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Diagnostic algorithm for syncope

Roberto Mereu [a], Arunashis Sau [b], Phang Boon Lim [c,*]

[a] Department of Internal Medicine, University of Pavia, Italy
[b] Imperial College London, UK
[c] Imperial Syncope Diagnostic Unit, Imperial College Healthcare NHS Trust, London, UK

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ABSTRACT

Syncope is a common symptom with many causes. Affecting a large proportion of the population, both young and old, it represents a significant healthcare burden. The diagnostic approach to syncope should be focused on the initial evaluation, which includes a detailed clinical history, physical examination and 12-lead electrocardiogram. Following the initial evaluation, patients should be risk-stratified into high or low-risk groups in order to guide further investigations and management. Patients with high-risk features should be investigated further to exclude significant structural heart disease or arrhythmia. The ideal currently-available investigation should allow ECG recording during a spontaneous episode of syncope, and when this is not possible, an implantable loop recorder may be considered. In the emergency room setting, acute causes of syncope must also be considered including severe cardiovascular compromise due to pulmonary, cardiac or vascular pathology. While not all patients will receive a conclusive diagnosis, risk-stratification in patients to guide appropriate investigations in the context of a diagnostic algorithm should allow a benign prognosis to be maintained.

1. Introduction

Syncope is a complex symptom with many potential causes. The history is essential for diagnosis and clinical features of the patient should be used to guide further investigations, where necessary. The use of a broad range of screening investigations to elucidate the cause of syncope is neither productive nor cost-effective, and is stressful for the patient. One important aspect of the initial evaluation of a syncopal patient is an assessment of the risk of sudden cardiac death (SCD). Diagnostic tests for syncope generally have a low yield (Linzer et al., 1997a, 1997b) and should be used in a focused manner to confirm a diagnostic suspicion obtained at the initial evaluation.

2. Initial evaluation

The initial evaluation of a patient with transient loss of consciousness (TLOC) should include a detailed history, physical examination (including lying and standing blood pressure measurements over a 3 minute period) and an ECG (Moya et al., 2009). Typical historical features include: a prodrome of feeling sweaty or warm before a faint, and lightheaded spells with prolonged sitting, standing or during episodes of pain or in a medical setting. Some features less suggestive of vasovagal syncope are: late onset of syncope (over 35 years of age), history of arrhythmia or diabetes, turning blue during the faint, or remembering behavior during the syncopal event (Sheldon et al., 2006). From the initial evaluation, it is possible to reach a conclusive diagnosis in 50–63% of cases with a high diagnostic accuracy of approximately 90% (Linzer et al., 1997a; van Dijk et al., 2008). Table 1 shows the situations in which a conclusive diagnosis can be made after initial evaluation (Moya et al., 2009). In these circumstances further investigations are not required.

In 50% of patients it is not possible to reach a conclusive diagnosis on initial evaluation, due to the absence of clear triggers and because the history and ECG are inconclusive (Linzer et al., 1997a). In these cases the ongoing diagnostic management depends on the patient's risk of SCD. Table 2 summarizes high-risk ECG features. Several studies have identified several risk factors for SCD in patients with syncope (Martin et al., 1997; Colivicchi et al., 2003; Quinn et al., 2004; Reed et al., 2007). There are three main conditions that set the patient in a high risk of SCD: the presence of severe structural or coronary heart disease (heart failure, low left ventricular ejection fraction, previous myocardial infarction), clinical or ECG features suggestive of arrhythmic syncope (syncope during exertion or when supine, palpitations at the time of syncope, family history of SCD or ECG abnormalities described in Table 2) and co-morbidities such as anemia or electrolyte disturbance. Patients at high risk may require prompt hospitalization and an intensive evaluation to exclude a cardiac cause of syncope (Moya et al., 2009). These patients should be admitted to a bed with continuous cardiac monitoring, or have a 24 hour Holter monitor attached in order to document significant arrhythmias. Cardiac syncope is associated with a 20–30% mortality reported at 1–2 years from several studies

* Corresponding author at: Imperial Syncope Diagnostic Unit, Ground Floor, B Block, Cardiac Investigations Centre, Hammersmith Hospital, Imperial College Healthcare NHS Trust, Du Cane Road, London W12 0HS, UK.
E-mail address: pblim@imperial.ac.uk (P.B. Lim).

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Table 1
Conclusive diagnoses that can be made on initial evaluation.

Neurally mediated syncope (NMS):
- Classical vasovagal syncope precipitated by emotional distress (strong pain, fear, invasive medical procedures, blood sight) or orthostatic stress and preceded by prodromal symptoms as nausea, warmth, light-headedness, dizziness, fatigue, cold sweat, blurred and fading vision or "sounds coming from a distance"
- Situational syncope occurring during or immediately after micturition, defecation, coughing, sneezing, laughing, or playing brass instruments.

Orthostatic hypotension:
- Syncope occurring in the standing position and documentation of orthostatic hypotension within 3 min of upright posture during the physical examination.

Arrhythmic syncope
- Persistent sinus bradycardia <40 bpm
- Repetitive sinoatrial block or sinus pauses >3 s
- Atrioventricular block Mobitz II second degree or third degree
- Alternating left and right bundle branch block
- Ventricular tachycardia or supraventricular tachycardia
- Non-sustained episodes of polymorphic ventricular tachycardia
- Long or short QT interval
- Pacemaker or ICD malfunction with cardiac pauses.

(Kapoor et al., 1983; Eagle et al., 1985; Kapoor, 1990; Sarasin et al., 2001; Del Rosso et al., 2008).

3. Diagnostic algorithm for syncope

The first step in assessing a patient with TLOC is to exclude a non-syncopal event. In these cases, TLOC is not related to temporary global cerebral hypoperfusion, as occurs in syncope, but is due to other causes such as epilepsy or psychogenic pseudo-syncope. A corroborative witness history is important to establish, particularly when the patient has minimal recollection of the events surrounding syncope. Fig. 1 is a diagnostic flowchart to guide management of patients presenting with TLOC. The most common cause of TLOC is neurally mediated syncope (NMS) with a particularly high prevalence between 10 and 30 years of age, compared to the rarer differential diagnoses of TLOC including psychogenic pseudosyncope (6%) and epilepsy (3.5%) (van Dijk et al., 2008). It is important to be aware of clinical features that can be used to differentiate between these diagnoses, to focus the work-up strategy for TLOC.

3.1. Differentiating syncope from epilepsy

There are several aspects in the history that differentiate epilepsy from syncope; some history based questionnaires showed a diagnostic accuracy of 96% with a sensitivity of 94% and a specificity of 96% for differentiating syncope from epilepsy (Sheldon et al., 2002). Typical features before, during and after the LOC help to differentiate these two conditions. Before LOC, both syncope and epilepsy can be preceded by prodromal symptoms; in syncope, vagal symptoms such as sweating, nausea, light-headedness and dizziness predominate, while in epilepsy the common features are feelings such as "déjà vu", abdominal discomfort, unpleasant taste or smell. During collapse in epilepsy, the patient is usually stiff with a hypertonic muscular tone while in syncope, muscular tone is usually hypotonic. Both epilepsy and syncope can result in clonic movements but in epilepsy they usually start before or at the moment of the LOC, are generally symmetric, synchronous and can last several minutes. On the other hand in syncope, clonic movements are secondary to severe hypoxia and occur after the onset of LOC, are generally asymmetric, asynchronous and last only a few seconds. During an epileptic crisis patients appear cyanotic, and can suffer from respiratory pauses and lateral tongue biting, while in syncope they appear pale with normal respiration. Tongue biting is not commonly seen in syncope, and if present, usually affects the tip of the tongue. Both syncope and epilepsy can cause urinary incontinence.

The state of the patient after recovery is also important; in syncope the patient is alert, well orientated but often feels weak, while in epilepsy the patient presents with a post-ictal period usually lasting more than 5 min characterized by confusion, drowsiness and sometimes transient focal neurologic signs. A neurology referral is recommended following LOC suggestive of epilepsy, and patients would then normally undergo brain imaging, and an electroencephalogram. However, the indiscriminate use of these neurological investigations in a patient with undifferentiated TLOC is expensive and low-yield.

Table 2
ECG abnormalities in a patient with syncope which may indicate high risk of sudden cardiac death.

- LBBB or RBBB combined with left anterior or posterior fascicular block
- Sinus bradycardia (<50 bpm or sinoatrial block in the absence of negative chronotropic medications or physical training)
- Extensive Q waves indicating previous large myocardial infarction
- Pre-excited QRS complex (i.e. presence of an accessory pathway)
- Prolonged or short QT interval
- RBBB pattern with ST-elevation in leads V1–V3 (Brugada pattern)
- Negative T waves in right precordial leads, epsilon waves, and ventricular late potentials suggestive of ARVD

3.2. Differentiating syncope from psychogenic pseudo-syncope (PPS)

The second most common cause of non-syncopal TLOC is psychogenic pseudo-syncope (PPS) (Raj et al., 2014). It is often not easy to differentiate PPS from syncope, and these patients generally receive many tests before a final diagnosis is made. The history is essential, and there may be history of previous psychiatric or psychological problems. Studies have shown that these patients are often young women, more symptomatic and had more disability compared with other patients who have unexplained syncope. Despite this high frequency, trauma is rarely reported (Linzer et al., 1990a). Episodes are usually witnessed, the TLOC is sudden and the duration can be longer than other causes of syncope. Moreover pre-syncopal episodes are also commonly reported (Tannemaat et al., 2013).

The investigation of choice for PPS is video-electroencephalography (EEG), with continuous blood pressure and ECG monitoring. Reproduction of typical clinical symptoms in the absence of significant EEG or hemodynamic changes confirms the diagnosis. When it is not possible to perform this test, or if this test is negative, other investigations for PPS include: hyperventilation test, head-up tilt test (HUTT) and continuous ECG monitoring. In the first two tests, it is possible to reproduce the episode of TLOC under medical supervision while the third proves the absence of significant arrhythmia during the spontaneous clinical events. In the hyperventilation test, the patient is asked to hyperventilate, which can induce TLOC with a predictive value of 59% for psychiatric causes (Koenig et al., 1992).

The prevalence of PPS amongst patients undergoing HUTT is 5.4% (Tannemaat et al., 2013), with up to 81% of patients suffering from PPS being identified on HUTT, likely due to typical clinical symptoms provoked by the emotional stress caused by the test in the absence of hemodynamic changes (Zaidi et al., 1999).

4. Investigations for syncope

4.1. History taking

History taking should be considered *the most important diagnostic tool* in evaluating patients with syncope. The clinical consultation should be conducted in a suitable environment with sufficient time for an open and trusting physician–patient rapport to be established. The physician concerned should be able to attend to the patient with undivided attention, to ascertain events and symptoms occurring immediately before, during and after each attack, and be able to probe psychosocial aspects of the history in an open-ended manner. This allows a complete clinical picture to be obtained and processed within a clear syncope diagnostic framework, to identify the cause of syncope where