Journal of Cardiology Cases (2011) 3, e149—e153

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Case Report

# An unique case suffering from repetitive syncope episodes due to ictal asystole

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Received 26 December 2010; received in revised form 19 February 2011; accepted 23 February 2011

**KEYWORDS**
Cardiac asystole;
Tradycardia;
SUDEP;
Temporal lobe epilepsy;
Pacemaker

**Summary** We present the case of a patient with syncope with repetition over 12 years, with a clinical profile not clearly related with a cardiogenic origin, who was studied by several medical specialties without any accurate diagnosis. After subcutaneous loop recorder implantation, we were able to demonstrate how seizures acted as a trigger in the genesis of an exaggerated cardio inhibitory reflex.

A new entity has been described, known as ''ictal asystole'', in patients with focal epilepsy mostly from the temporal lobes and has been implicated as a cardiac cause of sudden unexplained death in epilepsy.

We think this case could add new information about some patients who are at high risk of death but they are misdiagnosed.


## Introduction

Ictal asystole is a weird and unknown entity which could be behind sudden unexpected death in epilepsy. The etiopathological mechanisms involved are still discussed and not clear. We present an interesting case related to this pathology which could be interesting and helpful to other cardiologist in recognition and treatment of patients with similar clinical presentation.

## Case report

We introduce the case of a 69-year-old man with hypertension and dyslipidemia as risk factors. He was admitted to the hospital in 1997 because of chest pain accompanied by left bundle branch block and he therefore underwent thrombolysis with recombinant tissue plasminogen activator. At that time, he reported having suffered up to three episodes of transient loss of consciousness. An ischemia test

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doi:10.1016/j.jccase.2011.02.007

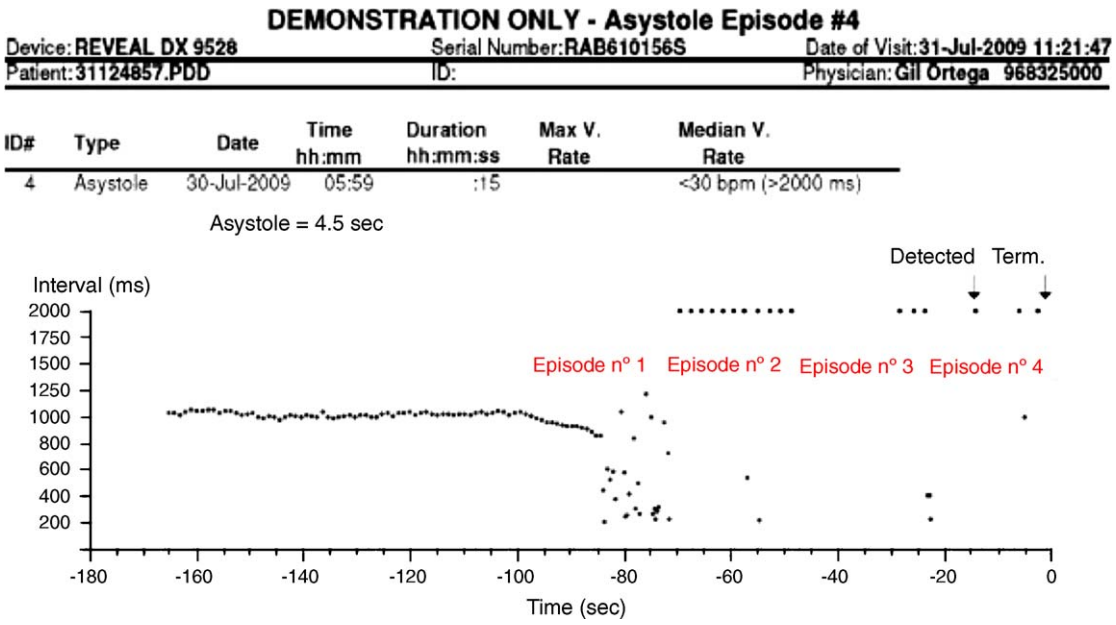


**Figure 1** Diagram showing the rate profile of the patient and how it is split into four different episodes.

was performed with an inconclusive result and the coronary angiogram did not show any significant lesions. The transthoracic echocardiogram showed left ventricular dimensions in the upper limit of normality. The 24-h electrocardiogram-Holter recorded sinus rhythm as baseline without significant pauses or rhythm disturbances. Finally, those episodes of unconsciousness were attributed to a vasovagal mechanism.

In 2000, he was assessed by the service of neurology for a further syncopal episode. On this occasion an electroencephalogram and nuclear magnetic resonance imaging (MRI) were performed, and both of them were normal. The patient continued to have several episodes of loss of consciousness, some of them accompanied by tongue biting. Suspecting seizures, a computerized tomography was requested, which was also normal. During 2001 and 2002 the patient continued with the same clinic. Several other tests were requested. A vertebral- and angio-MRI were normal and positron emission tomography (PET) showed evidence of a focal metabolism asymmetry at the level of the medial and inferior left temporal lobe, without a clear epileptogenic focus. Treatment with neosidantoine was started with transient improvement.

In June 2009, the patient was readmitted for new syncopal episodes preceded by dizziness. A new transthoracic echocardiogram showed no differences compared with the one performed several years before.

During a multidisciplinary session, this case was presented to the arrhythmia unit. An electrophysiology study was requested, in which an interval HV of 73 ms was measured [first-degree infrahisian atrioventricular (AV) block]. The Wenckebach point was demonstrated to be suprahisian and it was registered at 390 ms. The recovery time of the sinus node was also normal. There was no demonstration of double nodal physiology. The effective refractory period of the AV node was 500/310 ms. No other arrhythmias were induced by a complete stimulation protocol.

Because the clinical cause of the syncopal episodes was not clearly related to a cardiogenic origin (although some of them could be), we decided not to implant a pacemaker (although the HV interval was prolonged, there was a good behavior at high frequencies) until a more accurate diagnosis could be established. So, we decided to implant a subcutaneous loop recorder (Reveal™, Medtronic, Minneapolis, MN, USA) in July 2009.

In August 2009, the patient consulted the emergency room because of a new syncopal event while he was sleeping. His wife discovered him cold, cyanotic, and with guttural sounds. She tried to activate the device, but it was unsuccessful.

When the cardiologist on duty interrogated the device, there was one unique automatic episode which had been split into four different episodes cataloged as long asystole (Fig. 1). There were no manual activations.

At the beginning of the automatic episode (called Episode 1), a clear sinus rhythm could be distinguished at a heart rate of 65 beats per minute, followed by some noise of a probable muscular origin where it was still possible to check the persistence of at least three or four more beats in sinus rhythm before continuing with asystole (Fig. 2). Finally, in the fourth episode recorded, we checked the initial return to the previous sinus rhythm of the patient (Fig. 3).

The diagnosis of asystole was made, so the patient was referred to the intensive care unit. A final report from the Medtronic Company was requested in order to confirm the diagnosis and to rule out channel saturation due to a seizure event. Characteristic signals of poor contact or channel supersaturation (i.e. 6 Hz) were not present after applying a mathematical model of signal analysis, and it could be possible to confirm the sinus activity before and after the asystole.

We hypothesize that maybe a focal abnormality in the temporal lobe highlighted by the PET could act as a trigger which could cause an exaggerated cardioinhibitory reflex followed by prolonged asystole. A Cylos DR-T (Biotronik, Berlin, Germany) pacemaker programmed in DDD-CLS stimulation was implanted due to its potential benefits on the prevention and treatment of the vasovagal syncope [1].