Simultaneous EEG and ECG Recording During a Stokes-Adams Attack

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ABSTRACT

Simultaneous electroencephalographic and electrocardiographic recordings were obtained from a 77-year-old patient during a Stokes-Adams attack. The recordings showed a clear temporal relationship between symptoms, electroencephalographic and electrocardiographic changes during the Stokes-Adams attack. This case shows the usefulness of simultaneous EEG and ECG recordings in the investigation of patients with unexplained episodes of disturbed consciousness.

Keywords: Stokes-Adams attack, simultaneous electroencephalographic and electrocardiographic recordings

INTRODUCTION

The eponymously named "Stokes-Adams attack" is characterised by a sudden lost of consciousness unrelated to posture and is usually due to episodic intermittent high grade AV block, profound bradycardia or ventricular standstill. These bradyarrhythmias may be mediated by vagal reflex mechanisms and can be seen in a variety of disease states like cough, neuralgias and during a variety of diagnostic procedures like endoscopy, thoracenterosis and cardiac cathetherisation[1]. Stokes-Adams attacks can also develop in patients with acute myocarditis, athlete's heart and mesothelioma of the atrioventricular node. This is a case report of simultaneous electroencephalographic (EEG) and electrocardiographic (ECG) recordings obtained from a patient during a Stokes-Adams attack, showing the temporal relationship between symptoms, EEG and ECG changes.

CASE REPORT

A 77-year-old woman with a two months' history of "dizzy spells" was admitted for investigations. She presented with light headedness, blurring of vision, generalised weakness, unsteadiness and a sensation of swaying of the surroundings during an attack of these spells. These attacks were sudden in onset and were as abrupt in their recovery. She did not experience any syncope during these spells which could last from a few seconds to a quarter of an hour. They appeared randomly and were not related to neck movements,

posture changes, prolonged standing, micturition or coughing nor was she conscious of any palpitations during these dizzy spells. She was well in between attacks and had decided to seek medical advice only because these episodes were getting more frequent and persistent.

No cardiovascular or neurological abnormalities were detected on clinical examination. Resting 12-lead ECG showed sinus rhythm of 76 bpm with non-specific T inversions in the anterior leads. Simultaneous electroencephalographic and electrocardiographic recordings were performed as her symptoms were suggestive of a Stokes-Adams attack. Fortuitously, she had one of her "dizzy spells" during the simultaneous recordings. She was witnessed to have complained of dizziness just before becoming unconscious and was unresponsive a few seconds later with intense facial pallor. She was pulseless and apneic throughout the episode. She was unconscious for about a minute before spontaneous respiration and a pulse returned.

Simultaneous EEG and ECG tracings were obtained during the syncopal attack. These showed that she was in normal sinus rhythm with a rate of 70 bpm prior to her loss of consciousness. Her cardiac rhythm quickly degenerated into complete cardiac standstill after a couple of junctional escape beats. This lasted for 27 seconds. During the period of sinus rhythm, her EEG tracings showed prominent moderate voltage of 10 per second alpha rhythm of symmetrical distribution with no consistent focal changes. Ten seconds following ventricular standstill, this background EEG rhythm was replaced by a three per second burst of high voltage delta activity in all areas. This was followed six seconds later, by a flat record of no electrical activity in all the 20 scalp electrodes. It was during this period that the patient was noted to have lost consciousness.

Recovery from the episode was evident first by the return of a junctional beat followed by sinus bradycardia for five seconds before sustained normal sinus rhythm was re-established. Electroencephalic waves appeared three or four seconds after the return of sustained normal sinus rhythm and ten seconds after the reappearance of cardiac electrical activity. The initial electroencephalic waves were those of high voltage delta waves. These persisted for a few seconds to be replaced by theta frequencies waves lasting a

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minute. Normal brain wave activity was noted subsequently. The patient probably regained consciousness during the period of delta or theta waves activities as it was at this stage that the movement artefact of a wandering baseline was noted.

A Teletronic VVI permanent pacemaker was implanted and she was free from further "dizzy spells" or "blackout" when reviewed one year later.

DISCUSSION

Robert Adams was the first person to recognise the link between cardiac rhythm disturbances and cerebral symptoms when he characterised a form of apoplexy associated with a slow pulse in 1827[2]. The patient he described was an officer in the revenue with a pulse of 30 per minute with frequent apoplectic attacks of complete insensibility during which the pulse became even slower. This patient eventually died from his apoplectic attacks. William Stokes in 1846, recalled Adams' cases, added his own and defined a combination of fainting attacks with a slow pulse and degeneration of the heart which has been known ever since as the "Stokes-Adams attack"[3].

Although simultaneous EEG and ECG recordings have been commonly employed in the investigations of syncope and unexplained seizures with a variety of cardiac arrhythmias having been documented as a secondary effect of seizures, reports of continuous EEGs and ECGs tracings obtained during a Stokes-Adams attack are rare[4-5]. After a search on Medline, four case reports of Stokes-Adams attacks with documented simultaneous EEG and ECG recordings were found[6-9]. Three of these reported ventricular asystole as the primary trigger event. In these reports, complete electrocerebral silence was recorded 13 to 45 seconds after the onset of ventricular asystole, preceded in all cases by a marked slowdown of the electroencephalic wave form frequency and an increase in wave amplitude. Lai reported that his patient became unconscious when ventricular asystole persisted for longer than 5.5s[7]. However the EEG recording of his case showed slowing of the brain waves only, without complete electrical silence[7]. Schraeder reported that his patient became unconscious and then went on to develop a generalised tonic-clonic seizure a few seconds after the onset of a 'flat' EEG tracing. Ventricular asystole in his patient persisted for 13s and the seizure continued until after the return of heartbeat and brain wave activity[8]. In all these cases, recovery from electrical silence was generally rapid, with alpha waves returning 5 to 35 seconds after the return of cardiac rhythm. Tucker reported a case of chronic complete atrio-ventricular block and spells of Stokes-Adams attacks precipitated by paroxysms of ventricular tachycardia of varying duration[6]. He noted that abnormal EEG patterns appeared only when the paroxysm of ventricular tachycardia persisted for over 10 seconds. The first indication of an abnormal pattern was the presence of slow brain rhythms and the appearance of high voltage theta activity. The appearance of these large amplitude waves were often preceded clinically by a

short episode of confusion, pallor, and staring. The patient developed tonic convulsions when the runs of ventricular tachycardia lasted 18 seconds or longer. When the duration of ventricular tachycardia lasted more than 3 minutes, the patient invariably became unconscious and complete electrocerebral silence on the EEG was recorded.

Our case of fortuitous simultaneous EEG and ECG recordings during a Stokes-Adams attack showed a similar temporal association between the cessation of cardiac contraction, onset of syncope and the changes in brain electrical activity. The sudden appearance of ventricular asystole was followed soon after with a slowing of the frequency of the brain electrical activity, syncope and the rapid appearance of complete cerebroelectrical silence. It took 10 seconds of cerebral hypoperfusion for abnormal EEG waves to appear and an additional 6 seconds for the EEG to become completely flat. Normal alpha waves returned one minute after the re-establishment of cardiac rhythm.

This rapid loss of neuronal function following complete cessation of cerebral circulation was also demonstrated in lightly anaesthetised patients undergoing induced circulatory arrest for implantation of an automatic internal cardioverting defibrillator[9]. A decrease in mean arterial BP and changes in haemoglobin saturation of blood in the brain vasculature was followed, on average, 6.5 seconds later by EEG changes, with near isoelectric tracing evident 10 seconds after induction of ventricular fibrillation[9]. It is the abrupt manner in which ventricular asystole or bradyarrhythmia can occur and the rapid loss of cerebral function which follows the profound reduction or complete cessation of cerebral blood flow that resulted in the suddenness of the syncope that characterised the Stokes-Adams attack.

The usefulness of simultaneous EEG and ECG monitoring in the investigations of patients with unexplained episodes of disturbed consciousness, in patients with tonic anoxic seizures secondary to cardiac arrhythmias and in patients with cardiac arrhythmias triggered by epileptic seizures had been reviewed by Blumbart[4]. He showed that up to 25% of tonic anoxic seizures secondary to a cardiac arrest may have been misdiagnosed and treated as having generalised motor seizures[4]. Cardiac arrhythmias may also be implicated in some reported cases of sudden deaths that occurred in some epileptic patients. Conversely, virtually any type of cardiac arrhythmias may occur as a secondary effect of seizures, with even relatively minor complex seizures being shown to be capable of having a profound effect on cardiac rate and rhythm and unless simultaneous EEG and ECG data were available, cardiac arrhythmias which coincide with non-specific symptoms could be interpreted as causative and erroneously treated as Stokes-Adams syndrome.

Hence simultaneous EEG and ECG recordings can be helpful in the differential diagnosis of cardiac and cerebral dysrhythmias.

In summary, our case demonstrated clear temporal changes in cardiac rhythm, brain wave activity and

symptoms during a Stokes-Adams attack. It showed that brain wave activity and hence cerebral symptoms deteriorates rapidly following cessation of cerebral blood flow. Furthermore, simultaneous EEG and ECG monitoring have a role to play in the investigations of unexplained episodes of disturbed consciousness.

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