chogenic is very difficult. We agree with Dr. Sudo, stated in our manuscript, that additional documentation of the attacks with PET or SPECT studies would have been helpful. However, these studies were not possible in our center at the time these patients were observed. With regards to the EEG recordings, however, all four patients were recorded with both scalp and sphenoidal electrodes.

In our cases, the diagnosis of psychogenic basilar migraine rested principally upon four factors[1]: failure to respond to aggressive antimigraine (or antiepilepsy) medication in all four patients; extensive diagnostic studies, including MRI, MRA, cerebral angiography, and interictal PET scans, failed to document any abnormalities; ability to provoke typical symptoms with suggestion and provocation in all four patients; and dramatic improvement in two patients with psychiatric treatment (similar to improvement rate) are all consistent with a psychogenic etiology and strongly argue against an organic etiology for the majority of these patients' attacks. As stated in our paper, any or all of these patients may have had common, classic, or basilar migraine attacks. However, the disabling symptoms for which they had sought very extensive neurologic diagnosis and therapy were most consistent with psychogenic attacks.

Diagnosing pain or other neurologic symptoms as psychogenic must be made with great caution. However, we believe that underdiagnosis of conversion symptoms is common. Epilepsy may serve by way of analogy. Before the introduction of video-EEG monitoring units, the diagnosis of nonepileptic psychogenic seizures was much less common. For example, during my residency in New York City between 1983 and 1986, only two or three patients had nonepileptic psychogenic seizures in the differential diagnosis out of at least 200 patients with inpatients and outpatients with "seizures" that I evaluated under the supervision of attending neurologists. Nonepileptic psychogenic seizures now represent approximately 20% of our inpatient unit and have been identified in over 30% of seizure patients in a general neurology setting using provocative testing with suggestion.[7] Regardless of the exact percentages, nonepileptic psychogenic seizures are now commonly recognized. Among patients with headache that is chronic and responds poorly to aggressive medication trials, it is likely that a surprising percentage have psychogenic factors contributing to, or largely accounting for, their symptoms. The challenge will be to accurately identify such patients.

Finally, the issue of distinguishing conversion disorder from malingering is raised. We agree that these disorders are quite distinct. However, their differential in clinical practice can be quite difficult despite psychiatric consultation and follow-up.

Orrin Devinsky, MD

New York, NY

References

1. Sanchez VF, Devinsky O, Hainline B, Weinreb H, Luciano D, Vazquez B. Psychogenic basilar migraine: report of four cases. Neurology 1995;45: 1291–1294.
2. Sturzenegger MH, Meienberg O. Basilar artery migraine: a follow-up study of 82 cases. Headache 1985;25:408–415.
3. Battistella PA, Ruffilli R, Dalla PF, et al. 99mTc HM-PAO SPECT in pediatric migraine. Headache 1990;30:646–649.
4. Seto H, Shimizu M, Futatsuya R, et al. Basilar artery migraine. Reversible ischemia demonstrated by Tc-99m HMPAO brain SPECT. Clin Nucl Med 1994;19:215–218.
5. Kristensen O, Sindrup EH. Psychomotor epilepsy and psychosis. II. Electroencephalographic findings (sphenoidal electrode recordings). Acta Neurol Scand 1978;57:370–379.
6. Sperling MR, O'Connor MJ. Comparison of depth and subdural electrodes in recording temporal lobe seizures. Neurology 1989;39:1497–1504.
7. Bazil CW, Kothari M, Luciano D, et al. Provocation of nonepileptic seizures by suggestion in a general seizure population. Epilepsia 1994;35: 768–770.

Corrections

In "Unilateral spatial neglect recovery after sequential strokes" by Vuilleumier et al., which appeared in the January issue (Neurology 1996;46:184–189), a sentence appeared incorrectly in the second paragraph of the **Discussion** section on page 187. The sentence reads "The cortical area in the dorsal angular gyrus, constituting the ventral lip of the intraparietal sulcus, corresponds precisely to the architectonic pneumoencephalographic (PEG) area, which Eidelburg and Galaburda[20] found to have a rightward size lateralization in human brains." It should have read: "The cortical area in the dorsal angular gyrus, constituting the ventral lip of the intraparietal sulcus, corresponds precisely to the PEG area, which Eidelburg and Galaburda[20] found to have a rightward size lateralization in human brains."

In "Delayed emergence of a parkinsonian disorder in 38% of 29 older men initially diagnosed with idiopathic rapid eye movement sleep behavior disorder" by Schenk et al., which appeared in the February issue (Neurology 1996;46:388–393), a sentence appeared incorrectly in the first paragraph of the **Methods** section. It read: "In a series of 96 RBD patients (mean age, 52 yr; range, 9–81 yr),[5] 33 men ≥ who had idiopathic RBD at the time of initial diagnosis; 88% of these men were available for follow-up." It should have read: "In a series of 96 RBD patients (mean age, 52 yr; range, 9–81 yr),[5] 33 men ≥ 50 years who had idiopathic RBD at the time of initial diagnosis; 88% of these men were available for follow-up."

In "Brain choline-containing compounds are elevated in HIV-positive patients before the onset of AIDS dementia complex: A proton magnetic resonance spectroscopic study" by Tracey et al., which appeared in the March issue (Neurology 1996;46:783–788), a second corresponding author's address was omitted. The additional corresponding author is: Dr. B.A. Navia, Department of Neurology, Massachusetts General Hospital, Boston, MA 02118.

Correction

Neurology 1996;46;1787-1787-a
DOI 10.1212/WNL.46.6.1787-a