Fifteen-minute consultation on limiting investigations in the fainting child

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ABSTRACT
Syncope can be a dramatic and frightening experience for the patient and onlookers. It can also be a confusing area for doctors, with a lack of diagnostic certainty and a differential that ranges from the completely benign to the life-threatening. This is a tricky area for clinicians, who can find it difficult to reassure their patients and their families. The aim of this article is to present a clear scheme for appropriate investigation and referral of paediatric patients with syncope. The aim of investigations in such cases should be to confirm or exclude serious, life-threatening causes of syncope.

WHAT IS SYNCOPE?
Syncope is a transient, but complete, loss of consciousness (with associated loss of muscle tone) due to global cerebral hypoperfusion. By definition:
- It is of rapid onset
- It is of short duration
- Recovery is – quick – spontaneous – complete

Therefore, by definition, this excludes:
- Falls, which usually do not involve loss of consciousness
- States of altered, but not loss of, consciousness
- Coma, which, by definition, is not transient loss of consciousness
- Aborted sudden cardiac death
- Epileptic seizures
- Psychogenic causes
- ‘Near’ syncope, in which patients do not experience loss of consciousness, but can have several symptoms including lightheadedness, etc.

WHAT CAUSES SYNCOPE?
Syncope can be divided on an aetiological basis, into
- Syncope due to cardiac causes
- Syncope due to reflex mechanisms, which includes vasovagal syncope
- Orthostatic hypotension.

WHY DOES SYNCOPE HAPPEN?
Syncope occurs when the systolic blood pressure (BP) falls below around 60–70 mm Hg, although this exact figure is age-related.

BP is the product of cardiac output and systemic vascular resistance. Therefore, anything that affects one or both of these determinants will cause an alteration in BP. Examining cardiac output and systemic vascular resistance in turn.
Cardiac output can be affected by:
- Cardiac mechanisms
  - Changes in heart rate and rhythm
  - Structural heart problems, for example, aortic stenosis
- Reflex (nervous) mechanisms
  - Slow heart rate due to reflex nervous activity (vagal)
- Orthostatic mechanisms
  - Inadequate venous return

Systemic vascular resistance can be affected by:
- Reflex mechanisms
  - Vasodilation due to inappropriate reflex activity
- Orthostatic mechanisms
  - Impairment, due to a variety of primary and secondary causes, of the autonomic nervous system
- Generally cardiac factors do not affect systemic vascular resistance (figure 1).

WHAT CAUSES CARDIAC SYNCPE?
The serious, life-threatening causes of syncope are generally cardiac in nature. Cardiac causes of syncope result in low cardiac output. Cardiac syncope may be due to:
- Electrical disturbances (arrhythmias)
  - Bradycardia, which is uncommon in children
    - Sinus node dysfunction
    - AV node dysfunction
    - Implanted device (eg, pacemaker) malfunction
  - Tachycardia
    - Supraventricular
    - Ventricular
      - Long QT syndrome (see figure 2)
      - Brugada syndrome (see figure 3)
      - Catecholaminergic polymorphic ventricular tachycardia (see figure 4)
  - Drug-induced, which may act via the above mechanisms.
- Structural cardiac problems
  - Hypertrophic cardiomyopathy
  - Dilated cardiomyopathy
  - Arrhythmogenic right ventricular cardiomyopathy
  - Coronary artery anomalies

Figure 1  Mechanisms of syncope.

Figure 2  ECG of long QT syndrome showing the ‘Tangent’ method for measuring QT interval.

- For example, left coronary artery coursing between the aorta and the pulmonary artery; anomalous origin of the left coronary artery from the pulmonary artery
  - Aortic stenosis, resulting in an obstructed left ventricular outflow tract
  - Myocarditis
  - Pulmonary hypertension, although there is usually a history of exertional dyspnoea rather than syncope in this condition
  - Cardiac tumours

The non-life-threatening causes of syncope, therefore, fall into the remaining two categories, namely:
- Reflex syncope, which includes ‘vasovagal’ episodes
- Orthostatic hypotension.

HOW SHOULD YOUNG PEOPLE WITH SYNCPE BE ASSESSED?
Assessment begins with an accurate history to confirm syncope, to identify a possible causal mechanism (cardiac, reflex, orthostatic—see box 1 for further characteristics) and to ascertain any relevant background information, including family history. Assessment should
continue with physical examination. For completeness, BP can be recorded at this time. A 12-lead ECG should be performed as the initial baseline investigation in syncope. On the basis of these initial actions, further investigations or onward referral can be considered.

Studies have confirmed that the sensitivity of screening using history, examination and 12-lead ECG findings can be 100% sensitive for the cardiac causes of syncope.¹ ⁵

HISTORY
The mainstay of diagnosis is in the history. First and foremost, loss of consciousness must be confirmed. Many patients have funny turns but not true syncope. The event should be transient, of rapid onset and short duration. There should be loss of postural tone. Recovery should be spontaneous, complete and without sequelae. Lack of an eyewitness can make diagnosis more challenging. If not all of these features can be confirmed, then it will be necessary to exclude other causes of loss of consciousness. Seizures may be a sequelae of syncope. However, it is important to note that a number of people diagnosed with epilepsy may in fact have a cardiac cause of syncope. In a study from New Zealand, several patients had a delayed diagnosis of long QT syndrome and were instead diagnosed as epileptic. On review, several of these patients had had baseline ECGs that were abnormal, but because the corrected QT had been miscalculated initially, the diagnosis had been missed.⁶ The corrected QT interval should be calculated using Bazett’s formula, which corrects for heart rate. The end of the T wave can be difficult to define, but using the ‘Tangent’ method, this uncertainty is much reduced⁴ (see figure 2).

Once syncope is confirmed, the history must seek to elucidate the possible cause, that is, cardiac, reflex or orthostatic. A good history of the event, the situation and position of the patient before, during and after the event should be sought (see boxes 1–4). If mobile phone or video footage of the event exists, then this is often invaluable.

In the background, a family history of sudden death, arrhythmias at a young age (less than 40 years) and previous cardiac disease should be confirmed or excluded. Of note, a study from the Netherlands published in 2009 showed that patients with long QT syndrome were significantly more likely to have a family history of syncope and sudden death.⁷ Neurological disorders, including epilepsy and other neurodisabilities, and metabolic problems, principally diabetes, should be sought. Medications, including vasoactive substances such as diuretics, and other drugs (including of abuse) should be assessed.

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**Box 1** Classical premonitory symptoms for syncope²

*Often evolving, gradual, progressive*

- Nausea
- Blurred/tunnel vision
- Hearing changes (muffled, buzzing)
- Dizziness
- Light-headedness
- Sweating
- Pallor
- Cold, clammy skin
- Weakness, tremulousness

**Box 2** Features suggestive of reflex (neurally mediated) syncope² ³ ⁸

- Nausea (unique to non-arrhythmogenic syncope)
- Vomiting
- After an emotionally stressful event
- Following prolonged standing in a hot, crowded area
- During or after a meal
- After (not usually during) exercise
- With head rotation

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Figure 4  ECG showing appearances of polymorphic ventricular complexes on exercise testing in a patient who is β-blocked and known to have catecholaminergic polymorphic ventricular tachycardia.
Red flags for cardiac syncope

More specifically, ‘Red Flag’ features in the history significant for a cardiac cause of syncope are shown in box 4. Some investigators have found that dizziness is a symptom in patients with arrhythmogenic (cardiac) syncope. However, nausea appears to be unique to non-arrhythmogenic syncope.

Reflex syncope is suggested by typical triggers and typical prodromal symptoms (see boxes 1 and 2). The European Society of Cardiology further subdivides reflex syncope into vasovagal, situational, carotid sinus and atypical syncope.

Vasovagal syncope occurs secondary to emotional stressors or the sight of blood, or a painful stimulus, among others, but can also occur secondary to orthostatic stress. For example, this would include syncope on standing from a lying or squatting position.

Situational syncope might occur secondary to ordinary activities such as stretching, hair brushing, coughing, sneezing, micturition, swallowing (especially cold liquids), defaecation, following meals, after prolonged standing, in warm or crowded environments, or immediately after (not during) strenuous exercise.

Carotid sinus syncope is presumably secondary to carotid sinus massage causing vagal stimulation. This might occur when wearing tight collars or neckties.

Atypical forms of reflex syncope also occur. These are, by definition, rather difficult to define, but occur without apparent triggers or with an atypical presentation.

Limiting investigations in young people with syncope

Cardiac causes of syncope can most likely be excluded with high sensitivity with appropriate history taking, clinical examination and a 12-lead ECG. Further investigations are probably inappropriate. It may be helpful, and indicated, to obtain an ambulatory ECG prior to referral to, or at the suggestion of, a paediatric cardiologist. However, ambulatory ECG monitoring has a low yield for capturing arrhythmia, of the order of 14% in some studies, even in those subsequently shown to have cardiac syncope. In the same
study, 15% of patients reported symptoms that had no correlation on ambulatory ECG monitoring. The low yield is likely due to the relative infrequency of arrhythmia events, which are too infrequent to occur contemporaneously with monitoring.

Further investigations that may be carried out by a cardiologist may include an echocardiogram, in the context of an abnormal clinical cardiovascular examination, exertional syncpe or family history of heart muscle disease. In cases of exertional syncope, an echocardiogram is probably warranted to exclude such rare anatomical anomalies as anomalous origin of the left coronary artery from the pulmonary artery. The diagnostic yield of echocardiography is low, as with ambulatory ECG monitoring, less than 50% in some studies, even in those with cardiac syncpe. In one study from several years ago, the sensitivity of echocardiography for detecting a cardiac cause of syncpe was 18%.

In the case of exertional symptoms or startle-related syncpe, exercise testing is mandated, as conditions such as catecholaminergic polymorphic ventricular tachycardia cannot be detected on a resting ECG. Other, further investigations might include:

- Longer-term ambulatory monitoring, dependent upon symptom type and frequency. Event monitors (patient activated) generally have no role in syncpe, but are useful in palpitations
- Implantable loop recorder
- Invasive electrophysiological studies, although these are rarely required
  - Tilt-table testing is not particularly useful in the paediatric population, and should be left in the hands of experts. In the context of limiting investigations in fainting children, tilt testing is not usually required for excluding the life-threatening causes of syncpe. It may be helpful, in the hands of experienced investigators, in discriminating between reflex syncpe, orthostatic hypotension, seizure disorders and psychiatric disorders. Protocols for tilt testing are not uniform, and as children have a higher propensity to orthostatic hypotension compared with the adults for whom the protocols were designed, there is a lack of specificity inherent in this investigation.

In summary, when looking to limit investigations in the fainting child, it is important to have as clear as possible a history of the events. If syncope is confirmed, then the object is to exclude serious, potentially life-threatening, causes of syncpe. These are essentially the cardiac causes of syncpe. History, examination and ECG are usually sufficient to confirm benign causes of syncpe, or to identify those patients who need referral for further assessment and investigation.

Fainting in children will continue to provoke consternation, and there will always be diagnostic dilemmas. However, the vast majority of syncopal episodes in children are benign in nature, and it is satisfying and resource efficient to be able to swiftly reassure children and their parents on the basis of history, clinical examination and some rudimentary investigations.

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