Neurological or Cardiological Assessment of Transient Altered Consciousness?

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SUMMARY: Cardiac arrhythmias are a recognised cause of transient alteration of consciousness and unless suspected a cardiac aetiology for apparent neurological symptoms may be overlooked. We retrospectively assessed the variation in the investigations of 122 patients who were referred to our hospital over a twelve month period for assessment of transient alteration of consciousness and we attempted to identify any factors which might have influenced the clinical outcome for these individuals. Eighteen patients were initially referred for cardiological opinion and 104 for neurological assessment. Sixty-one patients presented with syncope, 43 with seizures, and 18 with presyncope. A baseline electroencephalogram (EEG) was obtained in 104 (85%) and 65 (53%) were also subjected to a stress EEG. Cerebral dysrhythmia was documented in 14 patients. A standard electrocardiogram (ECG) was recorded in only 58 (58%) and sinus bradycardia (heart rate <60/min) was observed in 34. Twenty-four hour (Holter) ECGs were recorded in only 58 (48%). Ventricular pauses (at least 1.84 seconds) and/or profound bradycardia (heart rate <40/min) occurred during waking hours in 10 patients and were more likely to be detected in individuals who also exhibited bradycardia on standard ECG (8 patients) ($p < 0.05$).

After six months, 35 (29%) of the 122 patients still lacked a diagnosis and although cerebral dysrhythmia had been diagnosed with equal frequency in the bradycardia and non-bradycardia groups, 21 (62%) of those with bradycardia were still symptomatic compared with 11 (30%) of the non-bradycardia group ($p < 0.02$).

Our findings indicate that, at present, investigation of patients with transient altered consciousness lacks a coordinated approach and bradycardia may influence the diagnosis and morbidity of these individuals.

We suggest that greater attention should be paid to possible cardiac causes of transient altered consciousness and that only under joint direction of a cardiologist and a neurologist can efficient and thorough investigation of these patients be realised.

Introduction

Cardiac arrhythmias, especially transient ventricular asystole, are a recognised cause of dizziness, syncope and convulsions (1, 2, 3, 4, 5) and unless they are suspected, cardiac causes for apparent neurological symptoms may not be recognised. A diagnosis of epilepsy is often one based on clinical criteria (6) and can have profound career and social implications for an individual (7). However, cardiac arrhythmias, including transient ventricular asystole, if they are recognised and successfully treated, in certain circumstances may permit continuation of an individual's career and minimal restrictions on social activities such as driving a motor vehicle (8). Therefore, it is essential that medical officers are aware of possible cardiac causes for neurological symptoms and that patients who are referred to hospital for neurological assessment of transient altered consciousness should also undergo cardiological investigations particularly if no obvious neurological cause for their symptoms is detected.

The purposes of this study are: i) to assess the variation in methods of investigation of patients with histories of episodic disturbance of consciousness who were referred over a period of one year to the Queen Elizabeth

Military Hospital (QEMH); ii) to identify any factors which might have influenced the clinical outcome for these patients.

Methods

The hospital medical records of all patients over 16 years of age who were referred over a twelve month period to the QEMH for investigation of afebrile seizures (i.e. at least one episode), syncope and presyncope were retrospectively studied.

Patients who were receiving treatment for psychiatric disorder (including addiction to alcohol) at the time of referral were excluded from analysis. In addition, individuals with histories of significant head injury (i.e. associated with coma and/or evidence of intracranial haemorrhage, cerebral oedema, or other structural damage discernible on computerised tomography (CT) or magnetic resonance imaging (MRI) of skull) were omitted from this study. Other reasons for exclusion were: a history of craniotomy, CT evidence (at time of referral) of an intracranial space occupying lesion, concomitant meningitis or encephalitis, chronic dementia, clear clinical evidence of pseudoseizures, sleep disorders (including narcolepsy). Vestibular and labo-

rinthine disorders, including Meniere's disease, which were clinically evident at time of presentation also precluded patient entry. In addition, no clinically apparent cases of Parkinson's disease, chronic cerebellar or brain stem degenerative disorders, or peripheral neuropathic processes were included in this study. None of the patients whose data were acceptable for analysis in this survey suffered from: diabetes mellitus, chronic liver disease, chronic renal failure, electrolyte imbalances, thyroid gland disorders.

Patients were classified as having experienced seizures, syncope or presyncope depending on a combination of their clinical histories, the authors' initial clinical assessments and, where available, witnesses' accounts (including F Med 138) of episodes of disturbance of consciousness.

For the purposes of this study, a seizure was defined as an abnormal, paroxysmal discharge of cerebral neurones clinically apparent to the subject or to an observer.

Syncope was defined as a transient loss of consciousness accompanied by muscle weakness and inability to stand upright. Presyncope was defined as a sudden onset of transient sensation of impending loss of consciousness without actual syncope.

In assessing the management of these patients, particular attention was paid to the number of individuals who were investigated by means of a standard (i.e. twelve lead) electrocardiogram (ECG), a twenty-four hour ambulatory electrocardiogram (i.e. Holter ECG), a baseline or ambulatory electroencephalogram (EEG) and an EEG recorded under conditions of fasting and sleep deprivation (i.e. stress EEG). All EEG and ECG recordings were scrutinised by the authors.

An EEG was classified as either normal or abnormal. An abnormal EEG was defined as a recording which demonstrated changes supporting a diagnosis of primary cerebral dysrhythmia. The number of patients who exhibited an abnormal EEG was documented. In assessing standard ECGs, the number of patients with evidence of an unexplained sinus bradycardia (i.e. heart rate <60/min) was noted. Thus, for example, bradycardia was disregarded when it was observed in patients who were receiving betablockers or digoxin therapy.

On analysis of Holter ECG tapes, particular attention was paid to episodes of profound bradycardia (i.e. heart rate <40/min) and ventricular pauses (i.e. asystole) occurring during waking hours. Heart rates were measured between three consecutive QRS complexes on a printout and, for the purposes of this calculation, ECG strips containing abrupt pauses or rhythm disturbances were not considered. A ventricular pause was defined as a R-R interval >1.8 seconds and an abrupt change in R-R interval (excluding compensatory pauses following premature QRS complexes). Therefore, regular R-R intervals of >1.8 seconds during a slow stable heart rhythm were not classified as pauses — i.e. in individuals with heart rates of 33/min or less.

Sleep associated profound bradycardia and ventricular pauses were not considered to be significant findings and were therefore disregarded. Furthermore, bradycardia and ventricular asystole occurring in patients who were taking beta blockers or other medication known to reduce heart rate and affect sinoatrial and atrioventricular conduction were disregarded. All other significant arrhythmias detected on Holter ECG monitoring were documented.

Details of specific bedside clinical tests such as detection of carotid sinus hypersensitivity and documentation of postural hypotension, where considered appropriate, were also recorded.

Statistical Methods

Differences between mean age were evaluated using Student's *t* test. Either Fisher's exact test or the chi-square test with Yates correction, as appropriate, was used for analysis of other data. Statistical significance was defined as $p < 0.05$.

Results

Over a period of twelve months, a total of 188 patients were referred to the QEMH for assessment of episodes of disturbance of consciousness. Sixty-six of these individuals were excluded from analysis in this survey on account of their initial clinical presentations.

Therefore, 122 adults (99 males) were considered as suitable cases for inclusion in this study. They ranged in age from 16 to 78 (mean $\pm$ SD: 30.9 ± 17.96) years.

Eighteen of these individuals were referred initially for cardiological assessment and the remaining 104 patients sought a neurological opinion. Forty-three cases were referred with a history of at least one seizure, 61 on account of recurrent syncope, and 18 because of recurrent presyncope.

At the time of their first presentation to our hospital, 6 of the 43 patients with a history of seizure had experienced only a single episode but all of those referred for investigation of syncope or presyncope had suffered more than two events. None of those with a history of seizure was referred initially for cardiological evaluation.

Figure 1 demonstrates that at least a baseline EEG was performed in all 43 cases who presented with a history of seizure and a stress EEG was obtained in 31 (72%) of these individuals. A minimum of a baseline EEG was recorded in 104 (85%) of the 122 patients and 65 (53%) were also subjected to a stress EEG (Fig 1).

An abnormal EEG was demonstrable only after fasting and sleep deprivation in 11 cases and in 3 others a baseline EEG was abnormal. Therefore, an abnormal EEG was documented in a total of 14 (13%) of those undergoing this investigation (Fig 1). Twelve of these individuals presented with a history of seizure, one with syncope, and one with presyncope.

Fig 1. Patients investigated by electroencephalography (EEG) and exhibiting abnormal EEG.

Data relating to figure 1.

Clinical presentation	No. of patients	No. of patients with baseline EEG recorded	No. of patients with stress EEG recorded	Abnormal EEG
Seizures	43	43 (100%)	31 (72%)	12 (28%)
Syncope	61	52 (85%)	28 (46%)	1 (2%)
Presyncope	18	9 (50%)	6 (33%)	1 (6%)
Totals	122	104	65	14

Figure 2 demonstrates that a standard ECG was recorded in 71 (58%) of the 122 patients and sinus bradycardia was documented in almost half of these cases. All of the ECGs were otherwise unremarkable apart from one in the non-bradycardia group which demonstrated left bundle branch block.

A Holter ECG recording was obtained in 58 (48%) of the study group and pauses (6 cases) or episodes of profound bradycardia (8 cases) were evident in a total of 10 patients. None of these individuals claimed to be symptomatic during these recordings. The periods of ventricular asystole ranged from 1.84 to 5.2 seconds in duration and were associated with second degree Wenckebach type atrioventricular heart block in 3 cases and sinus node arrest in the remaining 3 patients.

If the 90 cases with a normal EEG are considered together with the 18 individuals from whom an EEG was not obtained, only a standard ECG was recorded in 12 (11%), only a Holter ECG was performed in 2 (2%), and both types of investigation were documented in 53 (49%) of these 108 patients.

All of the 10 cases with pauses or profound bradycardia on Holter ECG had normal EEG recordings. Holter ECG monitoring did not demonstrate any rhythm disturbances in 25 patients. The arrhythmias detected in the remaining 33 individuals are listed in Table 1. None of these patients reported any symptoms in association with their recorded rhythm disturbances.

Fig 2. Patients investigated by electrocardiograph (ECG) and exhibiting bradycardia on standard ECG.

Data relating to figure 2.

Clinical presentation	No. of patients	No. of patients with standard ECG	No. of patients with bradycardia on standard ECG	Holter ECG recorded
Seizures	43	16 (37%)	6 (14%)	10 (23%)
Syncope	61	42 (69%)	23 (38%)	37 (61%)
Presyncope	18	13 (72%)	5 (28%)	11 (61%)
Totals	122	71	34	58

Holter ECG monitoring on three patients with an abnormal EEG did not demonstrate any arrhythmias.

Table 2 demonstrates that patients with bradycardia on standard ECG did not differ significantly in either age or sex composition from those individuals in the non-bradycardia group (Table 2). In addition, there was no

**Table 1
Significant findings on Holter ECG monitoring of 58 patients**

	No. of patients
Sinus node arrest	3
Profound sinus bradycardia	8
Nodal bradycardia	1
Short PR interval	1
Ectopic atrial pacemaker	1
Paroxysmal atrial flutter/fibrillation	2
Paroxysmal supraventricular tachycardia	1
Ventricular ectopics (benign)	14
Ventricular ectopics (R on T)	2
Paroxysmal ventricular tachycardia	1
A-V Block:	
First-degree	1
Wenckebach second-degree	3
Complete	1
None of the above	25

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Table 2

Comparisons of age, sex, EEG and ECG results between patients with and without bradycardia on standard ECG

	No. of patients with bradycardia N = 34	No. of patients with no bradycardia N = 37	p
Males (%)	32 (94)	29 (78)	N.S.
Mean age $\pm$ S.D.	29.9 $\pm$ 17.9	37.7 $\pm$ 21.2	N.S.
EEG performed (%)	28 (82)	25 (68)	N.S.
Abnormal EEG (%)	3 (9)	4 (11)	N.S.
Holter ECG performed (%)	23 (68)	33 (89)	p<0.05
Normal Holter ECG (%)	9 (27)	16 (43)	N.S.
Pauses and/or profound bradycardia (%)	8 (24)	2 (5)	p<0.05

N.S. — not significant.

significant difference in the number of individuals in each group who were subjected to EEG assessment and the percentage of patients who demonstrated abnormal EEG recordings was also similar (Table 2).

Although less patients with bradycardia were investigated by Holter ECG (p<0.05), these individuals exhibited an increased tendency (p<0.05) to manifest pauses and/or profound bradycardia during waking hours (Table 2).

At six months after initial referral, a diagnosis of cerebral dysrhythmia had been ascribed with equal frequency in the bradycardia and non-bradycardia groups (Table 3). There was no significant difference between the two groups in the number of patients lacking a diagnosis for their symptoms. However, after six months of hospital based investigations and attempts at treatment, 21 (62%) of those with bradycardia on standard ECG were likely to be suffering from recurrent episodes compared to 11 (30%) in the non-bradycardia group (p<0.02).

After six months of investigations, response to carotid sinus stimulation had been assessed in only 8 patients who had continued to experience recurrent symptoms. Carotid sinus hypersensitivity (i.e. ventricular asystole of at least 3 seconds duration following carotid sinus massage for a period of less than 5 seconds) was demonstrable in three of the four individuals in the bradycardia group whose responses to carotid sinus stimulation were documented. In contrast, none of the four patients in the non-bradycardia group who were subjected to carotid sinus testing manifested a hypersensitive reaction.

Tilt testing was performed in only 2 patients, both of whom exhibited sinus bradycardia at rest, and in each case failed to provoke symptoms or ventricular asystole.

By six months after initial referral, all patients had

Table 3

Comparisons of prevalence (after six months of investigations) of diagnosis of cerebral dysrhythmia, no diagnosis, and continuing symptoms between patients with and without bradycardia on standard ECG

	No. of patients with bradycardia N = 34	No. of patients with no bradycardia N = 37	p
Diagnosis of cerebral dysrhythmia (%)	11 (32)	10 (27)	N.S.
No diagnosis (%)	13 (38)	10 (27)	N.S.
Continuing symptoms (%)	21 (62)	11 (30)	p<0.02

N.S. — not significant.

been screened for evidence of postural hypotension but only one individual in the non-bradycardia group demonstrated this clinical sign.

Two patients were referred for cardiac electrophysiological studies and in both cases the results did not yield any useful information in relation to the management of their symptoms.

Discussion

This study has revealed that although an EEG was performed in all cases presenting with seizures, standard ECG was recorded in only 58% and a Holter ECG was obtained in only 48% of 122 patients referred to our hospital for investigation of episodic disturbance of consciousness. This was despite a documented normal EEG in 90 of these individuals.

Ideally, a diagnosis of primary cerebral dysrhythmia should be based on a combination of clinical history and data derived from EEG investigations. However, in practice, assessment of the clinical situation, in conjunction with a trial of anticonvulsant therapy, may often be the only means whereby this diagnostic dilemma can be resolved (6). In our hospital, an abnormal EEG was recorded in only 7 (10%) out of 71 patients in whom a standard ECG was documented and a tentative diagnosis of primary cerebral dysrhythmia was ascribed to a further 14 of these individuals whose EEG recordings were normal.

There was no significant difference in the prevalence of this diagnosis between those exhibiting bradycardia on standard ECG and those in the non-bradycardia group.

Sinus bradycardia was not an unexpected finding in the predominantly military population which attends our hospital. Most of these soldiers are of young age and possess a high degree of physical fitness. However, patients who exhibited bradycardia on standard ECG did not differ significantly in age from those in the non-bradycardia group but were more likely to manifest profound bradycardia and ventricular pauses of up to

5.2 seconds during waking hours.

It is generally recognised that such cardiac rhythms are common, particularly during sleep, in asymptomatic young athletes (9, 10, 11, 12). However, our patients were being investigated for episodes of disturbance of consciousness and there is also evidence that athletic individuals with ventricular pauses of even 1.4 seconds in duration can benefit from implantation of a pacing system (12, 13). Therefore, the significance of Holter ECG evidence of bradycardia and pauses, particularly in healthy military personnel, can be difficult.

It is disappointing that 23 (32%) of the 71 patients in whom a standard ECG was recorded had still not received a satisfactory explanation for their symptoms six months after initial referral to our hospital. Although 35 (29%) of the total 122 referred individuals still lacked a diagnosis at six months, this compares favourably with non-diagnostic rates of between 39% and 48% reported elsewhere (14, 15).

However, of greater concern is that 32 (45%) of the 71 patients in whom a standard ECG had been documented were still continuing to suffer from their symptoms after six months of hospital investigations and this problem was significantly more prevalent in those with bradycardia. This suggests that vagal mediated factors may be of critical importance in these individuals (16). Three out of 4 cases with heart rates less than 60/min demonstrated a significant hypersensitivity response to carotid sinus stimulation and this may lend support to the presence of increased vagal tone in these patients (2, 17). A previous study on the prevalence of carotid sinus hypersensitivity in asymptomatic individuals has involved 52 subjects whose ages ranged from 36 to 87 years (2). Thus, at present, there are clearly inadequate data on the significance of this finding in young athletes with histories of episodic disturbance of consciousness.

Tilt testing for the malignant vasovagal syndrome should be more commonly performed (18) and we intend to introduce this procedure in addition to assessment of responses to carotid sinus stimulation as part of our routine investigations of patients with unexplained seizures, syncope and presyncope.

In conclusion, our findings suggest that patients with transient altered consciousness who exhibit an unexplained bradycardia on standard ECG are significantly more likely than non-bradycardia patients to suffer continuing morbidity after six months of assessment. Therefore, greater attention should be paid to possible cardiac causes of altered consciousness.

At present, investigations of patients who are referred to our hospital for assessment of transient altered consciousness appear to lack a co-ordinated approach. In addition, since the present trend for super-specialisation in postgraduate hospital career training inevitably results in one discipline being relatively ignorant of advances in clinical research and electrophysiology in the other, we propose that a more

efficient and a more thorough hospital based assessment of these patients would be achieved if their investigations were to be co-ordinated jointly by neurologists and cardiologists.

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