CASE REPORT

Asystole in Course of Epileptic Episode

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A subgroup of patients with epilepsy die suddenly and ictal bradycardia (IB) has been implicated. We describe a 63-year-old man with diagnosis of refractory epilepsy. The patient had long video and EEG/ECG recordings (48 hours) of wakefulness and abnormal sleep due to the presence of seven ictal events, which are associated with complex partial seizures that start on the temporal region. The patient presented a severe dysautonomia ictal, at 10–15 seconds of the start of the event is observed bradycardia and after 25 seconds occurs asystole for 15 seconds. Dual-chamber pacemaker was implanted with improvement.

Ann Noninvasive Electrocardiol 2013;18(6):590–592

pacemaker; ictal bradycardia; asystole; epilepsy

ASYSTOLE IN COURSE OF EPILEPTIC EPISODE

People epilepsy have a higher risk of mortality compared with healthy individuals, a subgroup of patients with epilepsy die suddenly. This phenomenon describes sudden unexpected death in epilepsy (SUDEP) with a reported occurrence of up to 18%, is the most frequent epilepsy-related cause of death.¹ The exact mechanism remains unknown, but ictal bradycardia (IB) has been implicated.

We describe a 63-year-old man with previous diagnosis of refractory epilepsy since age 25 years. Events were characterized by sucking, oral, and hand automatisms about 5 minutes without secondary generalization. Two years ago the ictal events were associated with cyanosis, severe pallor, sweating, and loss of consciousness without abnormal movements and severe facial trauma. The patient had long video and EEG/ECG (Nicolet LTM, Carefusion, San Diego, CA) recordings (48 hours) of wakefulness and abnormal sleep due to the presence of seven ictal events, which are associated with complex partial seizures that start on the temporal region of the left hemisphere in most of the time it radiates and continue on

the right hemisphere. The patient presented a severe dysautonomia ictal, at 10–15 seconds of the start of the event is observed bradycardia and after 25 seconds occurs asystole for 15 seconds with cyanosis and hypotonia (Fig. 1). The seizures radiating to the right temporal region most often presented asystole. Dual-chamber pacemaker (St. Jude Medical, Inc., St. Paul, MN) was implanted with improvement of the events. During follow-up of 2 years, he has not presented events of loss of consciousness and continues his treatment anticonvulsant. The baseline electrocardiogram was normal (Fig. 2).

Cardiac arrhythmias are frequently observed during epileptic seizures. The most common is sinus tachycardia, which occurs in >90% of seizures and is usually without consequence. Heart bradycardia has been reported in <6% and rarely a severe bradycardia-asystole has been documented. Bradycardia during epileptic seizure has been labeled IB syndrome. However, there are studies that suggest that the prevalence of IB may be higher, ranged from 2.1% to 25.5% calculated per number of monitored seizures.^{2,3}

IB usually starts 10–30 s after the EEG seizure onset (4), and apparently after seizures discharges become bilateral. However, the IB onset can be

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No conflicts of interest to be disclosed.

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DOI:10.1111/anec.12069

Figure 1. Complex partial seizures that start on the temporal region and occurs asystole for 15 seconds.

simultaneous with or can precede the EEG seizure onset. Quite often, IB progress to asystole that usually last 10–30 s. The longest reported ictal asystole lasted for 60 s.⁴

It has been hypothesized that seizures may lead to the stimulation of regions of the brain, such as the insula, cingulate cortex, amygdala, or hypothalamus that regulate cardiac outflow through connection to brainstem and spinal cord nuclei. Some have suggested IB may have lateralizing value as well. However analysis of the dynamic EEG/ECG recordings of patients with IB suggested that bitemporal activation was present at bradycardia onset in the majority of patients, providing no clear evidence for the existence of a consistently lateralized cardiac parasympathetic representation in the central autonomic network. Seizure-induced activation of the central autonomic nervous system can cause a direct effect on post-ganglionic discharges on the heart. This results in a synchronization of cardiac autonomic discharges with epileptogenic activity called the lock-step phenomenon and induces a lethal bradyarrhythmia or asystole.⁵

In patients with epilepsy SUDEP most probably results from cardiac or respiratory failure. The pulmonary mechanisms include central apnea

or acute neurogenic pulmonary edema. The most significant and widely discussed cardiac mechanism of SUDEP is cardiac arrhythmia precipitated by seizure discharges acting via the autonomic nervous system. IB may be a point of differentiation from sudden unexplained death. The atonia directly resulting from asystole may produce injury depending upon circumstance, but whether ictal asystole leads to SUDEP and has lethal implications is less assured without evidence of direct causation.⁶ Central apnea was more common than obstructive apnea. Daly has emphasized the risk of cardiac arrest in any clinical situation in which the patient's respiration is depressed either reflexly or centrally, a time of heightened vagal sensitivity.⁷ However at present, best evidence supports complete seizure control as the only effective prophylactic measure for SUDEP.

The necessity of pacemaker implantation in all IB patients is questionable. Kovakam et al.⁸ found in 10 patients with median follow-up of 102.5 months that IB can be managed successfully with antiepileptic drugs alone; only 2 patients with refractory IB may require dual therapy of antiepileptic drugs and a pacemaker. Nevertheless, it is not possible yet to predict if

Figure 2. The baseline electrocardiogram was normal.

and which patients with IB will remain without serious consequences or will develop into severe arrhythmias.

Our patient has the usual features of patients with IB. It is interesting to note that there was significant clinical improvement with the use of pacemaker suggesting that this patient may have sinus node dysfunction and many of his events could be syncopes. In the same context the prevention of bradycardia and cerebral hypoperfusion secondary prevent triggering epileptic. In this patient was not evaluated sinus node function as events of loss consciousness and ictal pauses suggesting benefit from pacemaker implant regardless of results of invasive test or Holter. However during implantation and 1 year later, tests were performed to evaluate sinus node function without finding disorders.

Since this condition is uncommon it is important to know the pacemaker results and our case shows how these devices in selected patients can improve quality of life for our patients. Only with more information we can guide that patients will benefit from pacemaker implantation (e.g., according to

the length of the pause) and case reports and series are by far the best alternative.

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