LESSON OF THE MONTH

Brain seizes, heart ceases: a case of ictal asystole

Erle CH Lim, Shih-Hui Lim, Einar Wilder-Smith

Epileptic seizures commonly result in changes of cardiac rate.¹⁻⁴ Although tachycardias are much more frequent, ictal bradycardia and asystole may be life threatening and contribute to the syndrome of sudden unexplained death in epileptic patients.⁵ Differentiation between primary cardiac and cerebrogenic bradyarrhythmia is possible with the use of simultaneous EEG and ECG recording. The correct identification of ictal bradycardia and asystole is important as treatment needs to consider both optimal anticonvulsant therapy and the implantation of a cardiac demand pacemaker.

We report ictal asystole in a patient with a left temporal lobe seizure identified by simultaneous ECG and scalp EEG recording.

Case history

A 74 year old Malay man was first admitted in January 1992 to the Neurosurgery Department for evacuation of bilateral chronic subdural haematomas sustained after several accidental falls. Recovery was uneventful until July 1992, when he was admitted for a possible seizure. Brain CT disclosed a right cerebral peduncular infarct. A clinical diagnosis of post-traumatic epilepsy was made, and he was started on 300 mg phenytoin at night. Over the next 5 years, he had one seizure, whereas between 1997 and 1998, he had four seizures. The four were attributed to poor compliance with medications.

In June 1999, he was admitted to the Neurology Department after another generalised tonic-clonic seizure. Neurological and cardiac examination were unremarkable. Clinically, he was in sinus rhythm with a rate of 80 beats/min. Blood pressure was 120/80 mm Hg. Investigations showed a low serum phenytoin concentration of 1.6 mg/l on the day of admission. With supervised medication in the wards, the level had increased to 7.0 mg/l 3 days later. A 12 lead ECG was normal. Brain CT showed an old infarct in the right internal capsule, bifrontal encephalomalacia, and mild cerebral atrophy. A routine 30 minute surface EEG showed normal background rhythm with intermittent 4-5 Hz activity over the left temporal region. An ictal event lasting 44 seconds was recorded. This was characterised by 3-6 Hz rhythmic discharges over the left mid-anterior temporal region (T1, F7, and T3). About 7 seconds after onset of the left temporal epileptiform discharge, the patient developed bradycardia with asystole lasting 6 seconds, punctuated by a ventricular beat, followed by another

period of 12 seconds of asystole. Subsequently, the bradyarrhythmia returned, continued for a further 20 seconds, and abruptly ended with termination of the seizure (figure). During the EEG seizure, the patient was unresponsive to verbal questioning, ground his teeth, and had brief dystonic posturing of the arms towards the last half of the seizure. Subsequent cardiac evaluation with 24 hour Holter monitoring did not show any evidence of cardiac arrhythmia.

Discussion

Since Russel⁶ first documented cardiac asystole during a seizure in 1906, a wide range of cerebrogenic cardiac arrhythmias have been reported, ranging from tachyarrhythmias such as atrial fibrillation, supraventricular tachycardia, prolonged QT, and torsade de pointes to bradyarrhythmias.⁷⁻⁹ Most cardiac arrhythmias occur with temporal lobe epilepsy,¹ suggesting that there may be a specific temporal lobe effect on cardiac rate and rhythm. However, this may be partly due to technical difficulties in obtaining a clear ECG reading during generalised tonic-clonic seizures. Sinus tachycardia is by far the most common accompaniment of partial seizures and is seen in 54% to 96% of cases.^{1 3 4} By comparison, ictal bradyarrhythmias are rare,¹⁰ seen in 3.3%¹¹ to 17% of partial seizures.²

Compared with tachyarrhythmias, little is known about the pathophysiology of ictal bradycardia. This case demonstrates some of the typical features that have been found in cases of ictal bradycardia.¹⁰ Males are much more commonly affected, and over 80% of the cases reported in the literature had temporal lobe epileptic discharges precipitating the bradyarrhythmia.¹⁰ In our male patient, the onset of the partial seizure was clearly localised to the left temporal region.

Identification of discrete cerebral loci in the generation of arrhythmias has been pursued avidly.^{4 8 12-13} Of the cerebral foci implicated in arrhythmogenesis, the insula is probably the most important, due to its profuse interconnections with the limbic system, the hypothalamus, and other areas of autonomic control.⁸

Although bradyarrhythmias have been documented on surface EEG during left or right temporal lobe seizures,¹⁰ intraoperative stimulation of the human insular cortex has shown asymmetric autonomic response patterns. Tachycardia and pressor responses are more frequent with right insular stimulation whereas

Department of
Neurology, Singapore
General Hospital,
Outram Road,
Singapore 169608
E CH Lim
S-H Lim
E Wilder-Smith

Correspondence to: Dr Einar
Wilder-Smith

gnnews@sgh.gov.sg

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Ictal asystole: simultaneous EEG and ECG shows epileptiform discharges over the left anterior temporal region (filled out arrow) with subsequent asystole commencing about 10 seconds later (unfilled arrow). Onset of tonic arm posturing is marked (curved arrow). Scalp electrode montage is according to the 10/20 system.

bradycardia and depressor responses are more frequent with left insular stimulation.¹⁴

Other brain loci have been implicated in the genesis of bradyarrhythmia. Stimulation of the amygdala, dorsal vagal nucleus, nucleus ambiguus, and nucleus of the solitary tract have all produced bradycardia.¹³ Kahane *et al*¹⁵ described how the frontocentral region and temporal neocortex contribute to the genesis of ictal bradyarrhythmia in a patient with a hypothalamic hamartoma.

It can be difficult to distinguish between cerebrogenic and cardiogenic causes of cardiac arrhythmias. Our report highlights that it is only with simultaneous EEG and ECG recording that differentiation between the two causes is possible. The primary cerebrogenic nature of the bradyarrhythmia is convincingly demonstrated in this case with EEG seizure activity occurring before the onset of bradyarrhythmia. As it is important to also exclude intrinsic cardiac disease, cardiac investigation including 24 hour Holter ECG monitoring should be performed.

This case brings up two important management issues. The first is choice of medication, as anticonvulsant drugs can affect cardiac conductive tissue. As our patient had no intrinsic cardiac disease, the choice of antiepileptic drug is not of paramount importance, as would be the case in a patient with intrinsic cardiac con-

ductive tissue dysfunction. Antiepileptic drugs that should be avoided in the presence of bradyarrhythmia include carbamazepine, phenytoin, barbiturates, and benzodiazepines. Valproate and the newer antiepileptic drugs like lamotrigine and gabapentin probably affect cardiac conduction tissue much less.

The second management issue involves the need for a cardiac pacemaker. The decision to insert a pacemaker is made based on the duration of asystole. Symptomatic cerebral hypoperfusion in this patient is suggested by the development of higher amplitude, less synchronous, waves, followed by generalised suppression on EEG (figure). Generally, asystole of more than 4 seconds duration is potentially life threatening, and implantation of a cardiac pacemaker should be considered. Our patient's non-compliance with anticonvulsant drugs strongly argues for such a measure, despite the fact that control of his seizures is the single most important factor in the prevention of cardiac arrhythmias.

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