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

—Ramaswamy Premkumar, Ph.D.,
Pichaimuthu Rajan, BOT,
Ebenezer Daniel, MS, MPH

Schieffelin Leprosy Research and TrainingCentre, Karigiri - 632106, Tamil Nadu, India.

REFERENCES

1. BELL-KROTOSKI, J. A. Sensibility testing: current concepts. In: *Rehabilitation of the Hand: Surgery and Therapy*, 4th edn. St. Louis: Mosby-Year Book, Inc., 1995. pp. 109–128.
2. BIRKE, J. A., and SIMS, D. S. Plantar sensory threshold in the ulcerative foot. Lepr. Rev. **57** (1986) 261–267.
3. KETS, C. M., VAN LEERDAM, M. E., VAN BRAKEL, W. H., DEVILLE, W., and BERTELSMANN, F. W. Reference values for touch sensibility thresholds in healthy Nepalese volunteers. Lepr. Rev. **67** (1996) 28–38.
4. PREMKUMAR, R., DANIEL, E., SUNEETHA, S., and YOVAN, P. Quantitative assessment of facial sensation in leprosy. Int. J. Lepr. Other Mycobact. Dis. **66** (1998) 348–355.
5. WEINSTEIN, S. Intensive and extensive aspects of tactile sensitivity as a function of body part, sex and laterality. In: *The Skin Senses*. Springfield, IL: Charles C. Thomas Publishers, 1968. pp. 193–218.

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

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.

—Elsa María Rada, M.Sc.,

Laboratorio Leprología y Patología Experimental
Instituto de Biomedicina,
Caracas, Venezuela

—Edgar A. Zambrano, Ph.D.,

Laboratorio de Bioquímica de Parásitos,
Instituto de Biomedicina,
Caracas, Venezuela

—Nacarid Aranzazu, M.D.

Clinical Section M.D., Instituto de Biomedicina
Caracas, Venezuela

—Jacinto Convit, M.D.,

Director, Instituto de Biomedicina
Caracas, Venezuela

REFERENCES

1. Brennan, P. J., and Barrow, W. W. Evidence for species-specific lipid antigens in *M. leprae*. Int. J. Lepr. Other Mycobact. Dis. **48** (1980) 382–387.
2. Convit, J., Aranzazu, N., Ulrich, M., Pinardi, M. E., Reyes, O., and Alvarado, J. Immunotherapy with a mixture of *Mycobacterium leprae* and