Clinical commentary

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Cardiogenic syncope diagnosed as epileptic seizures: the importance of ECG during video-EEG recording

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ABSTRACT – We describe four patients with a previous diagnosis of epilepsy. After reviewing the ECG during the video-EEG recording, cardiogenic syncope, resulting from a cardiac arrhythmia, was identified as the cause of the seizures. Epileptic disorders and cardiogenic syncope may both manifest with convulsions, loss of consciousness, and loss of postural tone, leading to a high level of misdiagnosis. The one-lead ECG during video-EEG monitoring is a key component, which may allow correct diagnosis and treatment.

Key words: cardiogenic syncope, epilepsy, electrocardiogram, ECG, video-electroencephalography, EEG

Epilepsy is a neurological syndrome with abnormal synchronous electrical brain activity that causes a variety of symptoms depending on the lobe compromised; the term "epilepsy" refers to the predisposition to recurrent unprovoked seizures (Chang and Lowenstein, 2003). Epilepsy syndromes can be classified according to the type of seizure, the presence or absence of neurological findings, and EEG findings. Despite the limited sensitivity of EEG (interictal EEG sensitivity ranging from 25 to 56% and specificity ranging from 78 to 98%), EEG remains an important tool in the diagnosis and assessment of patients with epilepsy (Smith, 2005). The one-lead electrocardiogram (ECG) (as part of the video-EEG assessment) allows the detection of syncope or cardiac arrhythmias.

Here, we describe four patients previously diagnosed with epilepsy, in whom ECG alterations revealed cardiac arrhythmias as the possible aetiology of their symptoms.

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Case reports

Case 1

A 91-old, right-handed man was diagnosed with pharmaco-resistant epilepsy for the last six months; the patient had been treated with three different antiepileptic drugs, with no response. The semiology of events consisted of loss of consciousness with eyes closed, preceded by an aura of lightheadedness, which worsened when doing physical activity; all of these symptoms occurred prior to any antiepileptic drug use. He was treated with valproic acid (1,250 mg/day), carbamazepine (800 mg/day), and phenytoin (300 mg/day). Events occurred at least two or three times a day, especially when doing any type of physical activity, such as eating or changing clothing. Cranial MRI revealed no structural abnormality. Typical risk factors for epilepsy (family history, febrile seizures, head trauma, or previous brain infection) were absent.

General and neurological examination was normal. Video-EEG monitoring was started and revealed no interictal EEG abnormality. During the recording period, the patient was not medicated. During the continuous video-EEG recording, the ECG derivation indicated bradycardia (heart rate: 30 bpm), occurring simultaneously with the lightheadedness previously described by the patient; there were also alterations in P wave suggested by a complete heart block (*figure 1*). After this finding, the patient was sent to another institution where a Holter examination was performed. It was concluded that the bradycardia was secondary to a second-degree atrioventricular (AV) block. Subsequent placement of a pacemaker by a cardiologist at another hospital stopped all symptoms/events. Antiepileptic drugs were immediately discontinued. Over a follow-up period of two years, during which time the patient was followed by a cardiologist, the patient was free of seizures, and no more seizure-like episodes occurred.

Case 2

A 52-year-old, right-handed man was diagnosed with "absence epilepsy" (even though the diagnosis was more likely to be dyscognitive seizures, this was the term used by the primary physician). The semiology of the events was characterized by a sudden onset of behavioural arrest, followed by confusion. Each episode lasted 30 seconds and was followed by various episodes of emesis. The patient was treated with carbamazepine (200 mg, three times a day) for four months. The dose was increased (200 mg at time 06:00 and 12:00, and 400 mg at night) after persistence of seizures (five in one day prior to consulting at our hospital). MRI of the brain revealed no structural abnormality.

This patient was admitted to our hospital with various episodes of clonic movements of the jaw, mydriasis, left eye deviation, and facial redness, with loss of consciousness. A diagnosis of focal seizures was entertained. Neurological examination was normal.

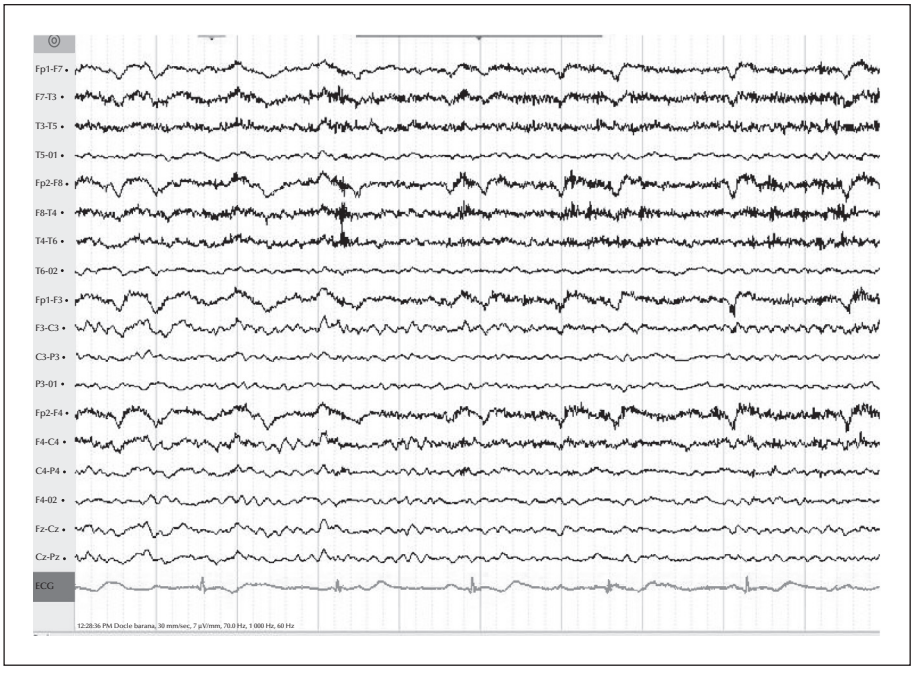

Figure 1. Case 1. Evidence of severe bradycardia based on the ECG recording during video-EEG monitoring; possible complete AV block. Heart rate: 30 bpm.

ECG performed during the admission revealed atrial fibrillation with an adequate ventricular response. Aspirin, at 100 mg daily, was started due to a CHADS2-VASc (congestive heart failure, hypertension, age, diabetes, stroke, vascular disease, age 65-74 years, and gender) of zero. Subsequent 24-hour video-EEG monitoring did not reveal any abnormality. However, on the continuous one-lead ECG, there was a pause of seven seconds, which occurred simultaneously with the patient's symptoms (*figure 2a*). Due to the known effect of carbamazepine causing bradycardia, the level of carbamazepine was measured (4.59 μg/ml; normal range: 4 to 12 μg/ml) and the results eliminated the drug as the cause of the symptoms. A diagnosis made by a cardiologist was of bradycardia due to a dysfunction of the sinoatrial node (*figure 2b*). The patient underwent a pacemaker implantation for control of the arrhythmia. After this, no more events occurred and all

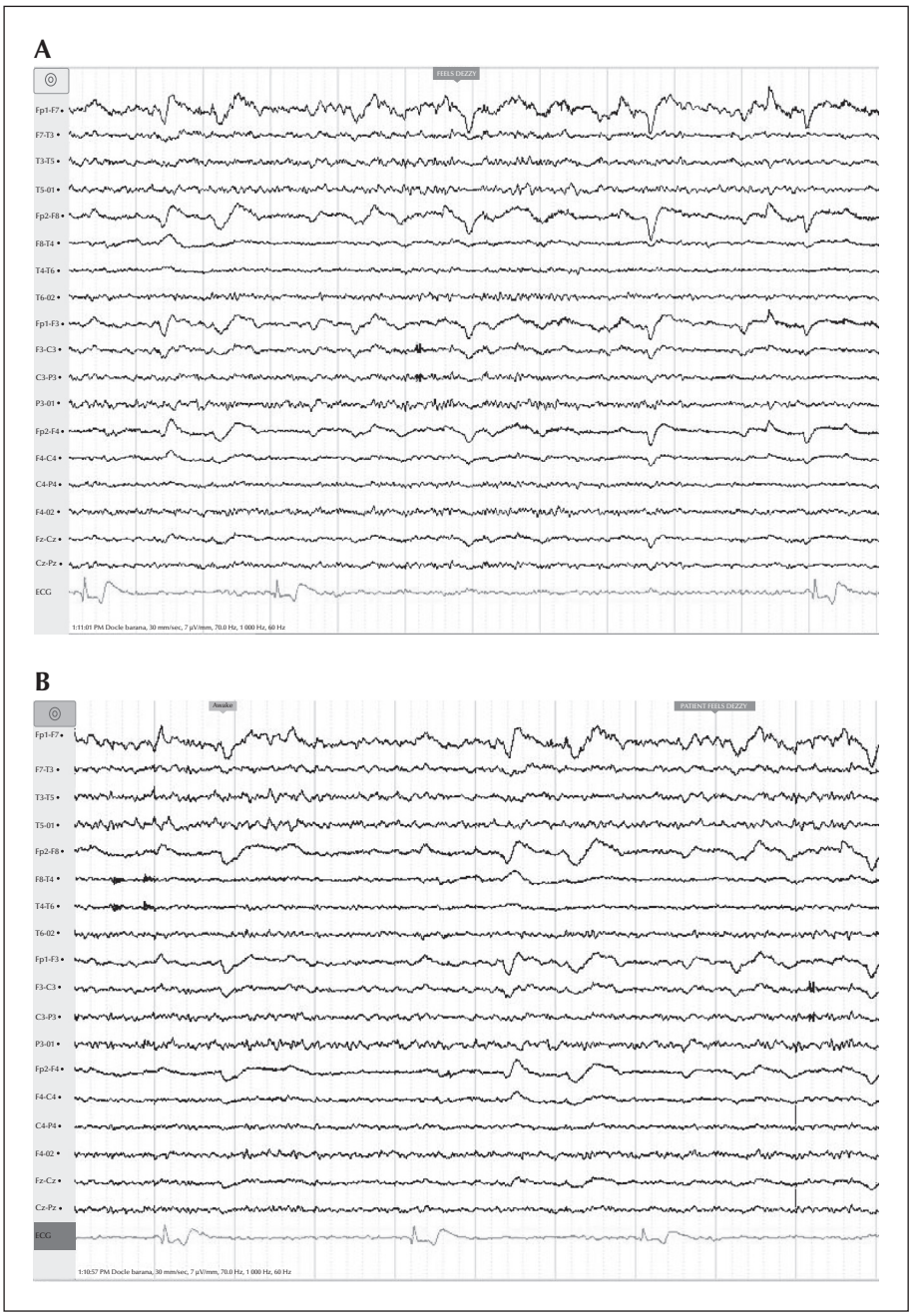

Figure 2. Case 2. (A) Evidence of symptomatic bradycardia based on the ECG recording during video-EEG monitoring. Note severe bradycardia corresponding to a seven-second pause during the ECG recording. Heart rate: 12 bpm. (B) Evidence of symptomatic bradycardia based on the ECG recording during video-EEG monitoring. Heart rate of 18 bpm with a two-second pause on the ECG.

antiepileptic drugs were discontinued. One month later after this episode, the patient returned to the emergency room due to dizziness and dyspnoea. Emergency doctors established that this was a problem associated with the pacemaker, which was reprogramed. A call was made to the patient a year later, and he stated that he was asymptomatic and that no more episodes, such as those described earlier, had occurred.

Case 3

A 46-year-old, right-handed woman had had a diagnosis of epilepsy since the age of 25. The seizure episodes consisted of prolonged expiration and involved brief, sudden lapses of unconsciousness with complete resolution. She had been off antiepileptic drugs since the age of 26 due to the absence of seizures. At the age of 46 years, after an episode of forced expiration and loss of consciousness for five minutes, she was admitted to our institution. Cerebral CT indicated a parietal calcification in the left hemisphere; likely due to neurocysticercosis.

On examination, she was found to have an irregular pulse with a frequency of 109 bpm. A one-lead ECG indicated an absence of P waves with spontaneous resolution; video-EEG monitoring showed no alterations, however, an absence of P waves (suggestive of atrial fibrillation) was noticed during two similar episodes of forced expiration and loss of consciousness on the ECG lead (*figure 3*). Holter monitoring performed at the hospital revealed episodes of tachycardia (from 120 to 180 bpm) with an absence of P waves that terminated spontaneously; these episodes were followed by a normal sinus rhythm with a heart rate of 102 bpm. The patient was diagnosed with paroxysmal atrial fibrillation and her symptoms were considered to be a consequence of the arrhythmia. Due to the unclear history based on the few seizures that had occurred, epilepsy diagnosis was considered to be equivocal. A call to the patient was made eight months after admission to the hospital revealing that she had been free of any of the symptoms previously described.

Case 4

A 90-year-old, right-handed woman was admitted to our institution due to events characterized by impairment of consciousness, behavioural arrest, and staring for five to ten minutes. The patient had a pacemaker due to a previous diagnosis of symptomatic bradycardia. Neurological examination was normal. An EEG performed at the time of admission did not reveal any epileptiform abnormality. Subsequent video-EEG recordings performed at the hospital confirmed the lack of EEG abnormalities, however, a rate of 150-180 bpm was noticed in the one-lead ECG, in association with a wide QRS during the episodes described above (*figure 4*). After cardiological assessment (an echocardiogram showing no structural abnormalities and a Holter examination showing QRS >0.12 s), a diagnosis of ventricular tachycardia was made. The patient's pacemaker was reprogramed which led to an improvement and the discontinuation of the epileptic treatment. One month later, the patient

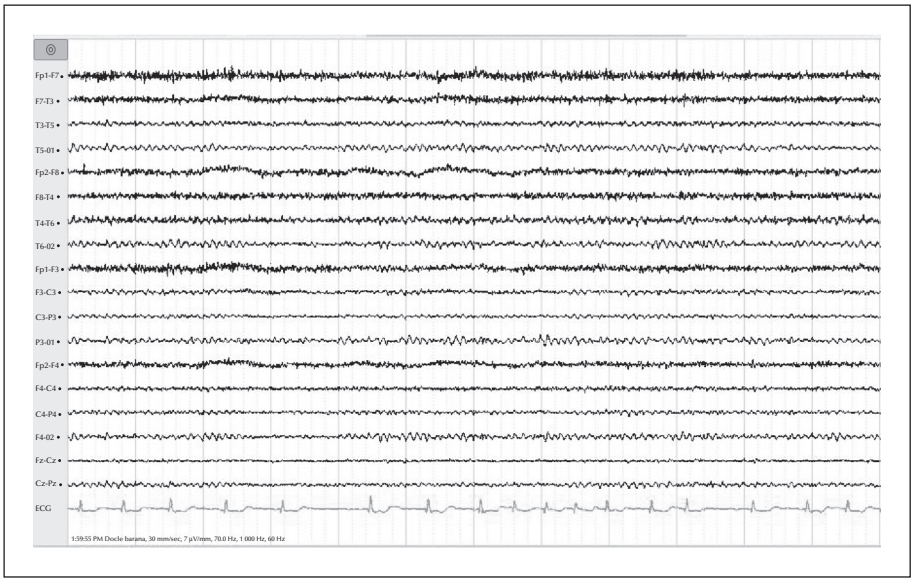

Figure 3. Case 3. Evidence of P wave alterations based on the ECG recording during video-EEG monitoring. Note irregular R-R interval and absence of P wave during the recording.

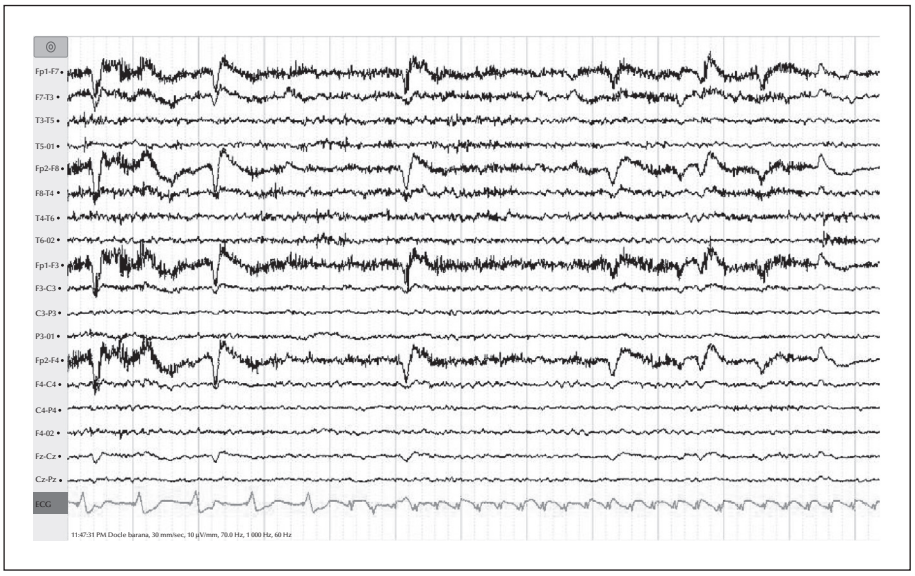

Figure 4. Case 4. Pace-maker rhythm, after six seconds, shows QRS alterations, suggestive of ventricular tachycardia based on the ECG recording during video-EEG monitoring. Note regular R-R interval and absence of P wave. Heart rate: 150-180 bpm.

died following cardiac failure. During this month, the patient did not have any symptoms, according to one of his first-degree relatives, and the cardiologist's assessment, 10 days after hospital admission, revealed an absence of tachycardia.

Discussion

Epileptic disorders and syncope both manifest with convulsions, loss of consciousness, and loss of postural tone (Grubb *et al*., 1991). Common causes of syncope include: vasovagal syndromes, cardiac arrhythmias, and carotid sinus hypersensitivity. Epilepsy and syncope are two entities with a high prevalence of incorrect diagnosis; one in four patients with epilepsy is misdiagnosed (Duplyakov *et al*., 2010). A cohort study of 74 patients with a diagnosis of epilepsy performed by Zaidi *et al*. showed that 31 patients (42%) had an alternative diagnosis; 19 patients had vasovagal syncope and two prolonged bradycardia (Zaidi *et al*., 2000). Furthermore, after 10 months of follow-up with the correct diagnosis and treatment, 19 of 31 patients were symptom-free. A number of published case reports and clinical commentaries also underscored the importance for differential diagnosis of clinical history taking and careful ECG interpretation (Stephenson *et al*., 2004; Spanaki *et al*., 2006; Fattouch et al., 2007; Gelisse *et al*., 2007; Ozkara *et al*., 2009; Doležalová et al., 2013; Xu *et al*., 2014).

In the present article, two of four patients showed significant clinical improvement after one year of follow-up. Rodrigues *et al*. also performed a cross-sectional study of 55 patients with faints, falls and convulsions, and normal EEG. They found that seven patients had a cardiac arrhythmia (*i.e*. complete AV blocks, sustained monomorphic ventricular tachycardia, ventricular fibrillation, atrial tachycardia, and AV node reentrant tachycardia) as the cause of the symptoms (Rodrigues *et al*., 2010).

In our hospital database, there are few cases similar to those described in this article, moreover, the clinical information is poorly described, limiting correct analysis. More importantly, cardiologists and neurologists from other institutions in Bogota confirm that these cases are relatively rare, but when they do occur, there can be serious consequences due to misdiagnosis and use of antiepileptic drugs. A search of our local databases revealed no clinical studies or any case report revealing the frequency of such cases in our local population.

Diagnostic errors are made because of the similarity of symptoms presented between cardiogenic syncope and epilepsy syndromes. When a cardiogenic syncope occurs (*e.g*. due to a bradyarrhythmia or tachyarrhythmia), there is a hypoperfusion to the brain that may cause myoclonic jerks, tonic spasms, eye movements, or loss of consciousness; signs that might orient to a diagnosis of epilepsy (Bergfeldt, 2003). Most patients with seizure-like episodes are diagnosed with epilepsy based on the clinical evidence. With no cardiac investigation and normal or diffuse changes in the EEG recording, patients are initiated with antiepileptic drugs that have no impact on the cardiac problem (or on the events of concern), and might even worsen the problem. As shown in Case 3, cardiac abnormalities may not always occur, limiting the use of sporadic ECG performed in the emergency room. The one-lead

ECG allows the physician to compare the abnormalities of the ECG or EEG recordings during the seizure-like episode, therefore facilitating the diagnosis. It also allows the physician to consider the need for continuous monitoring of electrical activity of the cardiovascular system by Holter examination or evaluate any structural abnormalities using an echocardiogram.

Conclusions

The importance of a clear clinical history, neurological examination, and EEG recordings are essential for accurate diagnosis. The one-lead ECG, as part of the video-EEG assessment, is a valuable tool to identify possible arrhythmias and proceed with better cardiological testing, such as a Holter examination or echocardiogram. Correct identification of cardiogenic syncope may lead to a treatment that will stop the convulsion and will avoid unnecessary use of anticonvulsant medications. Further clinical studies should be performed in order to establish the frequency of cardiogenic syncope, as well as possible strategies of diagnosis and treatment.

Supplementary Data.
Summary didactic slides are available on the www.epilepticdisorders.com website.

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TEST YOURSELF

(1) What is the physiological cause of convulsions occurring during a cardiogenic syncope?

(2) Which tests would you consider for a patient with episodes of loss of consciousness who has been treated with multiple antiepileptic drugs, with no improvement?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".