Symptomatic postictal cardiac asystole in a young patient with partial seizures

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CASE REPORT

This report describes a patient with complex partial seizures arising from the right temporal lobe who developed symptomatic sinus arrest following the end of his seizure activity. A ventricular pacemaker was implanted and was documented to function appropriately, preventing development of bradycardia associated symptoms during subsequent seizures. Possibly relevant cerebral structures are briefly discussed.

Introduction

It has been appreciated for some time that significant cardiac arrhythmias may occur during epileptic seizure activity. Sinus tachycardia is the most common arrhythmia observed but severe bradycardias due to sinus node arrest have also been described. We describe a patient who presented with a protracted episode of symptomatic sinus node arrest that began in the post-ictal phase of a complex partial epileptic seizure and which was subsequently prevented by implantation of a permanent ventricular pacemaker.

Case report

A 28-year-old male with a 12 year history of frequent partial seizures but otherwise in good health was admitted to our neurology laboratory for evaluation of surgical resection of the epileptic focus. His MRI showed a small cystic lesion in right basal temporal neocortex, most likely a dysplasias of the neocortex. His witnessed seizures were all psychomotor in nature with occasional verbal automatisms, lip smacking, and manual automatisms, sometimes evolving into tonic-clonic seizures. Because the disorder was refractory to several first-line antiepileptic medications, surgical treatment was considered. The patient therefore underwent withdrawal of his antiepileptic agent while under continuous simultaneous video, EEG and ECG monitoring as well as direct observation by a nurse. The sinus rate increased from 60/min at EEG-onset of the seizure (Fig. 1A) to 102/min by 20 s to a maximum of 126/min at 46 s after EEG-onset (Fig. 1B), while the right temporal activity became more and more distinct. Eight seconds after the EEG-termination of the seizure (61 s after ictal EEG-onset) the patient's heart rate abruptly slowed and sinus arrest, without an escape rhythm, was observed for the subsequent 31 s (Fig. 1C). Several junctional beats then appeared followed by sinus tachycardia at 102/min (Fig. 1D). Tonic-clonic activity developed shortly after the onset of the sinus arrest but stopped quickly after return to sinus rhythm. A ventricular pacemaker was implanted the next day and programmed to VVI mode 50 bpm, with a hysteresis rate of 40 bpm. Four days after the pacemaker implant and under identical monitoring conditions, the patient had a recurrence of his


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seizure and again developed sudden sinus bradycardia 9 s after EEG-termination of seizure activity (60 s after ictal EEG-onset). The pacemaker began to pace appropriately at that point until recovery of an adequate sinus rate 9 s later (Fig. 2). No tonic-clonic activity was observed during this seizure. Despite the right temporal lesion and localization of most seizures to the right temporal lobe, the presurgical work-up (neuropsychological exam, nuclear imagery, MRI, invasive EEG-monitoring with foramen ovale electrodes) revealed
significant bitemporal dysfunction underlying the partial epilepsy in this patient. A 24-hour ambulatory ECG recording obtained after the patient’s discharge, on antiepileptic treatment and without occurrence of epileptic seizures, showed normal sinus rhythm with a mean rate of 76/min, a maximum rate of 147/min and a minimum rate of 52/min. The patient has had recurrences of his partial seizures but has remained free of postictal tonic-clonic activity during the 6 months following his pacemaker implantation.
Discussion

Our patient suffered from drug refractory partial seizures that resulted in severe symptomatic sinus arrest — an infrequent finding. Additionally, this case is unique in that the sinus arrest appears after the end of all EEG-seizure activity whereas onset of sinus arrest in the previously described cases, cited in the references, occurred simultaneously with or soon after onset of the seizure. Consistent with previous clinical observations,
Figure 2  Postictal period (60 s after EEG onset; 9 s after EEG-offset) of a partial seizure of the same patient, after (VVI) pacemaker implantation, with appropriate ventricular pacing following the onset of severe sinus bradycardia. The EEG shows diffuse slowing with motion artifacts. Vp: ventricular pacing.
typical EEG changes such as generalized attenuation of the background activity were seen approximately 9 s following asystole and cessation of cerebral circulation (Fig. 1D)\textsuperscript{[12]}.

The beneficial effect of the permanent pacemaker in this patient was clearly documented by the recordings obtained during a subsequent seizure where the pause due to post-seizure sinus arrest was prevented, as well as the continued absence of tonic-clonic movements during the subsequent 6 months despite recurrence of partial seizures during that time.

The involved pathways by which epileptic seizures lead to a cardiac arrhythmia are not clear. Reports of well-documented cases noted that the large majority of such epilepsies are of temporal lobe origin\textsuperscript{[1,8]}. Most reports have not found a significant lateralization of the focus although a preponderance of right temporal epilepsy was noted by one investigator (and also is present in our patient)\textsuperscript{[1,8]}. Conversely, electrical stimulation of the left insular cortex in patients undergoing epilepsy surgery has been found to be associated more frequently with bradycardia compared with right-sided stimulation\textsuperscript{[14]}. However, despite the close proximity of the insular cortex to the temporal lobe structures most patients with temporal lobe epilepsy do not experience significant cardiac arrhythmias thus implying the presence of a complicated interaction between the temporal lobes and/or insular cortices in both hemispheres.

In conclusion, permanent ventricular or even dual chamber pacing may be required in this select group of patients with refractory temporal lobe epilepsy with associated symptomatic sinus bradycardia or asystole in whom the epilepsy cannot be completely controlled. Furthermore, only simultaneous EEG and ECG recording will reveal a possible cerebral origin of arrhythmias in these patients who are, typically, young.

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References