lesion of the lateral trunk of the sciatic nerve from a common peroneal neuropathy, EMG abnormalities in the short head of the biceps femoris muscle are significant.⁵

Despite the addition of the above-mentioned methods, sciatic nerve neuropathy may be a diagnostic problem. In some of our patients, space-occupying lesions compressing the nerve, such as hematomas or tumors, and nerve-sheath tumors were detected by MRI studies. The clinically and electrophysiologically guided use of neuroimaging methods such as ultrasound, CT, and MRI has an important impact on diagnosis and prognosis in sciatic nerve lesions.^{6,7}

Udo Zifko, MD
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Reply from the Authors: We appreciate the interest and comments of Dr. Zifko. We found his observation of hyperkeratosis and anhidrosis in patients with tibial division deficits particularly interesting. We have not made similar observations in our group of patients and wonder whether different clinical features in his patients, such as greater chronicity or severity, may explain his different experience.

With regard to our clinical grading system, several factors limit the utility of including hamstring strength as proposed by Dr. Zifko. We designed our clinical grading system to allow objective evaluation of the most debilitating aspects of sciatic neuropathy, namely, weakness of ankle dorsiflexion and plantar flexion. Hamstring weakness was rarely a severe feature in our patients. Furthermore, we do not believe that weakness of the hamstrings is a clinical hallmark of sciatic neuropathy. Indeed, fully one-half of our patients had clinically normal hamstring strength. As would be expected, the hamstring muscles are the first to show recovery, but we could not prove that early recovery of these muscles was a statistically significant prognostic factor for eventual recovery of ankle dorsiflexion or plantar flexion.

Regarding the clinical examination and EMG localization of sciatic neuropathies, our Methods section outlines our strict inclusion and exclusion criteria for selecting patients. These criteria excluded patients with evidence of neurologic deficits beyond the sciatic nerve distribution and patients with abnormalities in only peroneal or tibial distributions who did not have hamstring involvement. We are happy to reassure Dr. Zifko that our clinical evaluations include all the points raised by him and other points that can be found in any standard review of the topic.

Conventional nerve conduction studies of the distal peroneal, tibial, and sural nerves in proximal lower extremity nerve lesions serve only to document the extent of axonal loss and to localize lesions distal or proximal to the dorsal root ganglion. They alone are not sufficient in diagnosing sciatic neuropathy. We have not been convinced that either magnetic stimulation or electrical near-nerve-root stimulation is sufficiently sensitive or specific for the diagnosis of sciatic neuropathy, and we are not aware of any published study documenting the value of these techniques in this context.

Until magnetic stimulation and near-nerve electrical stimulation are better validated in the literature, conventional nerve conduction studies and EMG remain the cornerstone for diagnosing the vast majority of cases of sciatic neuropathy. Thus, we recommend obtaining the sural and superficial peroneal sensory nerve action potentials (SNAPs), the extensor digitorum brevis and abductor hallucis compound muscle action potentials (CMAPs), and the peroneal tibial F waves in all patients. The contralateral nerves may need to be studied for comparison. The saphenous SNAPs, tibialis anterior CMAPs, and soleus H reflex are also frequently helpful in selected cases. Needle EMG exam-

ination includes the peroneal and tibial innervated muscles above and below the knee, as well as several nonsciatic innervated muscles, including the gluteal muscles, femoral innervated muscles, and lumbosacral paraspinal muscles, in all patients with possible sciatic neuropathy.

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Correction

In the "Cuban Embargo" Correspondence exchange that appeared in the May issue (*Neurology* 1995;45:1032-1033), only one signatory (Oscar Papazian, MD, Miami, FL) was indicated for one of the "To the Editor" letters. The following names also should have appeared with that letter: Danilo Duenas, MD; Roberto Tuchman, MD; Jaime Baquero, MD; Kenneth Butler, MD; Israel Alfonso, MD; Marcel Deray, MD; Carlos Gadia, MD; Trevor Resnick, MD; Prasanna Jayakar, MD; Luis Alvarez, MD; Michael Duchowny, MD; Robert F. Cullen, Jr., MD; and Mark Epstein, MD. The editors apologize for the error.

Correction

In "Familial progressive subcortical gliosis: Presence of prions and linkage to chromosome 17" by Petersen et al, which appeared in the June issue (*Neurology* 1995;45:1062-1067), an author's name and degree were presented incorrectly in the byline. "T. Lynch, MRCPI" is the correct name and degree.

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Correction

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