

NEUROLOGY

Epileptic pseudodementia

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Neurology 1998;50;1472-1475

This information is current as of July 22, 2008

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knowledge in humans, but on the basis both of positron emission tomography⁷ and of lesion mapping studies,⁸ the posterior insula is very likely to be part of the vestibular system.

As regards our patient, we hypothesize that ictal discharges produced an imbalance in vestibular cortex projections to vestibular nuclei. In monkeys, vestibular cortex ocular motor efferences mainly project to the ipsilateral vestibular nuclei complex.^{6,9} A decreased activation in medial vestibular nucleus along the left horizontal canal possibly produces a right-beating nystagmus. Moreover, according to the hypothesis developed by Brandt and Dieterich,¹⁰ left-eye hypotropia might derive from decreased activity in the ipsilateral medial vestibular nucleus along the left utricular or vertical canal pathways.

Overall, we believe that vestibular cortex ictal involvement would produce signs of both dynamic (the right-beating nystagmus) and static (the skew deviation) vestibular imbalance.

To our knowledge, there has been no report of a skew deviation arising from excitation of, or lesions in, the cerebral cortex. Recently, Brandt et al.⁸ described stroke patients in whom lesions involving the vestibular cortex induced an altered subjective perception of verticality, which is a sign of otolith function imbalance.

In conclusion, this is the first clinical and electrophysiologic report of the occurrence of epileptic skew deviation as the main ictal manifestation in an otherwise neurologically intact subject. The phenomenon arose from a cortical disturbance that possibly produced an abnormal activation of descending pathways to vestibular nuclei.

Acknowledgment

The authors are grateful to Dr. David S. Zee for his helpful suggestions.

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Received April 21, 1997. Accepted in final form September 29, 1997.

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References

1. Stoltz SE, Chatrian GE, Spence AM. Epileptic nystagmus. *Epilepsia* 1991;32:910-918.
2. Kaplan PW, Tusa RJ. Neurophysiologic and clinical correlations of epileptic nystagmus. *Neurology* 1993;43:2508-2514.
3. Gibbs FA, Gibbs EL. *Epilepsy. Atlas of encephalography*, vol 2. Cambridge, Mass.: Addison-Wesley Press, 1952:222-224.
4. Fois A, Malandrini F, Tomaccini D. Clinical findings in children with occipital paroxysmal discharges. *Epilepsia* 1988;29:620-623.
5. Pierrot-Deseilligny C. Saccade and smooth-pursuit impairment after cerebral hemispheric lesions. *Eur Neurol* 1994;34:121-134.
6. Akbarian S, Grüsser OJ, Guldin WO. Corticofugal projections to the vestibular nuclei in squirrel monkeys: further evidence of multiple cortical vestibular fields. *J Comp Neurol* 1993;332:89-104.
7. Bottini G, Sterzi R, Paulesu E, et al. Identification of the central vestibular projections in man: a positron emission tomography activation study. *Exp Brain Res* 1994;99:164-169.
8. Brandt Th, Dieterich M, Danek A. Vestibular cortex lesions affect the perception of verticality. *Ann Neurol* 1994;35:403-412.
9. Akbarian S, Grüsser OJ, Guldin WO. Corticofugal connections between cerebral cortex and brainstem vestibular nuclei in the macaque monkey. *J Comp Neurol* 1994;339:421-437.
10. Brandt T, Dieterich M. Vestibular syndromes in the roll plane: topographic diagnosis from brainstem to cortex. *Ann Neurol* 1994;36:337-347.

Epileptic pseudodementia

Article abstract—Dementia is a frequent cause of memory loss with aging. The incidence of complex partial seizures sharply rises after age 60. Complex partial seizures that occur with subtle clinical signs or loss of awareness, or occur during sleep may defy identification. We report five elderly patients fearing dementia in whom memory dysfunction was due to unrecognized complex partial seizures.

NEUROLOGY 1998;50:1472-1475

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Dementia is a common neuropsychiatric condition occurring in 5 to 11% of the population by age 65 and up to 50% beyond age 85.¹ Alzheimer's disease (AD) is the fourth leading cause of death, a major cause of morbidity, and a significant concern for the aged population. Multiple medical conditions are known to result in dementia. Evaluation to exclude treatable causes of cognitive dysfunction is essential for proper diagnosis and treatment.

The prevalence of seizures is highest early in life and peaks again after age 60.² Complex partial seizures represent more than 50% of new seizure cases in the elderly. Complex partial seizures manifest some combination of altered awareness and amnesia during an attack. Recurrent partial seizures causing transient amnesia have previously been recognized.^{3,4}

We report five elderly patients who presented

Table Summary of clinical data on patients with epileptic pseudodementia

Patient no.	Age (y)	Symptom duration (y)	No. of EEGs done	EEG recording method	EEG onset	Seizures on EEG	Symptoms	Clinical signs	Response to AEDs
1	79	25	2	24-hr ambulatory	Left temporal	3	Memory lapses	Waxing and waning amnesia	Declined AEDs
2	56	15	1	24-hr ambulatory	Left temporal	4	Anxiety and memory loss	Morning confusion; disoriented, poor recall	Resolution of memory deficits
3	67	3	2	24-hr ambulatory video EEG	Artifact	1	Episodic memory loss	Recurrent transient global amnesia 30–45 min	Resolution of memory deficits
4	70	7	4	video EEG	Left temporal	6	Poor memory and feeling weird	Period over 4–5 days of depression, severe confusion, memory loss, depersonalized	Incomplete response
5	70	1	2	routine EEG	Left temporal	1	Confusion and memory loss	Period of “spacey” mumbling, facial flushing	Resolution of memory deficits

AEDs = antiepileptic drugs.

with memory impairment masquerading as dementia caused by serial complex partial seizures. Ictal recordings confirmed the diagnosis after initial EEGs were nondiagnostic. Multiple complex partial seizures were recorded in three patients. Four patients improved with antiepileptic drugs (AEDs).

Case reports. Five patients were identified at two centers. Summary of their clinical presentation is detailed in the table. The figure reflects an ictal EEG recording taken from Patient 2.

Representative case report. A 70-year-old man presented to a memory disorders clinic with memory loss and confusion. He had no significant past medical history and was taking no medication. He complained of increasing emotional lability with periods when he would feel “spacey” and mumble, with facial flushing. Impaired consciousness was absent. This occurred one to two times per month initially and then became continuous at the time of presentation. Neurologic examination was normal except for mild declarative memory deficits on mental status testing. Brain MRI revealed mild white matter changes and two lacunar infarcts. EEG demonstrated intermittent left temporal slowing. Repeat EEG captured an electrographic partial seizure emanating from the left temporal region appearing clinically as prominent amnesia. Treatment with carbamazepine resulted in resolution of memory loss and confusion.

Discussion. Amnesia for the ictus is a feature of complex partial seizures with variable periods of anterograde amnesia postictally. Blum et al.,⁵ during

presurgical evaluation of intractable epilepsy with inpatient video EEG, found that 30% of patients were unaware of their seizures. Gallassi et al.³ reported 13 patients with brief “epileptic amnesic attacks” that appeared to be a distinct form of temporal lobe epilepsy. Halgren et al.⁶ reproduced disruption of memory formation and retrieval with bitemporal electrical stimulation. Bridgman et al.⁷ using intracranial electrodes found that recurrent focal hippocampal subclinical seizures were capable of limiting memory recall.

Amnesia as an isolated manifestation of seizures is rare. Palmieri et al.⁴ postulated that “pure amnesic seizures” resulted from selective ictal inactivation of the mesial temporal structures without cortical involvement. Epileptic amnesia has been viewed as a Todd’s paralysis of the limbic system with neocortical recovery. Clinical assessment during long-term monitoring demonstrated persistent subjective memory complaints in our patients despite return of baseline EEG between electrographic seizures. Our findings agree with Tassinari et al.,⁸ who previously described “epileptic transient global amnesia” as a postictal phenomenon in a patient using video EEG monitoring. Various degrees of peri-ictal anterograde and retrograde memory disturbance may occur.

All our patients initially complained of memory difficulties fearing AD. Memory loss with a waxing and waning course was described by all. Patients 3 and 5 had more discrete episodes of memory impairment lasting from 30 to 45 minutes to several days.

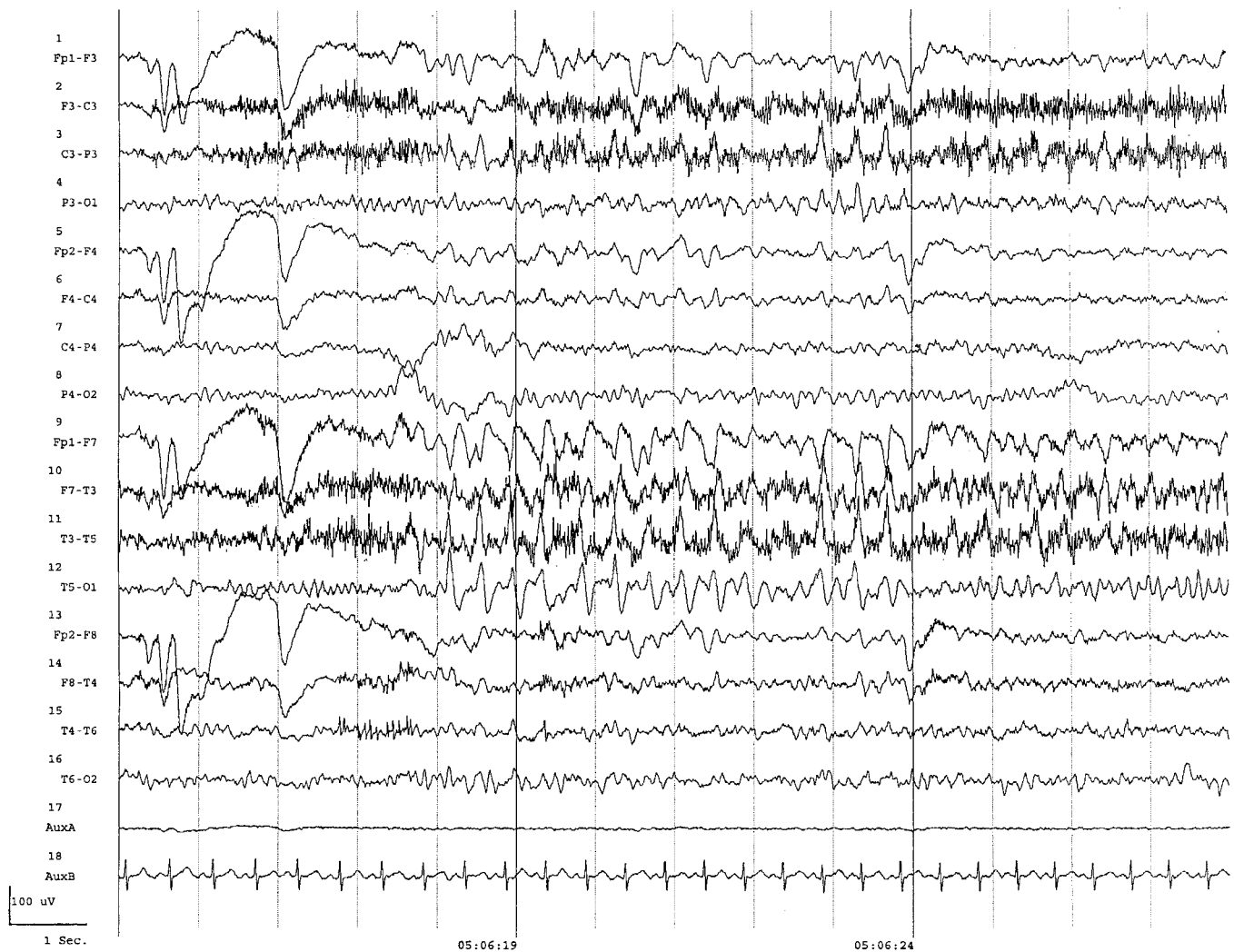


Figure. Representative EEG taken from Patient 2.

These identifiable episodes are similar to “epileptic amnesic attacks.” However, the duration of memory deficit in our patients was more prolonged and less discrete. Patients 2 and 4 had nocturnal seizures recognized only by the daytime sequelae. The heterogeneity of presentation supports the concept of an epileptic amnesic syndrome defined by Gallassi et al.³ Three of our patients had resolution of memory deficits with AED treatment. One patient improved with treatment, and one required an assisted care living facility because of memory impairment after declining AED treatment.

Formal neuropsychological testing in Patients 1 and 3 and the Mini-Mental State Examination in Patients 2, 4, and 5 demonstrated immediate memory deficits without global cognitive dysfunction. No progression of memory dysfunction was noted on follow-up testing in Patients 2 and 3, and no patient had objective evidence for dementia despite memory impairment.

Declarative memory likely involves hippocampal-entorhinal cortical connections for maintaining information; however, integration, indexing, processing, and storage depend on additional cortical projec-

tions. Hippocampal neuropathology coexisting with diffuse neocortical atrophy may be the neuroanatomic substrate for compromised retrieval and deficient storage mechanisms. Recurrent complex partial seizures may therefore lead to prolonged episodes of postictal memory dysfunction in elderly patients. Our first patient had right hippocampal atrophy on MRI with complex partial seizures originating from the left temporal lobe. Although atrophy may occur with aging, the possibility of bilateral hippocampal dysfunction created by both a structural and epileptogenic mechanism together may exist. Two of our patients demonstrated atrophy on brain MRI, and two patients had white matter ischemic changes. Such findings are frequent in elderly populations where unrecognized anatomic abnormalities may become clinically evident when complex partial seizures occur.

Seizures are usually diagnosed based on historical information supported with EEG. Approximately half of epilepsy patients have interictal epileptiform discharges (IEDs) on initial EEG. Still, some may not reveal IEDs despite repeated recordings.⁹ All our patients had initial nondiagnostic EEGs. Electro-

graphic partial seizures suggesting left temporal origin were captured in four patients. Seizure awareness has been noted to be lowest for left temporal seizures in one series.⁵ In one patient (Patient 3), ictal recording was obscured by artifact during an event designated as "memory lapse" on event diary. Subsequently, 24-hour inpatient video EEG captured no episodes.

We conclude that memory dysfunction may be caused by complex partial seizures and may present in two ways: discrete episodes of amnesia may occur or, alternatively, an insidious fluctuating course of memory dysfunction may simulate dementia. Prolonged EEG may help define an epileptogenic basis in patients with atypical or fluctuating dementia even when a routine EEG is normal. Subclinical, clinically subtle, or purely nocturnal partial seizures with amnesia may be detected by EEG. Frequent electrographic seizures may exist without apparent clinical signs. We recommend that a heightened level of suspicion for complex partial seizures in the elderly should be maintained. Epileptic pseudodementia represents a potentially reversible cause of memory dysfunction.

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Presented in part at the American Epilepsy Society meeting in San Francisco, CA, December 1996.

Received August 1, 1997. Accepted in final form November 18, 1997.

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References

1. Geldmacher DS, Whitehouse PJ. Differential diagnosis of Alzheimer's disease. *Neurology* 1997;48(suppl 6):S2-S9.
2. Hauser WA, Annegers JF, Kurland LT. Incidence of epilepsy and unprovoked seizures in Rochester, Minnesota 1935-1984. *Epilepsia* 1993;34:453-468.
3. Gallassi R, Morrcaie A, Di Sarro R, Lugaresi E. Epileptic amnesic syndrome. *Epilepsia* 1992;33(suppl 6):S21-S25.
4. Palmieri AL, Gloor P, Jones-Gotman M. Pure amnesic seizures in temporal lobe epilepsy: definition, clinical symptomatology, and functional anatomic considerations. *Brain* 1992; 115:749-769.
5. Blum DE, Eskola J, Bortz JJ, Fisher RS. Patient awareness of seizures. *Neurology* 1996;47:260-264.
6. Halgren E, Wilson CL, Stapleton JM. Human medial temporal lobe stimulation disrupts both formation and retrieval of recent memories. *Brain Cogn* 1985;4:287-295.
7. Bridgman PA, Malamut BL, Sperling MR, Saykin AJ, O'Connor MJ. Memory during subclinical hippocampal seizures. *Neurology* 1989;39:853-856.
8. Tassinari CA, Ciarmatori C, Alesi C, et al. Transient global amnesia as a postictal state from recurrent partial seizures. *Epilepsia* 1991;32:882-885.
9. Salinsky M, Kanter R, Dasheiff R. Effectiveness of multiple EEGs in supporting the diagnosis of epilepsy: an operational curve. *Epilepsia* 1987;28:331-334.

Carbamazepine responsive epileptic oral motor and ocular motor apraxia

Article abstract—We evaluated seven patients with oral motor apraxia and ocular motor apraxia. Apraxia in three patients (Group 1) with new-onset partial seizures and epileptiform discharges on EEG improved with carbamazepine. Four patients (Group 2) without seizures and nonepileptiform EEG findings had no change in apraxia after a trial of carbamazepine. Epileptic apraxia may precede clinical seizures and can respond to antiepileptic drugs.

NEUROLOGY 1998;50:1475-1477

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Apraxia is the inability to initiate skilled, complex, voluntary movements despite intact motor function, normal comprehension, and adequate cooperation. Oral motor apraxia (ORMA) has been reported with vascular and structural lesions of the anterior perisylvian regions.^{1,2} Patients with Landau-Kleffner syndrome and benign Rolandic epilepsy may also have ORMAs.³⁻⁵ Ocular motor apraxia (OCMA) may be caused by cerebellar⁶ and brainstem involvement in various disorders, including Joubert syndrome and Gaucher's disease, and acquired lesions of the frontal, supplementary, and the parietal eye fields (Balint syndrome).^{7,8}

A response of congenital apraxia to antiepileptic therapy has not been reported. After a parent re-

ported marked and rapid improvement of her child's chronic ORMAs after starting carbamazepine for seizures, we evaluated six other patients prospectively to assess the effect of this antiepileptic drug on ORMAs and OCMA.

Case reports. *Patient 1.* An 8-year-old boy presented with a history of a seizure characterized by a startle, deviation of the eyes and head to the right side, protrusion of tongue, gasping, choking, and jerking movements lasting for several minutes. He had a past history significant for prematurity, neonatal seizures, and hydrocephalus secondary to intraventricular hemorrhage at birth. The patient had chronic oral motor coordination problems with increased salivation, excessive drooling, and swallowing dif-

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