Evidence for reflex activation of experiential complex partial seizures

Article abstract—Reflex activation of seizures by thoughts or mental images is suggested by patients but has not been objectively demonstrated. The authors present a report of a man with experiential complex partial seizures reliably activated by thinking about his family home. During monitoring, such seizures were repeatedly induced in this way. Seizures were refractory to antiepileptic drugs, but ceased after left temporal resection. Pathologic examination showed cortical dysplasia.

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Patients often say that they can bring about seizures by certain thoughts, and even more frequently imply that they can interrupt or prevent attacks by breaking a train of thought. Such experiential reflex activation, though suspected, has only rarely been studied and, to our knowledge, never documented. We present a case report of a man who could reliably induce temporal lobe seizures by thinking about his family home, and whose reflex attacks were recorded during video monitoring.

Case report. This 36-year-old, right-handed man with no risk factors had intractable complex partial seizures that began at age 14. He had a rising epigastric sensation, an ill-defined feeling in his head, or a feeling of warmth followed by altered consciousness, lip smacking, and gestural automatisms. The seizures lasted about 1 to1.5 minutes. Postictally, he was drowsy and confused. There were no precipitating factors in the first several years, but he noted later that seizures were reliably precipitated by the thought of his family home, where he lived from birth to age 18, and more specifically by thinking of his father looking at him. He reported that about 90% of all seizures were triggered by this thought, and that the thought would always trigger a seizure. Seizures would occur if he thought spontaneously about it, or if he was asked to describe or recall it. He stated that he could also trigger seizures voluntarily by thinking about his family home. These were heralded by a feeling of fear. He noted that just after the triggering thought occurred, he would experience seeing his father, the rooms in the home, and other members of his family. He avoided walking by or visiting his parents' home because this would always trigger an attack. He described his childhood as happy, and recalling other childhood events or viewing photographs of his father, family, friends, or home did not precipitate seizures. Seeing or speaking to his father did not trigger attacks. He did not have seizures induced by mental stress without this thought. He found the seizures unpleasant and tried to avoid them by diverting his attention from the thought. He claimed to be able to abort some seizures in this way.

Several seizures occurred during interviews during which he was questioned about the seizure trigger. Neurologic examination was unremarkable. He was treated with several antiepileptic drugs in various combinations without success.

Routine EEG showed only sporadic left temporal slow activity. Neuropsychological evaluation showed mild verbal memory deficits, suggesting left temporal dysfunction. MRI with volumetric study of the temporal lobes showed no significant lateralized hippocampal atrophy. However, gray–white matter differentiation was indistinct in the left temporal lobe. Interictal SPECT showed mild left temporal lobe hypoperfusion. Ictal SPECT showed increased blood flow in the left temporal region.

Four typical seizures were recorded during video–EEG monitoring with scalp and sphenoidal electrodes (figure). All were triggered during interviews in which he was being asked about the seizure trigger or when he was talking about it. He reported his aura, and the seizures then occurred with staring, lip smacking, altered consciousness, and automatisms. The EEG showed ictal epileptiform activity initially over the left sphenoidal electrode and left temporal convexity. Just after the aura of one of these seizures, elicited while he was explaining what could trigger an attack, he said that he would have a seizure if he continued to think about it and then immediately repeated "la casa, la casa." He then said that the attack was coming, and then stared as the clinically evident seizure began.

He had a left temporal resection extending 5 cm from the temporal pole, including the amygdala and hippocampus. Pathologic examination showed cortical laminar disorganization, irregular clustering of neurons, balloon cells, occasional binucleate cells, and neurons showing abnormal polarity with misdirected apical dendrites. The amygdala and hippocampus were removed by aspiration and could not be studied pathologically. Adjacent white matter showed nonspecific astroglial changes.

He has had no seizures since the operation in May 1998, and takes 300 mg oxcarbazepine three times a day. He has been able to visit the family home and think about it spontaneously and on command without ill effects.

Discussion. The unusual feature of this patient's attacks is that almost all of his seizures were of an experiential nature, and for many years prior to surgery were generated by an action of mind, a quite specific recollection of emotionally meaningful people

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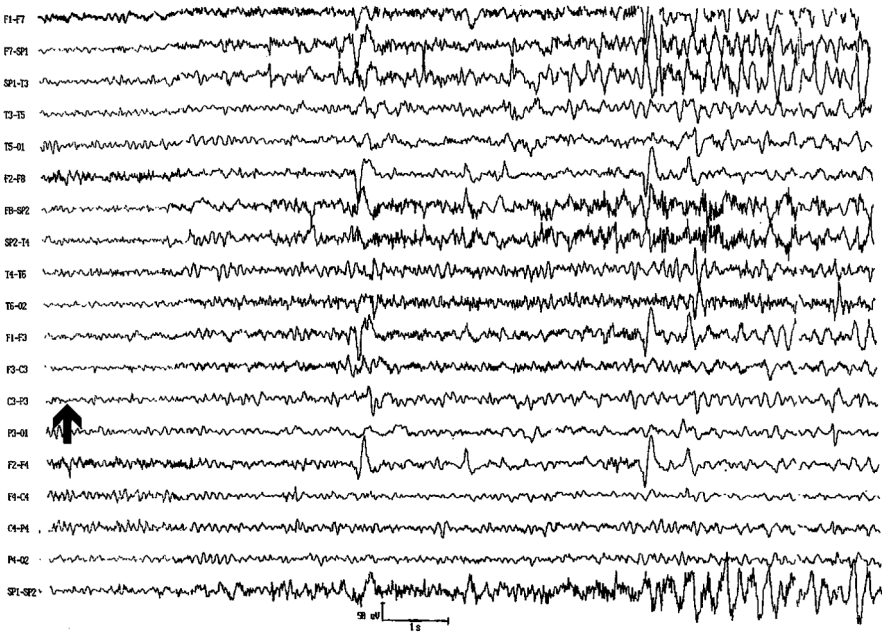

Figure. EEG of a reflex experiential seizure induced during video monitoring. Scalp and sphenoidal electrode recording. Seizure onset with ictal rhythmic activity seen on the EEG after the patient is asked (arrow) to think about his family home. Continuous tracing. Vertical marker = 50 uv; horizontal marker = 1 second.

and images, the thought of his family home. There were also features of forced thinking in the triggering of his attacks. He reported that he could abort some seizures by willfully diverting his thoughts from the trigger. Mental stress alone was not an effective trigger. After surgery he could think about the family home, view photographs of it, and visit with no ill effects. He was convinced that the triggering thought was the cause of the seizure and not a part of it. His ability to trigger seizures voluntarily, the sensitivity to the trigger seen during monitoring, and the results of surgery suggest that he was correct.

This patient had reflex seizures arising in the left temporal lobe with a cortical dysplastic lesion and has remained seizure-free since left temporal resection. The pattern of complex partial seizures and the history of seeing family members or the rooms of the home just before the visible seizure, as well as his exclamation during monitoring, confirm an experiential aura, classically associated with temporal cortical ictal discharge.

Patients often report that they can start or stop seizures by an act of mind, but documentation is scanty.[1] When specifically questioned, 22% of patients in a specialized epilepsy clinic claimed to be able to induce a seizure voluntarily by thoughts or moods, and 53% said that they could stop a seizure. The role of photosensitivity was not explored. Nonepileptic seizures were said to have been carefully excluded, but no EEG studies were reported.[1] It has been reported that 16% of children with refractory seizures in a specialized clinic could produce a seizure on demand, but EEG confirmation was not provided.[2] Aura interruption, which this patient used at times to prevent a full-blown seizure, is well described and discovered by patients on their own.[3] Seizures induced by an act of mind have been described as psychogenic[1], a term that has become attached to nonepileptic seizures. Although entirely correct, this term is best avoided at this time to prevent ambiguity and confusion. Reflex experiential seizures might be a better term to describe these attacks.

Dysplastic gray matter is inherently epileptogenic[4,5] and may have abnormal or reorganized connections with surrounding brain.[6-8] The hyperexcitable dysplastic region may have been easily triggered by afferents from normal limbic structures involved in bringing the critical memory to consciousness, and thus this dysplastic region could recruit temporolimbic tissue to discharge abnormally and produce a clinical complex partial seizure. However, an initial role for an abnormal hippocampus triggering a hyperexcitable dysplastic lesion is also plausible and cannot be excluded without depth electrode study and pathologic evidence. When dysplastic lesions are found, "dual pathology" commonly involves the hippocampus in the presence of neuronal migration disorders or gliosis.[9] The aura of fear and experiential phenomena is sometimes localized to mesial temporal structures, especially the amygdala; but temporal isocortex has also been implicated in experiential phenomena. Gloor, studying experiential auras, pointed out that different stimulation methods may account for this, but also suggested the existence of distributed neuronal networks with reciprocal connections between temporal limbic structures and isocortex. These psychical symptoms "could presumably be elicited from different locations within the temporal lobe, including temporal isocortex and various limbic structures."[10] A hyperexcitable and abnormally connected dysplastic region within such a network could well be involved in reflex seizures.

This documentation of such reflex seizures should lend credence to patients' statements that they can

bring on seizures by thinking about certain, often emotional, scenes or events; or inhibit seizures by changing their train of thought or interrupting forced thinking. Such activating or interrupting mechanisms may be more common than previously suspected.

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EEG discharges on awakening: A marker of idiopathic generalized epilepsy

Article abstract—In a series of 24-hour ambulatory EEG recordings from 1,000 consecutive adult outpatients (44.5% with generalized and 55.5% with partial epilepsy, one recording per patient), the authors found only 46 (4.6%) activations of epileptiform discharges on awakening. All recordings came from patients with idiopathic generalized epilepsy, predominantly with juvenile myoclonic epilepsy and generalized tonic-clonic seizures on awakening. Multiple spike discharges that develop with an unusually delayed onset after arousal (more than 10 minutes) might help to discriminate juvenile myoclonic epilepsy.

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The paroxysmal EEG discharges typical of generalized epilepsy often show circadian fluctuations. Clinical and EEG manifestations of juvenile myoclonic epilepsy (JME)—a type of idiopathic generalized epilepsy (IGE)—bear a strict relationship to the sleep–wake cycle, particularly during the transition phases.[1] Most patients with JME show myoclonic jerks on awakening, occasionally terminating in a convulsive seizure. Owing to its widely varying clinical presentations, JME sometimes remains undiagnosed even by experienced neurologists,[2-3] but its generally benign and drug-dependent course emphasizes the importance of a correct diagnosis. Therefore, a pressing diagnostic need is to identify a pattern of brain activity as a specific EEG marker of JME.

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Methods. We retrospectively reviewed 1,000 consecutive ambulatory EEG (A-EEG) recorded from 1,000 adult outpatients with epilepsy (aged 19 to 72 years, mean 30 years), one cassette per patient. A-EEG were recorded with an eight-channel cassette recorder (Oxford Medilog 9000, Oxford Instruments, Abingdon, Oxfordshire, UK) before, during, or after antiepileptic treatment. Recording lasted 22 to 25 hours and included one nocturnal sleep–wake cycle. Patients slept and woke naturally at home. Patients or their relatives marked the occurrence of clinical seizures on the recording by a push-button option.

One author (M.M.) classified patients as having generalized or partial epilepsy according to the International League Against Epilepsy criteria[4] (table 1). Patients with generalized epilepsy were further classified as having idiopathic (IGE), secondary, or cryptogenic generalized epilepsy. Patients with IGE were finally classified as having JME, epilepsy with generalized tonic-clonic seizures on awakening (A-GTCS), epilepsy with generalized tonic-clonic seizures at random (R-GTCS), or childhood absence epilepsy (CAE) (table 2). Patients with partial epilepsy were classified as having idiopathic, secondary, or cryptogenic partial epilepsy (see table 1). Patients with epilepsy of uncertain classification or with diagnoses other than epilepsy were excluded from the study.

We defined epileptic discharges on awakening (EDA) as the appearance of EEG discharges on awakening, or a 30%