Imaging in Cardiology: EEG Abnormalities During Cardiac Syncope and Spontaneous Recovery

M.J.J.A BOKERN
Waterland Ziekenhuis, Purmerend, The Netherlands

J.C.J. RES, A.J. PRAZSKY
Zaans Medical Center, De Heel, Zaandam, The Netherlands

Introduction

There is a close and intimate relationship between the heart and the brain. In many case reports, this relationship has been documented in the form of either bradycardia or tachycardia arrhythmias, following epileptic seizures. Many of these seizures originated in the temporal lobe [1-3]. Schott et al [4] found that arrhythmias occurred in 20% of patients who suffered from possible epilepsy. In those patients, tachycardia occurred 74% of the time, and bradycardia occurred 5.5% of the time. On the other hand, there is little or no systematic information correlating cardiac arrhythmias to EEG pathology. Based on data collected on patients with ventricular tachycardia [5] or induced asystole [6], there is a delay in the appearance of EEG changes after onset of the cardiac arrhythmia. This suggests that cortical function as measured by the EEG is preserved until well after onset of hypotension. And when it does occur, it is merely coincidental that the unfortunate moment of the cardiac arrhythmia has been captured on the EEG recording. Below is a case report on this topic.

Case Report

A 56-year-old man was referred to the outpatient clinic for evaluation of syncopal events. His history was not typical for epilepsy. He had not experienced any of the usual symptoms, such as post-ictal unconsciousness, but he did have some prodromal sensations, such as pre-ictal abnormal smells. Physical neurological and cardiac examinations were unremarkable. An electroencephalogram (EEG) was performed. During the test the patient had a similar syncopal event with marked loss of consciousness, accompanied by convulsions. The EEG during sinus rhythm showed "normal" alpha-activity (Figure 1), and during slowing of the heart rate there was "flattening" of the alpha-activity (Figure 2). At the beginning of syncope, the EEG showed an increase of theta and delta activity (Figure 3), followed by cortical silence and muscle activity (Figure 4). After restoration of the sinus rhythm, cortical silence proceeded for about 6 s until alpha activity reappeared (Figure 5).

Discussion

The EEG characteristics of cerebral anoxia are typical and can be distinguished in three phases: no EEG changes for short asystolic periods (< 6 s), then appearance of theta and delta rhythms for up to 13 s, followed by cortical silence, which was seen in this patient [5,7-9]. In many cases, muscle artifacts associated with syncope were observed. After the patient regained consciousness, the alpha rhythm was reestablished [9].

There is a potential for misdiagnosis in patients with epilepsy. Some patients with convulsive blackouts or syncope are referred to a neurologist. A multidisciplinary approach to the examination of these patients may help. Since this is not a commonly used approach in many clinics, it must be proposed for patients whose evaluation does not result in a clear and reliable diagnosis. The consequences of an incorrect diagnosis of epilepsy are severe, with respect to changes in patients' daily lives (such as driving) and increased insurance costs [10]. Furthermore, these patients may be treated with potentially harmful anticonvulsant drugs [11]. In this case a bradycardia was

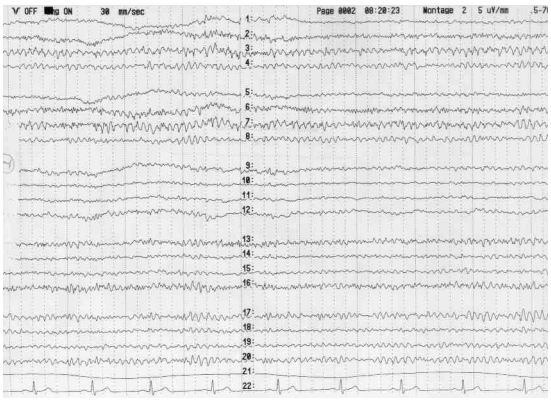

Figure 1. The EEG during sinus rhythm prior to the symptoms showes "normal" Alpha activity. Two minutes before syncope the sinus rate was 58 beats/min (lowest line in recording is the ECG). Time: 08:20:23.

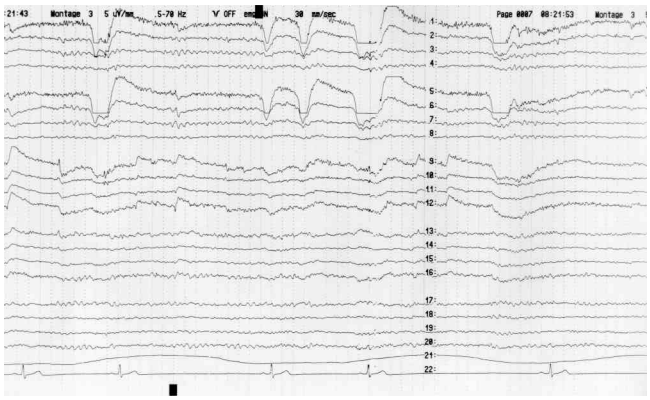

Figure 2. A definite slowing of the heart rate is documented by ECG. There is also a "flattening" of the cerebral Alpha activity. The patient is still conscious. Time: 08:21:53.

captured during the EEG recording and causes a symptoms in the form of syncope, which directs the therapy to pacemaker implantation. The patient did not experience another episode of syncope after DDD pacemaker implantation. If the EEG reveals a neurologic disorder, then a pacemaker implantation could be avoided [2].

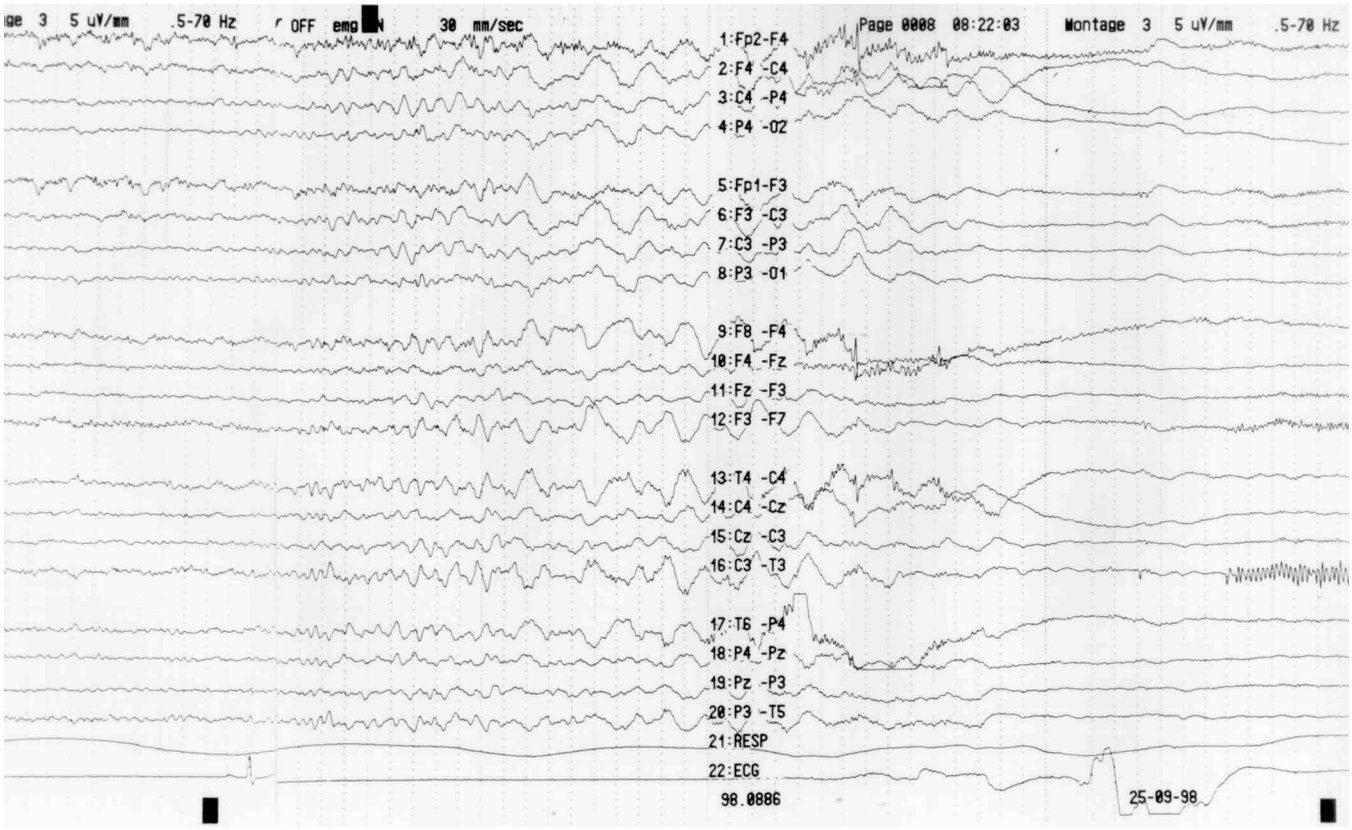

Figure 3. At the start (left side) of this part of the recording the last sinus beat was observed after 2 pauses, each lasting 4 s. Delta and Theta activity was present, which after additional 7 s changed into "cortical silence". Time: 08:22:03.

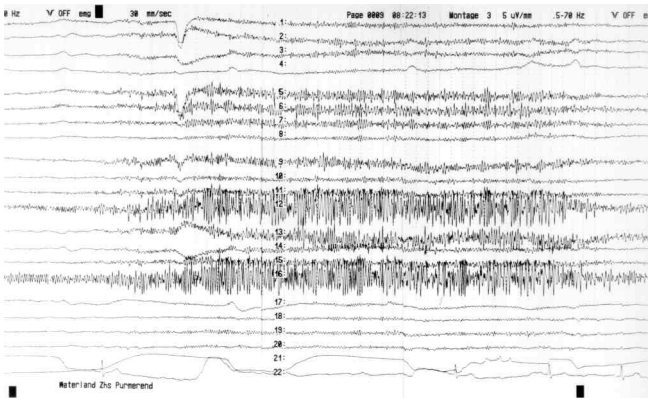

Figure 4. Together with Figures 2 and 3, this is a continuous recording showing muscle activity due to the convulsions induced by the cerebral anoxia. Time: 08:22:13.

Conclusion

Extensive evaluation by the neurologist and cardiologist may be recommended, and in dubious cases, video-telemetry monitoring with electroencephalography seems to be the ultimate test for obtaining a high sensitivity and specificity [12].

References

[1] Seeck M, Blanke O, Jallon P, et al. Symptomatic postictal cardiac asystole in a young patient with partial seizures. Europace. 2001; 3: 247-522.

[2] Van den Bergh PJPC, Fokke JEJ, Oude Ophuis AJM, et al. Temporal arrest. Neth Heart J. 2001; 7: 292-295.

[3] Liedholm LJ, Gudjonsson O. Cardiac arrest due to partial epileptic seizures. Neurology. 1992; 42: 824-829.

[4] Schott GD, McLeod AA, Jewitt DE. Cardiac arrhythmias that masquerade as epilepsy. Br Med J. 1977; 1: 1454-1457.

[5] Aminoff MJ, Scheinman MM, Griffin JC. Electrocerebral accompaniments of syncope associated with malignant ventricular arrhythmias. Ann Intern Med. 1988; 108: 791-796.

[6] Schraeder PL, Pontzer R, Engel TR. A case of being scared to death. Arch Intern Med. 1983; 143: 1793-1794.

[7] Gastaut H, Fischer-Williams M. Electro-encephalographic study of syncope: its differentiation from epilepsy. Lancet. 1958; 2: 1018-1025.

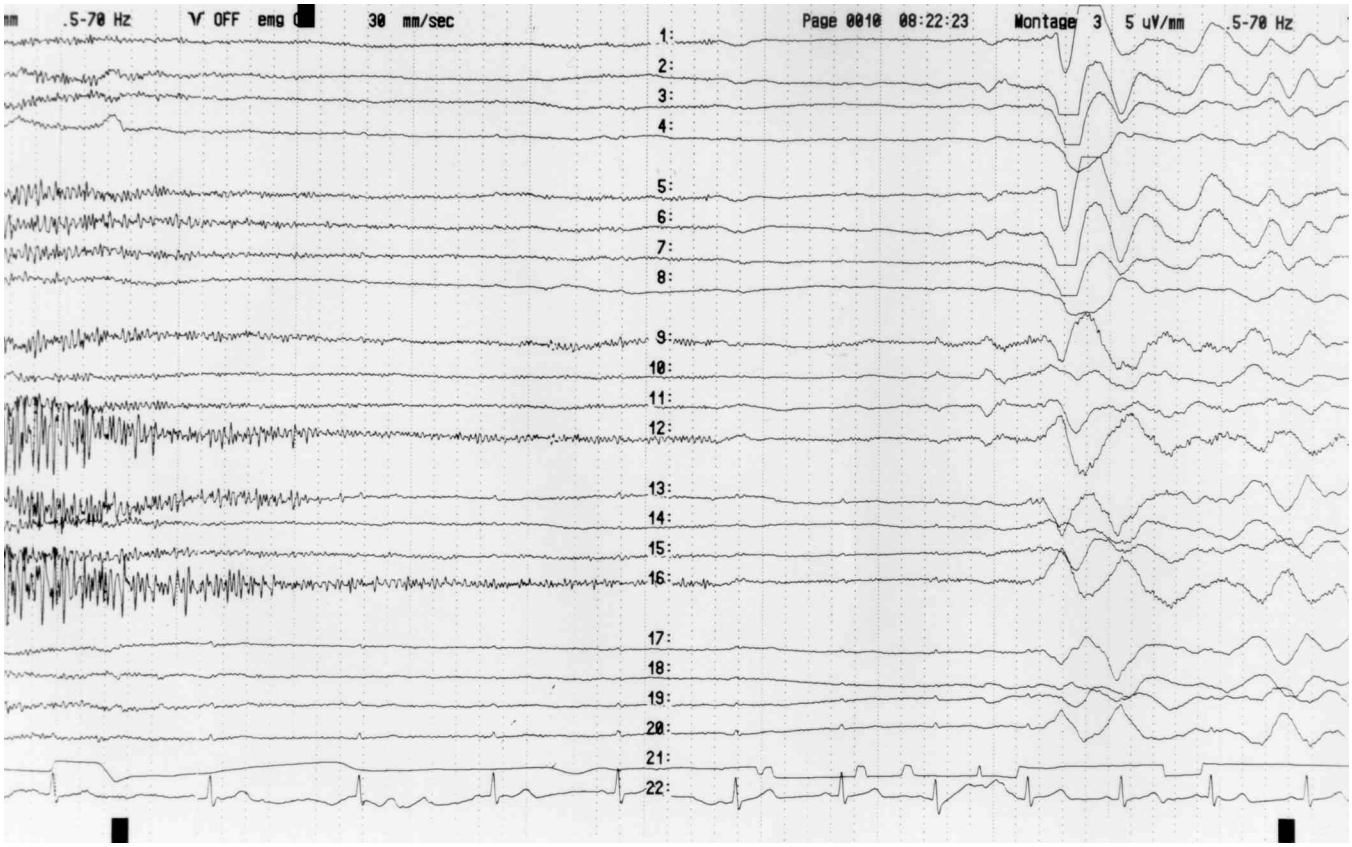

Figure 5. After the convulsions, the sinus rhythm is restored, starting at a rate of 50 beats/min and increasing to 75 beats/min. The EEG recordings still show "cortical silence" before the alpha activity reappears. ECG and EEG abnormalities ended after 30 s. After that period, there was a full recovery of the normal brain activity. Time: 08:22:23.

[8] Lempert T, Bauer M, Schmidt D. Syncope and near-death experience. Lancet. 1994; 344 : 829-830.
[9] Moss J, Rockoff M. EEG monitoring during cardiac arrest and resuscitation. JAMA. 1980; 244: 2750-2751.
[10] Shorvon S. Epilepsy and driving. Brit Med J. 1995; 310: 885-886.
[11] Scheuer ML, Pedley TA. The evaluation and treatment of seizures. N Eng J Med. 1990; 323: 1468-1474.
[12] Zaidi A, Clough P, Cooper P, Scheepers B, Fitzpatrick AP. Misdiagnosis of epilepsy: many seizure-like attacks have a cardiovascular cause. J Am Coll Cardiol. 2000; 36: 181-184.

Contact
Dr. Jan C.J. Res
Zaans Medisch Centrum "De Heel"
P.O. Box 210
1500 EE Zaandam
The Netherlands
Telephone: + 31 72 5817030
Fax: + 31 72 5817031
E-mail: janres@tref.nl