sensitivity tests on the hands, using both 1-g and 10-g filaments, in which some loss of sensation was recorded, classified by examiner (health assistants (HA) or supervisory staff). These tests relate to 79 patients in the AMFES project. Percentages are shown in parentheses

| | 1-g filament | | | 10-g filament | | |
|---|---|---|---|---|---|---|
| | HA | [all] | supervisor | HA | [all] | supervisor |
| Number of tests | 289 | | 140 | 289 | | 140 |
| Right median | 185 (64·0) | [61·5] | 79 (56·4) | 109 (37·7) | [35·2] | 42 (30·0) |
| Right ulnar | 211 (73·0) | [74·3] | 108 (77·1) | 136 (47·1) | [48·7] | 73 (52·1) |
| Left ulnar | 172 (59·5) | [57·6] | 75 (53·6) | 111 (38·4) | [37·1] | 48 [34·2) |
| Left median | 182 (62·9) | [61·5] | 82 (58·6) | 106 (36·7) | [35·9] | 48 (34·3) |

nerves, using both filaments. In 7 cases (7·1%) the observers differed only for findings with the 10-g filament; in 26 cases (26·5%) they differed only for the 1-g filament, and in 20 cases (20·4%) they had differences for both filaments.

### LONGITUDINAL ANALYSIS

From the overall group of patients, those 47 for whom 3 or more examinations were completed on different dates over a period of 3 months or longer, and in whom some insensitivity was recorded at least once, have been selected for longitudinal analysis of changing patterns of sensory loss to both 1-g and 10-g filaments. A total of 325 tests were performed on these patients. In cases where 2 examinations were carried out on the same patient on the same day, the one performed by the more senior examiner has been used.

In 46/47 patients, additional insensitivity was revealed using the 1-g filament on at least one occasion. For each nerve, for each patient, it is possible to categorize the overall pattern of insensitivity to testing with 1-g and 10-g filaments as follows:

A: nerves with any insensitivity to either filament;
B: some insensitivity to 1 g, but never to 10 g;
C: periods of insensitivity to 1 g, but not to 10 g, before and/or after periods of insensitivity to both filaments;
D: some insensitivity to both filaments throughout; and
E: a pattern different from any of the above

The results of these categorizations for the 47 patients in this analysis are shown in Table 4. Categories B and C together represent additional sensory loss detected by the use of the 1-g filament, and account for about half of the patients, for all nerves.

### EXTRAPOLATION TO PATTERNS OF INTERVENTION

From the above longitudinal analysis, it is possible to investigate the potential consequences of the choice of filament on interventions. If an arbitrary criterion for

**Table 4.** Longitudinal analysis of 47 patients in the AMFES study for whom sensory testing with both 1-g and 10-g filaments was performed on 3 or more separate occasions over at least one occasion. Results for each nerve in each patient are categorized by patterns of sensitivity observed with both filaments over time. Percentages for each category, for each nerve, are shown in parentheses

| Category | A | B | C | D | E |
|---|---|---|---|---|---|
| Nerve | | | | | |
| Right median | 11 (23·4) | 15 (31·9) | 8 (17·0) | 9 (19·1) | 4 (8·5) |
| Right ulnar | 9 (19·1) | 15 (31·9) | 9 (19·1) | 12 (25·5) | 2 (4·3) |
| Left ulnar | 15 (31·9) | 8 (17·0) | 9 (19·1) | 12 (25·5) | 3 (6·4) |
| Left median | 15 (31·9) | 8 (17·0) | 14 (29·8) | 5 (10·6) | 5 (10·6) |

A: never any insensitivity to either filament;
B: some insensitivity to 1 g, but never to 10 g;
C: periods of insensitivity to 1 g, but not to 10 g, before and/or after periods of insensitivity to both filaments;
D: some insensitivity to both filaments throughout; and
E: a pattern different from any of the above.

intervention with prednisolone is taken to be an increased loss of sensation at 2 or more points on 1 or more nerves, then with the use of the 10-g filament, 19/47 patients (40·4%) had 1 or more indications for intervention. Using the 1-g filament, 32/47 patients (68·1%) had 1 or more indications. The criterion was never met by 13/47 patients (27·7%). The mean number of indications per patient was also higher using the 1-g filament (1·25, sd 1·2) than with 10g (0·72, sd 1·1). Thus the overall effect of hypothetically using a 1 g filament instead of 10 g, would have come close to doubling the number of interventions, in this group of patients. Overall, 15 patients (31·9%) did not meet the criterion with a 10-g filament, would have been treated with the use of 1 g. Four other patients (8·5%) had additional indications for treatment using a 10-g filament while not for the 1g, but these were seriously impaired cases who had prolonged periods of total insensitivity to the 1-g filament.

## Discussion

The finding that the hands of 75 of these 159 leprosy patients were fully sensitive to the 1-g filament in all (376) tests, we consider to be evidence that a 1-g stimulus can be felt by normal hands. This was confirmed by the full response to 194 tests in 97 nonleprosy patients. From these findings we conclude that sensory loss to the 1-g filament indicates a real sensory impairment. This specificity of the 1-g filament as a tool for sensory testing of palms of hands is further supported by the 187 tests (563–376) in which both hands were found fully sensitive to the 1-g filament, in patients who at other times had a nerve function impairment with sensory loss to 1 g (or to both 1 g and 10 g).

In cases with any insensitivity, the fact that sensory loss to 1 g occurred in 61 more nerves than for the 10-g filament (an increase from 51·6% to 70·9%), indicates that the 1-g filament is a more sensitive instrument for the (early) detection of sensory loss in hands.

Interobserver variation was considerable, both for the 10 g and the 1-g filament and, not surprisingly, it was more so for the latter. Workers experienced in this kind of sensory testing will know that such differences may occur even when the same examiner repeats an examination within the same session on the same day. To some extent this is a characteristic of these tests, which are quite vulnerable to several influences.

Approximately twice as many interventions with prednisolone would have been indicated if the response to the 1-g filament had been taken as criterion in the same way as it is presently done for the 10-g filament. While this study does not allow any conclusions regarding the possible therapeutic effect of using this more sensitive criterion for initiating neuritis treatment, it is possible that it would be of benefit to patients.

As our study also indicates that the use of a 1-g filament would not lead to any significant proportion of false positive signs of nerve dysfunction, we conclude that for early detection of sensory loss to light touch on the palms of leprosy patients, the 1-g filament is an appropriate tool. With this filament, it should be possible to detect insidiously developing neuritis earlier than by using a ballpoint pen or a 10-g filament.

An important further concept is that, in the sensory testing of hands and feet, carried out now in so many leprosy control programmes, there are two distinct objectives:

(1) the *early detection of (insidious) neuritis*, for which a medical intervention would be indicated; and for monitoring the response to neuritis treatment. For this a sensitive instrument, providing a constant stimulus, is needed, but this test

should still be specific enough not to give false positive findings of nerve dysfunction; and

(2) the assessment of *loss of protective sensation*, i.e. the level of sensory loss that puts a hand or foot at risk for wounds, burns, etc. and consequent damage. This requires a much stronger stimulus.

Programmes should therefore address these two objectives separately. In addition, the level of stimuli required for (1) and (2) may differ between palms and soles of feet.* Whilst this approach to sensory testing would have considerable operational consequences, involving several instruments and revised methods of recording, we nevertheless believe it to be justified.

In our programme we now therefore recommend using a 1-g filament for the hands for objective (1), and a ballpoint pen for objective (2).

## Acknowledgments

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* Alert's studies on the testing of feet are not yet concluded.

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# Circulation and sensation at the fingertips of claw hands

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*Summary* Measurements of skin blood flow (by laser Doppler flowmetry) and temperature were made under environmental conditions promoting peripheral vasodilatation at the fingertips of a disfigured 'clawed' hand in 12 leprosy patients long-resident at Baba Baghi Leprosy Hospital, Tabriz, Iran. Sensory function was assessed by measuring the responses to light touch, pain and temperature of each finger, and peripheral autonomic function was gauged by estimating palmer sweating and by measuring skin vasomotor reflexes in response to inspiratory gasp.

In 2 patients all measured fingers had laser Dopper flux (LDFlux) values and skin temperatures lower than the 95% confidence limits for the mean of 20 healthy controls, i.e. were impaired; in 2 patients all fingers had normal values for LDFlux and temperature; and in 8 patients there was a combination of impairment with most fingers normal for these parameters but with the small finger most commonly impaired. There were 10 (67%) fingers with impaired LDFlux and temperature values who had significant sensory impairment, whereas only 5 (18%) of the fingers with normal LDFlux values and temperatures had a similar sensory deficit. Overall, the fingers with the most impaired sensation had significantly ($P < 0{\cdot}05$) lower LDFlux and temperature values than those with no sensory deficit. Microcirculatory impairment was not related to disordered skin vasometer reflexes or dysfunction of sweating.

We concluded that the relationship between motor (skeletal muscle) nerve paralysis and any subsequent sensory neuropathy and/or microcirculatory impairment is more complex than might be expected from previous understanding of the disease.

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## Introduction

The effect of *Mycobacterium leprae* infection on blood flow to the extremities has not been intensively investigated up to now, although several studies employing radiological arteriographic techniques have reported impairment of flow in the terminal vascular loops of the fingers[1] or tapering and tortuosity of the digital arteries of the hands.[2,3]

Recently noninvasive estimates of blood perfusion through the fingertip skin have been obtained in Indian leprosy patients.[4] These studies showed that measurements of blood flow by laser Doppler flowmetry (LDFlux) were lower than those found in normal subjects. Further studies in Indian patients with a variety of orthopaedic complications of the lower limb resulting from long-standing leprosy, but with undisfigured hands, have shown that these patients' fingers were comparatively cold and had low blood flows despite conditions conducive to peripheral vasodilatation:[5,6] this abnormality was most prominent in the multibacillary patients but it was also found in some people with paucibacillary disease. Since severe hand deformity results from damage to the mixed sensory and motor peripheral nerve trunks, and becomes established permanently if prompt ameliorative physiotherapy is not provided, it was expected that LDFlux would be impaired at least to the same extent, and possibly to an even greater one, in long-term patients with claw hands, thus reflecting the dominant role of autonomic control over blood flow through the peripheral microcirculation.

This short report describes the measurement of fingertip blood flow, skin temperature and sensation in a small group of leprosy patients with characteristic claw hand. The findings show that the relationship between functional neuropathy and microcirculatory deficit is more complicated than was expected from previous understanding of the disease.

**Table 1.** Clinical details for the 12 leprosy patients, and the results of fingertip temperature, LDFlux and sweat function for the claw hand of each

| Patient number | Hand: right (R) left (L) | Years at leprosy hospital | Age (years) | Type (multi/ paucibacillary) | Leprosy finger* impaired for temp (T) or flux (F) or both | | | | Impairment of sweat (S) function |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | index | middle | ring | small | |
| 1 | R | 32 | 47 | P | TF | TF | TF | TF | † |
| 2 | L | 32 | 44 | M | T | TF | TF | TF | S |
| 3 | R | 32 | 51 | M | TF | TF | † | TF | † |
| 4 | R | 19 | 57 | M | | | F | F | |
| 5 | R | 17 | 25 | P | | | TF | TF | |
| 6 | R | 31 | 60 | P | | | | TF | S |
| 7 | L | 21 | 31 | M | | | | TF | |
| 8 | L | 35 | 65 | P | | | | F | S |
| 9 | L | 35 | 52 | M | | | | TF | S |
| 10 | R | 29 | 48 | P | | | | TF | S |
| 11 | L | 26 | 43 | M | | | | | † |
| 12 | R | 28 | 47 | P | | | | | S |

* All fingers were tested: a blank space is left where no abnormality was found.
† Indicates that the investigated parameter could not be reliably obtained in this subject.

## Methods

### SUBJECTS

We selected 12 patient volunteers from the long-term residents of Baba Baghi Leprosy Hospital, Tabriz, Iran on the basis of severe disfigurement of at least 1 hand. Table 1 presents the age, length of residence and clinical classification of the disease (paucibacillary or multibacillary) in these patients. Disfigurement was defined as severe flexion of the fingers at both the distal and proximal interphalangeal joints. The ulnar nerves were involved most prominently in these patients but all exhibited median involvement to a greater or lesser extent. All 12 hands studied were functionally very disabled and none of the fingers had a normal range of movement. None of the hands were swollen or callused to visible inspection but thenar and hypothenar wasting was prominent in subjects 2 and 8.

We chose 20 people, mean age 31·5 (range 16–70) as controls: 10 were the offspring of treated leprosy patient who, as adults, continued to live and work within the large hospital compound; the other 10 were apparently healthy members of the hospital staff.

### MEASUREMENT OF BLOOD FLOW AND VASOMOTOR REFLEXES

A laser Doppler flowmeter (model PF2, Perimed, Stockholm, Sweden) was used to measure the blood flow through the skin over the pulp of the distal phalanx in the manner and with settings described previously.[4,6] The laser Doppler flowmeter measures movement of erythrocytes in the most superficial 1 mm of skin from changes in the frequency of coherent light reflected out of the tissue and gives an integrated measurement of microvascular blood flow (LDFlux) expressed in Volts.[7]

This method also permits assessment of vasomotor reflex responses at the fingerpulp, determined from the fall in LDFlux signal following a physiological challenge such as a deep inspiratory gasp (IG).[4] Figure 1(a) shows the kind of trace obtained from a healthy subject: LDFlux is high and the IG response is large. Figure 1(b), from a leprosy patient, shows a reduced LDFlux signal and IG response. In this study a reflex response was attempted on each fingerpulp of the hand under investigation.

### MEASUREMENT OF FINGERTIP SKIN TEMPERATURE

A platinum skin thermistor attached to an LCD output device was used to measure skin surface temperature in the manner described previously.[6] The probe (Model 4098, 9 mm diameter, Yellow Springs Instrument Co Inc, Yellow Springs, Ohio, USA) was held in close contact with the skin on the pulp of the distal phalanx close to the fingertip with a single strip of Millipore adhesive tape.

### SENSORY TESTING

Sensation in the upper limb was examined for integrity of light-touch sensation with cottonwool, for sharp tough/pain by pin-prick and for temperature sensation using a

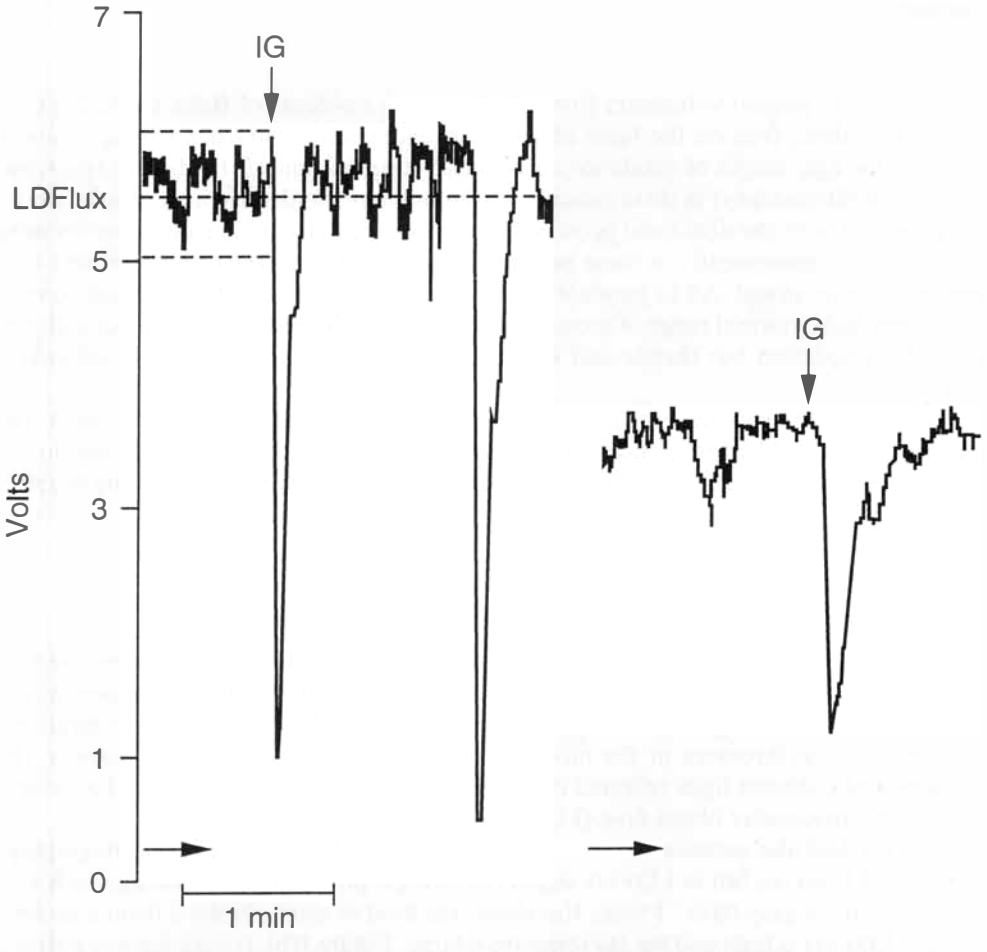


**Figure 1.** Typical LDFlux traces from (a) a healthy person, and (b) a leprosy patient. Under conditions of near-maximal vasodilatation the signal is pulsatile though stable around a mean value: this value is termed the LDFlux and can be recorded for analysis. Here, the leprosy patient has a reduced LDFlux and vasomotor reflex response to inspiratory gasp.

thermal sensibility tester.[8] We tested 3 sites on the palmer aspect of each finger, namely, at the fingerpulp and the middle and proximal phalanges. The results of all sensory tests on each finger were recorded on a 3-point scale: 1, absent or consistently mistaken; 2, partial sensation, i.e. variation from area to area on the volar aspect of the finger; and 3, unimpaired sensation (a positive result at all 3 sites on 1 finger). For convenience of analysis the final summary of results on each subject was the mean score (out of 3) combining the results for the 3 types of sensation tested.

In addition sweat function was crudely assessed from the subject's own report and was also confirmed by the experimenter from the appearance and texture of the skin of the relevant hand. Dry, hard and cracked hands reported by the subject as having swearing dysfunction were classed as impaired.

### EXPERIMENTAL PROTOCOL

Each subject was seated comfortably with the forearm and hand resting on a table at heart level at an ambient temperature of 25–29°C, maintained by a large paraffin stove. These conditions would be expected to induce near maximal peripheral vasodilatation and a stable blood flow through the fingertips in healthy subjects.[9] Each subject was allowed to equilibrate under these conditions for at leas 20 min before measurements were started. All 4 fingers were studied on the hand under investigation. Skin temperature was recorded and then laser Doppler measurements of blood flow and vasomotor reflexes were made. At the end of the experiment, which lasted at least 1 h, the LDFlux measurements were repeated to check for any effects of warming over a longer period. On a separate occasion sensation was tested in the order of light touch, pin-prick, and finally, temperature discrimination.

### STATISTICAL ANALYSIS

Differences between categories of patients were assessed using 2-way ANOVA taking intraindividual differences between fingers into account. The relationship between LDFlux and skin temperature was assessed by linear regression analysis.

## Results

### NORMAL SUBJECTS

Figure 2 illustrates the local fingertip skin temperature and corresponding LDFlux value for all 8 fingers (L2–L5, R2–R5) in the 20 normal subjects. The relationship between these 2 measurements was significant ($r = 0{\cdot}28$, $P < 0{\cdot}001$). The lower and upper 95% confidence limits[10] for the mean values of these subjects were 3·1–10·0 Volts, respectively, for LDFlux (mean 6·6), 31·0–35·0°, respectively, for skin temperature (mean 33·1)°C and 43–100%, respectively, for vasomotor gasp reflex (mean 77%). Figure 2 also shows that there were no significant differences in LDFlux and temperature between the index, middle, ring or little fingers in these healthy subjects. All had unimpaired sensory function and were without evidence of any disorder of palmar sweating function.

### LEPROSY PATIENTS

Table 1 presents the distribution of impairment to fingertip temperature and LDFlux on the deformed (claw) hand of each of the 12 subjects. Impairment was defined as LDFlux, skin temperature and vasomotor reflex measurements of less than 3·1 Volts, 31·0°C and 43%, respectively (the lower limits for the 95% confidence limits for the healthy controls above). Patients Nos 11 and 12 had normal values, and Nos 1 and 3 had abnormal values for each parameter on all measured fingers. The remaining 8 patients had a combination of impaired and unimpaired fingers, the small finger being most commonly affected. On patient No. 3 the ring finger was clamped to the palm, so the volar aspect was inaccessible.

Abnormalities of sweating function were clearly visible in 6 of the patients; in 3 others

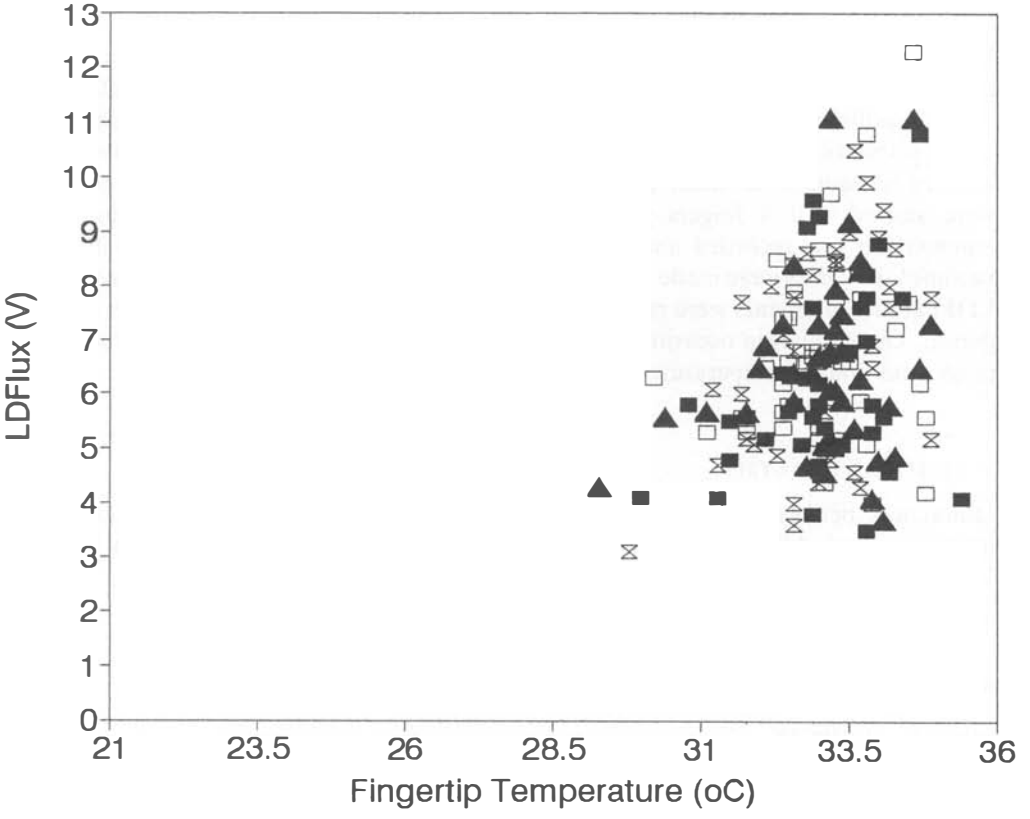


**Figure 2.** The temperature and LDFlux measured on 8 fingertip of 20 healthy subjects. There were no significant differences in the measured values between the index (■), middle (□), ring (▲) or small (⧖) fingers in this control group.

(Nos 1, 3 and 11) disorder of sweating could not be confidently confirmed by visual and tactile inspection.

Vasomotor reflexes were impaired or absent in all fingers studied in 7 patients; 2 patients (Nos 8 and 12) had normal grasp reflex responses in only 1 finger; and only patient No. 11 had a normal grasp response on 3 fingers. In patients Nos 1 and 3 the LDFlux measurements at the fingers were too low for assessment of vasomotor grasp reflex responses.[4]

There was no significant difference in temperature, LDFlux or sensation between the fingers of patients with multibacillary or paucibacillary types of lepsory. Figure 3 shows the significant relationship ($r = 0{\cdot}81$, $P < 0{\cdot}00001$) between temperature and corresponding LDFlux in the patient group. Of the 47 fingers studied 27 had both temperature and LDFlux values within the normal range and 15 fingers were impaired for both parameters. In addition it can be seen from this scattergram that those fingers with a low mean score for sensation (0–1·5) tend towards low LDFlux and low temperatures. Low sensory scores were seen in 10/15 (67%) fingers with impaired LDFlux and temperature but in only 5/28 (18%) fingers which had normal LDFlux and temperature. Overall, there was a significant difference ($P < 0{\cdot}05$) between those fingers

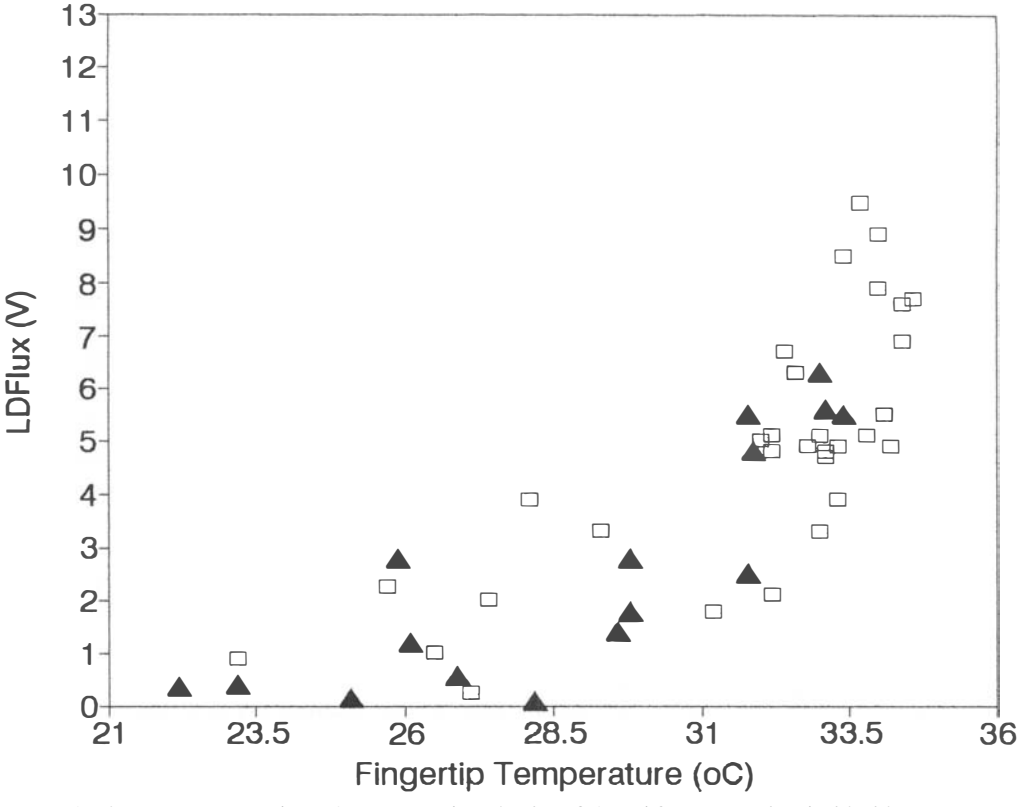


**Figure 3.** The temperature and LDFlux measured on the tips of clawed fingers associated with either poor (▲) or good (□) sensory function in 12 leprosy patients.

with a low mean score for sensation (0–1·5) and those with a high mean score (1·6–3) as regards fingertip temperature (28·9 *vs* 31·8°C, respectively) and LDFlux (2·61 *vs* 4·82 V).

## Discussion

The typical claw hand deformity of leprosy results from peripheral neuropathy producing paralysis of the intrinsic muscles of the hand. In paucibacillary patients the most likely immediate cause is the inflammatory process within the nerve trunks during a reversal reaction but slower paralysis may result from the gradual development of intraneural granulomata causing pressure and ischaemia within the perineurium.[11] Similar deformity in multibacillary patients results from damage to nerve trunks by bacillary multiplication within them, sometimes complicated by erythema nodosum leprosum. The distribution of the paralysis in these patients indicates that damage has occurred at both the median and ulnar mixed nerve trunks where it is possible that the smaller sensory fibres and the nonmyelinated autonomic fibres are also affected. The recent evidence that the combination of orthopaedic complication and sensory deficit can be associated with impaired digital circulation in some long-term patients with both

multibacillary and paucibacillary disease[6] makes it feasible that patients with claw hands could also have a disorder of fingertip microcirculatory control.

The hands of the present 12 subjects have had ulnar and medial nerve paresis of long standing, but most have retained both the capacity for relatively normal blood flow in most fingers under warm environmental conditions and a relatively unimpaired digital sensation. While some autonomic deficit in the form of vasomotor reflex impairment or crude estimate of sweating dysfunction was seen in all patients, low flows/low temperatures tended to occur in fingers exhibiting paresis in association with sensory deficit, confirming previous studies.[6]

The pathogenesis of these mixed nerve lesions is not clear. The subjects we have studied have all been treated with full courses of multidrug therapy and it is possible that, following initial damage to all fibre types, selective regeneration of the small sensory fibres could have occurred. This would have restored sensory and/or autonomic function without restoration of motor functions which are dependent on intact larger fibres. The duration of the motor denervation was probably prolonged in these patients. Absence of innervation for more than 3 years leads to irreversible changes in contractile tissue with no possible return of functional activity even when nerve fibres have returned to the grossly atrophic and fibrotic muscle.[12]

Wide-ranging and patchy nerve lesions can be produced by focal leprosy granulomata or bacillary infiltration due to the complicated pattern of intraneural fascicle branching[13] or by the spread of existing lesions in the manner postulated in the Dehio concept[11] with migration of organisms from a skin patch to the nerve branches coursing beneath the patch.[14] An alternative explanation for our findings is that large myelinated fibres are more susceptible than the smaller fibres to damage at the site of such lesions. There is good evidence that motor fibres are less resistent to the effects of compression than sensory fibres:[15] an example is the 'Saturday-night' paralysis in which muscle function is lost while sensation is spared or only transiently affected.[16] In the leprosy patient without the benefits of prompt ameliorative physiotherapy muscle paralysis could become established without impairment of sensory or sympathetic function.

Whatever the explanation, the cases reported in this short report provide evidence that a claw hand, though cosmetically ugly and functionally very inadequate, may be spared the gross microcirculatory and sensory impairment found in some undisfigured hands.

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# Silent neuropathy in leprosy: an epidemiological description

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*Summary* This paper presents epidemiological data on silent nerve function impairment in leprosy based on a retrospective study of 536 patients registered at Green Pastures Hospital, Pokhara, West Nepal. Because of the multiple possible aetiologies it is proposed that the clinical phenomenon should be named 'Silent Neuropathy' (SN). We defined this as sensory or motor impairment without skin signs of reversal reaction or erythema nodosum leprosum (ENL), without evident nerve tenderness and without spontaneous complaints of nerve pain (burning or shooting pain), paraesthesia or numbness. The functioning of the main peripheral nerve trunks known to be affected in leprosy was assessed using a nylon filament to test touch thresholds and a manual voluntary muscle test to quantify muscle strength.

Almost 7% of new patients had SN at first examination. The incidence rate of SN among the 336 new patients who were available for follow-up was 4·1 per 100 person years at risk. In total, 75% of all SN episodes diagnosed after the start of chemotherapy occurred during the first year of treatment. During steroid treatment the sensory and motor function in nerves affected by SN improved significantly ($p = 0{\cdot}012$, Wilcoxon matched-pairs signed ranks test) over a period of 3 months. The patients with more extensive clinical disease (3/9 or more body areas involved, more than 3 enlarged nerves or a positive skin smear) were found to be at increased risk of developing SN.

We discuss 4 different possible aetiologies of SN: 1, Schwann cell pathology; 2, nerve fibrosis; 3, cell-mediated immune reaction; and 4, intra-neural ENL. Some epidemiological evidence is presented that suggests that SN cannot be equated with a 'reversal reaction expressing itself in the nerves'.

It is recommended that all patients should have a nerve function assessment at every visit to the clinic at least during their first year of treatment. Regular nerve function assessment is essential to detect SN at an early stage and to prevent permanent impairment of nerve function.

## Introduction

Impairment of sensory or motor nerve function, without symptoms of neuritis has been described as a common phenomenon by a number of authors.[1–4] Various terms have

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been used to describe this phenomenon, including 'quiet nerve paralysis',[1] 'silent neuritis'[2,3] and 'nerve reaction.'[5] However, we were unable to find any publication on the epidemiology of this phenomenon in a population of nonselected patients. There is no consensus about its aetiology and in many leprosy clinics where no routine nerve function assessments are carried out, it is missed altogether.[3,6] Assessment of nerve function refers to testing of sensory and motor function of structures innervated by the major nerve trunks commonly affected by leprosy (facial, ulnar, median, radial, lateral popliteal and posterior tibial nerves) and their branches.

Because we regularly encountered SN at Green Pastures Hospital (GPH), in a sizeable proportion of patients requiring steroid treatment for neural impairment, we decided to study this phenomenon in more detail. This was done in the context of the retrospective cohort analysis on leprotic reactions and neural impairment which was carried out at our hospital.

GPH is a 100-bed mission hospital in Pokhara, West Nepal, run by the International Nepal Fellowship under its Leprosy Control Project, which is a joint venture with His Majesty's Government/Nepal. GPH is the main leprosy referral hospital for the West of Nepal.

This study addresses the following questions: 1, what is the prevalence at first examination and incidence rate of SN in the patients studied?; 2, can any risk factors be identified that are prognostic for an increased risk of SN?; 3, how well does it respond to steroid treatment?; and 4, what are the possible aetiologies of SN?

## Methods

### STUDY DESIGN

The current study was part of a retrospective (historic) cohort study of all leprosy patients registering at GPH between January 1988 and January 1992. Both previously treated and untreated patients were included, although, for most purposes, their results were analysed separately.

### CRITERIA FOR INCLUSION AND EXCLUSION OF PATIENTS

All new, previously untreated patients who registered for treatment in GPH during the abovementioned period were included in the study. Patients referred to GPH for treatment of reaction/neuropathy were included, but those whose treatment was started elsewhere more than 1 week before arrival at GPH were excluded.

### OUTCOME MEASURES

The number of patients with SN among all patients in the cohort stratified by classification and time of onset of neural impairment.

Odds ratios pertaining to the following potential risk factors: age, sex, classification of leprosy, extent of disease (numbers of skin lesions, nerves and body areas involved in the disease), bacteriological index, PGL-1 serological results and type of leprosy treatment (multidrug therapy (MDT) *vs* dapsone monotherapy (DDS)).

Voluntary muscle test (VMT) and touch sensibility test (TST) scores at various times during and after steroid treatment.

### DIAGNOSIS AND CLASSIFICATION OF LEPROSY

Details on diagnosis and classification, which included clinical examination, counting of the number of body areas involved in the disease, skin smears from routine sites and at least 1 active lesion, PGL-1 antibody testing and histopathology for a limited number of patients, were described in a previous publication.[7] Briefly, the body area system is based on a count of the number of body areas out of a total of 9 (head, 4 extremities, front and back, both divided in left and right side) that shows primary or secondary signs of leprosy. These may include skin lesions, enlarged nerves, clawing of fingers and ulcers.

### DIAGNOSIS OF REACTIONS AND NERVE FUNCTION IMPAIRMENT

#### REVERSAL REACTION (RR)

The diagnosis 'reversal reaction' was based on the presence of skin signs, but a patient could have some or all of the following clinical signs:

Skin: redness and swelling of (usually already existing) lesions, sometimes tender in the lesions.
Nerves: often signs of neuritis with/without swelling, nerve pain, tenderness or nerve function impairment.
General: sometimes oedema of hands, feet or face, occasionally fever.

#### SILENT NEUROPATHY (SN)

A patient was diagnosed as having SN if he showed the following clinical signs and symptoms: sensory or motor impairment (see below) without skin signs of reversal reaction, ENL, without nerve tenderness that had been noticed by the patient and without complaints of nerve pain (burning or shooting pain) or paraesthesia that were mentioned by the patient without specifically asking for them.

There are 3 common clinical scenarios. The first is of a patient who presents with skin signs of leprosy without complaining of his nerves, who during nerve function assessment is found to have sensory and/or motor impairment. The second is the patient who presents because of the consequences of impaired sensory or motor nerve function such as weakness, clawing or a painless ulcer of recent onset, but who does not recall reactive skin lesions or nerve pain. The third is that of a patient who is already taking antileprosy treatment, but who is found to have new neural impairment during a routine follow-up sensory or motor assessment. In this study we called SN a 'reaction'; this does not imply any inference concerning the aetiology.

#### CROSS-OVER REACTIONS

Some patients develop more than 1 type of reaction during their time of registration. For example, a borderline lepromatous patient who first has an episode of reversal reaction, followed after some time by an episode of ENL. We called this phenomenon 'cross-over reactions'.

### VOLUNTARY MUSCLE TEST (VMT)

The VMT score consisted of the sum of individual scores (0–5; 0 = paralysed, 5 = normal strength) for the following muscles: Facial nerve: orbicularis oculi (only)—maximum score: 5; Ulnar nerve: first dorsal interosseus ('index finger out') and abductor digiti minimi ('little finger out')—maximum score: 10; median nerve: abductor pollicis brevis and opponens pollicis ('thumb up')—maximum score: 10; lateral popliteal nerve: extensor hallucis longus and peroneus longus & brevis ('lateral foot up')—maximum score: 10.

### TOUCH SENSIBILITY TEST (TST)

Static touch sensibility of the ulnar and median nerves was tested on the palm of the hand, using a nylon monofilament giving a force of approximately 10 gm when pressed until it bent. The result was recorded as felt or not felt for each of the sites mentioned below. If the patient sometimes felt the touch and sometimes not, the result was recorded as partial. When necessary the test was repeated until the examiner was confident about the patients response. Touch sensibility of the posterior tibial nerve was tested in a similar way using a thicker monofilament, giving a force of about 75 gm. The size of these filaments was far from ideal (see Discussion below) but they were the only 2 sizes available in Pokhara at the time. During a normative study of touch sensibility thresholds of healthy Nepali subjects in Pokhara, about 99% were able to feel a 200 mg monofilament on the hand and more than 95% a 2 gm monofilament on the sole of the foot (Kets *et al.*, in preparation). The TST score consisted of the sum of touch sensibility test scores given for individual sites (2 = monofilament felt, 1 = doubtful, 0 = monofilament not felt); the number of sites depending on the nerve tested.

Ulnar nerve: 3 points on the pulp of the little finger, over the 5th metacarpophalangeal (MCP) joint and on the hypothemar eminence respectively—maximum score: 6; median nerve: 4 points on the pulp of the thumb, over the 2nd MCP joint, the pulp of index and middle fingers, respectively—maximum score: 8; posterior tibial nerve: 10 points, on the tip of each toe, over the 1st and 5th metatarsophalangeal joints, the instep, the lateral border and the heel—maximum score: 20. Further details of the testing methodology were reported in a separate paper.[8]

### IMPAIRMENT OF NERVE FUNCTION

A patient was diagnosed as having impairment if there was a deterioration of more than 2 points in the VMT score of an individual nerve or, similarly, 2 points or more in the TST score of a sensory nerve compared to the previous result, or maximum result in case previous results were not available. A reported onset of the neural impairment of less than 6 months previously was recorded as **recent**; otherwise it was recorded as **old**. The motor and sensory scores were further divided into 4 functional categories. Most patients had more than 1 nerve affected but in this paper treatment results are illustrated using only data on 1 ulnar nerve per patient. The functional categories are shown in Table 1.

### TREATMENT

Only patients with recent neural impairment (see above), and who had no other

**Table 1.** Functional scoring system for the ulnar nerve

| | Sensory score | Motor score |
|---|---|---|
| Anaesthetic/Paralysed | 0–2 | 0–2 |
| Bad | 3 | 3–4 |
| Moderate | 4 | 5–7 |
| Good | 5–6 | 8–10 |

concurrent severe illness, such as untreated tuberculosis, were considered eligible for steroid treatment. They received 1 of the following corticosteroid regimens:

1. Dexamethason 6 milligrams (mg) once daily (od) starting dose, tapering approximately 0·5 mg every 2 weeks, depending on the progress of the patient, thus giving a duration of treatment of about 6 months.
2. Prednisolone 30 mg twice daily (bd) starting dose, tapering approximately 5 mg every 2 weeks, depending on the progress of the patient, thus giving a duration of treatment of about 6 months.
3. Prednisolone 60 mg od starting dose, tapering approximately 5 mg every 2 weeks, depending on the progress of the patient, thus giving a duration of treatment of about 6 months.
4. Prednisolone 40 mg od starting dose, tapering approximately 5 mg every 2 weeks, depending on the progress of the patient, thus giving a duration of treatment of about 4 months.

### STATISTICAL METHODS

Risk factors were examined using logistic regression and the results are expressed as odds ratios. An odds ratio may be interpreted as the increase in risk in patients who have the risk factor compared to those who do not. For example, an odds ratio of 1·9 for bacteriological index means that patients with a positive skin smear had almost twice the risk of developing silent neuropathy as those with a negative skin smear. The difference between 2 paired samples was tested using the Wilcoxon matched-pairs signed-ranks test.[9] A $p$-value of less than 5% was used as the level of statistical significance. Incidence rates were calculated as the number of patients developing new nerve function impairment during the follow-up period divided by the cumulative person years at risk. Patients were censored from the denominator as soon as the first episode of SN had occurred. Patients lost to follow-up due to death, defaulting or transferral only contributed person years to the denominator for as long as they were still followed up. Prevalence and incidence were only calculated for the sub-group of new patients. The 95% confidence interval is given in parentheses, e.g. 4·2 (2·1–8·2) means that there is 95% chance that the ratio actually lies between the values 2·1 and 8·2. Analysis was done using Epi Info software, version 5.01[10] and SPSS for Windows, version 6.0.

## Results

### PATIENTS

We included 536 patients in the study—396 were new patients and 140 were old patients who either registered at GPH prior to the study period or were treated elsewhere before

being referred to GPH. The mean age for the new patients was 41 years (range 2–88) and for the old patients 39 years (range 8–72). Among the new patients 70% were male against 81% among the old patients. The average follow-up time for new patients at the start of data analysis was 21 months (range 1–49). There were 2 TT, 202 BT, 7 BB, 133 BL, 42 LL and 10 pure neuritic (PN) new patients, against 38 BT, 6 BB, 40 BL, 40 LL and 6 PN 'old' or referred patients—71 patients were or had been on DDS monotherapy, 344 patients had been or were on the multidrug regimen recommended by the World Health Organisation (WHO MDT).[11] The regimen for paucibacillary patients consists of daily dapsone 100 mg and once-monthly rifampicin 600 mg (dosages are for adult patients). For multibacillary patients clofazimine is added in a dose of 50 mg daily and 300 mg once a month. The duration of each regimen expressed in monthly doses is 6 months and 24 months, respectively (1 patient had taken a different type of MDT).

## PREVALENCE AND INCIDENCE RATES

Table 2 shows the prevalence of SN at first examination and the incidence rates during follow-up amongst the new patients. On average 6·8% of new patients presented with SN of recent onset at the time of diagnosis. More SN was diagnosed at the start of treatment than during or after treatment. Overall 52/396 new patients (13%) developed 1 or more episodes of SN.

The overall incidence rate of SN was 4·1 per 100 person years at risk (PYAR). The rate difference between Ridley–Jopling classification groups was not significant statistically, but the numbers in each group were only small.

Table 3 presents the incidence of SN episodes by time of onset. The majority of episodes diagnosed after registration occurred during the first year of treatment (75%).

## CROSS-OVER REACTIONS

The phenomenon of 'cross-over reactions' is shown in Table 4: 18 patients (12 BT and 6 BL) had a reversal reaction episode followed by SN. The reverse occurred in 6 patients (4 BT, 1 BL, 1 LL). Cross-over between ENL and SN occurred in 7 LL patients (4 × ENL → SN, 3 × SN → ENL).

**Table 2.** Prevalence and incidence rates of SN among 396 new patients of leprosy at GPH

| | Prevalence at registration | | Incidence rates | |
|---|---|---|---|---|
| Classification | Number | % (95%Cl)[a] | Number | per 100 PYAR[b] |
| TT | 0/2 | | 0/2 | |
| BT | 11/202 | 5·4 (2·3–8·6) | 13/183 | 4·0 (2·4–7·0) |
| BB | 0/7 | | 0/4 | |
| BL | 12/133 | 9·0 (4·2–14) | 5/106 | 3·0 (1·3–7·3) |
| LL | 2/42 | 4·8 (0–11) | 4/31 | 9·1 (3·4–24) |
| PN | 2/10 | 20 (0–45) | 1/10 | 6·7 (0·94–47) |
| Total | 27/396 | 6·8 (4·3–9·3) | 23/336 | 4·1 (2·7–6·2) |

[a] 95% confidence interval, [b] incidence expressed as the number of episodes of SN per 100 person years at risk (PYAR).

**Table 3.** Occurrence of silent neuropathy episodes by time of onset among 396 new patients at GPH

| | BT | | BL | | LL | | PN | |
|---|---|---|---|---|---|---|---|---|
| Period | Number | % | Number | % | Number | % | Number | % |
| At registration | 12 | 46 | 13 | 72 | 2 | 33 | 1 | 50 |
| 0–6 months | 5 | 19 | 1 | 5·6 | 3 | 50 | | |
| 7–12 months | 5 | 19 | 3 | 16 | | | 1 | 50 |
| 2nd year | 3 | 12 | 1 | 5 | 1 | 17 | | |
| 3rd year | 1 | 4 | 0 | 5 | | | | |
| Total | 26 | | 18 | | 6 | | 2 | |

### TREATMENT

Table 5 shows the progress of nerve functional scores during treatment. The median value of the sensibility score improved from 0 to 2·5 ($z = -2{\cdot}65$, $p = 0{\cdot}008$) over a period of 3 months, while the median value of the motor score improved from 6 to 7 ($z = 2{\cdot}53$, $p = 0{\cdot}012$) over the same period.

### RISK FACTORS

The only risk factor of statistical significance was the extent of clinical disease expressed as the number of body areas involved in the disease,[7] the number of enlarged nerves or the bacteriological index. If a cut-off point of 3 or more body areas (out of 9) was chosen to define 'extensive disease', the risk of SN was 3 times higher for patients with extensive disease compared to those with limited (= less than 3 body areas involved) disease (adjusted odds ratio 2·8 (1·3–6·0), $p = 0{\cdot}01$). The risk factors are summarized in Table 6.

## Discussion

### AETIOLOGY AND TERMINOLOGY

SN has been described as a separate clinical entity by several investigators.[1–3] As far as we are aware the aetiologies of SN have not yet been conclusively investigated histopathologically. Some characteristics suggest that 'silent neuropathy of recent

**Table 4.** Cross-over reactions among 536 patients registered at GPH

| | Classification | | | |
|---|---|---|---|---|
| Type of cross-over reaction | BT | BL | LL | Total |
| RR[a] → SN[b] | 12 | 6 | | 18 |
| SN → RR | 4 | 1 | 1 | 6 |
| ENL[c] → SN | | | 4 | 4 |
| SN → ENL | | | 3 | 3 |

[a] RR, reversal reaction; [b] SN, silent neuropathy; [c] ENL, erythema nodosum leprosum.

**Table 5.** Progress of nerve function scores during and after steroid treatment in patients with SN of the ulnar nerve

| | Start of treatment | 1 month[a] | 3 months[a] | 6 months[a] |
|---|---|---|---|---|
| Sensory function (ST) | | | | |
| Anaesthetic | 17 (94)[b] | 13 (76) | 9 (50) | 5 (42) |
| Bad | | | 1 (6) | |
| Moderate | 1 (6) | 3 (18) | 2 (11) | 1 (8) |
| Good | | 1 (6) | 6 (33) | 6 (50) |
| Total | 18 (100) | 17 (100) | 18 (100) | 12 (100) |
| Median of TST score (scale 0–6) | 0 | | 2·5 | 5 |
| Motor function (VMT) | | | | |
| Paralysed | 5 (19) | 2 (8) | 3 (145) | 1 (7) |
| Bad | 3 (12) | 2 (8) | 1 (4) | |
| Moderate | 18 (69) | 16 (67) | 9 (41) | 6 (40) |
| Good | | 4 (17) | 9 (41) | 8 (53) |
| Total | 26 (100) | 24 (100) | 22 (100) | 15 (100) |
| Median of VMT score (scale 0–10) | 6 | | 7 | 8 |

[a] The intervals refer to the time between the start of the steroid treatment and the date of the nerve function assessment, [b] number of patients; the numbers in parentheses are column percentages.

onset' is not necessarily the same as a reversal reaction in the nerve and, therefore, there may be several causes of SN.

First, the incidence rate of reversal reaction in borderline lepromatous patients was much higher than in lepromatous patients,[12] while the incidence rate of SN was higher in lepromatous patients (rate ratio 3·0 (0·81–11·2)). Secondly, 'cross-over reactions' occurred between reversal reaction and SN, as well as between ENL and SN. It would be conceivable that in some patients reversal reactions would only be manifest in the nerves, e.g. because skin lesions have 'burned out' during treatment. The latter could explain why some patients may have a reversal reaction at the beginning of their treatment, followed later by SN. But although this latter pattern is more common (Table 4), several patients first had SN followed by a reversal reaction with skin reaction. It is also difficult to believe that BL and LL patients would have more antigen in their nerves

**Table 6.** Risk factors for SN among 536 patients in GPH

| Risk factor | Odds ratio | *p*-value |
|---|---|---|
| Extent of clinical disease: | | |
| > 10 skin lesions | 1·2 (0·67–2·0)[a] | 0·60 |
| > 3 nerves enlarged | 3·0 (1·4–6·3)[a] | 0·004 |
| > 2 body areas involved | 2·8 (1·3–6·0)[a] | 0·010 |
| Bacteriological index | 1·9 (1·1–3·1)[b] | 0·015 |

[a] Odds ratio adjusted for age, sex and bacteriological index, [b] adjusted for age, sex and extent of clinical disease.

than in their skin, causing a neural rather than a skin reaction. The 'burned out lesion option' is unlikely because the majority of SN episodes were diagnosed at the beginning of treatment or during the first year (Table 3). If SN were just an expression of reversal reaction (RR) it remains difficult to explain why RRs sometimes occur in the skin, sometimes in skin and nerves and sometimes in the nerves only. The nature of SN can only be investigated (immuno-)histologically on nerve biopsies and by study of immunological markers during episodes of such neuropathy.

It has been shown that peripheral nerves of leprosy patients, particularly those of the lepromatous type, may already be affected subclinically at an early stage in the disease process.[13–15] Schwann cell pathology, particularly in unmyelinated fibres, is one of the first pathophysiological processes to occur in leprous neuropathy.[16–18] In addition, it is said that fibrosis following inflammation of the nerve tissue can be a further cause of neural impairment.[15] Many investigators have reported a marked intraneural increase in fibrous tissue in leprosy.[14,15,19–21] In advanced patients Job[14] found that nerve tissue was sometimes completely replaced by collagen, leaving the nerve as a nonfunctional fibrous cord. Charosky *et al.*'s[15] proposition that epineurial fibrosis leads to increased intraneural pressure and, thus, through ischemia to further 'neurological deficit', seems plausible. Despite this, there is no direct evidence that fibrosis actually leads to further nerve damage. Both these postulated early and late causes of functional impairment are strictly speaking not neuritis, i.e. inflammation of the nerve. We propose therefore to name the clinical phenomenon of silent impairment of nerve function with the descriptive term 'silent neuropathy' rather than to use the term 'silent neuritis'.

Besides Schwann cell degeneration and intraneural fibrosis there are at least 2 immunological processes that may cause SN. The first one is a cell-mediated immune reaction probably involving cytoplasmic antigens of *Mycobacterium leprae*. Although the immunological mechanism may be the same, lymphocytes from patients with a 'skin reaction' respond to different antigens (surface antigens) than those with a 'nerve reaction' (cytoplasmic antigens).[5] Because this neural reaction is a cell-mediated immune process, it has been classified by some investigators as a 'reversal reaction in the nerves'.[22] The second immunological reaction would be intraneural ENL. The occurrence of ENL lesions in peripheral nerves during an ENL reaction has been described by Pearson & Ross.[23] Whether this also occurs during episodes of SN has to our knowledge not been reported. As ENL lesions are usually tender, it seems unlikely that intraneural ENL would produce silent neuritis.

## TREATMENT

In the clinical setting it will often not be clear which pathological process is causing a given episode of silent neural impairment and, indeed, it may well be that one or more of the suggested processes occur simultaneously and contribute together to the observed impairment. For the leprosy worker the difference is not so important, providing that he or she is actively looking for SN, because neural impairment in both types of reaction responds to treatment with corticosteroids.

With a prevalence of 6·8% and an incidence rate of 4·1/100 PYAR, SN was a common phenomenon among our patients. We found that during a SN episode in BL/LL the patients' neural impairment sometimes responded well to treatment with

thalidomide only (unpublished observation). The SN patients who were treated with steroids showed good improvement in both sensory and motor function. The finding by Srinivasan *et al.*[1] that the extent of neural impairment has a prognostic value for the outcome of treatment, was confirmed by our data. Nerves with complete loss of function or which were classified 'bad' were much less likely to recover to 'good' function, than those whose function was still 'moderate' at the beginning of treatment (data not shown).

### RISK FACTORS

The extent of clinical disease as expressed by a count of the number of body areas involved in the disease, the number of enlarged nerves or the bacteriological index of the initial skin smear, was the only factor associated with a significantly increased risk of SN. This was not surprising as we have shown in a previous study that 'extensive disease' was a risk factor for any type of neural impairment. The suggestion by Parkhe *et al.*[4] that treatment with MDT (as compared to DDS treatment) would give an increased risk of silent neuritis was not confirmed by our data.

### ASSESSMENT OF NEURAL FUNCTION

Since regular assessment of neural function plays such a crucial role in the detection of silent neuropathy, the most sensitive methods that are operationally feasible should be employed. Recently, we have reported good results in the monitoring of sensibility of the hand and foot using Semmes–Weinstein monofilaments and moving 2-point discrimination.[24] We believe that such instruments should be made widely available and that training should be given in their appropriate use to all health workers involved in the treatment of leprosy patients. If standardized filaments are not available, they can be made from locally available materials such a suture or fishing nylon, but straight nylon is to be preferred.[25–27] Until the use of monofilaments has been implemented, the 'ballpen test' probably remains the best alternative.

## Conclusions

SN is a common complication in Nepali leprosy patients.

Some epidemiological evidence suggests that SN is not equivalent to a 'RR expressing itself in the nerves'. Further (histopathological) investigation is needed.

Patients with more extensive clinical disease were at increased risk of developing SN. These patients should have their nerve function assessed at every clinic visit at least during their first year of treatment.

Regular nerve function assessment, using the most sensitive tests available, is essential to detect SN at an early stage and to prevent permanent nerve function impairment.

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# Social problems of women leprosy patients—a study conducted at 2 urban leprosy centres in Delhi

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*Summary* Leprosy seems to afflict women less commonly than men, but for cultural reasons this difference may be more apparent than real. Unfortunately, the effects are as equally devastating, if not more so, in women than in men. This study, carried out at the Urban Leprosy Centres of Safdarjung Hospital and Dr Ram Manohar Lohia Hospital in Delhi, showed that the impact of stigmata attached to leprosy had more effect on educated women belonging to a higher socioeconomic group than on less fortunate women. Discriminative attitudes were more common in joint than nuclear families. Although many got support from their families, the disease had definite psychological effects. Because of the fear of infecting the family members, women sufferers kept themselves aloof and were constantly worried about divorce. Fear of social ostracism prevented the disclosure of disease to the community. Deformities and disabilities led to a deterioration in their functional capabilities and their psychological state of mind. Pregnancy did not affect regularity of treatment. Many women needed an escort to attend the clinic. Solutions to minimize some problems have been suggested.

## Introduction

Leprosy is a major endemic disease in India. It has the second highest prevalence rate of 3/1000 after Myanmar, where it is 15/1000.[1] Of the 10–15 million estimated leprosy patients in the world, India has the most, with 4 million.[2] Every year 470,000 new cases are detected in India alone.[3] But it should be emphasized that the magnitude of the problem is insufficiently reflected by mere statistics. Sensory-motor deficits in leprosy resulting from peripheral nerve damage manifest as anaesthesia, paralysis and the loss of the function of the hands, feet and eyes. Deformities are the main stigmata, leading to social, psychological and economic problems for patients and their families.

Although morbidity due to leprosy does not differ in men and women, it is the latter who form the most socially vulnerable group. Marriage is difficult and acceptance is not total.[4] The average rate of divorce that was mainly due to leprosy was 14·4% in Saudi

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women.[5] Under present Indian law leprosy is still grounds for divorce. The Muslim Marriage Act 1939, Special Marriage Act 1954 and the Hindu Marriage Act 1955 provide clauses for separation and divorce on grounds of a spouse suffering from the disease.[6] Leprosy has not only physical but also wider social implications associated with cultural, psychological and economic factors. In Turkish women,[7] besides social, cultural and economic problems, those with leprosy suffered more from stigmata.

In India[8] women could not work as efficiently as other female members of the family due to disabilities, deformities, or even opposition from family members. As a result, they lost their individuality within the family. Another study[9] reported that a sizeable proportion experienced problems if they had disabilities of grade II or more. Prejudice emanated mainly from neighbours, relatives and members of the immediate family.[10]

The presence of disabilities and deformities constitutes a major problem in the management of leprosy. However, disabilities were found to be less common in women than men,[11–13] the male : female ratio being 4 : 1.[13] This has partly been attributed to a lower incidence of lepromatous leprosy in women,[14] since the disability rate in lepromatous patients is the same in both sexes.[15]

Depression, anxiety and psychosomatic symptoms have been recorded,[16] and the associated social stigmata act as psychosocial stressors.[17] A higher suicide rate in leprosy patients as compared with the general population has been reported.[18] Community surveys in high endemic areas of India have reported higher psychiatric morbidity rates of 99/1000[19] and 63/1000,[20] arising mainly from physical disability. Specific concerns of leprosy patients include the disease outcome, future security, and loss or change of job. In addition, patients feel guilty, diffident and inadequate.

The present study attempts to identify the nature of the problems faced by women from the northern states of India suffering from leprosy.

## Hypotheses

The following hypotheses have been formulated and tested.

Women with leprosy, irrespective of their socioeconomic status, face social and psychological problems.

Leprosy causes strained relationships between the patient and her husband as well as with other family members.

The nature of the familial structure may influence the incidence of discrimination.

The psychological impact of the disease is directly related to the level of deformity and the visibility of patches.

## Subjects

This cross-sectional study was conducted on 50 women leprosy patients in 2 Urban Leprosy Centres located in New Delhi. Patients who had been treated for a minimum of 6 months, irrespective of the type of disease, whether paucibacillary or multibacillary, were included in the study. They were residents of Delhi, and the adjoining states of

Uttar Pradesh, Bihar and Rajasthan. There were 2 patients between 0 and 14 years old, 40 patients between 15 and 40 years old and 8 patients were over 40 years old.

## Method

Information was derived from interviews using a schedule designed to test the 4 hypotheses. The validity and reliability of the questionnaire was tested in a pilot study in which 5 patients from each hospital participated. Some amendments were made and the information gathered was also incorporated in the analyses. The final version is given in the Appendix. The interviews were conducted in the clinic by the first author who is a qualified medical social worker. The questions were translated into the local language and explained verbally to all the women leprosy patients included in the study so as to gather appropriate information.

The disability grading recommended by WHO[21] was used. Statistical analysis was carried out by the $\chi^2$ test using the statistical package TADPOLE III.[22]

## Results

The results are summarized in Tables 1–5. Table 1 gives details of family income, and the patient's educational, occupational and marital status—39 were married, 5 were widowed, and 5 were single, including the 2 children.

### RELATIONSHIP WITH MEMBERS OF THE FAMILY

Family means a group of people living together, related to each other through blood or marriage, e.g. including women's husband, children, parents and/or siblings.

As shown in Table 2, 35/39 of the husbands were aware of the disease. Of these 26/35 were cooperative, but relationships were strained with 9/35, irrespective of their educational status. In 41/50 the women's families were aware of the disease. Discrimination was faced by 10/41 patients. Out of 41 cases, 26 women were members of nuclear

**Table 1.** Profile of patients' education, occupation and marital status with family income

| Family income (Rupees/month) | Education | | | | Occupation | | | Marital status | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | I | P | S | G | Hw | Se | St | Si | Ma | Wi |
| < 1000 | 9 | 2 | — | — | 8 | 3 | — | — | 10 | 1 |
| 1000–2000 | 15 | 6 | 2 | — | 19 | 3 | 1 | 3 | 18 | 2 |
| 2000–3000 | 3 | 4 | — | 1 | 7 | 1 | — | 1 | 6 | 1 |
| > 3000 | 5 | — | — | 3 | 6 | 1 | 1 | 2 | 5 | 1 |
| Total | 32 | 12 | 2 | 4 | 40 | 8 | 2 | 6 | 39 | 5 |

I, illiterate; P, primary; S, secondary; G, graduate; Hw, housework; Se, service; St, student; Si, single; Ma, married; Wi, widowed.

**Table 2.** Educational level of patients and their family relationships

| Relationships to the patient | Awareness of disease | Attitude | |
|---|---|---|---|
| | | Cooperative | Discriminative/ strained |
| Spouse | 35/39 (89·74)* | 26/35 (74·28) | 9/35 (25·71) |
| Family members | 41/50 (82) | 31/41 (75·6) | 10/41 (24·39) |
| Education levels of patients | | | |
| Illiterate | 24/32 (75) | 18/24 (75) | 6/24 (25) |
| Primary | 11/12 (91·6) | 8/11 (72·7) | 3/11 (27·2) |
| Secondary | 2/2 (100) | 2/2 (100) | 0/2 |
| Graduate | 4/4 (100) | 3/4 (75) | 1/4 (25) |
| Family type | | | |
| Nuclear family | 26/32 (81·25) | 21/26 (80·26) | 5/26 (19·23) |
| Joint family | 15/18 (83·33) | 10/15 (66·66) | 5/15 (33·33) |

Difference in discriminative/strained attitude between nuclear and joint families not statistically significant at the 5% level. This was analysed using the $\chi^2$ test.

* Numbers in parentheses are percentages.

families, 5 of them meeting with discrimination. The remaining 15 belonged to joint families, and 5 of these faced discrimination.

### RELATIONSHIP WITH THE COMMUNITY

Communities of 16 (32%) patients were aware of their diseased condition, and 5 (31·25%) of these patients were not allowed to use common community places, 11/16 (68·8%) were allowed to interact with other members of the community, and 7/16 (43·8%) faced abuse because of the disease.

### PSYCHOLOGICAL IMPACT ON THE PATIENTS

In all, 25 out of 50 patients were afraid of infecting their families. Of the 50, 33 were illiterate and 17 had been educated (Table 3); 13 out of the 33 illiterates had this fear and 8/13 distanced themselves; 12 of the 17 educated women were afraid of infecting others, but only 6 distanced themselves.

### THE PSYCHOLOGICAL STATE OF MIND

In spite of a cooperative attitude from the husband, 10/39 (25·6%) women were worried about divorce, 31 (62%) women had a tendency to get angry over trivial matters and 35/50 (70%) were easily upset since contracting the disease; 15 (30%) of them also preferred to remain alone.

### THE PROBLEMS FACED IN TREATMENT COMPLIANCE

Out of the 50 interviewed, 30 (63·82%) had no difficulties in attending the clinic

**Table 3.** Correlation between psychological impact of leprosy and educational levels

| Educational level | No. of patients | Fear of infecting others | Self-distanced | |
|---|---|---|---|---|
| | | | Yes | No |
| Illiterate | 33 | 13 (39·39)* | 8 (24·24) | 25 |
| Primary and secondary | 13 | 8 (61·53) | 5 (38·46) | 8 |
| Graduate and above | 4 | 4 (100) | 1 (25) | 3 |
| Family type | | | | |
| Nuclear (N) | 32 | N-14 | N-8 | N-24 |
| Joint (J) | 18 | J-11 | J-6 | J-12 |
| Total | 50 | 25/50 | 14 | 36 |

Difference between illiterate and educated patients not statistically significant at the 5% level. This was analysed using $\chi^2$-test.

* Numbers in parentheses are percentages.

regularly; 18 faced the problem of travel expenses. A total of 16 (32%) adults needed an escort and 2 were dependent children.

There were 12 (24%) women who were not fully satisfied with the response to treatment due to the persistence of macules, a loss of sensation, reactions and deformities.

Of the 37 married women, 19 (51·4%) became pregnant, 16 during the course and 3 before beginning treatment; 5 (30·8%) discontinued treatment during pregnancy.

### DISABILITY GRADING AND EFFECTS ON EFFICIENCY AT WORK

There were 31 (62%) with disabilities, which were mild in 19, moderate in 9 and severe in 3; 28 women were certain that their efficiency would decline.

There were 29 women who felt ashamed or embarrassed by their physical imperfection, and 24 (77·4%) feared that people would question them about their deformity and skin patches and identify their disease. In 14 (45·6%) routine work was compromised by the disability, and 26 were concerned regarding the curability of the disease.

## Discussion

Nearly all (48/50) women in this study were of reproductive age and married. Economically, they belonged to low and lower middle-income groups, as reported in a WHO study.[23]

Illiteracy and the early drop-out of women at the elementary school level was seen in each socioeconomic stratum, indicating the low priority given to the education of women. There were 6 (out of 23) women in the middle, higher-middle and high-income groups families who attained higher education and therefore better opportunities of having economically sound positions (Table 1).

There were 42 (out of 50) women (including 2 students), who, irrespective of their socioeconomic or marital status, were engaged in household work and therefore financially dependent on their husbands and families.

**Table 4.** Relationship with other people in the family and community

| Author | Subjects | Strained intrafamilial relations | Fear of social ostracism | Social maladjustment |
|---|---|---|---|---|
| Sabesan *et al.*[17] (Madurai) | Men | 125/200 (62·5)* | — | 114/200 (57) |
| Kant[7] (Gujarat) | Men and women | 51/210 (24·2) | 72/210 (34·2) | — |
| Naik *et al*[8] (Bombay) | Women | 11/101 (11·0) | — | — |
| Naik *et al*[9] (Goa) | Women | 2/26 (8·0) | — | — |
| Kaur and Ramesh (present study) | Women | 10/41 (25·0) | 34/50 (68·0) | 6/16 (37·5) |

* Numbers in parentheses are percentages.

## RELATIONSHIP WITH FAMILY MEMBERS

Although many spouses and other family members were aware of the patients' illness and supported them in getting treatment, 9/35 had marital problems and 10/41 had strained intrafamilial relationships, as observed earlier.[8]

Over half the patients tended to hide the disease, mostly due to fear of ostracism, and had to attend to all matters relating to their treatment by themselves.

Strained intrafamilial relations adversely affected the patients. The effects varied, reflecting the social and cultural background, and literacy in a given locality. Our figures for social ostracism appeared high when compared to other studies, possibly because our study was confined to women while the other studies dealt only with males or both sexes (Table 4).

With better education there was increased awareness amongst the family members about the disease. This was lacking in patients from lower socioeconomic groups who were more concerned about their symptoms than the disease. Their low economic and social conditions did not elicit serious social reactions.

Whilst many families aware of the disease were understanding and supportive towards the patient, a significant number of patients from both illiterate and educated families faced discrimination. Families with more than 1 case of leprosy were more cooperative. In 5 such families studied, the husband who was being treated for leprosy brought his wife to the clinic when she developed it and this cooperation led to a better compliance with treatment.

There was no significant difference between patients coming from nuclear or joint families with regard to level of awareness of the disease. It appeared that there was a higher discriminative attitude to women in joint families. In South India leprosy has resulted in the break-up of joint families.[24]

## RELATIONSHIP WITH COMMUNITY

Because of the fear of social ostracism, almost double the number of the women, as compared to a previous study including both men and women,[8] hid their disease from society (Table 4).

Despite experiencing discrimination most of the patients mingled freely in the

**Table 5.** Psychological and psychosocial problems in leprosy

| Author | Subjects | Worried about divorce | Aggressive | Emotionally unstable/ solitary nature | Embarrassment due to deformity/ patches | Other people's concern | Decreased ability | Concerned regarding curability |
|---|---|---|---|---|---|---|---|---|
| Sabesan *et al.*[17] (Madurai) | Men | ND | 35/200 (17·5)† | 117/200 (58·5) | ND | ND | ND | ND |
| Price*[21] (Bombay) | Men and women | ND | ND | ND | 11/91 (12·08) | 11/91 (12·08) | 16/91 (18) | 6/91 (7) |
| Reddy[9] (Pondicherry, S. India) | Men and women | ND | ND | ND | ND | 2·91% | 19·36% | ND |
| Ramanathan *et al.*[12] (Jalma, Agra) | Men and women | 55% had psychiatric morbidity (including depression, anxiety, neurotic/somatic symptoms, etc.) | | | | | | |
| Kaur & Ramesh (present study) (Delhi) | Women | 10/39 (25·64) | 31/50 (62) | 15/50 (30) | 29/31 (93·54) | 24/31 (77·41) | 14/31 (45·16) | 26/31 (83·87) |

*Over 64/91 (70%) had the combination of these reactions.
†Numbers in parentheses are percentages.
ND, Not done in the study.

community. This reflects impact of health education through the mass media. However, there still is scope for improvement to root out discrimination.

## PSYCHOLOGICAL IMPACT ON WOMEN

In all, 50% of our respondents kept themselves aloof for fear of spreading infection. This fear, arising from self-incrimination for acquiring the disease, was greater in joint families. Although of no statistical significance this fear paralleled better education. Generally people from lower socioeconomic status had a fatalistic attitude and tended to ignore self-help measures.

Many worries about divorce due to leprosy were manifested in subtle ways such as becoming upset or angry over trivial matters. This tendency affected more women from joint rather than nuclear families. Apart from constraints due to poverty and large family size, this study shows that leprosy creates an additional psychological burden on women.

The different ways in which leprosy affects the psychology of the individual have been studied by other workers (Table 5) and showed much higher figure in women than men, accounting for their aggressive behaviour. The difference between the psychosocial aspects of other studies and the present study was found to be statistically significant, probably because our study was confined to women.

It has been noted[25] that a majority of leprosy patients were physically, socially and psychologically maladjusted. Patients were found to be withdrawn and isolated. In our study 15/50 of women preferred to stay alone.

The psychological impact of other chronic infections and deformities due to causes other than leprosy have also been studied. The victims of vitiligo experienced more psychological symptoms of free floating anxiety and depression than normal healthy individuals.[26] Compared to tuberculosis,[27] the fear of social ostracism was greater amongst leprosy patients because of visible stigmata. Cancer patients were also found to be depressed, using denial mechanism to ward off severe degrees of anxiety, tension and insecurity generated by the illness.[28] In a study of orthopaedically handicapped children and their families, there was a preponderance of denial reactions as a psychological defence mechanism in facing reality.[29] Recognizable psychological disturbances in children and their parents have been recorded after burns.[30]

## PROBLEMS FACED IN TAKING REGULAR TREATMENT

Treatment of leprosy is prolonged and raises several problems for ensuring compliance. To alleviate this, we provided railway concession forms and some monetary aid. However, this proved insufficient to cope with financial limitation in the lower socio-economic group.

Another important factor is the availability of a family member to take the patient to the clinic. In our study 16/50 patients were accompanied by a family member. This is particularly desirable during pregnancy since we found that nearly half of the women in the reproductive age group became pregnant during the course of the disease. Although the majority continued with their treatment, few (3/19) discontinued from fear of adverse effects on the child, leading at times to medical termination of pregnancy (MTP).

### PSYCHOSOCIAL PROBLEMS RELATED TO THE PRESENCE OF DEFORMITY/PATCHES

For a lay person leprosy is synonymous with disfigurement that sets the patients socially apart from others. Many (29/31) women in the present study with either patches or deformities felt embarrassed and feared attracting the attention of others to their physical imperfections. This proportion is a little higher than that conducted in a similar city in which 27% (25/91) of the sample group consisted of women.[31]

Almost half (14/31) the patients faced problems at work due to anaesthesia and deformities, worsened by complications like ulceration, paralysis, the wasting of muscles and the mutilation of body parts. Patients were suspicious whether the disease could be cured and expressed apprehension of progression of deformities.

### DISABILITY AND ITS EFFECT ON WORKING EFFICIENCY

Two-thirds of the patients with deformity had mild disability. The severity of disability, pain and repeated ulceration directly affected efficiency, similar to a previous report.[31]

## Solutions

It can be seen from the study that social and psychological problems faced by women suffering from leprosy were mainly due to the associated social stigmata. The attitude of the husband and family even after cure influences the psychological milieu of the patient immediately from the making of the diagnosis of the disease. Hence, it is essential to educate not only the patients, but also families and communities. Emphasis should be given to bringing about changes in attitude and practices. With the family and society accepting the patients, they are more likely to come forward for early and regular treatment, thereby minimizing or preventing deformities and disabilities.

Social and psychological support should be given importance. Counselling has been recognized as desirable but not been implemented adequately. It is essential in maintaining motivation to comply with treatment, and combating social and psychological problems of daily living since anaesthetic areas, hypopigmented patches and deformity that persist after treatment may raise social barriers.

Nearly half of the patients attending the ULCs have to travel a long way for treatment. In India, women face difficulties in continuing the treatment if it is not provided nearby. In any control programme facilities for treatment should be at accessible distances for women and children. In addition, financial assistance should be provided for reconstructive surgery and occupational therapy.

## Conclusions

Women with leprosy face definite social and psychological problems irrespective of their socioeconomic status.

Stigmata may result in strained relationships between the women, their husbands and family members.

Discriminative attitudes and fear of spreading infection are more common in joint families than nuclear ones.

Greater notice is taken of stigmata among the educated class.

The presence of deformities or visible patches have a bad psychological impact upon the patients.

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## Appendix

SUBJECT—SOCIAL PROBLEMS OF WOMEN LEPROSY PATIENTS

Interview Schedule

Case No. Date

Diagnosis

I. *Identification data*
A. *Individual information*

1 Name _____
2 Age_____ Sex _____
3 Address _____

4 Educational status_____
5 Marital status—Single/Married/Separated/Divorced/Widowed
6 No. of children (if any)_____
7 Occupation_____ Income (if any)_____
8 Status in the household_____
9 Caste _____
10 Religion _____
B. *Household information*
11 Household income (approx.)_____
12 Particulars of the household members _____

| Name | Sex/Age | Relationship | Education | Occupation | Income (if any) |
|---|---|---|---|---|---|

II *Information on disease status*

13 Deformity status due to leprosy (if any)
14 Presenting complaints
15 History of other chronic diseases (if any)

III *Interaction with household/society*
A *Interaction with household members*

16 Are members of the family aware or not aware of the disease?
Aware / Not aware
17 Is there any discrimination within the family due to the disease?
Discrimination / No discrimination

(i) Do they find fault only with you more frequently?
Yes / No

(ii) Are you abused (due to leprosy) while taking part in family matters?
Yes / No

(iii) Do the family members avoid taking food with you?
Yes / No

(iv) Are you prevented from using common articles of daily use?
Yes / No

(v) Are you allowed to participate in the family functions?
Yes / No

(vi) Do the family members hesitate in mixing with you?
Yes / No

(vii) Are you allowed to sleep in the same place as your family members?
Yes / No

(viii) Is your bedding kept with the bedding of the family?
Yes / No

(ix) Do the family members refuse to wash your clothes?
Yes / No

(x) Do the family members refuse to wash your utensils?
Yes / No

(xi) Do the family members ask you to cook separately?
Yes / No

(xii) Were you distanced by the family members/relatives after contracting the disease?
Yes / No

(xiii) When you are sick, do the family members take care of you or are you left alone?
Take care / Left alone

(xiv) Are you taking part in important decision-making matters in the family?
Yes / No

*Relationship with the spouse*

18 Does your spouse stay with you?
Yes / No

19 If yes, how are your conjugal relationships with him at present?

20 If divorced, give reasons?

21 Were you allowed or not allowed to breastfeed your children to be close to them?
Allowed / Not allowed

*Interaction with community people*

22 Are your friends/villagers/neighbours aware or not of your disease?
Friends —Aware / Not aware
Villagers —Aware / Not aware
Neighbours—Aware / Not aware

23 Do the community people prevent you from using common places? like,

| *Temple* | | | *Well* | | | *Tank* | | |
|---|---|---|---|---|---|---|---|---|
| Yes | / | No | Yes | / | No | Yea | / | No |

24 Do the community people allow you to sit with them when partaking feasts?

Yes / No

Few—

Mostly—

All—

25 Do the community people constantly abuse you or not, and do they or do they not ask about your disease?

Yes / No

*Interaction with the colleagues/employers at work place*

26 Are you continuing with the same job that you were doing before contracting the disease?

Yes / No

27 Are your colleagues/employers at the work place aware or not aware of your disease?

Colleagues— Aware / Not aware

Employers— Aware / Not aware

28 Do you or do you not face problems in getting work in the village/locality?

Yes / No

29 Does your employer pay you less or the same as other workers?

Yes / No

30 Do you get common facilities from your employer like the others?

Yes / No

31 Do your colleagues take or refuse to take food with you?

Yes / No

32 Do the colleagues maintain a distance in the work place?

Yes / No

33 Do your colleagues try to influence your employer to dismiss you from your job?

Yes / No

34 If yes, state reasons.

35 Are you or are you not allowed to take paid or unpaid leave to attend the clinic?

Allowed / Not allowed

IV *Psychological problems*

36 Do you think you are cured now (for those released from treatment)/getting cured (for those taking treatment)?

Yes / No

37 If no, why.

38 Are you or are you not worried about the infectivity of the disease?
Worried / Not worried

39 Have you or have you not distanced yourself from the family after contracting the disease?
Yes / No

40 If yes, state reasons.

41 Are you or are you not worried about divorce?
Worried / Not worried

42 Do you or do you not become angry over trivial matters?
Yes / No

43 Do you or do you not become upset easily?
Yes / No

44 Do you or do you not prefer to stay alone?
Yes / No

*Level of communication*

45 Do you hesitate to tell your problems to your family members/doctor/social worker?
Yes / No

46 Are you able to come to the clinic on your own or do you depend on others to bring you to the clinic
Alone / Accompanied

V *Problems of treatment compliance*

47 Are you attending clinic regularly?
Yes / No

48 If no, state reasons.

49 Are you taking your medicine regularly?
Yes / No

50 Do you take care of your hands, feet, eyes, as advised?
Yes / No

51 Are you able to perform your desired duties after contracting the disease? like
—Household work
—Job, if employed
Yes / No

52 Is there any chronic ulcer?
Yes / No

53 If yes, since when and how did you get it?

VI *Economic problems*

54 Do you get problems in attending clinic regularly because of the travel expenses?
Yes / No

55 If dependent, do the family object to giving you the money for travel?
Yes / No

56 If employed, have you been able to perform your required duties after contracting the disease?
Yes / No

57 Are you able to earn same/more/less after contracting the disease?

58 Are you losing your work days/daily wages for attending clinic/due to disease complications?
Yes / No

*Complications due to deformities*

59 Do you or do you not feel embarrassed by physical imperfection?
Embarrassed / Not embarrassed

60 Do you or do you not fear that people would ask about the deformity?
Fear / Not fear

61 Do you or do you not fear that people would recognize the disease by seeing the deformity?
Fear / Not fear

62 Do you find problems while working because of deformity? Explain.

63 Are you concerned about the future? Explain.

VII *Physiological problems*

64 Have you been pregnant since contracting the disease?
Yes / No

65 Did you continue with your treatment during pregnancy?
Yes / No

66 Did you breastfeed your child?
Yes / No

67 If no, state reasons?

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# Integrating leprosy control into primary health care: the experience in Ghana

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*Summary* Integration of leprosy control into primary health care is the most comprehensive and permanent system of delivering care to leprosy patients. But so far only a few countries have adopted this approach, largely on account of a fear of failure.

Over the past decade Ghana has developed a model approach towards the transition from a vertical to an integrated programme. The highlights of our approach included the development of the leprosy service as part of the overall development of the health service, increasing capacity building for leprosy control at the district and subdistrict levels as well as the establishment of a regular and effective monitoring to identify and correct operational problems early.

This paper describes the principles behind the integration, the strategies adopted and how they were implemented. It also includes the achievements made as well as the problems that were encountered and how they were solved.

## Introduction

The Ghana Leprosy Service has developed a model approach towards the integration of leprosy control into the mainstream primary health care programme. For most of the decade this new initiative has been in existence the programme has recorded a number of successes. During this period much experience has been gained in tackling the problems that accompany the transition from a vertical to an integrated programme. It is hoped that countries that have not yet taken the initiative towards integration and those which have already implemented this will benefit from these experiences.

## Background information

Ghana lies along the west coast of Africa. It is bounded by 3 francophone countries: Burkina Faso to the north, Côte d'Ivoire to the West and the Republic of Togo to the east. The country covers an area of 238,533 square kilometres.

The estimated total population in 1993 was 16 million. A little over 60% of these live in the rural areas and 46% are children aged under 15 years. The annual per capita income is US $400 and the doctor/population ratio is 1:12,500.


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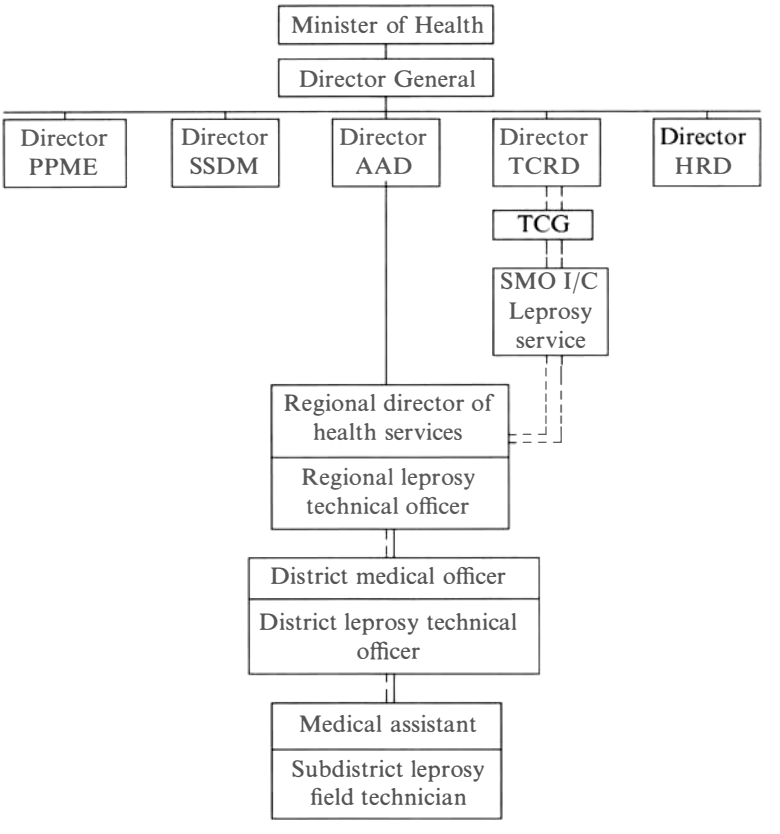


- - - - Line of technical supervision in the region. —— Line of administrative supervision. ==== Line of technical supervision at headquarters. PPME; Policy planning monitoring and evaluation; SSDM; Stores, supplies and drug management; AAD; Accounts and administration division; TCRD; Technical coordination and research division; HRD; Human resource division; TCG; Technical coordination group.

**Figure 1.** Structure of the Ministry of Health showing the new line of management of the leprosy service.

Administratively there are 10 regions with 110 districts; at the national level the country is governed by an elected government. In line with the decentralization policy the role of the districts has been strengthened by the creation of a district assembly. The assembly is made up of the executive and legislative wings. The latter comprises representatives from various constituencies in the district. Thus the district assembly is responsible for the overall development of the district, including formulation of programmes, strategies and resource mobilization.

## Ministry of Health

The Ministry of Health has undergone considerable organizational change in recent

times. The primary objective of these changes has not only been the decentralization of the health service but also to make services more effective and efficient. The current structure of the ministry is illustrated in Figure 1.

Under the current structure the former posts of Principal Secretary and Director of Medical Services have been combined in the Director-General of Health Services, who is responsible for the overall management and technical direction of Ministry of Health activities.

In the current structure the Director-General is directly served by 5 directors: Director for Accounts and Administration Division (AAD), Director for Policy Planning, Monitoring and Evaluation (PPME), Director for Technical Coordination and Research Division (TCRD), Director for Human Resource Division (HRD) and Director for Supplies, Stores and Drug Management (SSDM).

Each region is headed by the Regional Director of Health Services (RDHS), who is responsible directly to the Director-General. The district health team is headed by the District Medical Officer who is responsible to the Regional Director for Health Services.

## Leprosy Control Programme

The Ghana Leprosy Control Programme has been in existence since the late 1940s. From the onset, the programme was run vertically with its headquarters at the Ankaful Leprosy Hospital, which is about 150 km from the capital, Accra. There are 4 leprosy hospitals.

Under the vertical programme the administrative and technical aspects of the programme were the direct responsibility of the Senior Medical Officer in charge of the programme. With integration, many of these functions have been devolved to the regional, district and subdistrict levels. The prime responsibilities of the headquarters are now:

to assist in the development of national policies related to leprosy control;
to monitor and evaluate regional programmes and give technical advice and support where necessary;
to liaise between the Minister of Health and donors on the type and level of support needed, as well as channel donor support to the regions; and
to develop health education materials on leprosy as well as train trainers in the regions in leprosy control.

The prevalence of leprosy has decreased from over 20,000 in 1983 to 2,155 in 1993. This gives a current national prevalence rate of 1·4 per 10,000. The case detection rate fell from a level of 20 per 100,000 to 10 per 100,000 in 1988. Since then this level has been maintained. The proportion of new patients with disability grade 2 stands at 5%.

Multidrug therapy (MDT) was introduced in 1984 and 100% MDT coverage was achieved in 1991.

## Events leading to integration

In Ghana, a number of events necessitated the integration of leprosy control into the mainstream health care. These were:

In 1978, the Alma Ata Conference ushered in the primary health care concept. This concept was embraced wholeheartedly by Ghana. However, to fully implement the programme, it became necessary to reorganize the ministry structurally and functionally. The resulting changes effectively abolished the vertical programme, because many of their functions were devolved to the district, subdistrict and regional levels.

In 1981 the ministry stopped the training of specialized leprosy technical officers. The training of multipurpose technical officers for epidemiology had started, so it became unnecessary to continue training officers solely for leprosy control.

In the early 1980s the country suffered its severest setback in its balance of payments. Resources for health programmes were severely curtailed, thus bringing to light the inefficiency of the vertical programmes. It was, therefore, anticipated that unless alternative approaches were adopted to control leprosy, the programme could suffer a severe setback.

## Principles behind integration

The approaches that we adopted towards the integration of leprosy control were founded on the following principles. First, leprosy control is an integral component of the health service. Consequently, the changes that are made in the leprosy service should keep pace with changes in the health service as a whole. Secondly, regions have different health care needs and resources, different levels of health care development as well as different sociocultural environment and geography. Therefore, for activities which could be severely hindered by some of these factors, it is essential that some regions be allowed to move along at a slightly slower pace.

## Laying the foundation

For the effective implementation of primary health care it is essential to develop the health service at district level. It was recognized that the basic unit for health planning, budgeting and management was the administrative district, and all components of health work should be fully coordinated at district level.

In this regard, since the mid-1980s, a number of initiatives have been introduced by the ministry which have strengthened health care management at district level. These included:

the appointment of substantive district medical officers who would be responsible for the overall management of health programmes in the districts;

the creation of the district health management teams made up of the heads of health programmes and other health-related agencies;

strengthening capacity building at the district level. This included management courses for all members of the district health management teams;

strengthening logistical support to districts, especially the provision of transport for health activities;

introduction of management information systems for monitoring and evaluating performance as well as developing policies; and

training in health systems research for members of the district health management teams.

**About the Registration Numbers**

The first 2 digits, which range from 1 to 10, identify the regions, which are coded in alphabetical order.

The next 2 digits—i.e. the 3rd and 4th—identify a particular district; they range from 1 to 18, according to the number of districts in each region. Again, the districts are coded in alphabetical order.

The fifth digit identifies the health facility in the district, where a leprosy patient goes to receive anti-leprosy drugs. The coding is done according to whether there are leprosy patients within the catchment areas of the health facility. This digit ranges from 1 to 9, as there is no district with more than 9 recognized government health facilities.

The last 3 digits identify the patient.

**Ghana Leprosy Service, Ministry of Health, Epidemiology Division**

Leprosy Treatment Register, Region

About the registration numbers:

The first two . . . . . .

The next two . . . . . .

The fifth . . . . . .

The last three digits

No. ☐☐☐☐☐ DISTRICT ..... CLINIC ..... YEAR .....

| PT. NO. | NAME | SEX | Address House. No. Village | YOB | CLASS. | Date Start. | Att. Rate B/F | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | AH Rate TOTAL | Skin Smear Date | Skin Smear Results | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | | | | | | | | | | | | | |
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| | | | | | | | | | | | | | | | | | | | | | | | | |

**Figure 2.**

**Implementation**

The foundation for integration having been laid at the district level, the next task was the devolution of some of the functions of the headquarters of the leprosy service. To ensure a smooth transition, however, it was necessary that it was done in phases.

In the first phase, the administrative functions of the programme were devolved to the regions. This in effect meant that regions were made responsible for supply of fuel for leprosy programmes, staff salaries and promotion, discipline of staff, etc.

In the second phase the main area of emphasis was the preparation of health care managers and providers for the technical management of leprosy control within their respective regions. It was recognized that most health care managers and providers were not so well versed in leprosy control as to be capable of managing the disease within their areas of operation. Consequently, it became necessary to conduct training in leprosy control for all health workers whose duties were directly related to leprosy control.

The training focused on 4 key areas:

Training of trainers in health training institutions. Currently, leprosy control has been incorporated in the curricula of all health training institutions, including medical schools.

Training of health care managers in leprosy control. Training has been organized for Senior Medical Officers in charge of public health in the regions as well as members of district health management teams.

Training of health care providers at the subdistrict level. A manual had been developed specifying the roles each category of health worker would play in leprosy control as well as the knowledge and skill required to perform such roles. The staff who were selected for such training included Medical Assistants, Public Health Nurses, Community Health Nurses, Health Inspecting Assistants and Epidemiology Technical Officers.

Training of members of the Regional Training Units. The training was arranged so that the regional training units trained the District Health Management Teams, who in turn trained the subdistrict staff.

In the third phase of integration the focus has been the gradual devolution of some specific functions to the district and subdistrict level, clarification of the roles of specialized leprosy technical officers *vis-à-vis* 'non-specialized' leprosy care providers as well as a systematic monitoring and evaluation of regional, district and subdistrict programmes.

The first task which was devolved at the subdistrict level was the administration of antileprosy drugs. To assist record-keeping, however, a simple treatment card was designed for the health centres (Figure 2). Subsequently, these roles have been broadened to include the diagnosis of leprosy, health education, case-finding, case-holding, surveillance, and medical care. Because most Medical Assistants have not yet become fully competent in the diagnosis of leprosy, currently all suspected cases have to be confirmed by the district leprosy technical officers. In due course, however, patients will receive their treatment on the same day they are diagnosed at the health centre.

In the final phase, which has commenced in 1994, the emphasis is to increase the capacity of district hospitals to recognize and manage the complications of leprosy. With the current low bed occupancy of all the leprosy hospitals, a decision has been taken to admit general cases into all leprosy hospitals.

The role of the leprosy technical officer has been the drawing of district action plans

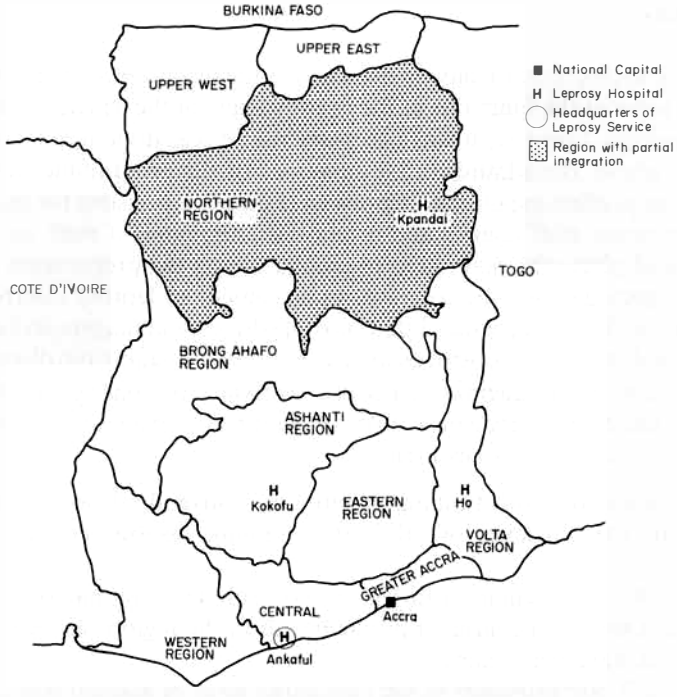


**Figure 3.**

for leprosy control, supervision of subdistrict staff, confirmation of diagnosis, performance of VMT & ST, record-keeping, supply of antileprosy drugs and footwear, slit-skin smear, etc.

## Monitoring and evaluation

During the transition phase towards full integration it is critical that an effective monitoring system be established to identify and promptly correct operational problems which may arise in the implementation of the programme. Unless regions, districts and subdistricts are given regular feedback on their performance all efforts made will be frustrated.

The headquarters monitors the regions twice a year, the regional technical officer monitors each district once every quarter and the district technical officer monitors each subdistrict once a month. A checklist was designed at the headquarters and regular feedback is given.

## Achievements

There have been a number of achievements since leprosy control was integrated into the mainstream health care:

Treatment for leprosy patients has been established in all health facilities and is administered by Medical Assistants. Currently most leprosy patients receive their drugs within 8 km of a health facility.

In total, 9 out of the 10 regions operate a fully integrated programme. In the last region—the Northern Region—integration has been slow on account of a poor health infrastructure, sparse distribution of population over long distances and poor roads. For parts of this region, therefore, some monthly domiciliary service is still administered (Figure 3).

Increasing numbers of cases have been reported by general health workers who were trained in leprosy control.

Following the massive public educational campaigns and the training of health workers in leprosy there has been a significant reduction in the degree of stigma.

Training in leprosy has been incorporated in the curricula of all health training institutions.

## Problems

Like all innovative programmes there are bound to be initial problems. A number of the problems we encountered were minor but I shall highlight the major problems which threatened the programme and the measures which were taken to deal with them:

Some leprosy technical officers felt threatened by the change, and consequently attempted to thwart it. In 1 region, 9 months after a circular had gone round for the establishment of treatment centres at all health facilities, nothing had been done and the programme was still run vertically. A national workshop was organized for all leprosy technical officers at which the important role they would play in the integrated programme was highlighted. Thereafter, they felt less threatened. It was also realized that the system of drug distribution facilitated the running of the programme vertically, and change was required.

To ensure that the technical officers sent returns to District Medical Officers, and not directly to their regional technical officers, as under the vertical programme, drugs were issued from the Regional Medical Stores to the District Medical Officers.

Initially some Regional Directors of Health Services and District Medical Officers failed to come to terms with the new responsibilities that had been devolved to them. Some of them still expected the headquarters of the leprosy service to draw regional action plans on leprosy, monitor district programmes and even discipline staff. However, the regular monitoring of the progress of integration in the regions and the feedback given have largely overcome the problem.

## Conclusions

Several approaches have been adopted towards the integration of leprosy control. The approach was adopted, i.e. the development of the leprosy service at almost the same pace as the development of primary health care has been facilitated by a number of factors. First was the commitment of the ministry towards the strengthening of the

health service for the effective implementation of primary health care. Second was the incorporation of a regular system of monitoring from the national to the sub-district levels for the early identification of operational problems. Finally, the donors displayed a great deal of flexibility towards meeting our changing needs during the transition phase.

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# Advantages, indications, and the manufacturing of melted PVC waterpipe splints

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*Summary* There are several indications when to use splints in the treatment of leprosy. PVC waterpipe is a cheap and easily available material in developing countries. Its advantages, indications, and the manufacturing of splints are described.

## Introduction

There are several indications when to use splints in the treatment of the complications of leprosy, e.g. neuritis, arthritis, ulcers and after surgery. In the past Plaster of Paris (POP) was used for this purpose and lately several synthetic materials have been introduced, such as low density polyethylene granules[1] or materials like Orthoplast which can be moulded by heating.

Leprosy remains mainly a problem of developing countries, but for the local manufacturing of splints, financial restrictions and lack of availability of materials are often limiting factors.

In this article we present our experiences gained with melted PVC waterpipe from 1987 onwards and compare this with other materials. The authors have more than 15 years' experience in hand surgery and the manufacturing of orthotics. Basic principles which are safe to apply in less experienced hands are discussed.

### DISADVANTAGES OF SOME MATERIALS FOR MAKING SPLINTS

In Nepal we previously used locally made Plaster of Paris (POP) which was cheap but of poor quality. Imported POP like Gypsona was stronger but much more expensive.

A great disadvantage of POP is that it becomes soft when applied during the humid rainy season or when moistened by wound discharge. Low density polyethylene granules are slightly cheaper but not easy to obtain in Nepal. All the other heat-malleable materials are very expensive and need to be imported.

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ADVANTAGES OF MELTED PVC WATERPIPE AS GROUND MATERIAL FOR SPLINTS

In the search for a good alternative to the above-mentioned materials we have experimented at our hospital with PVC waterpipe for the past 6 years.

Apart from the fact that it can be moulded, PVC waterpipe has proved to have a great number of other advantages: 1, inexpensive (Figure 1); 2, lightness (Figure 2); 3, durability (Figure 3); 4, availibility in developing countries; 5, ease and simplicity in processing; 6, easy to clean; and 7, strength.

An ankle–foot back slab used as a resting splint costing US $0.93, and weighing 125 g, can be made within 15 min and with daily use will last at least 3 years.

Nowadays we only use POP splints for those indications where prefabricated PVC splints cannot suffice, as the latter cannot meet specific individual requirements.

## Indications for splints

It is beyond the intention of this paper to provide a detailed description of each type of splint but merely to suggest indications, applications and risk factors in different types of PVC splints. As with all splints the actual manufacturing will also depend upon your own inventiveness, the individual requirements for each patient and the availability of other local materials. When you are in doubt about technical aspects do consult a handbook on splints or feel free to write to the authors.

| Material | Gypsona POP | Polyethylene granules | PVC waterpipe |
|---|---|---|---|
| Ankle–foot Back slab | NRs. 185.00 (US $6.17) | NRs. 90.00 (US $3.00) | NRs. 28.00 (US $0.93) |

**Figure 1.** Cost price comparison for the ankle–foot back slab in Nepali Rupees in 1991.

| Material | Gypsona POP | Polyethylene granules | PVC waterpipe |
|---|---|---|---|
| Ankle–foot Back slab | 1150 | 680 | 125 |

**Figure 2.** Weight (grams) comparison for an ankle–foot back slab.

| Material | Gypsona POP | Polyethylene granules | PVC waterpipe |
|---|---|---|---|
| Ankle–foot Back slab | about 1 month | (*) 'durable' | at least 3 years |

* In the article referred to[1] it is labelled as 'durable' but no lifespan is mentioned.

**Figure 3.** Durability comparison for an ankle–foot back slab.

**Figure 4.** The ankle–foot back slab (L), rigid chappel (M) and footdrop inlay splint (R).

RESTING SPLINTS (NONWEIGHT BEARING)

*To assist in the healing of ulcers*

The most frequent complication of leprosy is the occurrence of ulcers due to loss of sensation and/or alteration of the muscle balance in an extremity.

It is of paramount importance first to diagnose WHY an ulcer has occurred and from this to draw a plan of HOW future recurrence could be PREVENTED.

Once this has been done a plan for treatment can be made. As a part of the treatment rest of the affected tissue is advised.

Examples are:

(a) the ankle–foot back slab (Figure 4) for ulcers of the foot and ankle. Holes can be drilled to promote ventilation;
(b) the cock-up splint (Figure 5) for ulcers of the wrist and dorsum of the hand; and
(c) the volar hand splint (Figure 5) for ulcers of the fingers and at the palmar side of the hand.

*To immobilize inflamed joints and rest nerves*

Arthralgia and neuritis in general occur in association with reactional episodes.[2] Rest can support the medical treatment.

Examples are:

(a) the ankle–foot back slab for synovitis of the ankle joints or neuritis of the posterior tibial nerve. The ankle should be rested at about 30° plantar flexion;

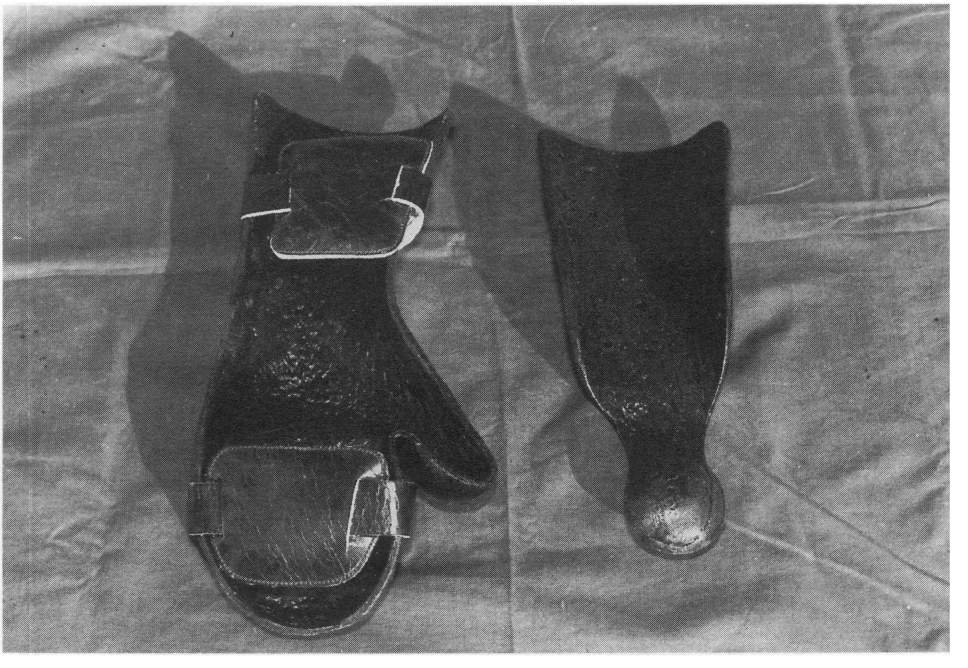

**Figure 5.** The volar hand splint (L) and cock-up splint (R).

(b) the long back slab for inflammation of the knee or neuritis of the popliteal nerve. The knee joint should be rested at about 30° flexion;
(c) the elbow back slab to rest the ulnar nerve. The best position is about 45° flexion; and
(d) the cock-up splint to rest the wrist joint and median and ulnar nerves at this level. In order not to overstretch the nerves a maximum of 10–30° dorsiflexion can be recommended.

*To immobilize (parts of) extremities after surgery*

In general this will be the case after septic surgery, arthrodesis, tendon transfers and tenotomy.

Examples are:

(a) the ankle–foot back slab to immobilize the joint either after septic surgery, tendon transfer, arthrodesis, or claw toe correction. After septic surgery a back slab is used which permits rest in 30° plantar flexion; after an arthrodesis or claw toe correction the ankle should be supported in neutral position while after tendon transfer for footdrop correction one would need 75° dorsiflexion; and
(b) the cock-up splint and volar hand splint after septic surgery on the wrist and hand.

In general muscle strengthening exercises of muscle groups of the affected limb will not be contraindicated and should even be encouraged as it promotes recuperation and can be an important psychological stimulus.

It should be remembered that stiffness will set in when normal joints are immobilized

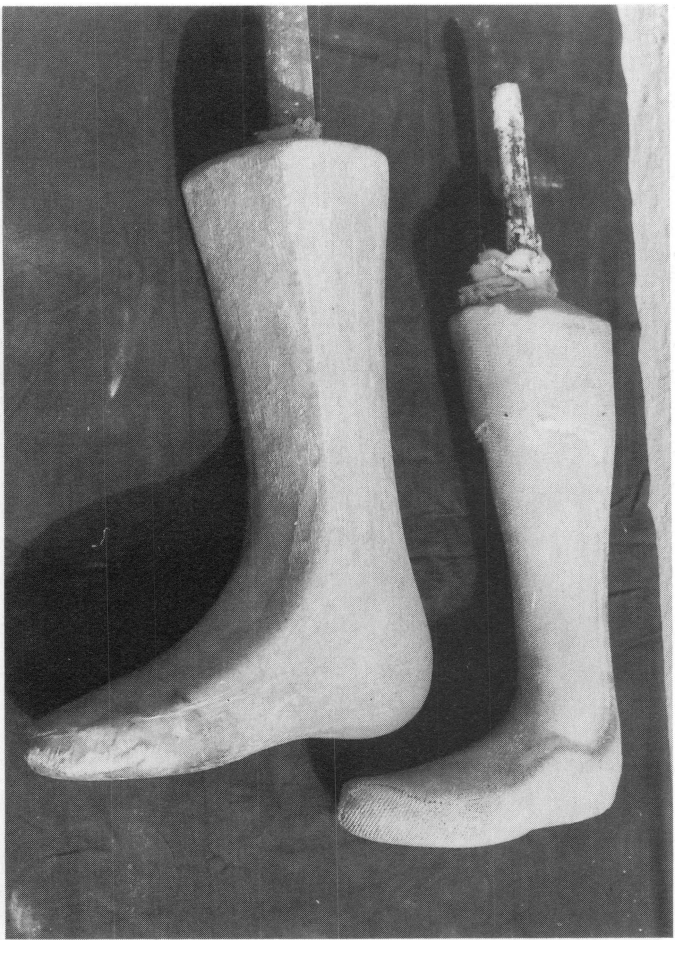

**Figure 6.** (a) Veneer impregnated POP forms for (L) ankle–foot back slab and (R) footdrop inlay splint. (b) The same for the volar hand splint and cock-up splint.

for longer than 3 weeks, and affected joints for longer than 2 weeks. When not contraindicated, controlled exercises of the immobilized joints are therefore advisable when splinting is indicated beyond these periods.

### POSITION MAINTENANCE SPLINTS

More than for any other indication one should be aware of the increased risk of undue pressure when splints are used for the reduction of contractures in extremities with diminished sensation.

The splints are applied at the end of the exercises to overcome the contracture in order to maintain the achieved result. Avoidance of sharp edges and acute bends in any material used, adequate padding, a protocol for the application and regular inspections are mandatory.

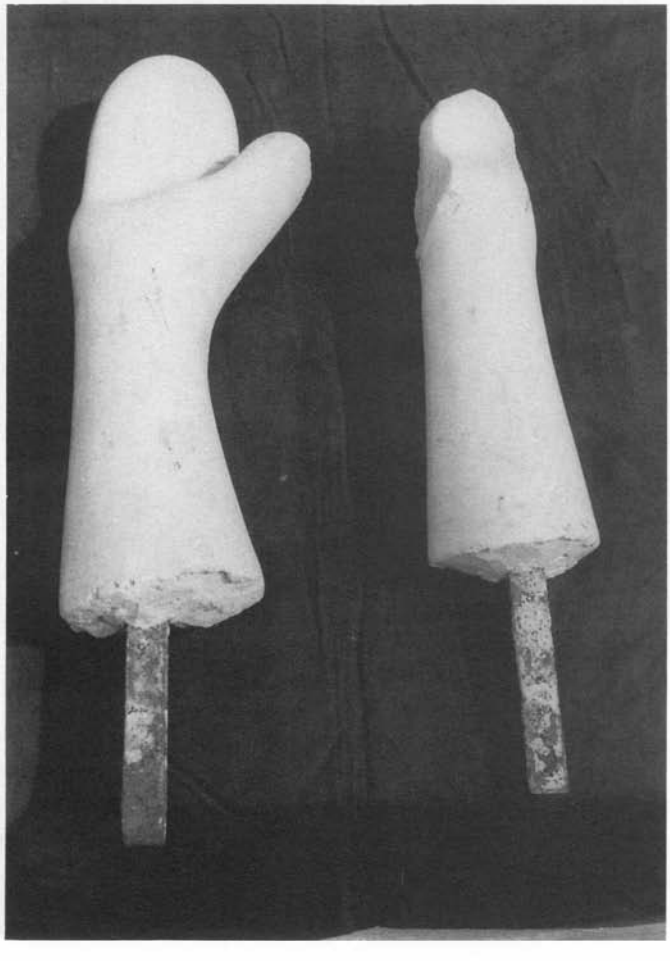

**Figure 6b.**

*To correct the contractures of soft tissues and joints*

This is mostly done in preparation for reconstructive surgery. Two types can be differentiated; the passive splints in which correction is obtained by remoulding the splint in which the hand is immobilized (types (a) to (c)), and the dynamic splints (type (d)) with springs and elastic bands which provide the correcting forces and in which movement in the joints and tendons remains possible.

Examples are:

(a) the lumbrical splint holding the fingers with the metacarpophalangeal joints flexed and interphalangeal joints extended in order to correct flexion contractures of the fingers in the metacarpophalangeal and the proximal and distal interphalangeal joints and soft tissues. Potential pressure points are at the fingertips and the dorsum of the finger joints;
(b) the volar hand dorsiflexion splint to stretch contracted flexor muscles (which often shorten in the claw hand), the wrist joint and the metacarpophalangeal joints;

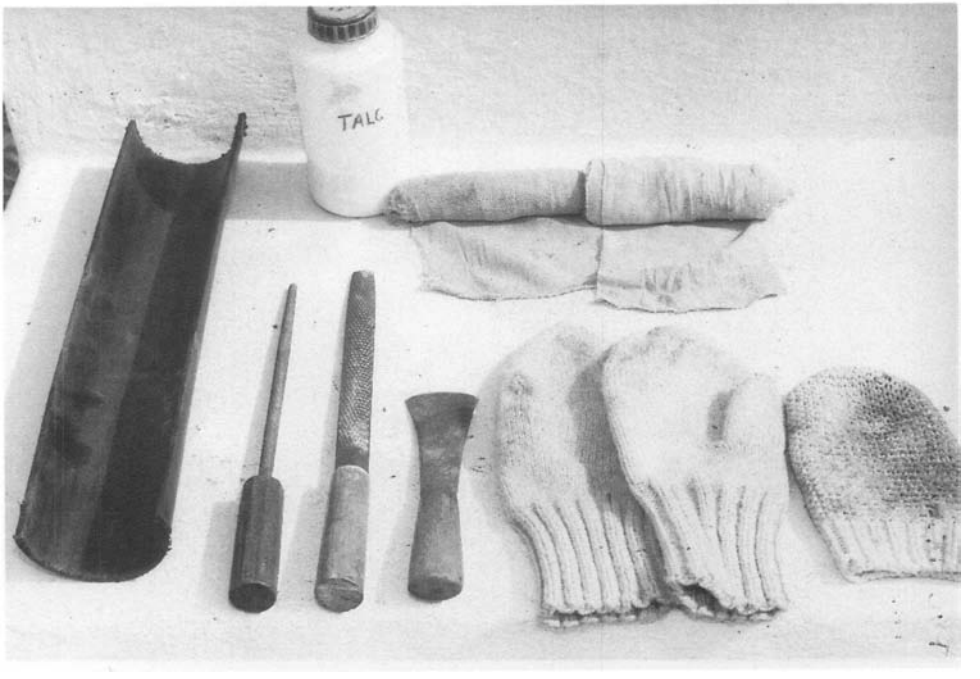


**Figure 7.** PVC waterpipe cut in half, files, knife, oven mittens (2 pairs), talc and elastic bandage.

(c) the thumb abduction splint to maintain the width of the first web-space after stretching exercises of the adductors and mobilization of the contracted trapezometacarpal joint, e.g. in preparation of an opponens plasty. A potential pressure point is the volar side of the basic phalanx; and
(d) the dynamic extension splint for dynamic extension traction of the interphalangeal joints. Potential pressure points are at the distal margin of the splint. The springs are attached with rivets about 2 inches distance from elbow level to the PVC.

## DYNAMIC SUPPORT SPLINTS

*To limit movement in specific joints while other adjacent joints are normally used*

Examples are (see Figure 4):

(a) the foot drop inlay splint to support the ankle. This splint can be inserted in most canvas shoes and a MCR insole is placed upon the splint. Further padding can be introduced by the wearing of medium thickness cotton socks. Also in warmer climates this will be kinder to the skin as it absorbs moisture that would otherwise be trapped between the skin and the PVC. Please check the remaining space left for the foot. The calf part is fixed to the lower leg with a leather strap;
(b) the rigid chappel with MCR footbed to rest the forefoot. Rigidity is obtained by drilling holes in a piece of flattened PVC after which it is stitched between the margins of

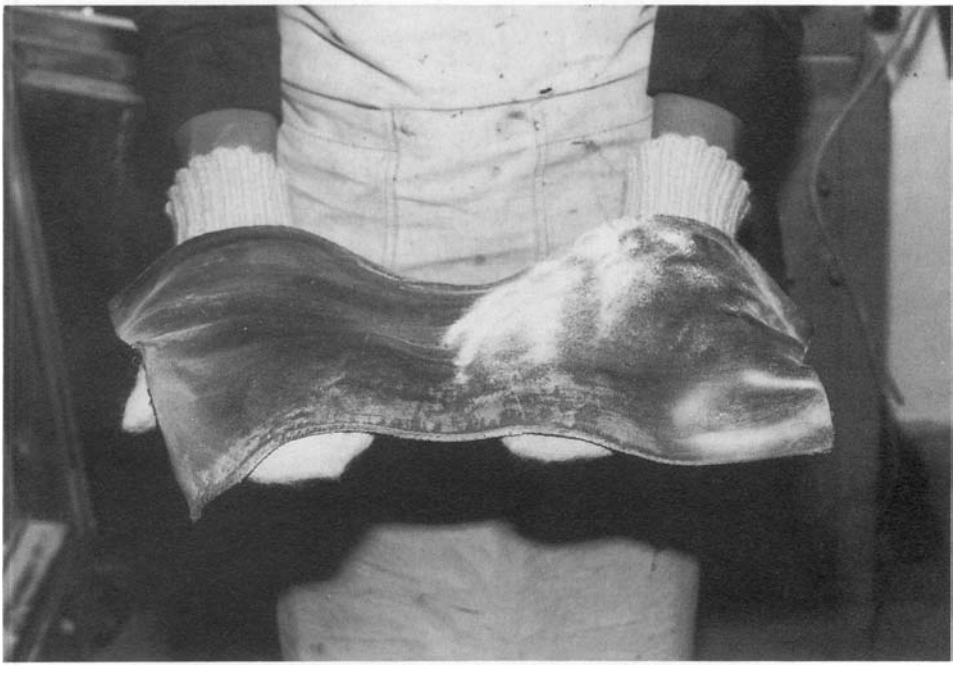

**Figure 8.** The melted PVC sprayed with talc.

the piece of tyre and the MCR as in normal MCR chappels. When used on even ground a rocker sole can be added. It is important to give a well-padded heel strap as much more strain is placed upon the skin of the ankle. In another article we hope to share our experiences with a semi-rigid moulded insole, which can be placed inside deep canvas shoes. This as an alternative for moulded boots.

## The manufacturing of splints from melted PVC waterpipe

### MATERIALS

Veneer impregnated POP or wooden forms of different sizes (Figures 6(a) and (b)). The first is used when a tailor-made splint is required and the latter for custom-made splints manufactured in series. One can veneer-impregnate POP by painting it with a veneer diluted with 5% kerosine.

PVC waterpipe of 4- to 7-inch diameter cut in half, files, knife, oven mittens (2 pairs), talc and elastic bandage (Figure 7).

Oven providing a temperature of 300°C. We use a simple, locally made oven.

### PROCEDURE

Place the PVC in the oven heated to 300°C for 15 min. Make sure that there is good ventilation in the room.

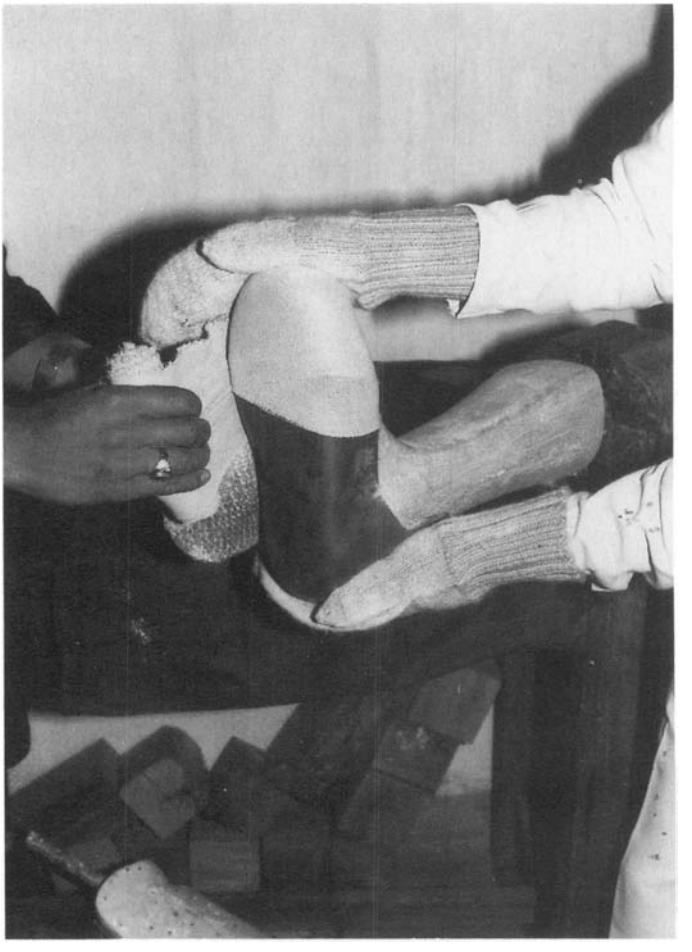

**Figure 9.** The PVC slab laid over the form and moulded to it with elastic bandage.

Take out the soft PVC slabs with the mittens and spray with talc (Figure 8).

The PVC will maintain its thickness but can be made thinner by stretching.

Place the PVC slab over the form and mould it by fixing it to the form with the elastic bandage for 5 min (Figure 9).

Cut and smooth the edges while the PVC is still warm (Figure 10).

In this phase the edges can still be slightly bent outward.

Drill holes in the splint for ventilation.

Line the splint with a layer of old blanket before applying it.

## Results and discussion

In 1987 we made 126 PVC ankle–foot back slabs which have since been in continuous

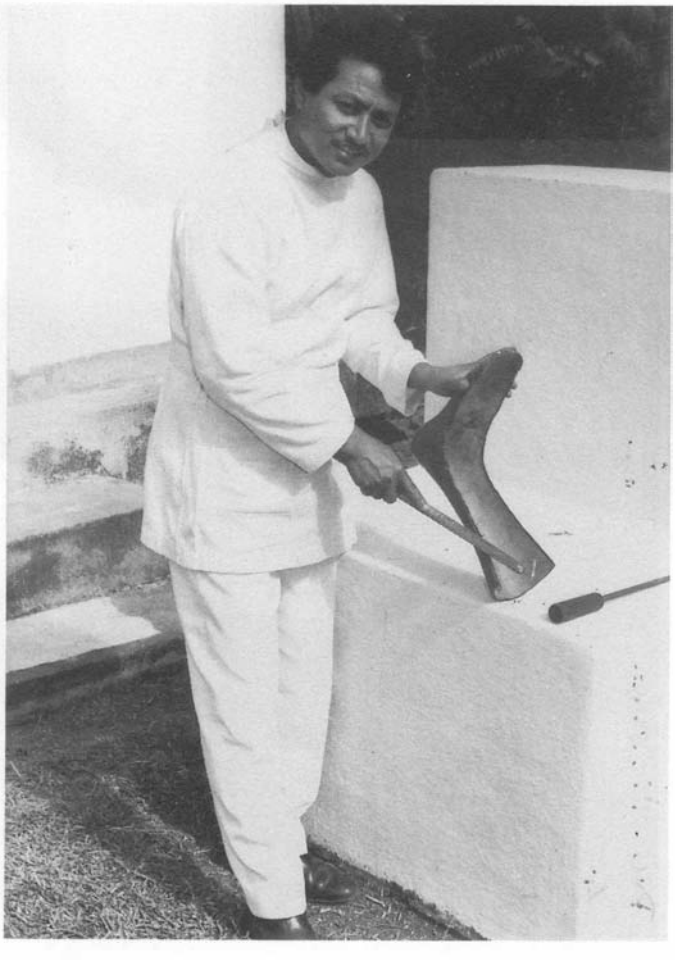

**Figure 10.** The cut edges are smoothed while the PVC is still warm.

use—6 of these were taken home by patients. We sold 25 back slabs to other centres in Nepal, and 85 ankle–foot back slabs are still in use at the Anandaban Leprosy Hospital. About a third of these are now thin at the heel as patients who cannot use crutches sometimes use their back slab for pushing their wheeled stools.

We keep using these old splints for protecting forefoot ulcers. The PVC appears not to be affected by any of the ointments commonly in use.

The footdrop inlay splints only last about 3 months, which is the same as the lifespan of a footdrop spring. Because it is a dynamic splint, not a resting splint, it undergoes more stress, especially at the ankle where it often breaks.

The great advantage of the footdrop inlay splint is the much better cosmetic aspect of the affected foot as the inlay is not visible to other people. Another great advantage of PVC is that it does not create sharp edges when it breaks. Still, just as indicated for the foot and the footwear, regular inspections of the splints are mandatory.

The footdrop inlay splints have been used in flat and hilly areas with the same result and lifespan.

Other types of resting and correcting splints of PVC are used in smaller numbers. Only those we have gained enough experience of are included in this paper.

We have not been able to use PVC for the resting splints for immobilization after tendon transfers in the hand, as balancing the transferred slips is a delicate issue calling for individual tailoring of the splint. It is of paramount importance to create a correct anatomical support for the transverse palmar arch in order to avoid undue stress to the intermetacarpal ligaments.

The introduction of PVC splints instead of Gypsona POP has resulted in a 40% reduction in the cost of making splints. The technique is now used in several centres throughout the country.

## Conclusion

Splints made at Anandaban Hospital out of melted PVC waterpipe have proved in our experience to have major advantages in many indications over splints made from other materials.

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*Lepr Rev* (1994) **65,** 396–406

# Letters to the Editor

## ANALYSIS OF COMPETITIVE EXAMINATION IN LEPROSY FOR MEDICAL UNDERGRADUATES IN BOMBAY OVER 22 YEARS OLD

Sir,

Apathy to and dislike of leprosy is still prevalent in Indian society as well as the medical profession. The attitude of the medical fraternity toward leprosy at all levels—medical students, teachers, general practitioners and consultants—needs to change totally for leprosy control programmes to be meaningful. In the past, a few sporadic efforts have been made by voluntary organizations to overcome this situation. In 1978 and 1979, 2 workshops on the 'Training of undergraduate medical students in leprosy' were conducted by the Acworth Leprosy Hospital Research Society and the Gandhi Memorial Leprosy Foundation with the Indian Council of Medical Research. The recommendations of the Workshops were submitted to universities and the National Medical Education Board, but no concrete steps have been taken except for the individual efforts of a few medical teachers. Refresher courses in leprosy for general practitioners and consultants are held by some organizations periodically. Discussing the role of 'The medical student and leprosy', in an editorial, McDougall[1] remarked that, 'it would not cost much money to radically upgrade their involvement with leprosy at the undergraduate stage'. Concerted efforts to involve medical colleges are indeed lacking on the part of the leprosy agencies even in endemic countries.

In India the number of medical colleges imparting education in indigenous medicine is 3 times those teaching allopathic medicine. In these non-allopathy medical colleges teaching of leprosy is not always done in tune with the modern scientific knowledge now being rapidly acquired on leprosy. Most of the graduates from these medical colleges practise their profession at the grass roots level, where they would have been more useful for the detection of early cases of leprosy, if proper teaching had been imparted. Recently the Hind Kusht Nivaran Sangh—Maharashtra Branch (1992) undertook training of students of such non-allopathy colleges in Maharashtra on an experimental basis. This was only in a small unit but it can be considered as a good beginning.

In this context the Acworth Leprosy Hospital Research Society has been making efforts since its inception to communicate the leprosy message to different strata of the medical profession in Bombay, such as the holding of competitive examinations for medical students. These examinations, which were initiated in 1972 in 4 allopathy medical colleges in Bombay City, were restricted to undergraduates. In 1979 they were expanded to intern groups and in 1988 further expanded to the students of 5 non-allopathy medical colleges in the city. These 3 groups were dealt with separately for judging. The students who passed and secured the first 10 ranks underwent a practical examination of leprosy on the pattern of regular medical examination.

Over the last 22 years, 710 students appeared for the competitive examination and this represents 2% of the total admission to these medical colleges during this period. In 1987 and 1993 an attempt was made to obtain a feedback from these students to judge the utility of the examination, and 82 and 67 students respectively responded (Table 1). Most respondents in 1987 were relatively well settled in their profession, whereas the same cannot be said of the 1993 sample.

**Table 1.** Results of feedback

| Questionnaire | 1987 | | 1993 | |
|---|---|---|---|---|
| | Response (%) | No Response (%) | Response (%) | No Response (%) |
| 1 Nature of examination | Complete and adequate 71 | 6 | Complete and adequate 81 | 1 |
| 2 Does examination fulfil its objects? | Yes 82 | 5 | Yes 83 | 2 |
| 3 Necessity of teaching session before examination | Yes 73 | 2 | Yes 84 | 1 |
| 4 Appearing in examination was beneficial | Yes 95 | — | Yes 93 | — |
| 5 Enough number of reference books in college library available | Yes 50 | — | Yes 51 | — |
| 6 Teaching of leprosy in medical colleges | Adequate 18 | — | Adequate 25 | — |
| 7 Knowledge acquired for preparation of examination was useful in future career | Yes 70 | — | Yes 90 | 1 |
| 8 Wish to participate in leprosy control programme | — | — | Yes 85 | 15 |

The analysis of feedback from medical students suggests that the majority of the students are happy with the nature and conditions of the examination and 90% of them found that by taking part in these examinations, they got an opportunity to obtain adequate knowledge of leprosy, and it is of special significance that 70% of them felt that they used it in their medical practice. The study shows that 76% of them were unhappy with the inadequate teaching of leprosy prevailing in their medical colleges, and 49% of them complained about the lack of reference material and textbooks on leprosy in their libraries. The results are somewhat similar in both the feedbacks obtained in 1987 and 1993, though there is a gap of 5 years between these studies.

The analysis of the special remarks obtained by the respondents suggests that special training is required by the participants before examination and the questions asked should be more objective. They further pointed out the need for the gearing up of health education for the public, especially to the medical profession and the motivation of leprosy patients for regular treatment. They also seem to be impressed by the need for vaccine and new drugs fore-shortening the duration of therapy for leprosy.

### IMPRESSIONS

This study gives the impression that though the percentage of students who took part in the competitive examinations is negligible, those that did benefited them as individuals. The leprosy programme may also have benefited in the areas of care detection and treatment. The static stage of the standard of medical education in leprosy for the last 22 years in Bombay City seems to have deprived the leprosy programme of the full potential of these undergraduate students. In view of the target to eliminate leprosy fixed as 2000 AD in our country, we feel that there is a necessity to work out the strategies to cover the entire population of medical students (allopathy and non-allopathy) adequately in respect of leprosy.

## Acknowledgment

We are indebted to LEPRA for the financial support given to hold competitive examinations for a period of 5 years between 1985 and 1991, out of the total period of 22 years.

We thank Mrs Samruddhi S. Ghosalkar and Mr P. Radhakrishnan for secretarial assistance.

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# INOCULATION OF THE *MYCOBACTERIUM LEPRAE* INTO THE HAMSTER CHEEK POUCH

Sir,

The lack of *in vitro* techniques for the cultivation of *Mycobacterium leprae* and the fact that *M. leprae* multiply and produce disease only in a limited number of species represents an important barrier to progress in leprosy research. The inoculation of mycobacteria into the footpads of immunologically intact mice remain the basic tool for assessing the activity of drugs against the bacilli. Unfortunately, this animal model has limitations because of the long duration of the experiments due to the very slow rate of growth of *M. leprae*. Immuno-deficient animals are little used in experimental leprosy due to the high cost of the animals and difficulties of their maintenance; furthermore, mortality is high before dissemination of the disease.[1]

In view of these data, we decided to study the behaviour of viable *M. leprae* inoculated into the cheek pouch of hamsters. This structure is an invagination of oral mucosa, where the lack of lymphatic drainage cuts the afferent arm of immune response.[2] In addition, we compared the histological aspects of lesions induced by viable *M. leprae* inoculated into the pouch and into the footpad, an area rich in lymphatics.

Suspensions of viable *M. leprae* were prepared from lepromatous nodules, as described by Shepard.[3] The mycobacterial identification was done through bacteria inoculation in a culture medium (Loewenstein–Jensen) and into the footpads of balb/c mice.[3]

Two-month-old male hamsters (*Mesocricetus auratus*) were divided into 2 groups. Group 1 (34 animals) were inoculated, under anaethesia (sodium nembutal, 40 mg/kg) into the submucosa of the everted pouch with 0·1 ml of a bacilli suspension containing $5 \times 10^6$ viable bacilli/ml. Group 2 (18 animals) were inoculated into the footpad with the same dose of bacilli. A minimum of 3 hamsters were killed by ethyl ether inhalation 30, 60, 120 and 150 days post-inoculation (pi). After death, samples from the pouch and inoculated footpads were collected, formol fixed, embedded in paraffin, cut and stained by hematoxin–eosin and Fite–Faraco.

No gross alterations were observed in the footpad of group 2 animals. Histologically, in 5 out of 8 hamsters studied 30 days pi, the mycobacteria evoked focal epithelioid granulomas, with giant cells, lymphocytes and very few, or no, bacilli. No macroscopic or histological alterations were observed in the footpad of animals killed after 30 days.

In 7 out of 34 hamsters inoculated into the pouch there was nodular infiltration 3–5 mm in diameter that were removed for histological study. From animals which did not present gross alterations, 3 random fragments were collected.

Histological alterations were observed in 16 out of 34 of the pouch-inoculated hamsters; it is possible that the absence of lesions in the remaining animals was related to the lack of gross alterations and that the fragments submitted to histology did not represent the inoculation site. In order to confirm this possibility, further experiments are being done, i.e. tattooing with Indian ink 1 cm above and 1 cm below the site of inoculation.

In the pouches that showed lesions, the reactions were represented by accumulations of large grossly vacuolated macrophages containing numerous bacilli, without any epithelioid transformation. This pattern persisted up to 150 days pi and were similar to that observed in anergic forms of human disease.

The ability of *M. leprae* to evoke epithelioid cell granulomas in the footpad, but not in the cheek pouch, an immunoprivileged site, confirms that, in leprosy, the epithelioid granulomas are directly related to the development of immune response to *M. leprae*.[1]

Moreover, since *M. leprae* grows easily and rapidly (about 30 days) in the pouch,

this model may represent a good alternative for the study of new antileprosy drugs and drug resistance.

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## PROTECTIVE FOOTWEAR FOR LEPROSY PATIENTS WITH SOLE SENSORY LOSS OR ULCERATION OF THE FOOT

Sir,

For many years it has been accepted that the management of patients with sole sensory loss and/or ulceration of the foot must include the wearing of suitable protective footwear, usually on a lifetime basis. This advice appears in publications from the World Health Organisation[1,2] and is included in recent guidelines from the International Federation of Anti-Leprosy Association (ILEP): *Prevention of Disability. Guidelines for Leprosy Control Programmes.*[3] At the recent 14th International Leprosy Congress in Orlando, Florida[4] a number of papers supported this view and many different types of protective footwear were on display.

In this Institute, the need for protective footwear has long been recognized and our staff includes a full-time shoemaker with appropriate tools and equipment. During the past 10 years, we have attempted to provide a pair of shoes made from microcellular rubber, tyre soles and soft leather straps, for all patients with significant sole sensory loss or ulceration of the foot. They have been instructed in the proper care and use of the shoes and on the need to report back when repair is needed, as also on the self-care of their feet, essentially as described in the above ILEP document.

We have recently reviewed our results with regard to footwear, with particular attention to the provision of 158 pairs of shoes during the past 4 years, including the necessary repair services. The results have been far from satisfactory. Enquiries amongst our health staff and social workers have revealed that many patients do not wear the shoes once they leave hospital, whilst others wear them for a short time and then discard them, or fail to report back when repair is obviously needed. Our re-admission rate for foot ulceration is high, doubtless related to deficiencies in self-care and the proper use of shoes. Interestingly enough, however, there are a number of patients who, from their own account and from the observations of field workers, have used the shoes as directed, thus suggesting that footwear does not give protection under all circumstances.

We have discussed the possible reasons for these disappointing results with staff members and come to the conclusion that there are, at least in this part of India, a number of factors which seriously undermine the potential effectiveness of the advised strategy. These include:

*Design.* The 'MCR design' has been shown to be technically satisfactory in many parts of India and elsewhere, but the use of such shoes in a village is unusual in that they do not resemble

footwear which is locally available, thus tending to identify people as leprosy patients. Closely allied to this is the next factor.

*Customs, traditions*. In general, people in villages in India do not wear shoes of any kind and in the case of females, the wearing of shoes in front of elders is unacceptable. Furthermore, women spend a great deal of the day around, or in the house, where shoes are never worn.

*Occupation*. Because 76% of the Indian population lives in villages[5] and many work with rice, paddy, sugarcane or other crops, where they are frequently ankle-deep in muddy water, it is unrealistic to advise patients to wear protective footwear under such circumstances.

*Maintenance and repair*. Under the rough conditions of village life and the distances many of our patients have to walk from their house to any health facility, standard MCR shoes do not last more than 6 months in this area, often less. Apart from the distances and transport costs which may be involved, repair or replacement of shoes involves delay and possibly the need for a second visit.

*'Release from treatment'*. The release of patients from treatment following satisfactory courses of multiple drug therapy of relatively short duration has, understandably, given many of them the impression that the disease is cured and that further self-care and follow-up are unnecessary; they become overconfident and tend to forget or ignore the advice we have given concerning foot inspection, soaking and the use of protective footwear.

As seen in this area and from this Institute, we reluctantly conclude that the advised policy of providing protective footwear to patients in need and of ensuring that they continue to use it on a long-term basis is both unsuccessful and unrealistic. Our impression is that it works no better in other parts of this State. Furthermore, the experience of those who have taken part in successive Independent Evaluations of the National Leprosy Eradication Programme in India[6] confirms that many patients are still being admitted, at considerable cost, to Temporary Hospitalization Wards (there are nearly 300 of them in the country) or other medical facilities for recurrent ulceration, but without an effective programme for the provision of protective footwear. We judge the problem to lie mainly with the strategy and the providers, rather than with the patient, but it is disconcerting to record that no obvious solution seems to be forthcoming. Better results might be obtained by markedly increasing the input of time, effort and money but, as shown by Becx-Bleumink, based on experience in Ethiopia,[7] it is extremely doubtful if this would be justifiable. This is certainly likely to be the case in India, where many thousands of patients are still in need of multiple drug therapy. Meanwhile, we are investigating the potential of an intensified programme of education of patients in self-care and the identification of measures which can be taken at village level, such as the use of zinc oxide plaster for wounds, cracks and ulcers,[8,9] to minimize further damage to the insensitive or ulcerated foot. We would welcome an exchange of views from others who have encountered similar problems in rural communities.

**Acknowledgment**

During a recent assignment for the Department of Medical Education & Training, Govt. of Orissa, Dr A. Colin McDougall (Oxford, UK), visited Aska and discussed some of the above problems with us. We are grateful for his encouragement and help with the preparation of this letter for publication.

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## PLANTAR LESIONS IN TUBERCULOID LEPROSY: A REPORT OF 3 CASES

Sir,

We report 3 histologically confirmed tuberculoid leprosy (TT) cases involving the sole of the foot which were detected at the outpatient clinic and in the field operational area of the Central Leprosy Teaching and Research Institute (CLTRI), Chengalpattu.

The plantar surface of the foot is an area of the body that is rarely affected by leprosy. Rarity of occurrence of such lesions and paucity of reports in the literature necessitated this report.

### CASE A

A 26-year-old male presented to the outpatient clinic of CLTRI with a history of having a single patch of 6 months' duration over the right foot extending halfway onto the sole. On examination

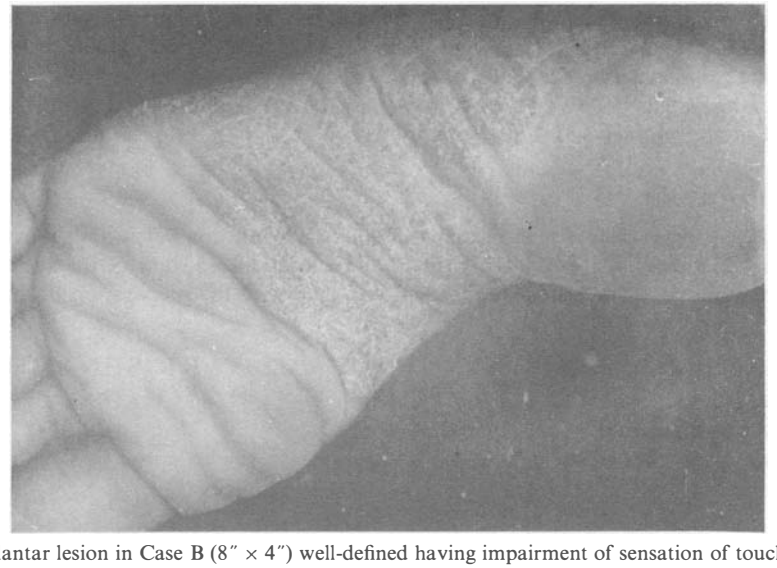

**Figure 1.** Plantar lesion in Case B (8″ × 4″) well-defined having impairment of sensation of touch on the left foot extending into the sole.

the lesion was found to be well-defined and erythematous, measuring 6″ × 5″ over the medial side of the dorsum of the right foot extending well onto the sole. There was definite sensory loss for all the modalities (touch, pain and thermal). Response to lepromin was 9 mm with ulceration. Mantoux was 0 mm. A biopsy from the lesion showed histopathology consistent with tuberculoid leprosy and was immunoperoxidase positive for *Mycobacterium leprae*.

CASE B

An 18-year-old male presented at the outpatient with 2 skin patches, 1 over the left elbow and the other over the left foot extending onto the sole. The lesion on the elbow which had first been noticed by the patient about 3 months ago was located over the lateral aspect of the left elbow joint. It was 5″ × 2″, erythematous and anaesthetic. The lesion over the left foot which had been noticed about 6 months earlier measured 8″ × 4″, was erythematous, well defined with a raised margin, and had impairment of sensation for touch, pain and thermal modalities (Figure 1). All the peripheral nerves were normal. Histopathology results were consistent with a diagnosis of tuberculoid (TT) leprosy (Figure 2). Lepromin was 9 mm and Mantoux was 0 mm. Treatment with MDT for PB leprosy was started. About 2 months after the start of treatment the patient developed acute neuritis of the left common peroneal nerve. He was prescribed steroids for which the response was partial and therefore necessitated decompression of the nerve. The lesion on the elbow resolved completely but not the one on the foot, which showed a histopathology picture on a repeat biopsy of BT leprosy.

CASE C

An 8-year-old boy was brought to the field clinic by his father with a history of a patch on the right foot which had been noticed about 2 months earlier. The lesion on examination was 5″ × 4″, erythematous, anaesthetic, well defined with raised margins, and situated on the medial side of the

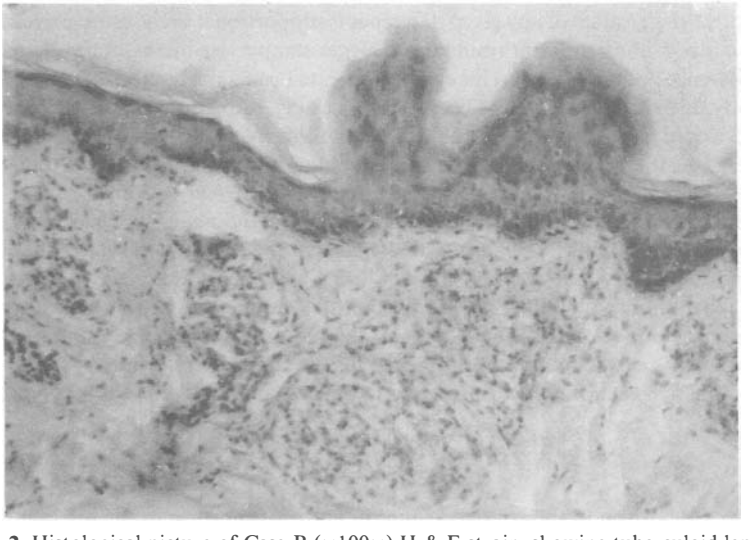

**Figure 2.** Histological picture of Case B (×100×) H & E strain, showing tuberculoid leprosy.

dorsum of the foot and extending onto the sole. The histopathology result was consistent with tuberculoid (TT) leprosy.

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*CLTRI* P. KRISHNAMURTHY & B. SEKAR
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## COMMENT: REVERSAL REACTION IN MULTIBACILLARY LEPROSY PATIENTS FOLLOWING MDT WITH AND WITHOUT IMMUNOTHERAPY WITH A CANDIDATE FOR AN ANTILEPROSY VACCINE, *MYCOBACTERIUM W*. H. K. KAR *ET AL.*

Sir,

It was with much interest that I read the above paper published in *Lepr Rev* (1993) **64,** 219–26. The immunotherapy described holds promise, and in particular the apparent rapid clearing of *Mycobacterium leprae* from the tissues is a very interesting phenomenon. It is also commendable that Kar *et al.* have not only thought of the possibility that such therapy might increase the risk of reversal reaction, but that they have actually set up a trial to investigate this possibility. The conclusion they draw from the trial seems reassuring: the difference between the proportions of patients that developed a reversal reaction in each group, 22·6% in the vaccine group *vs* 15·1% in the control group, was not statistically significant. Similarly, the proportion of severe reactions was only 'marginally higher' in the vaccine group (43·7% *vs* 33·3%). This leads the authors to conclude, 'Thus, the vaccine did not precipitate *any* additional neurological complication—an important observation in the context of introducing an immunemodulator' [italics mine].

I fully agree that the latter observation is essential not only when introducing an immunemodulator, but for any new leprosy treatment that is introduced.[1] The problem with the above study is that they *did* find an increase in risk of reaction over a 2-year period of 7·5% overall, 10·3% in the BL/LL group and 10·4% in risk of severe reaction. These differences were not statistically significant *with the given sample size*, which was only 53 patients in each group.

'Significantly' (the *z*-value) of any given difference is proportional to the sample size: a small sample size is likely to give a nonsignificant result and a bigger sample size increases the chance of finding a significant difference if it truly exists. This can be illustrated using the number of reactions observed in the above study. If the whole study had been 10 times as big, the observed number of reactions in the vaccine group would have been 120/530 (22·6%); in the control group 80/530 (15·1%). The difference would still be 7·5%, the *z*-value is now 3·12, corresponding to a *p*-value of 0·0018, a highly significant result! The difference in the BL/LL group would have been 80/390 *vs* 40/390, giving a *z*-value of 3·99, $p < 0{\cdot}0001$. The conclusion of the study would have been very different. The relative risk of vaccine *vs* control would have been 1·50 (1·13–1·97). This means that the vaccine seems to be associated with an increase in the risk of reversal reaction of 50% (95% confidence interval 13–97%). It would be unlikely that the authors would have concluded that the vaccine 'can be safely used'.

For a study such as conducted by Kar *et al.* the required sample size should be calculated in advance on the basis of the minimum difference that is clinically relevant to detect. The formula giving the sample size *in each of the trial groups* in the case of a difference between proportions is:[2]

$$n = \frac{p_1 \times (1 - p_1) + p_2 \times (1 - p_2)}{(p_2 - p_1)^2} \times f_{(\alpha,\beta)}$$

where $p_1$ is the proportion in the control group, $p_2$ is the proportion in the intervention group (in this case the vaccine group), and $f_{(\alpha,\beta)}$ is a constant value that depends on the type I and type II

error size that is acceptable.[2] Usually, these are 5% and 20%, which correspond to an *f*-value of 7·9. Thus, if we say that it would be important to detect a 7·5% increase in risk of reversal reaction (as in the above study), the equation would read:

$$n = \frac{0{\cdot}151 \times (1 - 0{\cdot}151) + 0{\cdot}226 \times (1 - 0{\cdot}226)}{(0{\cdot}226 - 0{\cdot}151)^2} \times 7{\cdot}9 = 426$$

We would, therefore, need 426 patients in each group, or more than 8 times the study size of the above study! With the given group size of 53 even an increase in risk of 15% in the vaccine group would not have been significant at the 5% level.

Since this 'adjuvant' vaccine would potentially be used on a large scale, caution is called for. If, say, 10,000 BB, BL and LL patients were to be treated with the vaccine, this might result in an *extra* 750 cases of reversal reaction. Applying the 'neuritis proportion' found in the study (25%) then 188 patients would have a severe reaction with neuritis, needing steroid treatment. Kar *et al.* report that 1 patient out of 7 (14%) failed to recover on steroid treatment and needed reconstructive surgery. This corresponds with our experience, but may be a conservative estimate (the failure rate being higher under operational conditions). Applied to the above numeric example, 26 patients would need reconstructive surgery as a result of the vaccine.

This may be a far too pessimistic a view since the actual increase in risk due to the vaccine may well be lower than 7·5%. The reported data are, after all, compatible with the null hypothesis of no difference between the groups. But the point is that we cannot tell, the results did not *prove* the null hypothesis, they just failed to reject it.

A much larger trial (preferably done blind) is therefore urgently called for, before this vaccine can be used on a large scale on the basis of an unjustified sense of safety.

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W. H. VAN BRAKEL

## References

[1] *Consensus development statement on the chemotherapy of leprosy.* ALM International 1992.
[2] Pocock SJ. Clinical Trials. *A practical approach.* John Wiley & Sons (Publ), Chichester, 1988.

## COMMENT: LEPROSY CONTROL THROUGH GENERAL HEALTH SERVICES AND/OR COMBINED PROGRAMMES. P. FEENSTRA

Sir,

The analysis provided in the editorial 'Integration of leprosy control' by P. Feenstra (*Leprosy Review* **64,** Number 2, June 1993, pp. 89–96) was admirable. I have 2 observations:

* The reference to the prerequisite—cited twice in the article—for 'an adequately functioning general health service infrastructure' represents a very, very big 'if' in most leprosy-endemic countries;
* The integration of leprosy control activities into even an 'adequately functioning general health service infrastructure' is morally and ideologically sound, even laudable.

In practice, however, it is more often discovered that while general health workers in an integrated health service soon cope well with MDT administration and even the demands of data collection imposed by the 'specialized . . . planning and evaluation' services, what suffers is the active case searching, the interest specialized leprosy workers have in being dynamic in seeking early diagnosis. Ferreting out intradomiciliary contacts, promoting routine skin examination for

general clinic patients, advocating rural extension of skin examination into remote areas of infectious foci; these are all activities that are too infrequently pursued in an 'adequately functioning general health service infrastructure'.

The conclusion I sadly reach is that without these and other specialized activities, there is less early diagnosis and obviously more infection and deformities.

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## REPLY: 'RESULTS OF SURGICAL PROCEDURES FOR THE CORRECTION OF FOOT-DROP AND LAGOPHTHALMUS DUE TO LEPROSY'

Sir,

In *Lepr Rev* (1994) **64,** 282–3, Margreet Hogeweg expresses deep dissatisfaction with the results of temporalis transfer for the correction of facial paralysis with lagophthalmos.

As a plastic surgeon with extensive training in ophthalmology and more than 30 years' experience with reconstructive surgery in several countries, both Asian and African, I strongly disagree with this.

It is quite true that many, possibly most of the patients who come for correction of facial paralysis have a loss of corneal sensation. I have never considered this a contraindication to temporalis transfer. It is obvious that pre-existing corneal opacities will remain unchanged. It is also perfectly obvious that recent paralysis, traditionally of less than 6 months' duration, should receive full treatment with corticosteroids, even if no other evidence is present of reversal reaction.

Possibly Dr Hogeweg has encountered operations which were performed with a less satisfactory technique. It is important that the transferred strip of temporalis muscle reaches the outer canthus in an oblique direction, so that the lower lid will be lifted in the closure. It is also important that the facial strips after passage through the lids, close to the margin are crossed deep to the canthal ligament, thus pressing punctum lacrymale against bulbus so that the tear pump may be reactivated. It is equally obvious that it must be ensured that tear passages are open before the operation, and that any infections have been dealt with. The traditional wedge excisions of the lower lid to correct the frequently overslack and drooping lower lid are in fact often unsatisfactory, not least because the resultant scar tissue on the lid irritates the cornea. Instead I use a wedge excision away from the eye and a lateral transposition of the lid obliquely upwards.

Both of these procedures, that might be performed in a single session, are not unduly complicated or difficult for a trained surgeon. They do not require complicated surgical or anaesthetic equipment.

I grant her that competent postoperative physiotherapy is necessary, but even if the cornea is completely anaesthetic, it is possible to create a new reflex arc, using visual stimulus as the afferent branch and the temporalis muscle as the effector. Even if this is not possible, the results are generally good. Nearly all patients have permanently reduced palpepral fissure and closed eyes during sleep. Detailed descriptions of these techniques are given in *Lepr Rev*, **34** and *Brit J Plast Surg*, **14** and **31**.

*Braineparken 85* J. G. ANDERSEN
*Haderslev, Denmark*

*Lepr Rev* (1994) **65,** 407–409

# Book Reviews

## *Teaching tools for health professionals.* Luc Van Parijs and Betsy Abraham

This is a reinforced paperback of 275 pages, published by TALMILEP (Teaching and Learning Materials, International Federation of AntiLeprosy Associations), supported by the German Leprosy Relief Association (DAHW) and other ILEP members. The content is to some extent based on experience gained by LVP in the running of workshops on teaching methods and educational materials in various parts of Africa and Asia. The Foreword describes the main purpose of the book as being '. . . to provide busy health professionals with a guide to the selection and use of seven of the most widely available and effective tools for teaching. The book also provides an understanding of the principles of teaching and learning that are relevant for using these tools.' The text is divided into 2 main sections. Section 1 on Teaching and Learning ('Who is a teacher?', 'Teaching tools in teaching' and 'Teaching tools in learning') helps the student to choose specific tools for specific tasks. Section 2 on Teaching Tools (chalk board or blackboard, flip-chart and flash cards, real objects and models, hand-outs, overhead projector, slides and video) describes, in considerable detail and with a wealth of practical advice, how the various tools may be most effectively used. There is an extensive and valuable list of references (pages 253–9), including relevant books and chapters in books. Both authors have extensive practical experience of their subject and this book should be of great value to all who have responsibility for teaching health professionals, not only in leprosy, but in a wide range of other subjects.

*A. Colin McDougall*

Published by TALMILEP, London, 275 pp.

## *Cascade of workshops on teaching methodology*

The Report of the First Workshop held at Wardha, Maharashtra, India in February 1992, describes the main objectives and content of sessions on '. . . teaching methodology for trainers in leprosy training centres.' The 17 participants were all closely involved in teaching or training activities in various parts of India. The meeting took place at the Gandhi Memorial Leprosy Foundation in Wardha, Maharashtra, and was sponsored by the German Leprosy Relief Association (DAHW, PO Box 110462, Wurzburg D-8700, Germany). The Report includes—a review of GMLF's contribution to training activities in India; the current need for a revision of teaching methodology; aims of the workshop and a detailed description of the subject matter covered, including discussions, over a period of 6 days. The 23 appendices include a useful review of the scope of teaching aids (books, models, charts, blackboard, flannelgraph, video, etc.) in the teaching and learning process.

*A. Colin McDougall*

[Copies obtainable from: Secretary, GMLF, P.O. Hindinagar, Wardha 442-103, Maharashtra, India.]

***Intrinsic minus hand. (patho)kinesiology, rehabilitation and reconstruction.***
**J W Brandsma (1993)**

This book is the fruit of 15 years of practical experience of dealing with paralysis of the intrinsic muscles of the hand in patients with leprosy. There is a wealth of information based on careful observation, objective testing of muscles and ranges of movement. The full and accurate recording of findings and follow-up is excellent. As the author states, these findings can be applied equally well to other cases with differing aetiology.

The pros and cons of several reconstructive operations are discussed and pre and post-operative care is described. The approach is purely surgical, conservative treatment with well designed, lively or even fixed 'knuckle duster' splints with curved palmar bars are not considered. The great improvement in the function of the hand confered by these splints encourages use and therefore maintains mobility when combined with passive exercises to prevent contractures of muscles and consequent stiffness of joints. The author stresses the need to prevent these complications which may be so severe in some cases that they militate against successful reconstruction.

The chapter on anatomy does not clearly indicate the important role of the ulnar intrinsics and all the lumbricals in extension of inter-phalangeal joints and stabilization of the extensors of the metacarpo-phalangeal joints of the fingers and of the slip of abductor pollicis brevis which serves the same purpose for extensores pollicis longus et brevis. It was surprising to see no reference to the classical papers of Stack and Napier. The pitfall of failure to recognize anomalous innervation is described, but no mention is made of the communications between the ulnar and median nerves that may also occur in the lower axilla, upper arm and even in the hand itself.

A detailed index at the end of the book would be helpful and sadly the use of eponyms is no longer fashionable. These criticisms are relatively minor and the book is recommended warmly to all who deal with patients suffering from leprosy or other conditions which paralyse the intrinsic muscles of the hand.

*Ruth E. M. Bowden*

A Doctoral Thesis presented successfully to the Rijks University of Utrecht CIP-Gegevens Koninklijke Biblioteck den Haag, The Netherlands, pp. 181, 315 refs, 34 tables, 43 figs.

***Histoid leprosy*** **by Professor V. N. Sehgal (2nd edition, 1993) ISBN 81-7179-338-X**

It is unusual to encounter an academic book where the author describes his own previous work as 'scintillating' and 'authoritative'. So does Professor Sehgal justify these epithets in the second edition of his monograph on the puzzling entity of histoid leprosy?

I find several aspects of this book unclear. On the definition of histoid leprosy, is it purely clinical (multiple discrete nodules), or does histopathology have an inclusive/exclusive role? Whilst we accept the dermatofibroma-type histology as exemplifying the lesion, another worker's concept of a spectrum of histoid lepromas is quoted without comment. The latter has a histology indistinguishable from [ordinary] lepromatous leprosy. This is hardly helpful to those seeking definitive criteria, for clinical or research purposes. Certainly in my experience clinicians often call all discrete multibacillary nodules 'histoids'. Should we go along with that irrespective of histology?

In an illustrative clinical case of histoid leprosy, I am puzzled by the ascription of a *downgrading* type I reaction in histoid leprosy in a patient on MDT. In another patient, the histological description of the necrotizing histoid does not describe the necrosis, which is a pity since it might illuminate the concept of augmented cell-mediated immunity (CMI), which is held to be the aetiology of histoids.

The histoid is said to be 'a young leproma in special reactional field'. Does that phrase actually mean anything? I find nothing in the evidence quoted—blood T-cell distributions, serum immunoglobulin levels, or histopathology–that supports the claim that histoids represent augmented CMI. What are the 'early T-cells' referred to here? Why do spindle-shaped macrophages with high bacillary loads *per se* indicate active local CMI? A histological analogy not referred to in the book is with the *M. avium-intracellulare* histoid-like lesions (i.e. dermatofibroma-type) seen in immunocompromised patients. In those patients we know, from blood CD4+ T-cell data, that their cellular immunocompetence is effectively zero. In leprosy, I would not quibble with the evidence that many histoids represent regrowth of drug-resistant bacilli. But why the lesions develop as they do is still a mystery.

The clinical photographs of histoid leprosy here are variable; a few look like satellite weather maps. The histopathological photographs are very poor indeed, and electron micrographs are not included although EM appearances are said to show significant differences between histoids and lepromatous leprosy.

All in all, I doubt that anyone who has seen patients with histoid leprosy and read some of the original articles on the topic will come away much the wiser after reading the book.

*S. B. Lucas*

Published by Jaypee Brothers Medical Publishers, New Delhi, India, 72 pp, Rs 75.00.

## Errata

Serum lactoferrin in lepromatous leprosy patients, Om Parkash, B. K. Girdhar & U. Sengupta, *Lepr Rev* (1993) **64,** 295–301.

Page 296, Measurement of serum lactoferrin, line 2

for '2.0 μg/ml' read '20 ng/ml'

Disabilities of hands, feet and eyes in newly diagnosed leprosy patients in eastern Nepal, A. Schipper, W. J. Lubbers, Margreet Hogeweg & R. de Soldenhoff, *Lepr Rev* (1994) **65,** 239–47.

The following correction should be made to the above paper.

Page 241/2, The illustrations for Figures 1 and 2 should be transposed so that they appear with the correct caption.

*Lepr Rev* (1994) **65,** 410–415

# Leprosy Review Index

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