Volume 73, Number 3
Printed in the U.S.A.
(ISSN 0148-916X)

CORRESPONDENCE

This department is for the publication of informal communications that are of interest because they are informative and stimulating, and for the discussion of controversial matters. The mandate of the JOURNAL is to disseminate information relating to leprosy in particular and also other mycobacterial diseases. Dissident comment or interpretation on published research is of course valid, but personality attacks on individuals would seem unnecessary. Political comments, valid or not, also are unwelcome. They might result in interference with the distribution of the JOURNAL and thus interfere with its prime purpose.

Quantitative Measurement of Sensory Impairment in Referral Centers[1]

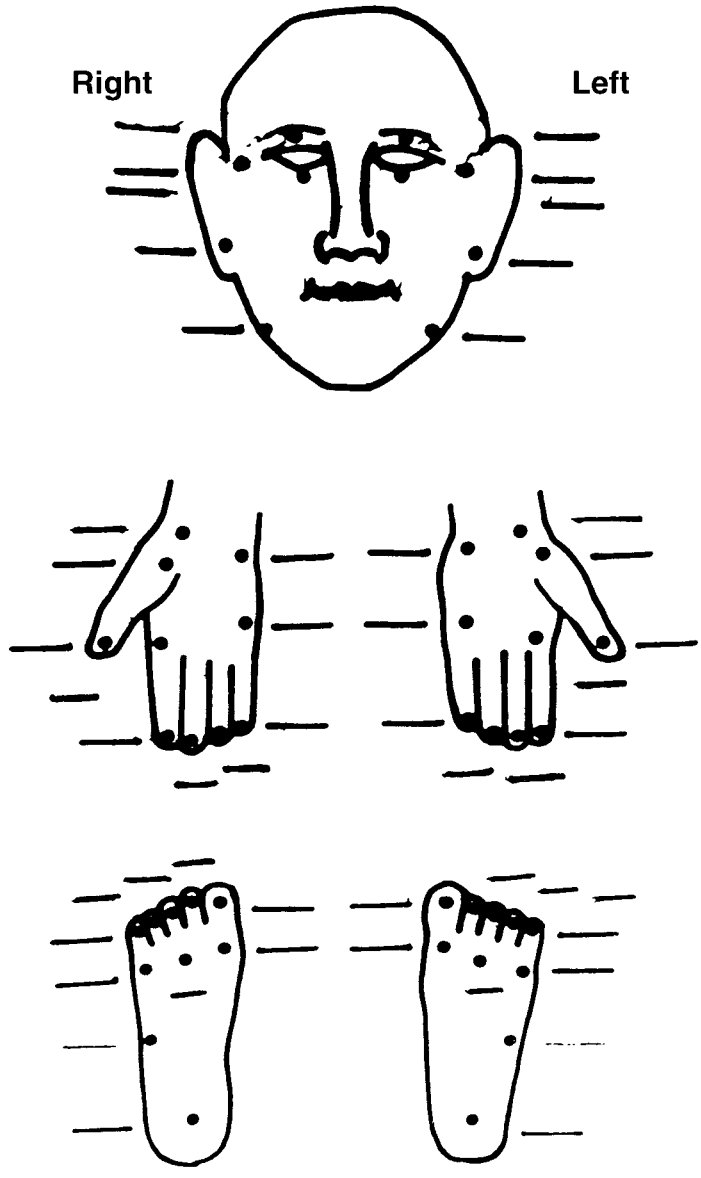
TO THE EDITOR:

In 1998, we had suggested the quantitative method of sensory assessment of face and testing sites for the limbs ([4]). The following improvement to the original method may be required and this is based on our clinical experience in using this technique in a referral center with specialists and time available.

The 10 sites for testing sensation on the face, hands, and feet are unchanged. We suggest two changes with the Semmes-Weinstein monofilaments as a result of the recent understanding of the normal sensation of the hands and feet. Similarly, the method to score sensory nerve status is also altered. This is because the clinicians expressed that the norms for muscle grading are: "zero" indicating flaccidity, and a maximum score of 5 given for normal musculature; this was reversed in our quantitative sensory testing. In order to have a uniformity between sensory and muscle testing, we recommend the changes depicted in the following assessment form. In the revised form, 0 to 4 sensory grading system is followed for the hands. For the foot, 0 to 3 grading system is used because their sensory function is less than that of hands, which have to manipulate objects and require well developed sensory nerve endings. For the face, a 0 to 3 grade sensory threshold scale was used with the interpretations suggested by Premkumar, *et al*. ([4]).

The interpretations presented for the foot and hand are also based on the following previous scientific studies. Krotoski published the details on interpretation for the hands ([1]). Similarly, Birke, *et al*. interpreted 10 g filament as the level of protective sensation in leprosy patients ([2]). Kets, *et al*. study demonstrated that the touch sensibility monofilament threshold screening in healthy Nepalese population were 0.2 g for hands and 2 g for feet ([3]). Since all of the South Asian population is likely to be similar to that of Nepalese, we had taken the interpretation of this study and made a small modification to Krotoski's hand sensory battery by removing 0.05 to 0.07 g filament as an instrument to test normal sensation. In the original neurological mappings by Weinstein demonstrated the higher sensitivity in the face; the mean threshold of males to be 0.02 g; females, 0.018 g ([5]). Despite the above work in neurology, in the facial sensation assessment we suggest using a filament that gives a force of 0.05 to 0.07 g. It will be higher than the threshold for the face and will avoid false negative responses for the following reason: The lowest sensory threshold in normal individuals quoted in the Weinstein article is in the laboratory situation, which cannot be duplicated in clinics. Therefore, the next higher threshold may be required to increase the test sensitivity.

We are also aware that more studies are needed to answer the following research questions arising from this work. For instance, the lack of testing the corneal sensation to an extent limits the usefulness of testing facial sensation. Since this study



QUANTITATIVE SENSORY IMPAIRMENT MEASUREMENT

Summary Table.

Body Part	Nerves	Number of Sites Tested (per nerve)	Score Nerve		Maximum Score For Each Body Part
			Maximum For Each *Right*	*Left*	
Face	Trigeminal	3	/9	/9	/18
	Auricular	2	/6	/6	/12
Hands	Ulnar	4	/16	/16	/40
	Median	6	/24	/24	
Foot	Posterior Tibial	10	/30	/30	/30

"Zero" score indicates maximum sensory loss. The denominator indicates normal sensation.

Source: Int.. J. Lepr. Other Mycobact. Dis. ***66*** *(1998) 348-355 with revisions in 2004.*

THE FIGURE.

KEY FOR GRADING

FACE

Not Felt	Felt	Interpretation	Score
	0.05 g	Normal superficial sensation	3
0.05 g	0.2 g	Normal superficial sensation diminished	2
0.2 g	2.0 g	Loss of normal superficial sensation—deep sensation intact	1
2.0 g		Total loss of pressure sensation	0

FOOT

Not Felt	Felt	Interpretation Score	
	2 g	Normal superficial sensation	3
2 g	10 g	Normal superficial sensation lost—protective sensation intact	2
10 g	300 g	Protective sensation lost—deep pressure sensation intact	1
300 g		Total loss of pressure sensation	0

HAND

Not Felt	Felt	Interpretation Score	
	0.2 g	Normal superficial sensation	4
0.2 g	2 g	Normal superficial sensation diminished	3
2 g	4 g	Superficial sensation lost—protective sensation intact	2
4 g	300 g	Protective sensation lost—deep pressure sensation intact	1
300 g		Total loss of pressure sensation	0

SUMMARY TABLE.

Body Part	Nerves	Number of Sites Tested (per nerve)	Maximum Score For Each Nerve *Right*	*Left*	Maximum Score For Each Body Part
Face	Trigeminal	3	/9	/9	/18
	Auricular	2	/6	/6	/12
Hands	Ulnar	4	/16	/16	/40
	Median	6	/24	/24	
Foot	Posterior Tibial	10	/30	/30	/30

"Zero" score indicates maximum sensory loss. The denominator indicates normal sensation.
Source: Int.. J. Lepr. Other Mycobact. Dis. ***66*** *(1998) 348–355 with revisions in 2004.*

confines in using the instrument of S-W filaments in testing only the skin in the limbs and face, and not the cornea, it is beyond the scope of this work. In the previous work on facial sensory testing, the authors hypothesized that the corneal sensation assesses only the ophthalmic branch of the trigeminal nerve ([4]). The other two branches of the nerve usually go unexamined. Facial sensory testing, we have suggested, will give quantitative sensory information for all three branches of the trigeminal nerve. Hence, the specific research question would be whether testing the facial sensation around the eyes could indicate corneal insensitivity?

There is also a research question related to the testing sites: whether further reduction in the number of testing points would be more beneficial than the 25 sites we proposed? Our suggestion for further testing sites reduction is to 10; for example, two each for facial, great auricular, ulnar, median and posterior tibial. A further scrutiny is also needed into the validity of the facial sensory loss and its interpretation to function that we have suggested in our previous work ([4]), in a larger population.

Method used to score sensory nerves supplying face, hand and feet

Ten testing sites have been selected for each hand, foot and face. Three testing points have been identified for each trigeminal and 2 for each great auricular nerve: 4 for ulnar, 6 for median and 10 for posterior tibial. If the patient feels 0.05 g filaments in the face and 0.2 g in hands or 2 g filaments in

feet on each point, three score is given to that site for face and foot. Four score is given to that site in hand as four filaments are used for the palmar surface: two for not feeling that filament in face and foot and so forth.

Thus, total loss of sensation at a point will be scored as zero for the face, hand, and feet; i.e., since there are 3 testing points for trigeminal, the maximum sensory loss per this nerve is scored, as 0 + 0 + 0, which is 0. Normal sensation will be scored as 3 + 3 + 3 = 9. The maximum score for normal sensation of the following nerves are stated below and these are indicated as denominators in the first Table.

Nerves	Maximum score per intact nerve
R. Trigeminal	9
L.Trigeminal	9
R.Great auricular	6
L.Great auricular	6
R.Ulnar	16
L.Ulnar	16
R.Median	24
R.Median	24
R.Posterior tibial	30
L.Posterior tibial	30

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Serologic Recognition of Low Molecular Weight Mycobacterial Protein Fractions in Lepromatous Patients with Type II Reactions (ENL)

To the Editor:

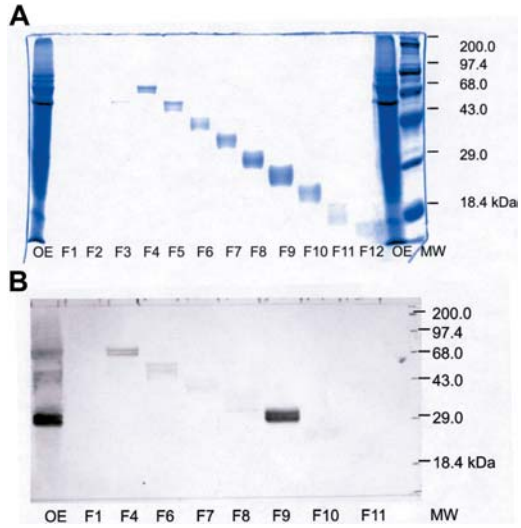
Hansen's disease is a mycobacterial infection that produces physical disabilities. The progression of the disease is slow and indolent but in some cases there are changes in the immunological status with the development of acute episodes represented by reactional states. Many of these reactional episodes occur after treatment has been finalized and, therefore, it is important to clarify whether they constitute relapses. We wished to determine if specific patterns of serologic recognition of mycobacterial proteins were associated with Type 2 reactional states in lepromatous patients. Serum samples were taken from 12 adult patients, mean age of 43 ± 16 yrs, with a predominance of males (80% M and 20% F), who were undergoing a Type 2 reactional episode (erythema, nodosum leprosum, ENL). These sera were divided in two groups of six sera each: sera

from Group I were antibody-positive to phenolic glycolipid (PGL-I), the other six (Group II) were negative. ENL reactions were characterized using histopathological criteria, including the presence of undifferentiated macrophages and relatively abundant PMNs, with or without acid-fast bacilli. The group of six patients that gave negative reactions for antibodies to PGL-I (Group II) had completed multidrug therapy; they presented an average of six episodes of ENL. Of the six patients in Group I with detectable antibodies to PGL-I, two were still being treated. ENL reactions were less frequent in Group I (average 4 episodes).

Soluble component fractions were obtained by an electroelution technique from *Mycobacterium leprae* soluble extract (MLSA) and *Mycobacterium bovis* soluble extract (MbSA) ([5, 6]). The soluble extracts were obtained by rupturing purified bacilli with the French Press ([2]). The extracts contain cytosol proteins as well as proteins freed from the cell walls. Insoluble material was eliminated by centrifugation. Protein concentration was determined by the BCA method ([7]).

Starting with a 10% SDS-PAGE preparative gel under dissociating and denaturing conditions, 1 mg of MLSA and MbSA was resolved in polypeptides of different mobilities (see The Figure), which were fractionated by electroelution in a mini BIORAD® 65-1256 electroelutor, according to the instructions provided by the manufacturer.

Twelve electroeluted fractions were obtained for both the MLSA and the MbSA antigens. ELISA tests were used to evaluate activity with the pooled sera, using IgG antibodies specific for the Fc gamma chain (Sigma A0170) as the second antibody ([4]).



THE FIGURE. OE, original extract (*M. bovis*). F1, F2. different electro-eluted fractions MW: molecular weight standards. (A, Coomassie brillant blue stain; B, Western blot with LL serum.) The typical ladder of eluted fractions corresponding to their molecular weights is shown, with successively smaller proteins from F1 to F12.

A clear difference in recognition was seen between the two groups of sera studied. In the ELISA tests with both MLSA and MbSA electroeluted fractions, we saw an immunodominant recognition of proteins with a relative mobility of 30 kDa, corresponding to Fraction 9 (see The Table). There was also serologic recogni-

THE TABLE.

Patients group	Fractions MbSA						
	F6	F7	F8	F9	F10	F11	F12
GI	<0.125	<0.125	0.241 ± 0.001	0.470 ± 0.01	0.256 ± 0.01	<0.125	<0.125
GII	<0.125	<0.125	<0.125	0.296 ± 0.003	<0.125	<0.125	<0.125
Patients group	Fractions MLSA						
	F6	F7	F8	F9	F10	F11	F12
GI	<0.125	<0.125	0.257 ± 0.004	0.501 ± 0.001	<0.125	0.313 ± 0.03	<0.125
GII	<0.125	<0.125	0.178 ± 0.025	0.483 ± 0.01	<0.125	<0.125	<0.125

The results are expressed as optical density (O.D.) at 492 nm. To establish the criterion of positivity, the value resulting from mean OD plus 3 times the standard deviation of 12 healthy subjects was used as the cut-off point (0.125 O.D. units).

tion of low molecular weight MLSA proteins (less than 30 kDa) in patients in group I which was not observed in Group II.

In preliminary studies we previously reported a clear difference between the IgG antibody levels directed towards soluble mycobacterial proteins (*Mycobacterium bovis* MbSA and *Mycobacterium leprae* MLSA) in an ENL active group (n = 4) as compared with the non-active group (n = 4) ([3]). In the ENL active patients we found IgG antibody levels towards MbSA and MLSA of 0.535 ± 0.24 and 0.731 ± 0.32, respectively, as compared with the non-active patients, whose values towards the same total proteins were zero. In this study using the electroelution technique we were able to demonstrate the immunodominant antigens found in patients in an ENL reactional state.

Many authors have shown a decrease of IgM antibodies directed towards phenolic glycolipid (PGL-I), which is an *M. leprae* structural component ([1]) in these reactional patients. To examine this, we separated the reactional patients in two groups, according to their PGL-I positivity. IgM antibodies against native PGL-I were measured in an enzyme linked immunosorbent assay using the method described previously ([8]).

In addition to the immunodominant recognition towards proteins with a 30 kDa relative mobility, both with MbSA and MLSA, we also saw that the recognition in Group I involves a larger number of protein fractions, including low molecular weight proteins (<30 kDa), compared to the patients in Group II.

We have recently increased the number of multibacillary patients (n = 70), and there have been no significant differences in the *Mycobacterium leprae* 30 kDa protein antibodies between patients who had Type II reactions and those who did not. In this larger group of 70 multibacillary patients, nine presented ENL reactions and the other 61 did not. Of the nine with ENL, eight (89%) gave positive reactions to the 30 kDa protein, average optical density 0.8816. Of the 61 remaining patients, 42 (69%) gave positive reactions to the 30 kDa protein, average OD 0.5885. This difference was not statistically significant, p = 0.42, but the observation suggests a trend toward stronger reactivity in patients with ENL. The sera of newly diagnosed multibacillary patients reacted with other peptides of both higher and lower molecular weights. In this population of 70 patients, 62.6% were in treatment and presented bacillary indices of less than 2+. Reactivity was strongly associated with bacillary load. Reactivity to the 10 kDa protein of *M. leprae* was lower in treated patients than in new cases (unpublished data).

In conclusion, both patients who had ENL as well as those who did not responded to the 30 kDa peptide of *M. leprae,* but the reactions tended to be stronger in the former group. Additional more detailed studies will be necessary to detect a clear marker for ENL, using individual proteins of the 85B complex or specific peptide sequences of other proteins that might discriminate between patients with or without reactional phenomena.

Acknowledgment. This research was supported by grant number S1-2001000859 from Fondo Nacional Ciencias y Tecnología (FONACIT) Caracas, Venezuela.

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Paucibacillary Treatment for Large Tuberculoid Lesions of Leprosy?

To the Editor:

Under the title "Should large lesions of leprosy be considered as multibacillary for treatment purposes even if the total number of lesions is less than five?" [Int. J. Lepr. 72 (2004) 173–174], Kumarasinghe and Kumarasinghe called attention to an interesting aspect regarding the treatment of big size tuberculoid lesions or borderline-tuberculoid lesions according to Ridley & Jopling classification.

Their arguments for the treatment of patients with large plaques are valid but the recommendation since the beginning of Multi-drug Therapy - M.D.T./World Health Organization/82 ([2]) was to treat patients upon a positive or negative bacilloscopy. The size or the number of lesions were not to be taken into account. Millions of patients have been treated since, with a relapse rate of less than 1%. We present a patient classified and treated as PB leprosy with a large plaque and five smaller lesions.

Patient. N.R.L, 45 years old, registered at the Fundação de Medicina Tropical do Amazonas, Manaus, Brazil.

The patient presented a large plaque lesion on the chest (Fig. 1) and five smaller lesions on the face, arm and posterior part of the trunk. No enlargement of the ulnar nerve or of other peripheral nerves could be found. The patient was clinically classified as reactional borderline tuberculoid leprosy.

The histopathology (Hematoxylin-Eosin) showed a granulomatous lesion with lymphocytes, histiocytes and giant cells (Fig. 2). The Wade stain was negative for acid-fast bacilli. The patient was classified as borderline tuberculoid (BT) leprosy.

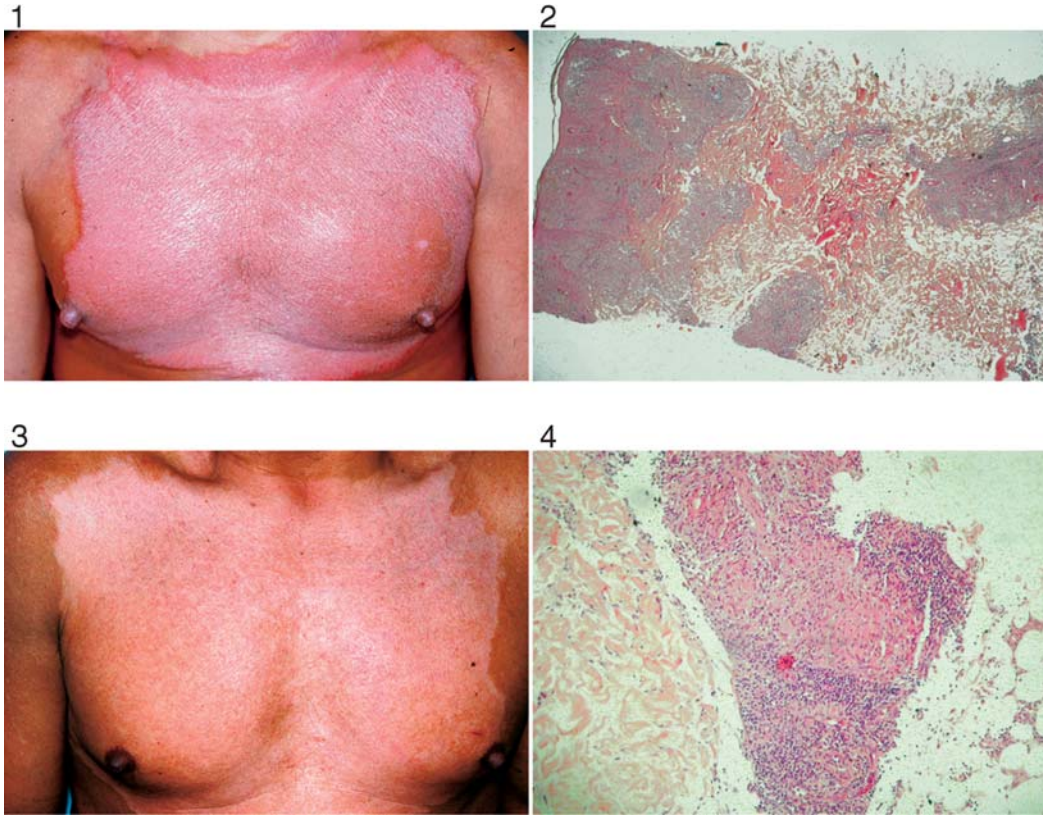
Paucibacillary M.D.T. according to the W.H.O. plus 60mg prednisone per day was started in March 2003. M.D.T. was stopped after 6 months of regular treatment and cortisone was slowly tapered off after 3 to 4 months.

All the lesions regressed leaving a hypopigmented area (Fig. 3). During the last clinical reexamination (10/11/04) no relapse was found. A new histopathology of the edge of the lesion showed a regressive infiltrate (Fig. 4).

COMMENTS

The W.H.O. recommendation to treat leprosy was based on bacilloscopy for many years on the bacilloscopy results, and the efficacy of M.D.T. has been the same worldwide: Less than 1% of relapses. The W.H.O. ([3]) recommendation to treat leprosy patients according to the number of lesions was mainly an operational decision to implement M.D.T. in the field. There was no recommendation related to the size of the lesion.

We think that a patient with a negative bacilloscopy and a histopathology consistent



FIGS. 1–4. **1.** Borderline tuberculoid leprosy. Large infiltrated plaque with well defined edges. **2.** Hematoxylin-Eosin—presence of granulomatous infiltrate with epithelioid cells, giant cells and lymphocytes. **3.** After nearly two years. Regression of the plaque, leaving a residual hipochromic lesion. **4.** Regression of the infiltrate.

with a tuberculoid granuloma with scarce or no bacilli in the Wade or Fite-Faraco stain must be classified as paucibacillary leprosy. We agree with Kumarsinghe and Kumarsinghe ([1]) that ". . . the larger the lesions of leprosy, the higher the number of bacilli that cause the pathology. . . .," but in such a lesion the clinical aspect is roughly the same with defined edges and very well established borders between the lesion and the normal skin. Besides the relatively uniform clinical aspect as observed in our patient, the skin smears and the histopathology were the same in repeated biopsies with the number of bacilli scarce or negative. We could not find any information in the literature to substantiate the statement of the authors, that tuberculoid leprosy could evolve to the lepromatous pole over several years with the lowering of patient´s cellular immunity ([1]).

We have been treating patients with multiple (more than 5) lesions as PB leprosy when the clinical aspect of the lesions and the histopathology showed a picture of tuberculoid leprosy.

We agree with Kumarasinghe and Kumarasinghe ([1]) that the W.H.O. recommendation is "particularly important in areas where treatment is initiated without any bacteriological and histopathological confirmation . . ." However, in referral centers and in universities with good laboratory support the present WHO guidelines to treat as PB leprosy or MB leprosy should not be followed. It seems there are no scientific data to justify a formal recommendation to treat leprosy according to the number or size of the lesions.

Acknowledgement. To Dr. Gotfried Schmer of the State University of Washington.

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Drs. Kumarasinghe Reply: *Should Large Lesions of Leprosy Be Considered as "Multibacillary" for Treatment Purposes Even If the Total Number of Lesions Is Less Than Five?*

TO THE EDITOR:

We thank Souza Santos, *et al*. for their interest in our article ([5]). While agreeing with some points made by them, it seems that they have misunderstood some of the points we made.

First, our recommendation of "considering large lesions of leprosy as multibacillary" was not aimed for teaching hospital settings where microbiological and histopathological facilities and good clinical expertise are available, but for field settings, in areas where treatment is initiated without any investigations, purely based on the number of hypopigmented lesions. At the teaching hospitals and tertiary care centers we also treat patients after considering the smear results and skin biopsy results, in addition to the clinical picture.

It is well known that even a single lesion can be multibacillary ([3, 4, 6]). The rationale of total number of lesions as the only criterion for deciding on the treatment type, as well as for scientific analyses has been questioned ([8]). However, in a retrospective study carried out in India, Gift, *et al*. have found that World Health Organization (W.H.O.) operational classification is a satisfactory method for deciding on the form of treatment ([2]). In this analysis, they have taken the smear examination as the gold standard for evaluating the sensitivity and specificity of the W.H.O. operational classification. However, where the larger lesions (>10 cm) were present they have found that the specificity was 91.2% although the sensitivity was low. As only 4.9% of smear positive cases have had records of the size of the lesion, that study appears to be inadequate to evaluate the validity of the size of lesions as an additional parameter.

Our recommendation for treatment of large plaque leprosy with three drugs for one year ("multibacillary treatment") is based on the observation of more relaspses in this group of patients who have been treated with two drugs for six months. In another study conducted in Sri Lanka it was shown that several patients with large lesions (>10cm) of leprosy were smear positive although the total number of the patches was less than five ([1]).

We do not dispute that many cases of paucibacillary leprosy have less than 5 patches. Although the authors agree on the W.H.O. operational classification based on the number of patches, the case described by the authors, with a large plaque of leprosy plus 5 other lesions on the face, would have been classified as "multibacillary", if the microbiological investigations were not done, going by the visual classification recommended by the W.H.O. It is known that some cases of leprosy may improve even with dapsone monotherapy (as was the practice before the advent of multi-drug therapy, M.D.T.), or single dose multidrug therapy. It would be interesting to see the long term outcome of the case presented by the authors. Even though a smear was negative, in the case presented by the authors, we would have not have been comfortable in administering paucibacillary treatment only for 6 months, in a patient with such extensive lesions. Cell mediated immunity is of paramount importance in the pathogenesis of leprosy ([7]). It is clear that patients progress in the leprosy spectrum towards the lepromatous pole when the immunity of the host is unable to overcome the infection by lepra bacilli. In cases of subpolar lepromatous leprosy some areas with typical hypopigmented semianaesthetic lesions can often be seen while other smear positive lesions coexist in the same patient. Clearly not all cases of multibacillary leprosy start as polar lepromatous leprosy. In our statement in the article we did not imply that "polar tuberculoid leprosy" would downgrade to "polar lepromatous leprosy" which are generally immunologically stable.

We agree with the authors that a larger scale study would be helpful to resolve the issue whether larger lesions due to leprosy should be treated with the "multibacillary drug regime" at least for one year.

A representative lesion should be microbiologically and histopathologically evaluated whenever possible, and the findings should be evaluated in conjunction with the clinical features before commencing on treatment. The current W.H.O. operational classification; while being useful in the community perspective, appears to be an over-simplification in some situations. The search for any additional features to fine tune the parameters should be continued.

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Has the Term "Elimination" Outlived Its Utility?

TO THE EDITOR:

Please permit us to make some more observations (arising from our combined experience of over 60 years in leprosy relief work) particularly relevant to India, which contributes 77% of active cases to the global pool of active leprosy cases. One to 1.5 million out of 2 to 3 million leprosy-disabled in the world are reported to live in India.

Dr. Yo Yuasa, who was the President of the International Leprosy Association for two terms, exhorted everyone to work towards a "World Without Leprosy" at the International Leprosy Congress, Beijing in 1998. He defined this state as "a world without leprosy-related problems, both medical and social, emphasizing the point that it is not the disease *per se* but its related problems, mostly social but some medical, which require attention."

This slogan was, however, pooh-poohed by the World Health Organization (W.H.O.) and the W.H.O.-influenced governments and the "program managers," who were obsessed with the term "Elimination." The target year was 2000, which is now revised to 2005, when the mean prevalence rate of 1 case per 10,000 is expected to be reached. Unfortunately by then, the world will also be free from the so-called "Leprologists." The enormous funds still needed to do justice to the clinical problems related to leprosy and the rehabilitation of patients would have dried up. The "pool" of leprosy patients with reaction, neuritis and its sequelae, and those needing rehabilitation contributing to the "disease burden" in the community will far out number the active cases needing multi-drug therapy (M.D.T.) As yet there is no evidence of the much talked about secondary level and tertiary level "Referral Centers" easily accessible to patients living in areas deprived of even basic health services, where the primary health centers with which leprosy is "integrated." Most patients and the health providers are not even aware of the technology to prevent the adverse progression of complications and palliative care of irreversible disabilities, let alone the concept of "Community-Based Rehabilitation."

It is strange that the same public health specialists who talk about "Elimination" have now started fighting for "Human Rights" of leprosy patients without even attempting to formulate a mass-based strategy for addressing the clinical problems of patients "released from control."

Perhaps they are waiting to celebrate the eventful day of 31 December 2005 to announce their "Victory over Leprosy" before thinking of planning the secondary and tertiary level referral centers! It is time that the people, patients, and particularly the donors are made aware that this victory is by no means a victory over all leprosy-related problems, as enshrined in the definition of "World Without Leprosy." The donors are made to believe that with the magic word "Elimination," the disease is already on the verge of being wiped out.

Has not the jargon "Elimination" of leprosy outlived its utility? Though it is rather late, should we not devise a more patient-friendly term for "Elimination" that truly reflects the sincere attempt at the eradication of all ills afflicting the persons who have contracted specially the progressive forms of the disease?

—Dr. R. Ganapati,
—Dr. V. V. Pai

Bombay Leprosy Project