

Investigations for Syncope; Role of EEG and the Specialty of the Ordering Physician

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Syncope is a common condition and constitutes 1-3% of all emergency department and 6% of hospital admissions in the United States¹. It is characterized by a sudden transient loss of consciousness, loss of postural tone and relatively rapid recovery. The diagnosis of epilepsy is considered if there are associated abnormal body movements, prolonged loss of consciousness, tongue biting or incontinence. Syncope occurs in all age groups however the incidence rises in the elderly, especially after the age of 70. The assessment and investigation of patients with episodic and transient loss of consciousness remains a challenging task particularly if the history and investigations are not entirely supportive of the diagnosis of epilepsy or a cardiac cause.

Most patients with syncope have no warnings while others may experience premonitory symptoms that can be helpful in differentiating syncope from seizures or other conditions. When establishing a diagnostic strategy for a patient with syncope, one must rely heavily on clinical information however; important information and witness description are often missing. In addition, there can be disagreement about the significance of clinical symptoms between physicians.

Seizure like activity in the context of syncope can be the result of the global cerebral hypoperfusion². Observations of the clinical manifestations of syncopal attacks in a video study showed that myoclonus is common in syncope and other seizure-like events such as head turning and even automatisms can occur. This makes diagnosis even more difficult³.

In an editorial by Hunter and Moss in a recent issue of *Neurology*⁴, the authors discussed the commonality between the channelopathies associated with long QT interval and CNS that could cause seizures. They referred to the recently reported presence of cardiac potassium channels in CNS raising the question of whether seizures in these patients are cardiogenic in origin or whether they are epileptic seizures due to impaired ion channels in CNS. They indicated that patients with LQTS have a higher incidence of seizures compared to general population of 0.5%-1%.

Seizures can also infrequently provoke arrhythmias. Keilson⁵ reported that in 338 patients with the diagnosis of epilepsy, ambulatory electro-encephalogram (EEG) for 20-24 hours revealed 56 episodes of electrographic seizures (focal and generalized) lasting at least for ten seconds, with no recorded arrhythmias. However, ictal bradycardia syndrome and ictal asystole have been described in rare patients with epilepsy mostly related to temporal lobe seizures⁶.

One pitfall when attempting to determine the etiology of syncope is misdiagnosing a cardiac dysrhythmia as an epileptic seizure. Appropriate treatment, such as placement of cardiac pacemaker, may then not be instituted. Fortunately, syncope and

seizure disorder in the same patient rarely co-exist⁷. On the other hand, treatment of patients with heart disease with anti-seizure drugs such as carbamazepine could have serious medical and psychosocial outcome such as conduction defects, potential hypersensitivity reaction to anti-seizure drugs, driving restrictions, and cost as indicated in the discussion by Poliquin-Lasnier and Moore published in this issue⁸.

In order to direct physicians in diagnostic approach, in 2002, Sheldon et al in a prospective study addressed the historical criteria that distinguish syncope from seizures⁹. Six hundred and seventy one patients with history of loss of consciousness completed a 118-item historical questionnaire. In this so called Syncope Symptom Study, the data set was statistically analyzed. The causes of loss of consciousness were known in 539 and included seizures. One hundred and two patients had complex partial seizures and primary generalized epilepsy and the other 437 had other causes, mostly cardiac. The point score based on symptoms alone correctly classified 94% of patients and the authors concluded that a simple point score of historical features distinguishes syncope from seizures with a high sensitivity and specificity. In another study, the authors validated patient characteristics such as San Francisco Syncope Rule to predict the likelihood of serious outcomes¹⁰.

Most studies report a good prognosis for syncope (often in those with vasovagal attacks); however, the morbidity and mortality are not negligible in patients with cardiac or neurologic causes. Sarasin et al in their prospective study analysed the outcome of the 650 patients presenting with syncope and found 9% mortality in 18 months period¹¹. In fact, in this study, patients with underlying cardiovascular disease had a mortality of 30% in one year. Considering the risk of significant morbidity or mortality in a subgroup of patients with cardiogenic or neurologic cause, patients presenting with transient loss of consciousness often undergo a battery of tests including ECG, 24 hour ambulatory ECG monitoring, tilt table test, carotid sinus massage and EEG.

In a recent study the yield of diagnostic tests in evaluating syncopal episodes in older patients was reported¹². The authors studied 2106 patients over age 65 years. Cardiac enzymes, CT head, echocardiogram, carotid ultrasound and EEG all affected the diagnosis or management in less than 5% of cases and helped in determining the etiology of syncope in less than 2%. The cost per test for diagnosis and management was highest for EEG and CT head. Postural BP recording performed in only 38% of patients had the highest yield in the diagnosis and management of syncope.

In this issue of the Canadian Journal of Neurological Sciences, Poliquin-Lasnier and Moore⁸ reviewed the records of patients who were referred with the diagnosis of syncope over

the previous five years. The goal was to determine the yield of EEG in patients with suspected syncope and also the yield of EEG if ordered by neurologists, a question that had never been addressed before. They also sought to determine whether an abnormal EEG significantly changed the management of these patients. The authors found that only 57 out of 517 EEGs (11%) were reported abnormal. There were no epileptiform discharges and only nine (1.6%) showed potentially epileptic abnormalities. Their findings are similar to the previous studies that report very low diagnostic yield for EEG in this group of patients.

A retrospective study reviewed EEGs performed in a hospital in the previous four years with the question of syncope. They found diffuse slowing in 28% of patients and only 1.46% showed epileptiform discharges. This number was similar to the general population and did not change the management of these patients¹³. Bridges et al obtained 24 hour cassette ambulatory EEGs in 67 patients with syncope and only one revealed epileptic discharges (1.5%)¹⁴ and several other studies have reported similar results and little yield for EEG in patients with syncope¹⁵.

The authors in the current study also found that the yield of EEG was higher if it was ordered by ER physicians or internists compared to neurologists. The difference was likely due to the fact that most of the EEGs ordered by the ER/internists were on patients who were hospitalized; their EEGs were performed in less than 24 hours compared to a median of 14 days for the entire study population. The hospitalized patients could have also had other (e.g. metabolic) abnormalities not studied which could also lead to EEG abnormalities. In the group in which EEG was ordered by a neurologist, the patients usually had an office visit and the detailed clinical data was not reported.

Electroencephalogram may be normal in 12–50% of patients with epilepsy and serial EEGs increase the diagnostic yield¹⁶. In patients with syncope, if the history is clearly of a cardiac/homodynamic cause, EEG does not aid in the diagnosis, however, it can show abnormalities as reported in the literature¹⁷. In his review, Brenner describes EEG findings in four major categories of syncope i.e. neurocardiogenic, neurologic, decreased cardiac output and orthostatic hypotension. He concluded that regardless of the cause of syncope, EEG findings are similar during the episode and reflect cerebral hypoperfusion.

Initial slowing of background rhythms on EEG (presyncope) is followed by high amplitude delta activity. If the hypoperfusion persists, there is subsequent flattening of the EEG. None of the findings were distinctly epileptic. Convulsive syncope may occur in cases of severe and prolonged ischemia at the time of flattening of the EEG. Clinically, this is often mistaken for epilepsy. Conversely, epileptic seizures with ictal bradycardia syndrome or ictal asystole may occasionally mimic syncope. There also remains a small group of patients that have low seizure threshold and experience convulsions provoked by hypoxic/ischemic insult with epileptic abnormalities on their EEG.

Electroencephalogram should not be routinely performed in the evaluation of syncope. However, in a subgroup of patients with transient loss of consciousness and clinical presentations suggestive of epileptic seizure (e.g. typical auras, witnessed convulsions or automatisms, prolonged recovery, tongue biting or incontinence and amnesia) ordering EEG is justified. In this

group of patients if EEG is negative for seizure activity, serial EEGs including sleep deprived study and sometimes continuous video/EEG recordings are required.

The current publication agrees with the literature⁸. However, with the limitations of a retrospective study, the authors did not analyse the clinical history and impression of the ordering physician, which are important in such data analysis. In the management of patients presenting with a transient loss of consciousness, an approach should be taken using the basic principles of a careful history (using witness information where possible), and physical examination to formulate specific laboratory investigations to address the clinical differential diagnoses of syncope as suggested by McKeon¹⁸.

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