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CASE REPORT

Cardiac asystole during a temporal lobe seizure

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Partial seizure

Summary The association between temporal lobe seizures and cardiac arrhythmias has been anecdotally reported in the literature. Ictal bradycardia and cardiac asystole are rare, and maybe underestimated. The physiological mechanism is poorly understood. We report a patient with left temporal lobe seizures who developed ictal bradycardia and cardiac asystole during a complex partial seizure and required a subsequent placement of a pacemaker.

Introduction

Cardiac arrhythmias have been reported during epileptic seizures, particularly those of temporal lobe origin.[1] Ictal tachycardia is the most common. Bradycardia and cardiac asystole are very rare during an epileptic seizure, but is potentially life threatening. The underlying pathophysiology is not well known. Diagnosis of this potentially lethal condition is important since cardiac arrhythmias have been implicated to play an important role in sudden unexpected death in epileptic patients (SUDEP).[2,3] We report a patient with left temporal lobe seizures who developed bradycardia and asystole accompanying a complex partial seizure during a prolonged video-EEG monitoring for pre-surgical evaluation.

Case report

The patient was a 43-year-old Caucasian male with history of refractory complex partial seizures since the age of 16 years. He reported 3—4 seizures per week. He usually had an aura described as a ''strange feeling'', followed by decrease in awareness, becoming unresponsive and mute. There was some vague description of right hand tonic posture. He was usually aphasic postictally. His seizures were poorly controlled on combination of carbamazepine 1200 mg/day and leviteracetam 2000 mg/day. He had previously failed several antiepileptic medications including phenobarbital, phenytoin and topiramate.

He had no history of febrile seizures during childhood nor a central nervous system infection. He had a mild head injury with no loss of consciousness when he fell on the concrete while playing basketball at the age of 10 years. There was no history of hypertension, cardiac disease or diabetes. He denied any history of palpitations, chest pain, syncope or exercise intolerance. There was no history of smoking. Family history was negative for epilepsy. His father, who was 72, had transient sinus tachycardia due to ''emotional stress'', which was treated for a short period of time with beta-blockers. Pre-surgical investigations included head magnetic resonance imaging (MRI) with volumetric hippocampi measurements that showed evidence of left mesial temporal sclerosis (Fig. 1).

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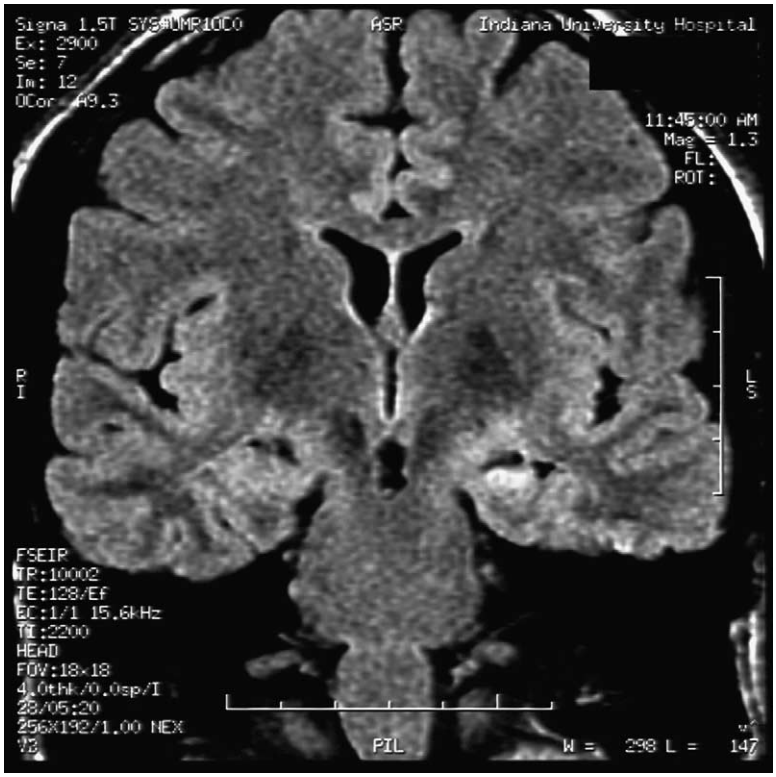

Figure 1 Coronal fast fluid-attenuated inversion-recovery (FLAIR) MRI showing atrophy and increase in signal of the left hippocampus.

An interictal single photon emission computed tomography (SPECT) using technitium-99m (^{99m}Tc) and hexamethylpropyleneamine oxime (HMPAO) revealed decrease blood flow in the left mesial temporal region. Electroencephalogram (EEG) demonstrated interictal epileptiform discharges over the left anterior temporal region.

The patient was admitted to the video-EEG monitoring unit to record his epileptic seizures and to determine their location of onset. Leviteracetam was discontinued and carbamazepine was decreased to 600 mg/day. On admission, blood pressure was 134/80 mmHg, heart rate was 76 per minute and respirations 18 per minute. The patient was afebrile with normal cardiovascular and neurological examination. Continuous video-EEG recording using scalp and sphenoidal electrodes was performed. During the first day of monitoring, the patient had several complex partial seizures and one secondarily generalized seizure lasting 2—3 min. Ictal activity had a localized onset from the left anterior temporal region. EKG during these seizures remained normal. There were very frequent interictal sharp wave discharges recorded over the left anterior temporal lobe, and at left sphenoidal electrode (SP1).

On the second day of hospitalization, the patient was lying in bed, watching TV, when he suddenly had a seizure during which he became unresponsive and mute. No automatic behavior was noticed. He was aphasic and confused for about 15 min postictally. Following this seizure, he denied any chest pain, shortness of breath or palpitations. EEG showed an ictal pattern characterized by rhythmic 7 Hz theta activity confined to the left anterior temporal region lasting 40 s typical of a brief complex partial seizure of the left temporal origin. Thirty seconds into the seizure the EKG revealed sinus bradycardia for 10 s followed by a cardiac asystole lasting 10 s (Fig. 2a—c). During the period of asystole the EEG showed mild diffuse slowing. The EKG, then, spontaneously returned to normal sinus rhythm of 88 beats/min. Blood pressure immediately after the seizure was 148/86 mmHg. Patient was restarted on levetiracetam, and his carbamazepine dose was increased to 1200 mg/day. Complete hematological examination showed normal electrolytes including magnesium, normal comprehensive blood cell count and liver enzymes. A random carbamazepine level on the next day was 14. A baseline 12-lead EKG was done and revealed no abnormalities. A transthoracic echocardiogram showed normal global left

ventricular systolic function without structural abnormalities.

The cardiology service was consulted and recommended a cardiac pacemaker placement. A pacemaker was implanted without complications, and patient was discharged to home on the current antiepileptic drugs. The patient continued to have complex partial seizures and ultimately underwent a left temporal lobectomy.

Discussion

Cardiac arrhythmias have been reported after complex partial seizures of temporal lobe origin.[1] In a retrospective study involving 81 patients, Zijlmans et al.,[4] reported sinus tachycardia in more than 90% of epileptic seizures, usually toward the end of the seizure. In a series by Blumhardt et al., isolated bradycardia was noted in 1 of 74 seizures

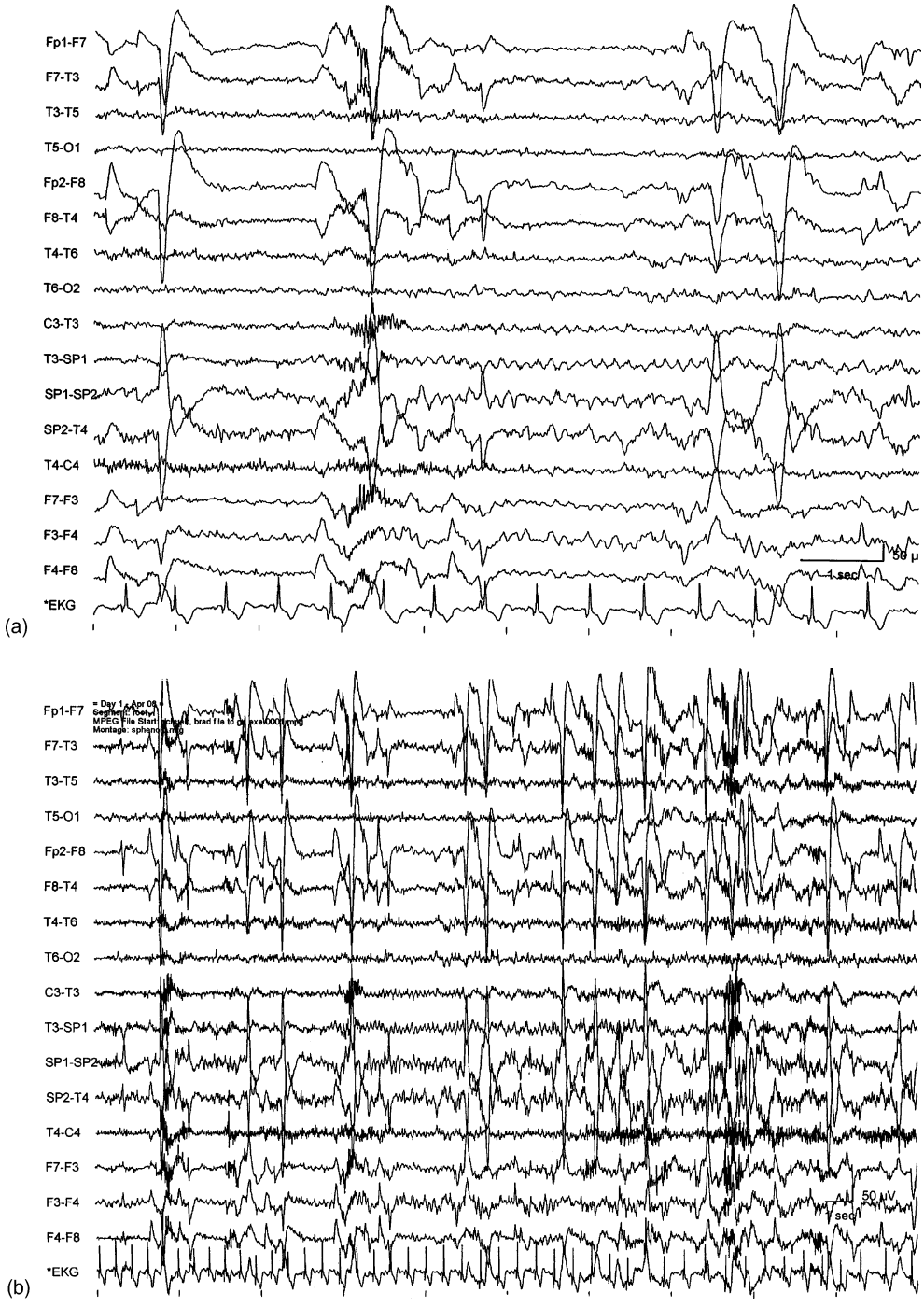

Figure 2 (a—c) Ictal EEG and EKG showing electrographic seizure onset and subsequent bradycardia and asystole.

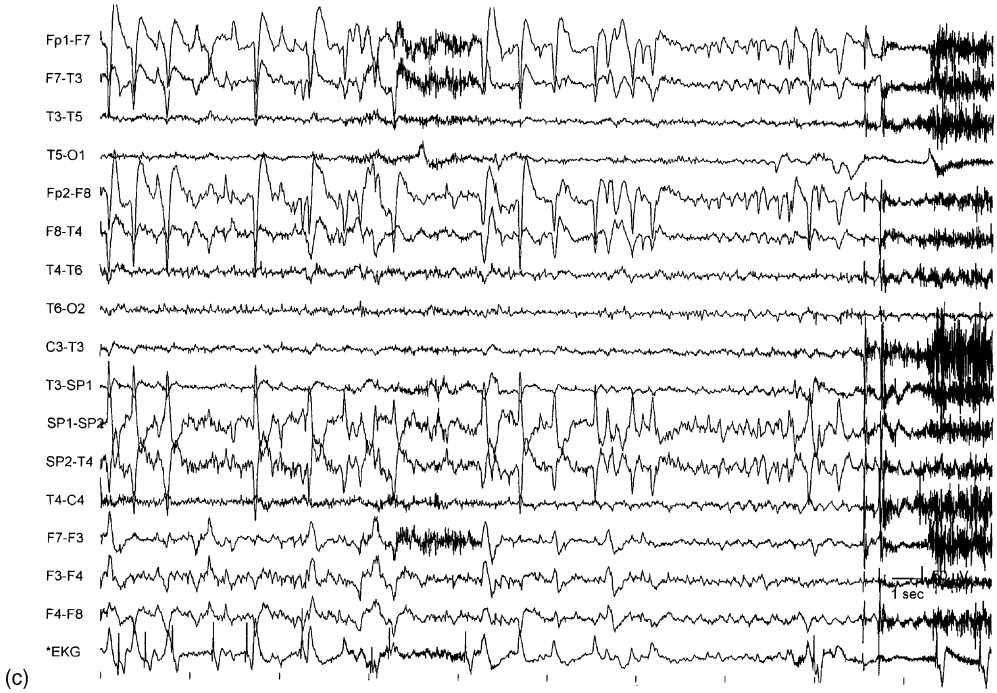

Figure 2 (*Continued*).

recorded.[5] Sinoatrial block and cardiac asystole have been rarely reported.

Cardiac arrhythmias have been linked to sudden unexpected death in patients with epilepsy.[2,3] However, in most of the reported patients with cardiac arrhythmias during a seizure, the duration of cardiac asystole is usually short averaging 5—10 s, with no major complications.[6] There was one reported patient with a 30 s asystole during a seizure.[4] Commonly, patients do not have any previous history of cardiac disease or others cardiovascular risk factors. The cardiac arrhythmia is detected incidentally during prolonged video-EEG monitoring as in our patient.[4].

The physiologic mechanism involved in the association between temporal lobe seizures and cardiac arrhythmias is poorly understood. Experimental studies demonstrated an important role of the insula in cerebrogenic cardiovascular disturbances, including cardiac arrhythmias in association with seizures.[7] Cardiac chronotropic organization has been shown to be located within the posterior insular cortex of rats. Oppenheimer et al.[8] demonstrated that stimulation of the insular cortex produces heart block leading to escape rhythms, ventricular ectopia and ultimately death in asystole. The insular and the limbic cortex project to several subcortical sites (stria terminalis, amygdala, lateral hypothalamic area, and paraventricular nucleus), brainstem (periaqueductal gray matter, locus ceruleus, nucleus tractus solitarius, dorsal vagal nucleus, nucleus ambiguous and ventrolateral medulla) and the intermediolateral column of the spinal cord. This neural circuitry complex is important in controlling the cardiac rhythm among other autonomic functions.[7] Whether the laterality of epileptogenic focus has any bearing on the type of cardiac arrhythmic, tachycardia or bradycardia, during an epileptic seizure is unknown. Oppenheimer[9] demonstrated that left human insular cortex stimulation increases the parasympathetic cardiovascular tone leading to bradycardia whereas right insular cortex simulation increases the sympathetic tone resulting in tachycardia.

There is no consensus regarding management of patients who develop cardiac arrhythmias during seizures. The majority of patients do not have underlying cardiac structural lesion or other cardiovascular risk factors. The cardiac arrhythmia is detected incidentally during a seizure recorded on prolonged Video-EEG monitoring. It is not known if pacemaker implantation is medically indicated in all patients with seizure induced bradycardia, however, in those with asystole it seems prudent to do so. The decision is even more controversial in cases like our patient who is a very good surgical candidate with a very high likelihood of becoming seizure free after resective surgery. It is not known whether these patients are at continued risk for bradyarrhythmias following a successful temporal lobe resection.

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References

1. Tinuper P, Bisulli F, Cerullo A, Carcangiu R, Marini C, Pierangeli G, et al. Ictal bradycardia in partial epileptic seizures: autonomic investigation in the three cases and literature review. *Brain* 2001;**124**:2361—71.
2. Opeskin K, Thomas A, Berkovic SF. Does cardiac conduction pathology contribute to sudden unexpected death in epilepsy? *Epilepsy Res* 2000;**40(1)**:17—24.
3. Leestma JE, Walczak T, Hughes JR, Kalelkar MB, Teas SS. A prospective study on sudden unexpected death in epilepsy. *Ann Neurol* 1989;**26**:195—203.
4. Zijlmans M, Flanagan D, Gotman J. Heart rate changes and ECG abnormalities during epileptic seizures: prevalence and definition of an objective clinical sign. *Epilepsia* 2002;**43(8)**:847—54.
5. Blumhardt LD, Smith PE, Owen L. Electrocardiographic accompaniments of temporal lobe epileptic seizures. *Lancet* 1986;**1(8489)**:1051—6.
6. Reeves AL, Nollet KE, Klass DW, Sharbrough FW, So EL. The ictal bradycardia syndrome. *Epilepsia* 1996;**37**: 983—7.
7. Cheung RTF, Hachinski V. The insular and cerebrogenic sudden death. *Arch Neurol* 2000;**57**:1685—8.
8. Oppenheimer SM, Wilson JX, Guiraudon C, Cechetto DF. Insular cortex stimulation produces lethal cardiac arrhythmias: a mechanism of sudden death? *Brain Res* 1991;**550**: 115—21.
9. Oppenheimer S. *Stroke* 1993;**24(Suppl I)**:I-3—I-5.