Short report

Cardiac asystole associated with epileptic seizures: a case report with simultaneous EEG and ECG

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SUMMARY Cardiac arrhythmias occurring in association with epileptic seizures are a potential source of diagnostic confusion and a possible cause of sudden unexpected death in epilepsy. A case is described in which simultaneous ambulatory electroencephalography and electrocardiography revealed periods of asystole coinciding with epileptic seizures. The asystole appeared to precede obvious changes in the scalp recorded electroencephalogram (EEG), but clinical attacks and EEG seizure activity were not altered by pacemaker correction of the cardiac arrhythmias.

It has long been recognised that changes in heart rate and rhythm may accompany epileptic seizures.¹⁻³ During the last few years a number of case reports of seizure-associated cardiac arrhythmias have been published⁴⁻⁸ which are of interest for two main reasons. Firstly, it has been suggested that these arrhythmias may be the cause of some sudden unexplained deaths in epileptics.^{9,10} Secondly, seizure-associated arrhythmias may lead to misdiagnosis. The symptoms of partial seizures and of cardiac arrhythmias overlap and can easily be confused. If episodes of disturbed consciousness in an undiagnosed epileptic are recorded by ambulatory electrocardiography (AECG or "Holter" monitoring), a cardiac arrhythmia which coincides with symptoms may be erroneously regarded as the primary causative event. Pacemaker insertion⁵ or inappropriate antiarrhythmic drug treatment¹¹ may result. Many clinicians are aware of the possibility of cardiac arrhythmias causing secondary anoxic seizures and may regard an arrhythmia associated with a seizure as aetiologically significant. Confusion may particularly arise when seizure-induced cardiac arrhythmias occur near the onset of attacks, perhaps before seizure activity is seen in the scalp-recorded EEG.

We report a case in whom simultaneous ambulatory electroencephalography and electrocardiography (SAMME) recordings of seizures were made before

and after successful treatment of ictal asystole with a cardiac pacemaker.

Case report

In 1972, a 55 year old man lost consciousness without warning. Six similar attacks occurred on the same day. A family practitioner who witnessed the attacks described pallor with twitching of the face and limbs. Recovery was associated with flushing, sweating, malaise, numbness of the left leg, headache and left facial weakness. If standing, he fell. An ECG was normal, except for occasional ventricular ectopics and a sinus bradycardia with RR intervals of up to 1.7 seconds. A routine EEG was described as "abnormal with transient generalised slow on one or two occasions during the recording". On these grounds a diagnosis of epilepsy was made and he was commenced on phenobarbitone and phenytoin. After three years free of attacks anticonvulsant drugs were discontinued.

He subsequently developed angina but otherwise remained well until four years later when he lost consciousness while driving and was involved in a road traffic accident which rendered him tetraplegic. In hospital he suffered another episode of loss of consciousness associated with tongue biting, cyanosis and rigidity. On recovery, he complained of chest pain. An ECG showed T wave inversion in leads II, III and aVF, but there was no change in serum cardiac enzymes. He made an almost complete recovery from his spinal injury but episodes of loss of consciousness continued despite the re-introduction of anticonvulsant drugs. Some involved generalised convulsions with clenched teeth, cyanosis, incontinence of urine and tongue biting. Others consisted of sudden falls with loss of consciousness, pallor, cyanosis and grinding of teeth, but not convulsive movements. He was unconscious for 5 to 10 minutes and confused for 1 to 2 hours afterwards.

In 1984, the patient complained that his heart would sometimes miss a beat. Episodes of sinus bradycardia noted on an ECG raised the possibility of Stokes-Adams attacks

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Fig (a) Simultaneous ambulatory cassette recording of EEG and ECG during a seizure. The left hemisphere electrode placements approximate to P3-T3 and the right to P4-T4. The ECG electrodes are positioned to produce a modified lead II. A nine second asystole precedes rhythmic seizure activity, which begins in the left channel at 0946.

and led to a cardiological referral. AECG showed a sinus bradycardia associated with both generalised convulsions and "blackouts" without convulsions. The bradycardia was thought 'insufficient to account for the episodes of loss of consciousness'. Carotid sinus massage had no significant effect on heart rate. He was therefore referred for neurological investigation.

A routine EEG showed generalised raised amplitude, episodic 1.5 Hz activity, most marked in the right frontal region. After withdrawal of anticonvulsant drugs, three attacks were recorded by SAMMEE. Two of the ictal recordings showed periods of sinus arrest lasting 8-9 seconds occurring near the onset of the attacks. The EEG traces appeared normal at this time but were followed within a few

seconds by chewing artifact and sharp and slow wave activity (fig a). In the third recorded attack, similar activity on the EEG was associated only with slowing of the heart rate. Two of the attacks were witnessed by ward staff. One consisted of generalised convulsions with cyanosis and tongue biting and one involved loss of responsiveness, cyanosis, facial twitching and elevation of both arms. All attacks were followed by several hours of postictal confusion. It was thought that the attacks in which asystole appeared to precede the EEG seizure activity might be cardiac in origin or, alternatively, that the period of asystole might be contributing to the duration and severity of primarily epileptic attacks. For these reasons a demand pacemaker was inserted.

The attacks subsequently continued apparently un-

Fig (b) Simultaneous ambulatory cassette recording of EEG and ECG during a seizure following pacemaker insertion. At 1305 seizure activity again begins on the left. Pacemaker function is satisfactory throughout, yet the EEG (and clinical) features of the attack are unchanged.

changed in frequency and severity and repeat SAMMEE recording during two generalised seizures (which were associated with incontinence of urine and prolonged postictal confusion) showed bilateral seizure discharges accompanied by correct pacemaker activation with no asystole or bradycardia (fig b). On neurological review after a year there had been no change in attack frequency or severity, despite pacemaker and continued medication.

In 1987, aged 69 years, he developed heart failure. A few months later, he had an episode of chest pain and fever. A chest radiograph showed shadowing at the left base and an ECG showed atrial fibrillation, left ventricular hypertrophy and inferior and lateral T wave inversion. There was no rise in serial cardiac enzymes. He was treated with antibiotics for a presumed chest infection but after a few days had further chest pain and died suddenly. A post-mortem examination was not performed.

Discussion

This case confirms that cardiac rate or rhythm changes caused by an epileptic seizure may, occasionally and confusingly, appear to precede the seizure activity observed in the scalp recorded EEG, as previous studies have shown.¹²⁻¹⁵ Arrhythmias more commonly occur at the end of the seizure as the sympathetic tachycardia and raised blood pressure are returning to normal levels, but vagal effects such as bradyarrhythmias may also predominate early in the course of some seizures.¹³⁻¹⁵ Recordings from depth electrodes may of course reveal that seizure activity is occurring before the onset of the ictal cardiac effects, but such information will seldom be available to clinicians using

ambulatory EEG for diagnosis.

We are aware of one previous report of ictal asystole in which both ECG and EEG were recorded during an attack. Katz and colleagues⁴ recorded EEG and ECG during complex partial seizures in two patients. The EEG showed temporal seizure activity becoming generalised while periods of sinoatrial arrest began shortly after the EEG seizure onset. The periods of sinus arrest lasted 8 to 10 seconds. Subsequent cardiological investigations were apparently normal, although details were not given in the report.

Several other reports of seizure-associated sinus arrest (generally without definitive simultaneous recording of ECG and EEG) have been published.^{5,6,8,11} In some cases loss of consciousness has been attributed to syncope occurring secondary to a seizure-induced asystole, and some patients do appear to have been partially responsive to therapy for their secondary arrhythmia. For example, Phizackerly and colleagues¹⁶ described a 71 year old woman with attacks of epigastric discomfort and a feeling of apprehension or impending doom, sometimes proceeding to loss of consciousness. An interictal EEG showed right anterior temporal spikes. The pulse which was normal at the onset of the epigastric sensation, sometimes ceased abruptly and the ECG showed "complete sino-auricular block". If this continued for more than five seconds, loss of consciousness occurred. Treatment with tincture of belladonna abolished the episodes of loss of consciousness, although the attacks of epigastric discomfort and apprehension continued. If we assume that the arrhythmia in this case was secondary to a temporal lobe seizure (as the authors suggest), the response to treatment suggests that loss of consciousness was not directly due to the seizure, but to the asystole caused by it.

In our case it was initially suspected that the periods of asystole might be contributing to the severity or duration of the attacks or even precipitating them. That this was not the case was suggested by some ictal recordings which revealed that identical attacks could occur without asystole (although they were all associated with some degree of cardiac slowing), while the failure of pacing to prevent or modify the frequency, severity or the characteristic features of the seizures confirmed that the periods of asystole were in fact secondary and non-contributory ictal arrhythmias.

A wide variety of other arrhythmias, both supraventricular^{5,11,17-19} and ventricular^{7,20} have been described in association with epileptic seizures. The potential for the misdiagnosis of epileptic seizures as cardiac arrhythmias therefore certainly exists,^{5,11} although it is difficult to know from presently available data how commonly this occurs. Simultaneous ECG and EEG recordings can assist but as our case demonstrates, the apparent temporal relationship of ictal ECG and scalp EEG changes does not necessarily indicate the

primary cause of the symptoms.

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