2. Linet MS, Stewart WF. Migraine headache: epidemiological perspectives. Epidemiol Rev 1984;6:107-139.
3. Tatemichi TK, Mohr JP. Migraine and stroke. In: Barnett HJM, Stein BM, Mohr JP, Yatsu FM, eds. Stroke: pathophysiology, diagnosis, and management. New York: Churchill Livingstone, 1986:845-863.
4. Broderick JP, Swanson JW. Migraine-related strokes. Arch Neurol 1987;44:868-871.
5. Rothrock JF, Walicke P, Swenson MR, Lyden PD, Logan WR. Migrainous stroke. Arch Neurol 1988;45:63-67.
6. Bogousslavsky J, Regli F, Van Melle G, Payot M, Uske A. Migraine stroke. Neurology 1988;38:223-227.
7. Welch KMA, Levine SR. Migraine-related stroke in the context of the International Headache Society classification of head pain. Arch Neurol 1990;47:458-462.
8. Selby G, Lance JW. Observations on 500 cases of migraine and allied vascular headache. J Neurol Neurosurg Psychiatry 1963;23:23-36.
9. Collaborative Group for the Study of Stroke in Young Women. Oral contraceptives and stroke in young women: associated risk factors. JAMA 1975;231:718-722.
10. Hart RG, Miller VT. Cerebral infarction in young adults: a practical approach. Stroke 1983;14:110-114.
11. Iglesias S, Visy JM, Hubert JB, Tehindrazanarivelo A, Tzourio C, Bousser MG. Migraine as a risk factor for ischemic stroke: a case-control study [abstract]. Stroke 1993;24:171.
12. Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias, and facial pain. Cephalalgia 1988;8:27.
13. Olesen J, Friberg L, Olsen SK, et al. Ischaemia-induced (symptomatic) migraine attacks may be more frequent than migraine-induced ischaemic insults. Brain 1993;116:187-202.
14. Oleson J. Some clinical features of the acute migraine attack: an analysis of 750 patients. Headache 1978;18:268-271.
15. Caplan LR. Migraine and vertebrobasilar ischemia. Neurology 1991;41:55-61.
16. Sacquegna T, Andreoli A, Baldrati A, et al. Ischemic stroke in young adults: the relevance of migrainous infarction. Cephalalgia 1989;9:255-258.
17. Featherstone HJ. Clinical features of stroke in migraine: a review. Headache 1986;26:128-133.
18. Webster AWI, Smith HJ, Sharpe DN, et al. Patent foramen ovale in young stroke patients. Lancet 1988;2:11-12.
19. Lechat P, Mass LJ, Laschault G, et al. Prevalence of patent foramen ovale in patients with stroke. N Engl J Med 1988;318:1148-1152.
20. Levine SR, Deegan MJ, Futrell N, Welch KMA. Cerebrovascular and neurologic disease associated with antiphospholipid antibodies: 48 cases. Neurology 1990;40:1181-1189.
21. Brey RL, Hart RG, Sherman DG, Tegeler CH. Antiphospholipid antibodies and cerebral ischemia in young people. Neurology 1990;40:1190-1196.

Migraine-related seizures in adults with epilepsy, with EEG correlation

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Article abstract—We studied the relationship between migraine and epilepsy in 395 adult seizure patients. Seventy-nine patients (20%) also had migraine syndrome, and 13 of these patients (3%) experienced seizures during or immediately following a migraine aura. Patients with catamenial epilepsy and patients with migraine with aura were at an increased risk for an association between these two disorders. In two patients, we recorded the entire sequence from migraine aura to partial seizure, and in both there were distinctive changes on the EEG during the migraine aura that preceded the onset of an electrographic complex partial seizure. Periodic lateralized epileptiform discharges were recorded in five other patients in close temporal relation to their migraine attacks. There was improved seizure control with combination antimigraine and antiepileptic drugs (AEDs) in six patients who failed to respond to AEDs alone.

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Gowers[1] remarked on the difficulty inherent in differentiating the symptoms of migraine from those of epilepsy. Lennox and Lennox[2] credited Dr. Douglas Davidson with the first use of the term "migralepsy" to refer to patients with classic migraine that subsequently evolved to an epileptic seizure. Despite many reviews of the relationship between migraine and epilepsy, the nature of this association is still unresolved.[3-7] The dilemma is compounded by the lack of objective diagnostic criteria for migraine, necessitating reliance on the patient's history to make the distinction.

The distinctive features of migraine have been found in several pediatric epilepsy syndromes. These include benign epilepsy of childhood with occipital spike-wave complexes,[8] benign rolandic

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epilepsy with migraine,[9] mitochondrial encephalomyopathy with lactic acidosis and strokes,[10] basilar migraine with seizures,[11] and migraine with primary generalized absence.[12] There is a less well-defined interaction in adults. Because of the high prevalence of each disorder, the relationship is often considered coincidental. The purpose of this study was to analyze the association between migraine and epilepsy in an adult population and to investigate whether migraine triggers seizures in patients with both disorders. In addition, we examined how migraine impacted on the management of epilepsy in patients with both disorders. Since many of our patients underwent continuous EEG and audiovisual monitoring, we analyzed the electrographic characteristics of acute migraine attacks.

Methods. *Fatient population.* The initial study population (n = 395) consisted of all patients (ages 16 to 76 years) who were referred to our seizure clinic and seen by one of us (B.L.E.) during the 6-year period between January 1, 1985, and December 31, 1990. This included 212 patients (92 women) with a diagnosis of definite epilepsy defined by clinical and EEG criteria, and classified according to the proposed International Classification of Epilepsy and Epileptic Syndromes,[13] and 183 patients (93 women) with a history of generalized tonic-clonic (GTC) seizures but without EEG abnormalities. Patients with atypical paroxysmal spells, sleep disturbances and parasomnias, and blackouts of uncertain type were excluded. From the initial 395 patients, we identified 79 (52 women) with a diagnosis of both a seizure disorder and migraine, and we studied the migraine-seizure relationship in these 79 patients.

Procedure and analysis. All 395 patients in our seizure clinic were questioned for symptoms of migraine, and if they met the criteria for migraine as defined by the Headache Classification Committee of the International Headache Society,[14] they were included in the final study group. Migraine was classified as *migraine without aura* (common migraine), *migraine with aura* of 5 to 60 minutes' duration or *migraine with prolonged aura* lasting 60 minutes to 7 days (classic migraine), *migrainous infarction* (symptoms of aura persisting longer than 7 days), *migraine aura without headache*, or *basilar migraine*. Patients with unilateral postictal headache but no other migraine characteristics were excluded since it is difficult to reliably differentiate migraine without aura from postictal headache.

Patients were asked to describe their migraine aura to determine whether this was distinct from their seizure aura. We reviewed the temporal relationship between the migraine and seizure events from clinical descriptions provided by the patient and any family member or observer. We inferred a direct relationship between the two disorders only if seizures occurred during the migraine or migrainous aura. Seven patients were admitted for prolonged EEG monitoring and simultaneous audiovisual recording. Hard copy EEG recordings of both the migraine and seizure attacks were reviewed. Seizure localization was determined from a combination of the clinical seizure characteristics, interictal and ictal EEG, and MRI findings.

Because of the high proportion of women in our study group, the temporal relationship of migraines and seizures to the menstrual cycle was examined. Catamenial migraine or catamenial epilepsy was defined as a clustering of greater than 50% of attacks during days 21 to 28 of an average monthly cycle (the week prior to onset of menstruation). The response of migraines and seizures to antimigraine medication was assessed to determine whether better migraine control resulted in better seizure control. Patients were routinely followed at 3-month intervals. A greater than 50% reduction in seizure frequency between visits constituted a beneficial response.

Results. *Patient population.* The table summarizes the characteristics of all 13 patients. Of 395 epilepsy patients (47% women), 79 (20%) had migraine; 52 (66%) of these 79 patients with both disorders were women. Thirteen of the 79 patients demonstrated a complex and direct interaction between their migraine attacks and seizures. In all 13 patients, seizures occurred during or at the end of a migraine aura. These 13 patients represented 3% of the original population of 395 seizure clinic patients. No definite temporal relation was observed between migraine and seizures in the remaining 66 patients, and the relationship between the two disorders in these patients remains unclear.

Seizure type and EEG characteristics. Five patients had complex partial (CP) seizures, which included three with a temporal lobe focus and two who were not localized. Four patients had primary GTC seizures, with a 3-Hz generalized spike-wave EEG pattern in three and a normal EEG in the other. Three further patients had simple partial (SP) seizures with rhythmic motor activity (epilepsia partialis continua [EPC]) and periodic lateralized epileptiform discharges (PLEDs) in the EEG. A final patient had brief SP motor attacks that rapidly evolved to secondary GTC seizures.

We recorded PLEDs in close association to the migraine attacks in five patients. No acute structural lesion was identified clinically or radiographically in three of these patients to account for the PLEDs. A frontotemporal glioma in remission (patient 4) and a migrainous infarct (patient 3) were present in the other two patients, but in both cases, despite their lesions, the PLEDs were absent on numerous 24-hour EEGs and were only found during an episode of migraine with prolonged aura. Four of the patients with PLEDs had catamenial epilepsy (patients 3, 5, 6, and 7).

Migraine type. All 13 patients who demonstrated a close association between their migraine attacks and seizures had migraine with aura, and the migraine prodrome preceded or was associated with seizure onset in all thirteen. This included three patients with basilar migraine, six with typical aura, and four with prolonged aura. Two of the patients with prolonged aura also sustained migrainous infarctions. Seizures and migraines increased during the week preceding menstruation in seven of the 11 women and occurred immediately postpartum in another. In patient 1, initial EEG moni-

Table. Patients with definite migraine-seizure relationship

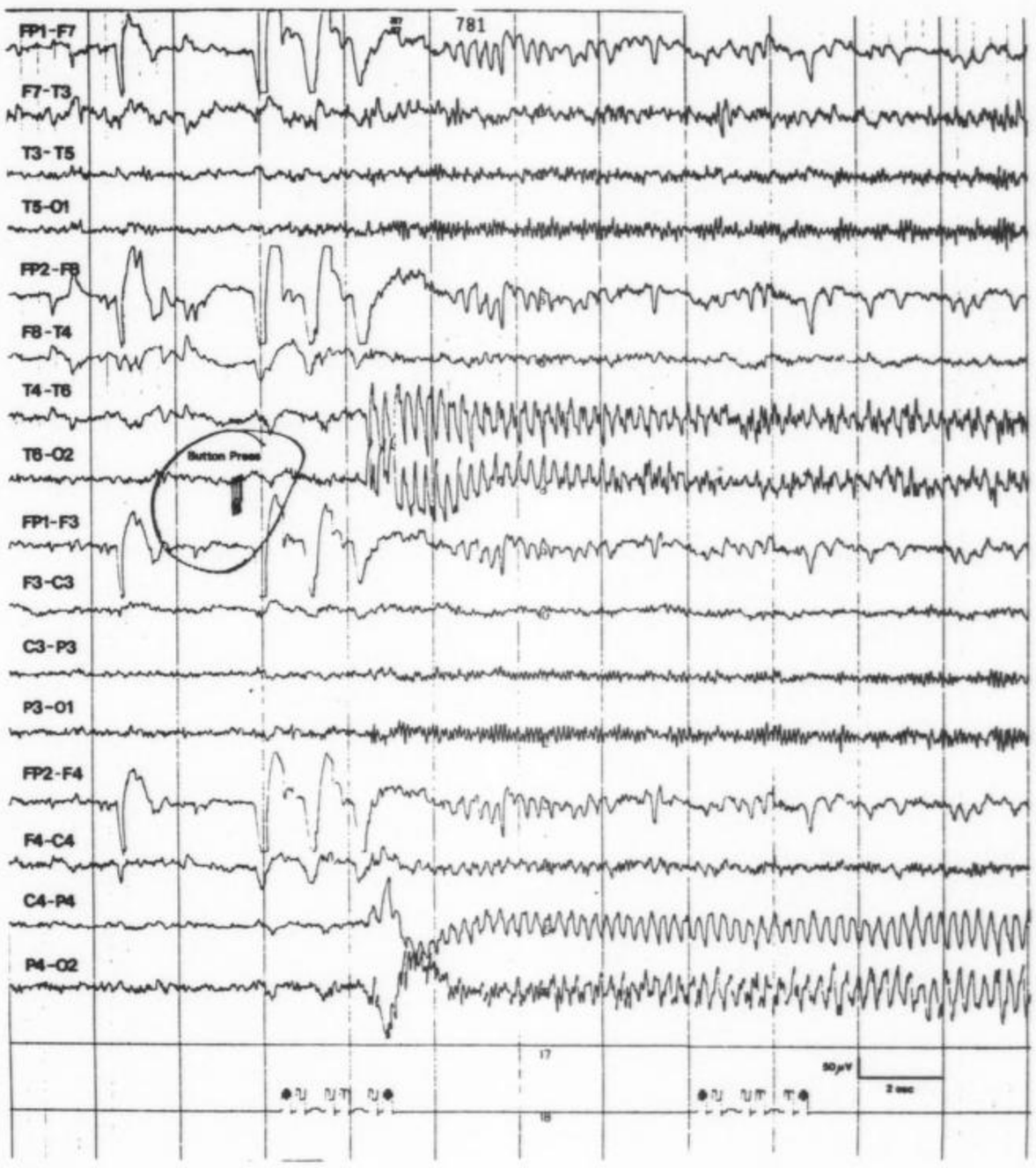
Pt	Sex	Age	Migraine type	Seizure type	EEG: interictal	EEG: during migraine	Relation to menses	Response to treatment
1	F	26	B	CP	L ant temp spike	Posterior temp rhythmic theta	No	AED + CCB
2	F	38	T	CP	L ant temp spike	Occipital rhythmic theta	Yes	Refractory
3	F	31	P, I	CP	Normal	PLEDs	Yes	AED alone
4	F	34	T, I	SP, EPC	Normal	PLEDs	No	AED + CCB
5	F	30	P	CP	R ant temp spike	(bi)PLEDs	Yes	AED + progesterone
6	F	21	T	SP, EPC	Generalized spike-wave	PLEDs	Yes	AED + CCB
7	F	49	P	SP, EPC	Normal	PLEDs	Yes	AED + CCB
8	M	33	T	Primary GTC	Generalized spike-wave	ND	Not applicable	AED + propranolol
9	M	16	T	Primary GTC	Generalized spike-wave	ND	Not applicable	AED + cyproheptadine
10	F	28	T	CP	Normal	ND	Yes	AED alone
11	F	21	B	Primary GTC	Normal	ND	No (postpartum)	AED alone
12	F	45	B	Primary GTC	Generalized spike-wave	ND	Yes	Refractory
13	F	35	P	SP	Normal	ND	No	AED alone

Migraine type: B = basilar, T = with typical aura, P = with prolonged aura, I = migrainous infarction.
Seizure type: CP = complex partial, SP = simple partial, EPC = epilepsia partialis continua, GTC = generalized tonic-clonic.
EEG: L = Left, R = right, ant = anterior, (bi)PLEDs = (bilateral independent) periodic lateralized epileptiform discharges, ND = no data, AED = antiepileptic drug, CCB = calcium channel blocker.

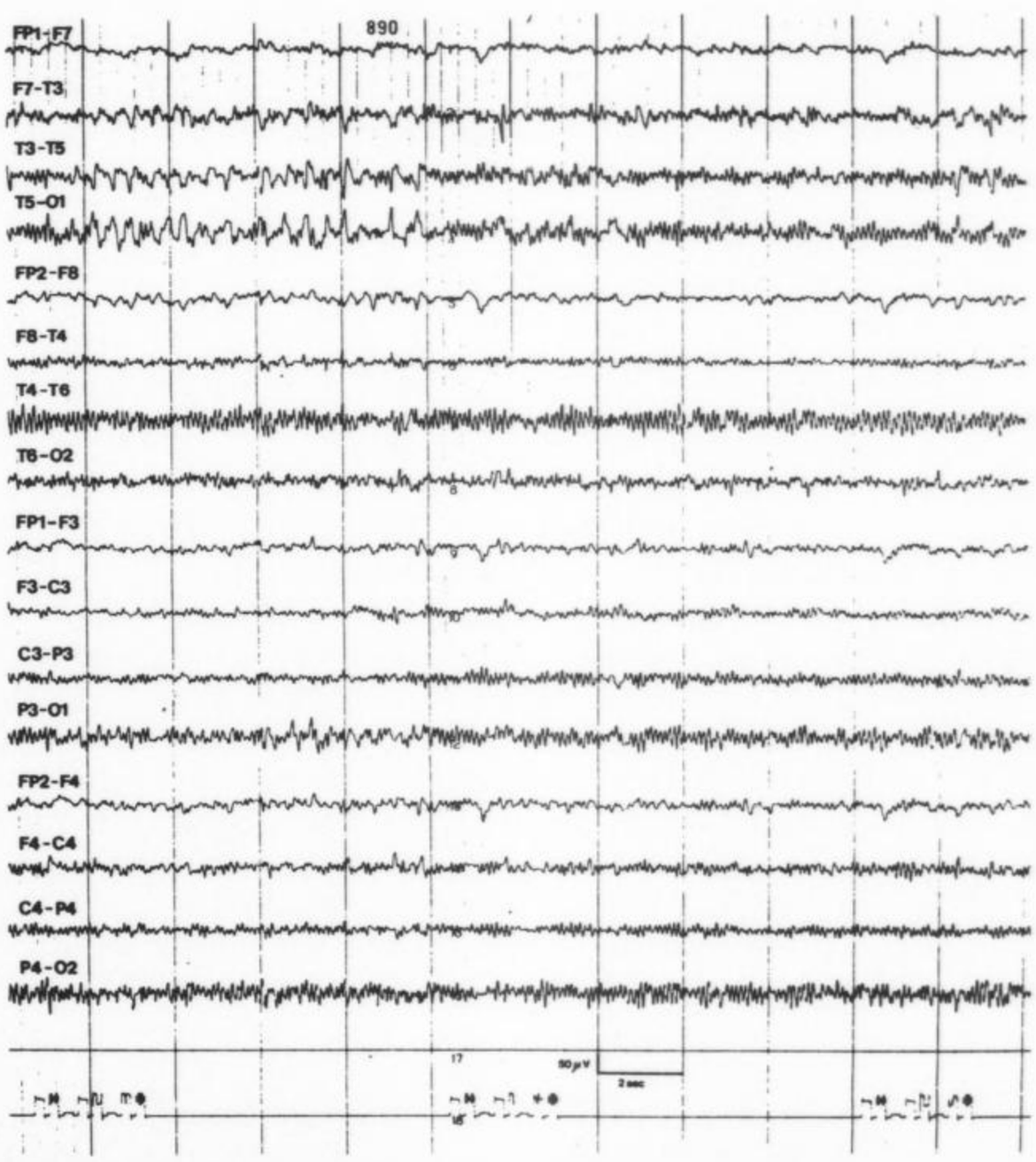
toring during basilar migraine auras showed only normal alpha activity. Subsequently, more intense spells were recorded in association with high-amplitude rhythmic 2.5- to 4.5-Hz sharp activity maximal in the right posterior quadrant (figure 1A), or, occasionally, the left (figure 1B). On occasion, such spells evolved into a CP seizure that rapidly generalized. Patient 2 experienced 15- to 30-minute episodes of decreased vision associated with nausea; these typical migraine auras were sometimes followed by a seizure. During EEG monitoring, bioccipital bursts of 5-Hz slowing accompanied the visual prodrome (figure 2A), which, after 18 minutes, evolved to a CP seizure with left anterior temporal buildup (figure 2B) followed by rapid generalization. In patients 3 and 4, PLEDs were recorded during the early stages of a prolonged migraine aura with a subsequent migrainous infarction. In patient 5, EEG monitoring during a premenstrual migraine showed PLEDs maximal in the left temporal region (figure 3A). A CP seizure followed 16 seconds later, when the PLEDs were superseded by a rhythmic left temporal ictal sharp-wave pattern. During another premenstrual exacerbation of headaches and seizures, the EEG showed bitemporal independent PLEDs (figure 3B). PLEDs were likewise recorded during attacks of migraine with aura in patients 6 and 7, both of whom also had EPC, although PLEDs occurred at other times without clinical accompaniment. In addition to patient 1, basilar migraine occurred in patients 11 and 12. Patient 11 had symptoms of basilar migraine that evolved to a GTC seizure on the second postpartum day; 3 days later, she developed transient paraplegia, and her MRI showed an increased T_2 signal in the central pontine region which later resolved as she clinically recovered.

Response to antimigraine treatment. Six of 13 patients (patients 1, 4, and 6 to 9) who were refractory to antiepileptic drugs (AEDs) responded to a combination of antimigraine medication and AEDs. Patients 1, 4, 6, and 7 responded to a calcium channel blocker (CCB) plus a single AED. Patient 8 was controlled on valproate and propranolol, while patient 9 responded to valproate and cyproheptadine. Patient 5 responded to a combination of a progesterone agent and AEDs. Four patients with infrequent seizures were controlled on AEDs alone, and two remaining patients have remained refractory to all pharmacologic manipulation with AEDs, antimigraine drugs, and progestational agents.

Discussion. The prevalence of migraine in early general population studies ranged from 1.7% to 63%,[4,5,15] with more recent studies still showing wide ranges of 5% to 20% for men and 15% to 29% for women.[16,17] Such variability among prevalence figures may be further reduced as the new uniform criteria for defining "migraine" come into widespread use.[14] The reported frequency of migraine in an epileptic population is less variable, with a range of 8.4% to 15%.[18] Our figure of 20%,



A



B

Figure 1. (A) Patient 1 had high-voltage rhythmic activity over right posterior temporal-parietal leads (T4-T6, T6-O2, C4-P4, P4-O2) beginning 4 seconds after she pressed the alarm button (see circled event marker) indicating onset of a basilar migraine aura. Note emergence of 9- to 10-Hz alpha activity on the opposite side (T5-O1, P3-O1) and reduced-amplitude eyeblink artifacts due to ptosis (Fp1, Fp2), all occurring after onset of the paroxysmal 3.5-Hz activity. (B) This 24-second sample, recorded during another basilar migraine episode in the same patient, shows waxing and waning paroxysmal 2- to 3-Hz slow and sharp activity in the left posterior temporal area (T3-T5, T5-O1). (All figures except figure 3B use half-standard graph speed and contain the time of day—written sideways—in the bottom channel.)

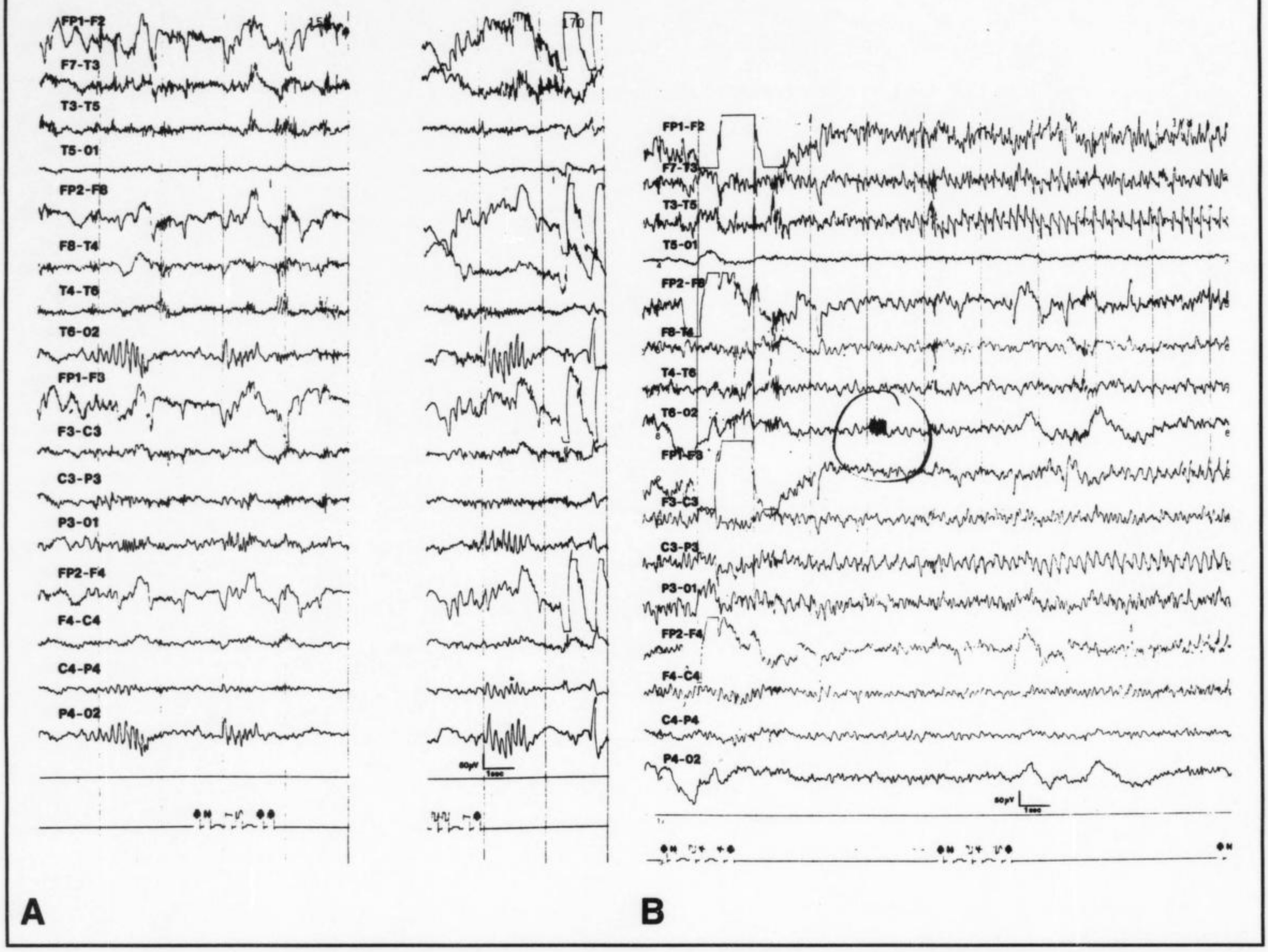
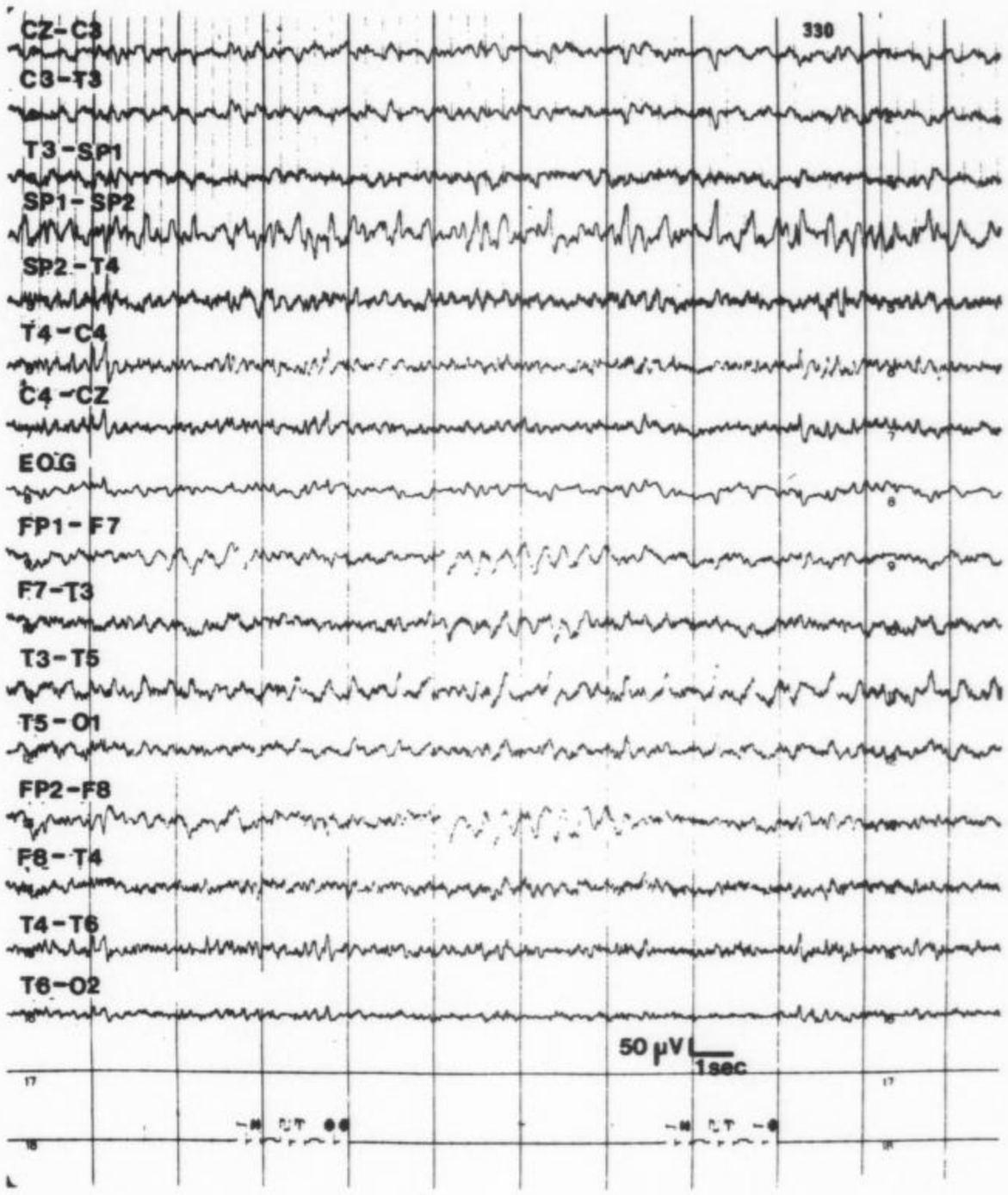


Figure 2. (A) Patient 2 had these EEG samples, taken 7 minutes apart, during an 18-minute episode of typical migraine aura with loss of vision and nausea. The EEG shows bursts of medium-high voltage 5-Hz rhythmic activity in the right occiput (O2).(B) This EEG segment was recorded 2.5 minutes from the end of the previous figure, at onset (see circled event marker) of a clinical complex partial seizure that subsequently generalized. The EEG shows rhythmic 3- to 4-Hz sharp activity maximal at T3-T5 and C3-P3. (Note: channel 4 [T5-O1] was disconnected.)

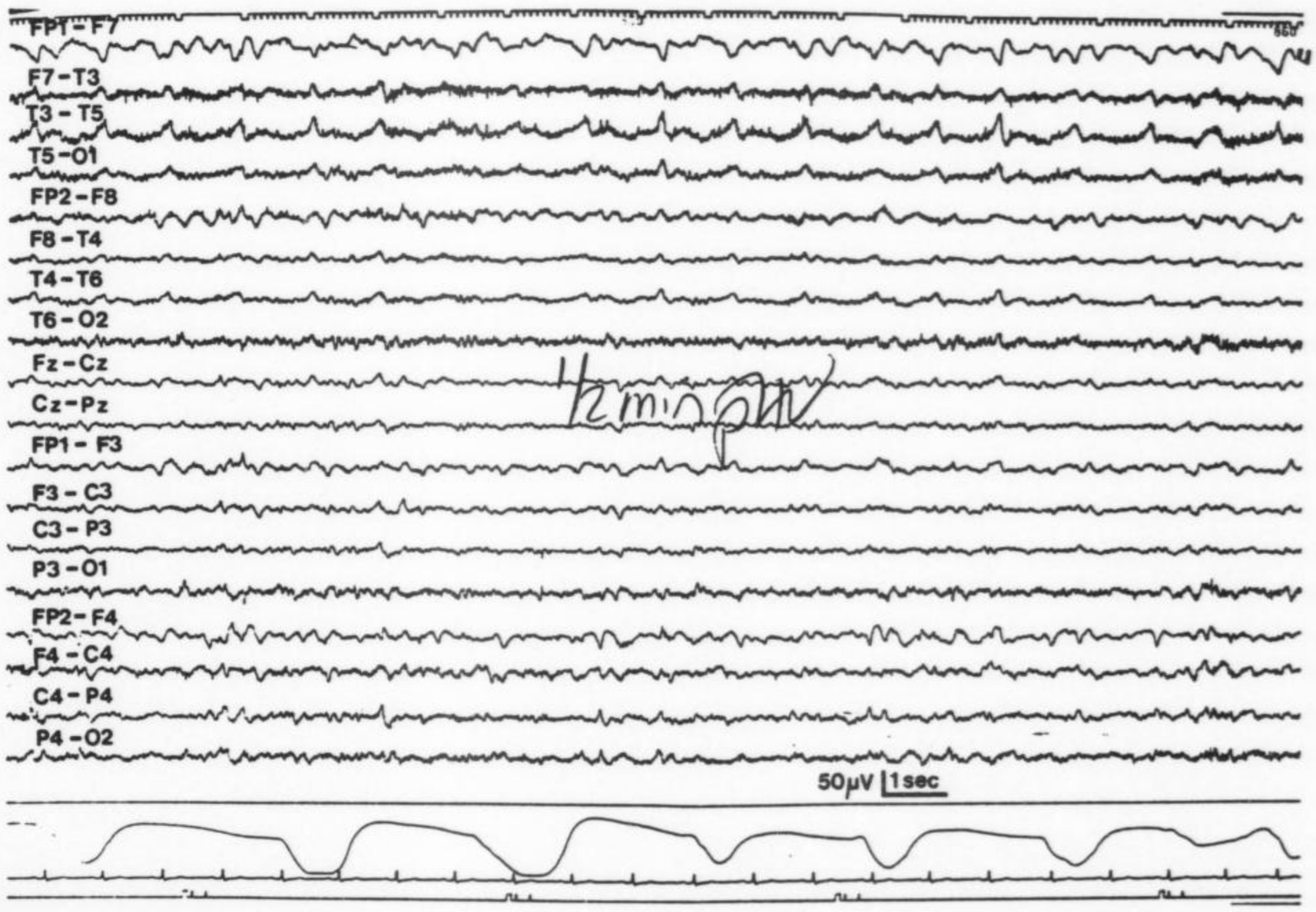
while reasonable for a general population, was higher than other epilepsy population studies[18] because we specifically looked for evidence of migraine in all seizure patients. The frequency of epilepsy in migraineurs is also variable, ranging from 1% to 17%,[18] with the higher figures possibly linked to over-interpretation of EEG abnormalities in migraine patients.[19-21]

We found a definite association between migraine and epilepsy in 3% of adult patients with seizures. Most patients who showed this relationship were refractory to medical management with routine AEDs using either monotherapy or combination therapy, and improved seizure control involved better suppression of the migraine component. A serum level of a CCB in the therapeutic range for hypertension (50 to 200 ng/ml for verapamil) was adequate for improved control of the migraine-seizure sequence in four of five patients when added to the existing AED regimen. CCB are reported to have antiepileptic properties,[22-24] and this may account for the improved seizure control in these four patients, but these drugs are not widely used in human epilepsy.

Only migraine with aura was associated with seizures, possibly because of the distinctive blood flow changes during the migraine aura. Regional cerebral blood flow (rCBF) is decreased during migraine with aura but not in migraine without aura.[25] The reduced rCBF present during migraine aura, or the associated spreading depression, may trigger the subsequent seizures. However, we may have biased our study toward patients who have migraine with aura and excluded patients who have migraine without aura since we rejected patients who had postictal headaches but no other migraine characteristics. In our patients, seizures occurred during, or at the conclusion of, the migraine aura and generally preceded any headache (as in so-called intercalated seizures[24]). The mi-



A



B

Figure 3. (A) Patient 5 had periodic lateralized epileptiform discharges (PLEDs) in the left hemisphere, maximal at Sp1-F7-T3, recorded during a prolonged migraine aura. There were also right-sided anterior temporal spikes. (Note: EOG is recorded from right to left outer canthi). (B) The same patient, during another migraine-seizure exacerbation, had EEG monitoring that showed bitemporal independent PLED-like activity. (Note different montage.)

graine auras were longer than SP seizure auras since they lasted at least 5 minutes (typical aura) and as long as several days (prolonged aura). Nausea was associated with the migraine prodrome and complicated the diagnosis, since nausea is a frequent aura of temporal lobe seizures.[26]

It is unclear why migraine should have a stronger influence in children with epilepsy compared with adults.[8-12] The explanation may be linked to the greater frequency of occipital lobe epileptogenic abnormalities in the pediatric compared with the adult population.[8] Because the metabolic and blood flow changes present during migraine preferentially involve the posterior cerebral region, these posterior epileptogenic foci may be triggered by the migrainous changes.[25]

The literature concerning EEG abnormalities in migraine patients is confusing because most studies did not make a distinction between migraine with and without aura, and there were no strict criteria to determine what constituted an EEG abnormality. A normal EEG or nonspecific slowing are the usual findings in patients who have migraine without aura, or interictally in patients who have migraine with aura.[19-21] During acute migraine with aura or acute basilar migraine, the EEG may be normal or show unilateral or bilateral posterior rhythmic delta or theta activity often reactive to eye-opening, absent posterior rhythms, periodic slow transients, or occipital interictal epileptiform discharges.[11,27-30] In the only study that captured the clinical transition to an epileptic seizure, Beaumanoir and Grandjean[31] illustrated an EEG (from their case 6) recorded during a nocturnal seizure; the clinical pattern suggested a link to daytime migrainous attacks, but there was no confirmation of the migraine aura since the patient was asleep until the onset of the seizure.

We captured the migraine-seizure transition in patients 1 and 2. During a basilar migraine in patient 1 (figure 1, A and B), there were prolonged bursts of repetitive posterior rhythmic sharp waves, alternating with a normal background. The paroxysmal activity sometimes changed sides between attacks but was only posterior. A seizure with left temporal onset and rapid generalization was recorded following one such episode. We also recorded a similar migraine-seizure transition in patient 2 (figure 2, A and B), characterized electrographically by rare bursts of right occipital rhythmic theta during a migrainous visual aura, followed 18 minutes later by a seizure with left temporal onset.

We also recorded PLEDs (figure 3A) in association with a migraine in five patients (table), and in one, they occurred independently on both sides (figure 3B), without associated lesions on imaging and with subsequent complete clinical and EEG recovery. The PLEDs were associated with prolonged focal motor status (EPC) in three other patients, again with complete recoveries. Since PLEDs are associated with acute brain lesions (usually vascular) and represent a dynamic EEG phenomenon,[32] we attribute the PLEDs to the acute metabolic and blood flow changes present during the migraine prodrome. Gastaut et al[33] recorded PLEDs during an attack of familial hemiplegic migraine, followed by complete clinical and EEG recovery.

The biochemical abnormalities and pathophysiologic mechanisms responsible for the migraine-seizure relationship are speculative, although certain observations among our patients provide clues to the possible mechanisms. Because we only observed an association in patients having migraine with aura, our data implicate the metabolic changes that occur during periods of reduced rCBF during the migrainous aura. Focal ischemia will reduce seizure thresholds in animals,[34] but the same degree of ischemia is unlikely in migraine, except in migrainous infarction. Seizure threshold is also reduced in animals during spreading depression, an effect thought to be due to increased extracellular glutamate and potassium.[35] Although spreading depression per se has not been documented on EEG in humans, this may be secondary to technical difficulties in recording slow direct current shifts, since dynamic magnetic field changes have been recorded using magnetoencephalography.[36] Hormonal and endocrine changes prior to menstruation may lower both migraine and seizure thresholds, and our patient group showed a striking catamenial pattern.[37-39] Among our patients, 10/13 had easily detectable occipital photic following responses at 25 Hz or above. Since both migraineurs and patients with primary generalized epilepsy often have an increased driving response during photic stimulation, this may represent similar cortical hyperexcitability in both conditions.[20,21] Therefore, in migraine patients, a spike-wave EEG pattern, photoconvulsive response, or family history of primary generalized epilepsy may represent a subgroup at increased risk for seizures.

Migraine symptomatology in patients with epilepsy should be analyzed since there is a confusing overlap of clinical and electrographic features between these two entities. Better seizure control may be obtained by specifically emphasizing the treatment of the migraine component, but a controlled prospective study is needed to make a more definitive conclusion. It should be emphasized that a strong causal relationship between migraine and epilepsy is uncommon, identified in only 3% of our tertiary referral hospital's adult epilepsy clinic population, contrasting with 20% who carried both diagnoses. Nevertheless, since a high index of suspicion is needed to identify any such patients, it is recommended that seizure patients be thoroughly questioned for migraine symptomatology.

References

1. Gowers WR. The borderland of epilepsy: faints, vagal attacks, vertigo, migraine, sleep symptoms and their treatment. London: Churchill, 1907.

2. Lennox WG, Lennox MA. Epilepsy and related disorders. Boston: Little, Brown, 1960:451.
3. Basser LS. The relation of migraine and epilepsy. Brain 1969;92:285-300.
4. Barolin C. Migraine and epilepsy—a relationship. Epilepsia 1966;7:53-66.
5. Andermann F. Clinical features of migraine-epilepsy syndromes. In: Andermann F, Lugaresi E, eds. Migraine and epilepsy. Boston: Butterworths, 1987:3-30.
6. Bruyn GW. Migraine and epilepsy. Funct Neurol 1986;1:315-331.
7. Ehrenberg BL. Unusual clinical manifestations of migraine, and "the borderland of epilepsy"—reexplored. Semin Neurol 1991;11:118-127.
8. Gastaut H. A new type of epilepsy: benign partial epilepsy of childhood with occipital spike-waves. Clin Electroencephalogr 1982;13:13-22.
9. Kinast M, Lueders H, Rothner AD, Erenberg G. Benign focal epileptiform discharges in childhood migraine (BFEDC). Neurology 1982;32:1309-1311.
10. Dvorkin GS, Andermann F, Carpenter S, et al. Classical migraine, intractable epilepsy and multiple strokes: a syndrome related to mitochondrial encephalomyopathy. In: Andermann F, Lugaresi E, eds. Migraine and epilepsy. Boston: Butterworths, 1987:203-232.
11. Camfield PR, Metrakos K, Andermann F. Basilar migraine, seizures, and severe epileptiform EEG abnormalities: a relatively benign syndrome in adolescents. Neurology 1978;28:584-588.
12. Baier KW, Doose H. Migraine and petit mal absence: familial prevalence of migraine and seizures. In: Andermann F, Lugaresi E, eds. Migraine and epilepsy. Boston: Butterworths, 1987:293-311.
13. Commission on Classification and Terminology of the International League Against Epilepsy. Proposal for classification of epilepsy and epileptic syndromes. Epilepsia 1985;26:268-278.
14. Headache Classification Committee of the International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgias, and facial pain. Cephalalgia 1988;8(suppl 7):1-96.
15. Allen W. Inheritance of migraine. Arch Intern Med 1928;42:590-599.
16. Abramson JH, Hopp C, Epstein LM. Migraine and non-migrainous headaches. A community survey in Jerusalem. J Epidemiol Community Health 1980;34:188-193.
17. Waters WE, O'Connor PJ. Prevalence of migraine. J Neurol Neurosurg Psychiatry 1975;38:613-616.
18. Andermann E, Andermann F. Migraine-epilepsy relationships: epidemiological and genetic aspects. In: Andermann F, Lugaresi E, eds. Migraine and epilepsy. Boston: Butterworths, 1987:281-291.
19. Hockaday JM, Whitty CWM. Factors determining the electroencephalogram in migraine: a study of 500 patients according to clinical type of migraine. Brain 1969;92:769-788.
20. Ziegler DK, Wong G. Migraine in children: clinical and electroencephalographic study of families—the possible relation to epilepsy. Epilepsia 1967;8:171-187.
21. Slatter KH. Some clinical and EEG findings in patients with migraine. Brain 1968;91:85-98.
22. Wauquier A. Is there a common pharmacological link between migraine and epilepsy? Funct Neurol 1986;1:515-520.
23. Greenberg DA. Calcium channels and calcium channel antagonists. Ann Neurol 1987;21:317-330.
24. Terzano MG, Pietrini V, Parrino L, Milone FF. Migraine and intercalated seizures with occipital EEG paroxysms: observations on a family. Headache 1986;26:509-512.
25. Olesen J, Friberg L, Skyhøj OT, et al. Timing and topography of cerebral blood flow, aura, and headache during migraine attacks. Ann Neurol 1990;28:791-798.
26. So NK. Epileptic auras. In: Wyllie E, ed. The treatment of epilepsy: principles and practices. Philadelphia: Lea & Febiger, 1993:371-375.
27. Beaumanoir A, Jekiel M. Electrographic observations during attacks of classical migraine. In: Andermann F, Lugaresi E, eds. Migraine and epilepsy. Boston: Butterworths, 1987:163-180.
28. Panayiotopoulos CP. Basilar migraine? seizures, and severe epileptic EEG abnormalities. Neurology 1980;30:1122-1125.
29. Manzoni GC, Terzano MG, Mancia D. Possible interference between migrainous and epileptic mechanisms in intercalated attacks: case report. Eur Neurol 1979;18:124-128.
30. Swanson JW, Vick NA. Basilar artery migraine: 12 patients, with an attack recorded electroencephalographically. Neurology 1978;28:782-786.
31. Beaumanoir A, Grandjean E. Occipital spikes, migraine and epilepsy. In: Andermann F, Lugaresi E, eds. Migraine and epilepsy. Boston: Butterworths, 1987:97-110.
32. Markand ON, Daly DD. Pseudoperiodic lateralized paroxysmal discharges in electroencephalogram. Neurology 1971;21:975-980.
33. Gastaut J-L, Yermenos E, Bonnefoy M, Cros D. Familial hemiplegic migraine: EEG and CT study of 2 cases. Ann Neurol 1981;10:392-395.
34. Kogure K, Schwartzman RJ. Seizure propogation and ATP depletion in the rat stroke model. Epilepsia 1980;21:63-72.
35. Van Gelder NM. Calcium mobility and glutamic acid release associated with EEG abnormalities, migraine and epilepsy. In: Andermann F, Lugaresi E, eds. Migraine and epilepsy. Boston: Butterworths, 1987:367-378.
36. Simkins RT, Tepley N, Barkley GL, Welch KMA. Spontaneous neuromagnetic fields in migraine: possible link to spreading cortical depression [abstract]. Neurology 1989;39(suppl 1):325.
37. Backstrom T. Epileptic seizures in women related to plasma estrogen and progesterone during the menstrual cycle. Acta Neurol Scand 1976;54:321-347.
38. Backstrom T, Zetterlund B, Blom S, et al. Effects of intravenous progesterone infusion on the epileptic discharge frequency in women with partial epilepsy. Acta Neurol Scand 1984;69:240-248.
39. Schachter SC. Hormonal considerations in women with seizures. Arch Neurol 1988;45:1267-1270.