Evaluation of the child with syncope

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Abstract

Transient loss of consciousness (TLOC) is spontaneous loss of consciousness with complete recovery; there is temporary impairment of brain function associated with loss of awareness, loss of responsiveness, and loss of memory. Syncope is the most common cause of transient loss of consciousness in children. Whilst syncope is potentially easy to understand using applied physiology, therapeutic opportunities are often missed, leading to avoidable morbidity and disability. In this article we will discuss the causes of TLOC and focus on the evaluation and management of syncope and orthostatic intolerance in children and young people.

Keywords fludrocortisone; head-up tilt test; midodrine; PoTS; slow sodium; transient loss of consciousness

Introduction

Syncope is a symptom rather than a disease. It is caused by cerebral hypoxia and/or hypoperfusion. Up to 15% of children will experience an episode prior to the end of adolescence. Pre-syncope (the feeling of lightheadedness and associated symptoms as though one might lose consciousness) is even more common. The aetiology of pre-syncope and syncope is most often benign. There are, however, life-threatening (usually cardiac) causes.

The semiology of syncope can be very variable, and includes convulsive, hypotonic unresponsive, and unresponsive stare. Pre-syncopal symptoms may precede the loss of consciousness, and post-ictal symptoms, especially confusion, distress, and sleepiness, may follow.

Causes of syncope

1) Cardiac

Cardiac syncope is caused by an abrupt drop in cardiac output caused by an arrhythmia (typically a tachyarrhythmia) or structural heart disease. Examples of primary electrical disturbances which can cause syncope are: Long QT syndrome, Brugada syndrome, and pre-excitation syndromes. Structural heart disorders include hypertrophic obstructive cardiomyopathy (HOCM), severe aortic stenosis, and arrhythmogenic right ventricular dysplasia. The first two can cause sudden left ventricular outflow tract obstruction, while the latter can lead ventricular tachyarrhythmias. Paradoxically, because there are also runs of tachycardia in sleep, Long QT is well known to cause episodes of convulsive syncope in sleep. These can be easily misdiagnosed as epileptic seizures.

2) Neurally mediated or reflex syncope

In neurally mediated syncope (NMS) there is an abnormality of autonomically mediated alterations in vasomotor tone and heart rate which are normally responsible for maintaining blood pressure and cerebral perfusion, under different circumstances. NMS can be as a result of

a) Cardio-inhibition (sudden bradycardia or asystole)
b) Vasodepression (inappropriate peripheral vasodilatation reducing vascular resistance and systemic blood pressure)
c) A mixture of the two

Examples of cardio-inhibitory syncope are reflex asystolic syncope (RAS also known as reflex anoxic seizures or infantile vasovagal syncope) and blood injury phobia. RAS (Figure 1) is the same as malignant cardio-inhibitory syncope, a term preferred by adult cardiologists. Somewhat confusingly, in North America RAS is conflated with Expiratory Apnoeic Syncope (see Apnoeic Syncope below) and both are called "breath-holding spells". Vasodepressor or mixed NMS is by far the most common type of syncope and is typically associated with pre-syncopal symptoms. It is widely known as vasovagal syncope (a term used interchangeably with neurocardiogenic syncope or simple fainting). NMS is generally thought of as a benign condition, but if severe or frequent can be associated with marked morbidity and disability. Therapeutic options are discussed below.

3) Apnoeic syncope

Examples of apnoeic syncope include upper airway obstruction (UAO), Valsalva related syncope, and Expiratory Apnoea Syncope (EAS), also known as "blue breath-holding attacks". In the latter, there is reduced venous return as the chest is held in expiration, so cardiac output is impaired. The child becomes deeply cyanosed within a couple of seconds, probably due to intra-pulmonary shunting. This then reduces the oxygen delivery on top of the reduced cerebral perfusion.

4) Vascular syncope

This is syncope caused by reduced circulating blood volume, leading to reduced cardiac output and hence impaired cerebral perfusion. Examples include hypovolaemic shock, septic shock, anaphylaxis, and systemic hypotension.

5) Cerebral syncope

Obstruction to cerebral blood flow can occur acutely e.g. in adolescent stretch syncope, and in vertebrobasilar syncope.

6) G-force induced loss of consciousness

G-force induced loss of consciousness is an unusual type of syncope which occurs from excessive and sustained G-forces e.g. pulling blood away from the brain. We are yet to hear of a paediatric case.

7) Mixed mechanisms

It is likely that in some cases multiple factors contribute to the syncope: in someone prone to NMS or OI (see below), a low

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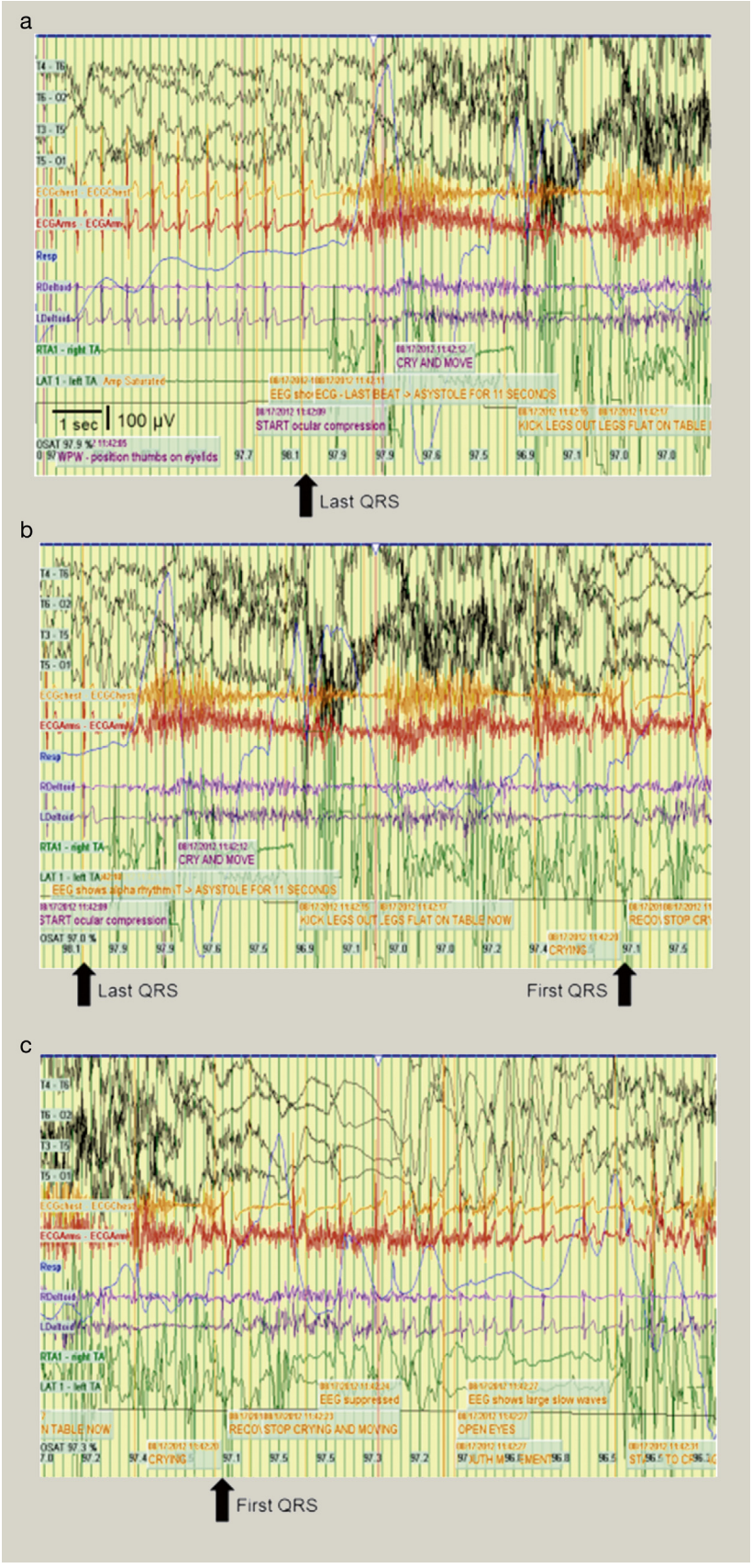

Figure 1 Ictal ECG–EEG before (**a**) during (**b**) and after (**c**) a reflex asystolic syncope precipitated by ocular compression. The heart rate can be seen in the ECG signal in the orange, red, and purple lines. The four black lines at the top are EEG showing brain activity, and the two green lines at the bottom are tibialis anterior surface EMG showing muscle activity. There is significant movement artefact during the syncope.