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Syncope or seizure? The diagnostic value of synchronous tilt testing and video-EEG monitoring in children with transient loss of consciousness

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ABSTRACT

Syncope and seizure are frequently encountered problems in daily neurology practice, and they also share common findings such as transient loss of consciousness and atonia. Sometimes, it is difficult to make a differentiation between the two entities using only clinical findings. In this study, nineteen patients aged between 5 and 20 years who had recurrent transient loss of consciousness and occasional atonic events were examined with synchronous tilt testing and video-EEG recordings. Eleven patients were initially diagnosed with epilepsy, and they were given antiepileptic drugs. Eight patients displayed neurally mediated syncope during examination. Four of the eight patients had cardioinhibitory syncope type 2B. Three-fourths of the patients with syncope had been initially diagnosed with epilepsy and were prescribed antiepileptic drugs. One patient with cardioinhibitory syncope who had prolonged asystole and frequent attacks needed a cardiac pacemaker. Following implantation, she had no new attacks. Synchronous tilt testing and video-EEG recordings give more information than doing them separately, and they are helpful in the differential diagnosis of syncope and seizure.

1. Introduction

Syncope and seizure are neurologic conditions that may have similar clinical findings like transient loss of consciousness and postural tone. The development of clonic or jerky movements with an attack that otherwise suggests syncope makes the differential diagnosis much more difficult. The problem is aggravated by the fact that both conditions have no gold standard laboratory tests. Electroencephalogram (EEG) is the most important tool for diagnosing epilepsy, but it has limited sensitivity. The EEG has a sensitivity of no more than 50% and might reach 80% with appropriate activating measures [1]. Therefore, a normal EEG does not rule out epilepsy. It is well-known that 23–30% of patients who are referred to pediatric epileptology centers actually have nonepileptic events [2,3]. Reflex (neurally mediated) syncope is one of the most frequent causes of paroxysmal nonepileptic events.

Syncope is a transient loss of consciousness due to a transient global cerebral hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery. A low or inadequate peripheral resistance can be due to inappropriate reflex activity causing vasodilatation and bradycardia manifesting as vasodepressor, mixed, or cardioinhibitory reflex syncope. Abnormal movements (e.g., myoclonic jerks) may develop as a consequence of cerebral hypoperfusion [4]. Although clinical guidelines were reported to distinguish syncope from epileptic seizures, misdiagnosis is still a problem [5]. Those patients having recurrent transient loss of consciousness and postural tone may be diagnosed with epilepsy by even well-trained physicians.

In this study, we aimed to evaluate the diagnostic value of synchronous tilt testing and video-EEG monitoring in pediatric patients who had transient loss of consciousness and postural tone where the diagnosis remained unclear.

2. Materials and methods

2.1. Subjects

This cross-sectional study was conducted between March 2010 and February 2011 at Ege University, Faculty of Medicine, Child Neurology Department. Nineteen children aged between 5 and 20 years were included in the study. All patients experienced at least two attacks of transient loss of consciousness with or without loss of postural tone. Eleven patients were diagnosed with epilepsy. Standard neurologic and cardiac examinations were done. 12-lead electrocardiography (ECG), total blood count, awake and sleep electroencephalogram (EEG) recordings and cranial magnetic resonance imaging (MRI) were obtained in all patients. Inclusion criteria for patients were as follows:

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1. Experienced at least two transient loss of consciousness and postural tone attacks.
2. Normal neurologic and cardiac examinations.
3. Normal ECG and cranial MRI.
4. Normal or mild nonspecific changes on EEGs.
4. Normal hemogram.

2.2. Tilt testing

Tilt testing and video-EEG monitoring were simultaneously applied to all patients. Tilt testing was performed in a single laboratory in the Cardiology Department according to a local protocol. Patients were rested supine for 15 min (baseline period) then underwent head-up tilt on a powered tilt table to 60° for up to 45 min (tilt period) or until symptoms occurred. Blood pressure, heart rate and pulse oximetry values were recorded every 5 and 3 min during baseline and tilt periods, respectively. Tilt testing responses were evaluated with a modification of the original Vasovagal Syncope International Study classification [6,7]:

Type 1 mixed: Heart rate falls at the time of syncope, but the ventricular rate does not fall to less than 40 beats min^{-1} or falls to less than 40 beats min^{-1} for less than 10 s with or without asystole of less than 3 s. Blood pressure falls before the heart rate falls.
Type 2A: Cardioinhibition without asystole. Heart rate falls to a ventricular rate less than 40 beats min^{-1} for more than 10 s, but asystole of more than 3 s does not occur. Blood pressure falls before the heart rate falls.
Type 2B: Cardioinhibition with asystole. Asystole occurs for more than 3 s. Heart rate fall coincides with or precedes blood pressure fall.
Type 3 vasodepressor: Heart rate does not fall more than 10% from its peak at the time of syncope.

2.3. Electroencephalogram (EEG)

Simultaneous video-EEG (Nihon-Kohden, 9100) recording was also obtained during the tilt test. An international 10–20 electrode placement system was used. Data were recorded with the placement of reference electrodes on earlobes, and one channel was used for heart rate. Video-EEG recordings were reviewed by two authors (S. G. and S. Y.) independently. Observed movements were of myoclonic or tonic nature during syncope attack. Movements similar to twitching lasting for shorter than a few seconds were accepted as myoclonic jerks. Sustained muscle contractions lasting for at least 2 s were evaluated as tonic movements.

Before testing, informed consent was obtained from each family. The Ethics Committee of the Medical Faculty at Ege University approved the study.

3. Results

Nineteen patients (twelve female and seven male) were enrolled in the study. Their mean age was 12 ± 4 years. They experienced at least two attacks of transient loss of consiousness with or without atonia within 2 months–10 years. The attack numbers ranged between 2 and 100. In their histories, limb movements of either tonic or myoclonic pattern were described by 6 patients (32%). During attacks, abdominal pain was found in 3 patients, and urinary incontinence was found also in 3 patients. Vomiting was a rare finding; it was only found in two patients. Their first EEGs, taken at the beginning of their symptoms, were normal in all except for five patients. At admission, eleven patients had been diagnosed with epilepsy, and different anticonvulsants were given to them. Nine of them had used antiepileptic drugs for 1.5–5 years. At admission, three patients were still taking anticonvulsant drugs. Their ECG, awake/sleep EEGs and cranial MRI findings were normal.

During tilt testing, mixed syncope was observed in four patients, and cardioinhibitory type 2B syncope was observed also in four patients. Eight of the nineteen patients (42%) had displayed syncope. However, only one patient (case 3) complained about dizziness and nausea before syncope developed. In the cardioinhibitory group, asystole was observed ranging from 9 to 24 (mean: 15) s. During their syncope attack, all patients showed loss of consciousness and atonia. Two patients with cardioinhibitory syncope (cases 3 and 5) displayed asychronous bilateral myoclonic jerks of the upper extremities during asystole. Tonic flexor contractions of the arms following myoclonic jerks were observed in one patient (case 5). The video-EEG recording of case 3 displayed asystole that lasted for 27 s, and he also showed myoclonic jerks and atonia (Fig. 1). During the syncope attacks, video-EEG recordings revealed focal bilateral parietooccipital or diffuse delta activity. Generalized voltage suppression followed diffuse delta activity in three patients. Its duration was between 7 and 10 s. Two of the three patients had cardioinhibitory syncope. The demographic and laboratory findings of our patients are outlined in Table 1.

Six of the eight patients (75%) displaying vasovagal syncope had been diagnosed with epilepsy, and they used antiepileptic drugs for 1.5–5 years. Two of them were still taking the drugs. Their drugs were tapered within 2 months. One patient (case 2) who had four attacks during the last 2 months displayed cardionihibitory syncope with prolonged asystole. Since the events could be fatal, a cardiac pacemaker was implanted. She has not experienced a new attack for 1.5 years. The rest of the cases with cardioinhibitory syncope continue to be followed regularly.

During synchronous tilt testing and video-EEG recording, one patient displayed nausea and dizziness with changes in neither heart rate nor blood pressure. The test was ended at the 21 min of upright position. Tilt test was accepted as negative but symptomatic. In this patient, simultaneous video-EEG recording revealed normal waking rhythms. He was recommended preventive measures for vasovagal syncope.

The remainder of the patients (cases 10–19) had negative tilt testing response and normal first and study EEGs. Four patients (cases 10, 12, 16 and 18) experienced their last attacks between 18 and 30 months ago. They had used anticonvulsant drugs for 2–4 years. They had no further attacks after commencement of antiepileptic drugs. So, those patients were diagnosed with epilepsy. Case 14 had abused drugs, and it was thought that his attacks might be related to central adverse effects of the drug. Two patients (cases 11 and 15) described loss of consciousness immediately after a fall during playing or running. These attacks were presumptively diagnosed as pallid breath-holding spells, even though they were older than 4 years. Two patients (cases 13 and 17) suffered from loss of consciousness attacks when affected by illness, febrile status or hunger. One patient also described abdominal pain just before loss of consciousness. Therefore, we concluded that their attacks were similar to syncope, even though they had negative tilt testing response. Information about vasovagal syncope and its prevention was given to their parents. The last patient (case 19) had syncope-like epileptic (?) attacks and psychogenic attacks for 10 years, and she was followed-up by both the Child Neurology and Child Psychiatry departments. She was also still taking oxcarbazepine. Her presumptive diagnosis favored psychogenic nonepileptic seizures.

4. Discussion

During childhood, transient loss of consciousness with or without change in postural tone is a common finding for both epileptic events and neurally mediated syncope. Sometimes, it is difficult to distinguish between them based only upon clinical findings. Up to 64% of patients seen in epilepsy clinics may not have epilepsy, and syncope