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
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captured during the EEG recording and causes 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 1. The EEG during sinus rhythm prior to the symptoms shows "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.

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.
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


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