

Cardiac Asystole and Bradycardia as a Manifestation of Left Temporal Lobe Complex Partial Seizure

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Sudden cardiac death is almost always associated with coronary artery disease (1), but even in coronary artery disease, the autonomic nervous system plays a significant role in the genesis of arrhythmias (2). It is well established that the central nervous system can trigger sudden death by intense activation of the autonomic nervous system (3), the release of opioids, or the release of neuroactive peptides (4). Cortical stimulation studies suggest sympathetic predominance over the right hemisphere and a parasympathetic effect on the left hemisphere (5). Cortical activity, as seen in complex partial seizures with concomitant changes in heart rate, might help to validate the above findings.

We attempted to clarify the possible localization of cortical activity in patients who have asystole or bradycardia associated with complex partial seizures. We report on three such patients and review

the literature on seizure-associated asystole or bradycardia monitored by simultaneous video electroencephalography–electrocardiography.

Patient 1

A 44-year-old right-handed woman with a family history of epilepsy had had staring spells and episodes of loss of consciousness since early childhood. After two recent convulsions, therapy with valproic acid and carbamazepine was started. Despite anticonvulsant treatment, she continued to have frequent 1-minute staring episodes. Results of physical and neurologic examinations, computed tomography of the head, and electrocardiography were normal. During monitoring, epileptiform discharge over the left hemisphere was associated with a staring spell and random leg movements, followed by 26 seconds of asystole. Interictal electroencephalography showed an isolated epileptiform disturbance that was maximal over the left temporal and frontal area. An anticonvulsant drug regimen of lamotrigine and carbamazepine has kept the patient free of seizures and cardiac arrhythmias for 3 years.

Patient 2

A 52-year-old right-handed man with a history of left frontal lobe trauma had had complex partial

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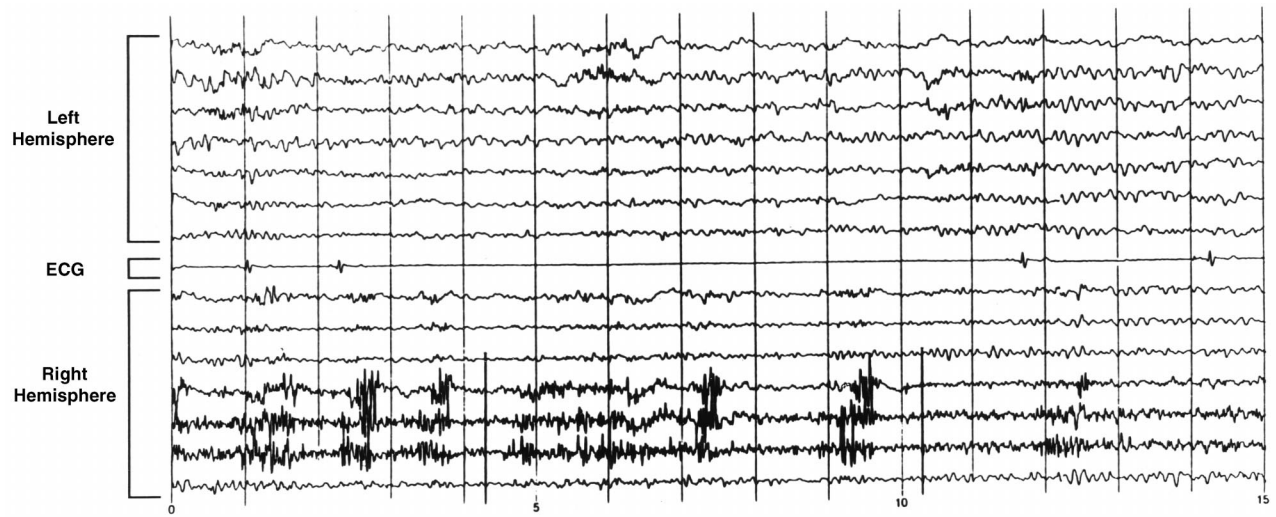


Figure. Electroencephalogram and electrocardiogram (ECG) from a patient with cardiac asystole (patient 2). In the first 6 seconds of the electroencephalogram, epileptiform discharges are seen in the left temporal region. These changes are temporally related to the 9-second period of asystole noted in the electrocardiogram. Muscle artifact is seen over the right hemisphere.

seizures and tonic-clonic seizures for 3 years. The seizures were well controlled with carbamazepine therapy. He began to experience drop attacks and fainting spells. Results of physical and neurologic examinations, electrocardiography, echocardiography, Holter monitoring, and tilt-table testing were normal. Magnetic resonance imaging of the brain showed extensive encephalomalacia of the left frontal lobe. During monitoring, he was aroused from sleep by left temporal epileptiform activity followed by 9 seconds of asystole (Figure). The patient receives a therapeutic dose of carbamazepine, but he continues to have auras of dizziness without loss of consciousness or syncope at 4 years of follow-up.

Patient 3

A 28-year-old left-handed man with a 16-year history of complex partial seizures had an increase in seizure frequency over 9 months despite antiepileptic treatment with phenytoin, carbamazepine, and lamotrigine. Results of physical and neurologic examinations and magnetic resonance imaging of the brain were normal. Routine electroencephalography showed left temporal epileptiform activity. During monitoring, the patient experienced an electrical disturbance over the left hemisphere followed by a drop attack associated with 24 seconds of asystole. A cardiac pacemaker was inserted to prevent asystole. The patient is free of seizures 1 month after pacemaker placement and is receiving carbamazepine and phenytoin.

Discussion

We present three patients who had cardiac asystole associated with complex partial seizures that

originated in the left temporal lobe. A review of the literature revealed 10 reports on 14 patients with complex partial seizures who underwent simultaneous video electroencephalography-electrocardiography (6–15). Nine patients had asystole (range, 5 to 40 seconds) and 5 had bradycardia. The seizure activity originated in the left temporal lobe in 9 patients, in the right temporal lobe in 2 patients, and in the right occipital lobe in 1 patient. In 2 other patients, asystole was associated with bitemporal epileptiform disturbances (10, 15). It seems, then, that cardiac asystole or bradycardia is associated with left temporal lobe epileptic activity.

In patients who undergo temporal lobectomy, stimulation of the right insular area causes tachycardia, whereas left stimulation causes bradycardia (5). Unilateral electroconvulsive therapy has similar effects (16). Intracarotid injection with amobarbital causes homolateral cerebral inactivation and produces different heart rate responses (17). Right-sided intracarotid injection results in bradycardia, whereas left-sided injection causes increases in the heart rate. The heart rate also increases with right-middle cerebral artery stroke, possibly because of disconnection of the insula from its cortical influence (18). These studies suggest a sympathetic predominance over the right hemisphere and a parasympathetic effect on the left hemisphere. Analysis of RR-interval variability in patients with temporal lobe epilepsy has shown similar results (19).

Bradycardia and asystole result from increased parasympathetic flow through the vagus nerve, which originates in the nucleus ambiguus and dorsal nucleus of the vagus in the medulla. The slowing of the atrial rate is greatest with stimulation of the right nucleus ambiguus (20). The connections of

the cerebral cortex and subcortical areas to the brainstem vagal nuclei are not well defined. However, because left cortical stimulation and right vagal stimulation affect the heart rate in a similar way, the fibers from the left cortex must cross to stimulate the right brainstem vagal nuclei.

Sinus node dysfunction and increased vagal tone are the most common causes of bradycardia and asystole. Our 3 patients and the 14 patients in the literature show that cortical stimulation of the temporal lobe can result in asystole or bradycardia, demonstrating the importance of cortical activity in the genesis of cardiac arrhythmias. Identifying the site of the abnormal activity (cerebral cortex, vagal tone, or sinus node) is paramount in the management of these patients. Many of the reported patients with asystole caused by temporal lobe seizures were effectively treated with anticonvulsant drugs. Arrhythmias that result from sinus node dysfunction or increased vagal tone may be prevented with a pacemaker. Seizure-induced asystole that is refractory to anticonvulsant treatment may also be controlled with a pacemaker.

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