LESSON OF THE MONTH

Complete atrio-ventricular conduction block during complex partial seizure

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Abstract

Cardiac arrhythmias are a well known cause of epileptic seizures. Epileptic seizures resulting in cardiac arrhythmias are less well recognised and cardiac arrhythmias are commonly presumed to be of primary cardiac origin. This paper describes a patient with complete AV heartblock during a partial complex seizure. Simultaneous EEG/ECG monitoring was used to show the secondary nature of the bradyarrhythmia.

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When seizures and cardiac arrhythmias coincide, clinicians often interpret the seizures as secondary to arrhythmia-induced cerebral anoxia. The converse, cardiac arrhythmias induced by seizures, has been recognised since the beginning of this century[1] and several recent reports draw renewed attention to the subject.[2-4] Simultaneous EEG/ECG monitoring can help to distinguish central neurogenic from primary cardiac arrhythmias.[4 6]

Case report

A 56 year old white woman presented with 5 episodes of loss of consciousness without specific symptoms of epilepsy. Past medical history was uneventful except for previously reported arterial hypertension. On admission physical examination was normal with a BP of 150/90 mm/Hg and a regular pulse of 88 bpm. Routine blood tests, chest *x* ray, ECG and cranial CT were normal.

Simultaneous EEG (16 channel) and ECG (1 channel) recording showed unremarkable 9–10 cs symmetrical medium amplitude alpha rhythm and sinus rhythm of 80 bpm, respectively. After hyperventilation the EEG displayed intermittent slowing with numerous theta and sharp waves in the left anterior and midtemporal electrodes (figure). Shortly after, in the same leads, an EEG discharge appeared in the form of continuous rhythmic sharp theta waves of slowly increasing amplitude. Ten seconds into this EEG discharge, the patient complained of vague discomfort in the epigastric region, and briefly raised both arms before slumping into the chair. She was unresponsive for the next 70 seconds. Fifteen seconds after the start of the left antero-medial temporal EEG discharge, sinus rhythm had slowed to 60 bpm. This was shortly followed by complete atrio-ventricular conduction block, with a ventricular escape rhythm at 30 bpm. (figure). Ten seconds after onset of the complete a-v block, high amplitude sharp-slow waves partially generalised by spreading to both fronto-temporal regions in the EEG. They were then replaced by high and subsequently low amplitude slow waves which in turn gave way to alpha rhythm. Five seconds after cessation of the high amplitude slow activity in the EEG, cardiac rhythm was restored to sinus rhythm.

The patient was diagnosed as suffering from complex partial seizures associated with cardiac autonomic features and was subsequently treated with carbamazepine. No recurrence of either epileptiform seizures or bradycardia has subsequently been encountered.

Discussion

Recent publications have highlighted central neurogenic factors in altering cardiac rate and rhythm.[2 3 6 7] Sinus tachycardia is the most frequently observed rhythm change associated with seizures, irrespective of the nature of the convulsion.[3 7] A study of cardiac rhythm changes during complex partial seizures (CPS) found that in 74 sequentially recorded seizures, more than 90% were accompanied by increased heart rate and only one was associated with sinus bradycardia.[4] Other studies confirm these results.[7 8] Bradyarrhythmias are less common and they are not linked to any one particular type of seizure.[3 6 9-12]

It can often be difficult to establish whether a cardiac arrhythmia or epileptic discharge is the primary event. The most helpful investigation to differentiate between the two is simultaneous EEG/ECG monitoring as it enables accurate assessment of the temporal relationship between the EEG and ECG. Even this, however, requires caution as well as experience. A cardiac rhythm change due to a cerebral cause can develop before evidence of epileptic discharge in the scalp EEG.[2 6] This is not surprising as deep subcortical epileptic discharges may take less time to reach the closer autonomic regulatory centres than the more distant scalp EEG. As a result cardiac rate changes may occur before changes in the scalp EEG.

In the study by Blumhardt *et al*[4] 57% of seizures (as detected by scalp EEG) were preceded by changes in heart rate by a mean of 10·2 s. In 36% EEG and ECG changes were approximately simultaneous and in 7% ECG

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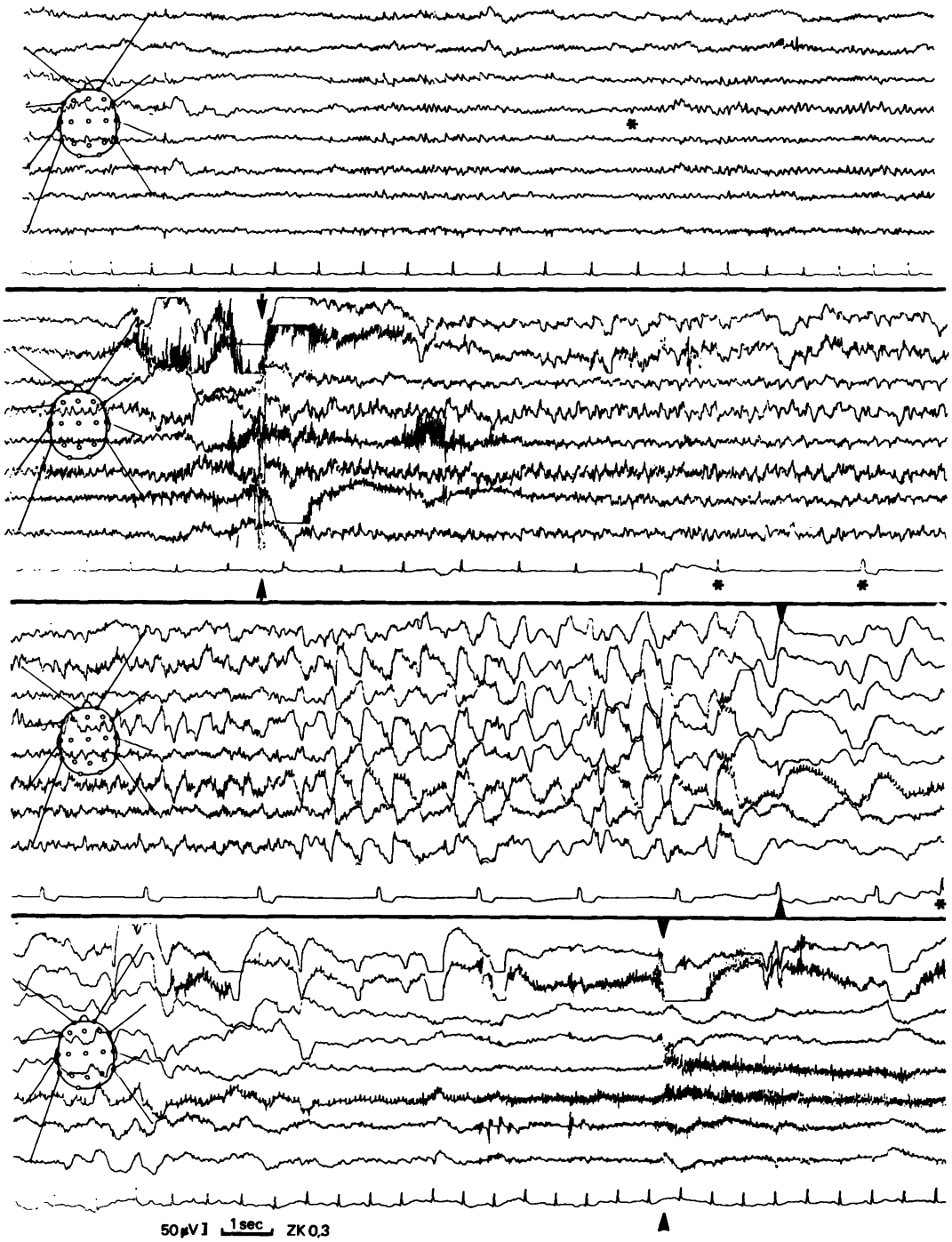

Figure Continuous recording of 8 temporal EEG channels and 1ECG channel during a complex partial seizure with cardiac autonomic features. Top column: Medium amplitude 8–9 cs alpha rhythm with occasional left anterior and midtemporal sharp-slow wave. The asterisk marks the beginning of the EEG epileptic discharge. Note the regular sinus rhythm of 80 bpm. Second column: arrows mark the beginning of the clinical seizure and the artefact caused by the lifting of the arms. The asterisks mark the last sinus beat and the first ventricular escape beat respectively with two isolated P waves appearing in between. Third column: arrows mark the end of the EEG epileptic discharge. The asterisk marks the return of the sinus rhythm. Fourth column: re-emergence of the pre-ictal EEG. Arrows mark the return of the patient's responsiveness. (Monopolar montage, time constant (ZK) of 0·3)

alteration followed 3–5 s later. However, depth electrode recordings may reveal the prior onset of epileptic activity in relation to changes in cardiac function. Unfortunately this is seldom practical. This sequence of events may be occasionally suspected when antiarrhythmic drugs or cardiac pacing fail to prevent symptoms.[2 6] Also, because conventional EEG incompletely covers the brain surface, not all epileptic discharges will be recorded. This may give the impression that the cardiac arrhythmia is not related to epileptic discharge.[6] An analogy can be drawn with epilepsia partialis continua, where in less than 50% of cases is there close correlation between clinical phenomena and scalp EEG discharges.[13]

In our patient, the temporal epileptic discharge clearly preceded the slowing in heart rate by 15 s and distinctly identifies the central neuronal origin of the bradyarrhythmia. As the cranial CT scan showed normal temporal lobes, the origin of the seizure cannot be more accurately localised than the left anterior to mid-temporal area, as indicated by the EEG. Although treatment with carbamazepine was effective, it is worth noting that this drug has on occasion been associated with slowing of atrio-ventricular conduction and so could possibly worsen atrio-ventricular conduction time.[14] In spite of the infrequency of this complication, it would be prudent to perform follow up Holter-ECGs or to consider the use of an antiepileptic such as valproate, which is known to be effective in complex partial seizures without affecting atrio-ventricular conduction.

With the increasing use of long-term ECG monitoring in the evaluation of syncope, it is important not to conclude that primary cardiac arrhythmias are the only possible cause of a patient's complaints.[4 6] Even when they coincide with clinical symptoms, cardiac arrhythmias may be secondary to seizure activity. It is worth noting that in one study of the common sources of error in the diagnosis of convulsive disorders, 83% of patients with complex partial seizures were initially incorrectly diagnosed.[15] It is quite likely that if our patient had been primarily investigated by ECG monitoring alone, a false diagnosis of primary cardiac bradyarrhythmia would have been assumed.

In conclusion, there needs to be increased awareness of arrhythmogenic seizures, which may be underdiagnosed because of a low level of clinical suspicion. Simultaneous EEG/ECG monitoring is superior to either test alone and should be used in uncertain cases.

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