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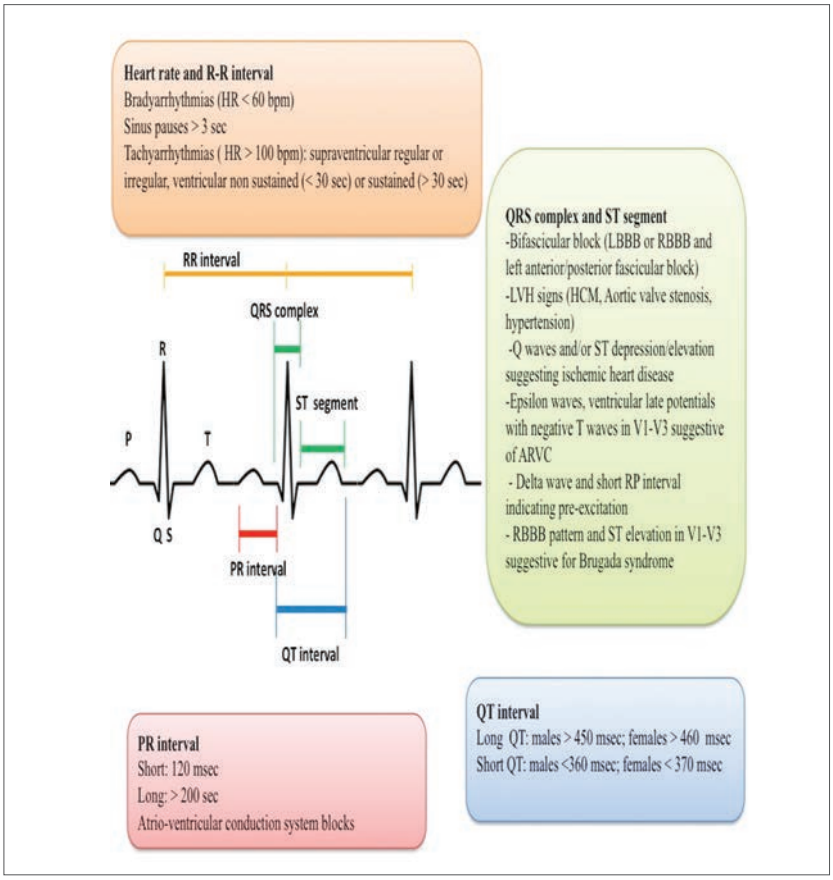


FIGURE 1
ECG features suggesting cardiac syncope

What are the causes of cardiac syncope?

How should patients be investigated?

How should diagnosis be confirmed?

SYNCOPE IS DEFINED AS A TRANSIENT LOSS OF CONSCIOUSNESS (T-LOC) AND POSTURAL TONE DUE to global cerebral hypoperfusion with rapid onset, brief duration and prompt spontaneous recovery.[1,2]

It is a relatively common clinical entity with 40% of people experiencing a syncopal event during their lifetime and accounts for 1% of emergency department attendances and 6% of hospital admissions.[3,4] The incidence increases with age, with two peaks in the second and eighth decades.

Syncope is often a source of anxiety and may lead to injuries.[5] Up to 13% of patients have recurrent syncope with a reported detrimental impact on quality of life similar to chronic conditions such as low back pain.[6] Moreover, the prognosis varies greatly with the diagnosis: ranging from benign in reflex syncope to life-threatening in those with structural heart disease.[2]

The assessment of syncope is built around identification of the small minority in which it is a marker of an underlying pathology that has a risk of sudden cardiac death, while reassuring the vast majority who have a benign cause.

CLASSIFICATION OF SYNCOPE

Syncope can be classified into three primary groups: reflex (neurally mediated), orthostatic hypotension and cardiac syncope.

Identifying the underlying cause of syncope is often challenging, but it is important to differentiate it from other causes of T-LOC which can be neurological (epileptic seizures, subarachnoid haemorrhage and migraine), metabolic (hypoglycaemia, hypoxia and hyperventilation) or psychogenic.

Reflex syncope is the most common cause of syncope, accounting for 50% of all episodes of T-LOC. Its hallmark is a precipitating event or trigger which elicits a centrally mediated response through withdrawal of sympathetic tone resulting in vasodilatation and hypotension (vasodepressive reflex), an increase in vagal tone with bradycardia and asystole (cardioinhibitory reflex) or a combination of both (mixed).

The main types of reflex syncope are: vasovagal, situational and carotid sinus hypersensitivity.[7] Vasovagal syncope occurs typically in young patients and the trigger is either emotional (e.g. the sight of blood or stress) or physical (e.g. pain or prolonged standing) leading to an abrupt reduction in blood pressure (BP) with or without bradycardia. Syncope is usually preceded by nausea, sweating, visual changes (typically tunnel vision) and dizziness. However, in as many as a third of patients, particularly the elderly, reflex syncope occurs without a prodrome.

In carotid sinus hypersensitivity there is an augmented vagal response to »

stimulation of the carotid sinus. Typical triggers include head turning, neck extension, shaving and tight neckwear. It is a common finding in the asymptomatic elderly patient and up to 25% of patients following a fall. Thus, diagnosis must only be made if symptoms occur during carotid sinus massage.

Situational syncope is characterised by distension of mechanoreceptors in the bladder (micturition), rectum (defecation) or oesophagus (swallowing).[8]

In orthostatic hypotension, blood pooling in the lower extremities upon standing leads to a reduction in venous return and cardiac output. Defined as a drop of at least 20 mmHg in systolic BP or 10 mmHg in diastolic BP within 3 minutes of standing, it typically occurs in elderly patients due to a combination of polypharmacy with vasoactive/diuretic drugs, reduced baroreceptor sensitivity, renal sodium wasting and impaired thirst.

'Cardiac syncope accounts for 10% of presentations. The incidence rises significantly with age'

Cardiac syncope accounts for 10% of presentations. The incidence rises significantly with age. A drop in cardiac output and subsequent cerebral hypoperfusion results from either an arrhythmia (tachycardia or bradycardia) or from an anatomical obstruction to cardiac output (aortic stenosis, pulmonary embolism, hypertrophic cardiomyopathy), see table 1, below. Syncope may occur at rest or supine with arrhythmia or on exertion with obstructive causes.

Table 1

Causes of cardiac syncope

Anatomic
- Aortic stenosis
- Atrial myxoma
- Cardiac tamponade
- Hypertrophic cardiomyopathy
- Myocardial ischaemia
- Pulmonary embolism

Arrhythmias
Bradyarrhythmia: Sinus node dysfunction, atrioventricular block
Tachyarrhythmia: Ventricular and supraventricular tachycardias

INITIAL EVALUATION

Establishing a definitive cause can be challenging as syncopal episodes are infrequent, unpredictable, brief and often unwitnessed. Physicians can rarely examine patients or capture the underlying rhythm during a syncopal event unless there are reproducible triggers.

Assessment should therefore be systematic: a thorough history, physical examination including lying and standing BP measurements and 12-lead electrocardiogram (ECG).

The European Society of Cardiology (ESC) 2018 syncope guidelines propose a framework on how to evaluate patients following an episode of T-LOC.[2]
- Establish if the episode of T-LOC was due to syncopal or non-syncopal causes
- Identify the underlying cause through accurate history of events before, during and after the episode of T-LOC (see table 2, below)
- Risk stratify patients into low- and high-risk groups to inform decisions regarding admission to hospital and further investigations (see table 3, p17).

The medical history and physical examination are key components which, when used in combination, can distinguish a syncopal event from other causes of T-LOC in 60% of cases and identify the probable cause of syncope in a quarter of cases.[9,10]

Every effort should be made to obtain a history from a witness if available.

In addition to a comprehensive history of the events surrounding the syncope, the medical history should focus on:
- Identifying and characterising any prior presyncopal or syncopal episodes
- Personal or family history of cardiac disease and sudden cardiac death.

Conditions with a higher risk profile include ischaemic heart disease, conduction disorders, hypertrophic cardiomyopathy, and inherited cardiac conditions such as Brugada syndrome and long QT syndrome.

Table 2

Key features in the medical history when assessing patients with episodes of T-LOC (Adapted from Saklani et al)[5]

Circumstances
Vasovagal: Classical triggers include pain, medical interventions, prolonged standing in warm environments, post-exertion
Situational: Cough, sneeze, micturition and defecation
Carotid sinus syncope: Head rotation, extension, shaving
Orthostatic: Shortly after standing, worse in the morning and after meals, recent introduction of new medications, prolonged standing in warm environments
Cardiac: Rest, supine or on exertion. Known history of structural heart disease or family history of sudden cardiac death. Provocation by sudden noise suggests long QT

Prodrome
Vasovagal: Long prodrome (> 5 sec), consistent in the same individual. Typically, nausea, diaphoresis, light-headedness and blurred vision. May be absent in the elderly
Cardiac: Shorter prodrome (< 5 sec) or absent. Palpitations or chest pain may be present but non-specific
Seizures: Sudden onset or brief aura (olfactory, gustatory or déjà vu)

Observations during T-LOC
Syncope is characterised by brief episodes of T-LOC generally < 5 minutes and usually < 30 seconds. Incontinence and brief clonic movements may occur
Vasovagal: Pallor, diaphoresis, dilated pupils, low blood pressure
Cardiac: Cyanosis but not pallor
Seizures: Prolonged syncope (> 5 minutes), cyanosis, tongue biting, horizontal ocular deviation, incontinence more likely, frothing at the mouth, tachycardia and high blood pressure, tonic-clonic movements
Psychogenic: Atypical features with bizarre movements and resisting attempts to open eyes

Recovery
Vasovagal: Quick recovery but prolonged fatigue is common (often hours)
Cardiac: Quick recovery without any residual symptoms in tachyarrhythmias and bradyarrhythmias
Seizure: Post-ictal confusion and transient neurological deficit

- Identifying medication that may cause or exacerbate symptoms

A general examination may provide clues to the aetiology and uncover complications such as laceration, fractures and injuries. A complete cardiovascular examination is recommended to identify any features suggestive of structural heart disease, particularly abnormal rates or rhythms and heart murmurs. Neurological assessment for focal deficits should also be undertaken.

During examination it is essential that supine BP is measured, followed by standing BP every minute for at least 3 minutes to uncover orthostatic hypotension. Several studies report a high prevalence of delayed orthostatic hypotension and, if history suggests symptoms occurred only after several minutes, standing measurements should continue for at least 10 minutes to unmask a delayed drop in BP.[11,12]

Although an ECG has a low diagnostic yield, it can identify several abnormalities such as conduction disturbances, arrhythmias, ischaemic changes, pre-excitation and prolonged QT interval (see figure 1, p15) and must be performed routinely.[7] In contrast, a 'normal' ECG has a very high negative predictive value (> 99%) for a serious underlying cardiac cause and provides reassurance.

A broad panel of blood tests is commonly requested but there is often little added diagnostic value or data to support their use. Instead, they should be targeted either to exclude or confirm a diagnosis, for example, if the patient is hypoxic and has chest pain there is value in adding a D-dimer. A routine transthoracic echocardiogram is also not recommended unless initial assessment suggests structural heart disease or there is a known cardiac disorder.

Finally, carotid sinus massage should be performed routinely in patients 40 years or older with syncope of unknown origin. The left and right sides are sequentially assessed in supine and sitting positions by a firm massage for 5-10 seconds with continuous ECG and BP monitoring. A positive response (>3 second pause and/or a > 50 mmHg drop in systolic BP) and reproducible syncope is diagnostic of carotid sinus syncope.[1,2] Contraindications include the presence of a carotid bruit, TIA or stroke within the past 3 months and a > 70% carotid stenosis on ultrasound.[13] If carotid examination is normal the chance of carotid sinus massage causing a transient ischaemic attack (TIA) or stroke is very low (0.14-0.28%).[13,14]

Table 3

Diagnostic criteria with initial evaluation (Adapted from the ESC syncope guidelines)[2]

Dagnosis	Typical features
Classical vasovagal syncope	Typical precipitating events (fear, severe pain, emotional distress, or prolonged standing) associated with typical prodromal symptoms
Situational syncope	Syncope during or immediately after urination, defecation, coughing or swallowing
Orthostatic hypotension	Syncope or presyncope with: ● ≥ 20 mmHg drop in systolic BP or systolic BP < 90 mmHg
Cardiac ischaemia-related syncope	ECG demonstrates ongoing ischaemia or previous myocardial infarction
Arrhythmia-related syncope	ECG demonstrates the following: ● Bradyarrhythmias: sinus pause > 3 seconds, persistent bradycardia < 40 bpm, 2nd degree Mobitz type II or 3rd degree AV block ● Alternating bundle branch block ● Tachyarrhythmias: Ventricular tachycardia or rapid SVT

CONFIRMING DIAGNOSIS

Following initial evaluation, the diagnosis may be graded as likely/confirmed, probable, or remain uncertain. The ESC guidelines have set out criteria to aid diagnosis, see table 3, below.[2]

It is not uncommon for the cause of syncope to remain unknown despite several investigations. In 36% of patients with syncope in the Framingham Heart substudy the aetiology remained unexplained following investigations.[7] With greater access to specialised syncope units the number of unexplained syncopal episodes has dropped significantly and can be as low as 10%; many studies report concomitant reduction in hospital admissions and overall investigations.[15] The frequency, severity and presence of high-risk features will dictate further diagnostic and treatment strategies.

RISK STRATIFICATION

Ten percent of patients presenting with syncope to the emergency department will have a serious event within the next 30 days and the mortality at one year is 1%.[2] Thus, if a diagnosis remains elusive despite a comprehensive initial evaluation, risk stratification is warranted.

Several risk scores have been put forward to help inform decision making and identify those at greater risk for adverse outcomes by mainly combining ancillary and biochemical tests with vital signs and medical history.[16-18] The ESC guidelines propose a simplified risk stratification tool based on medical history, syncope characteristics, physical examination and ECG (see table 4, p18).[2]

Low-risk features are most commonly present in reflex syncope; the prognosis is benign and patients can be reassured and discharged.[1,2,7] Indeed, unnecessary admission is harmful and a burden to the healthcare system.[15,19]

Conversely, high-risk features are more likely to be due to an underlying cardiac condition. One year mortality following cardiac syncope may be up to 30% and therefore requires rapid and intensive investigations.[20]

'One year mortality following cardiac syncope may be up to 30% and therefore requires rapid and intensive investigations'

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key points

SELECTED BY

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Syncope is defined as a transient loss of consciousness (T-LOC) and postural tone due to global cerebral hypoperfusion with rapid onset, brief duration and prompt spontaneous recovery. Around 40% of people experience a syncopal event during their lifetime. Syncope accounts for 1% of emergency department attendances and 6% of hospital admissions. The incidence increases with age, with two peaks in the second and eighth decades. Syncope can be classified into three primary groups: reflex (neurally mediated), orthostatic hypotension and cardiac syncope. Reflex syncope is the most common cause of syncope, accounting for 50% of all episodes of T-LOC.

Cardiac syncope accounts for 10% of presentations. Incidence rises significantly with age. A drop in cardiac output and subsequent cerebral hypoperfusion results from either an arrhythmia (tachycardia or bradycardia) or from an anatomical obstruction to cardiac output (aortic stenosis, pulmonary embolism, hypertrophic cardiomyopathy). Syncope may occur at rest or supine with arrhythmia or on exertion with obstructive causes.

Assessment should be systematic: a thorough history, physical examination including lying and standing BP and 12-lead ECG. The medical history and physical examination are key components which, when used in combination, can distinguish a syncopal event from other causes of T-LOC in 60% of cases and identify the probable cause of syncope in a quarter of cases.

Although an ECG has a low diagnostic yield, it can identify several abnormalities such as conduction disturbances, arrhythmias, ischaemic changes, pre-excitation and prolonged QT interval and must be performed routinely. A 'normal' ECG has a very high negative predictive value (> 99%) for a serious underlying cardiac cause. A routine transthoracic echocardiogram is not recommended unless initial assessment suggests structural heart disease or there is a known cardiac disorder.

Prolonged ECG monitoring should be employed when there is a high pretest probability of capturing an arrhythmia. The value lies in correlating the heart rhythm with the presence of symptoms. It can therefore be diagnostic but also confidently rule out arrhythmic aetiology if no abnormalities are observed during typical episodes. Monitors should be selected according to the frequency of syncope and the likelihood of recording the events.

If following initial assessment cardiac syncope is suspected patients should be referred for urgent specialist cardiovascular assessment or emergency department admission. NICE recommends referral within 24 hours in patients with T-LOC and any of the following features: any ECG abnormality; heart failure; heart murmur or dyspnoea; syncope on exertion; family history of sudden cardiac death. The treatment of cardiac syncope is mainly directed at the underlying cardiac disease.

ADDITIONAL TESTING AND INVESTIGATION

Additional investigations should be tailored to suspected aetiology. It is important to bear in mind that in many cases, especially in the elderly, syncope is multifactorial, arising from an interaction of various coexisting conditions, rather than a single cause and results of a test must be interpreted with caution.

Tilt testing is an orthostatic challenge to assess susceptibility for vasovagal response and determine the haemodynamic pattern (cardioinhibitory, vasodepressive, mixed). It is a safe procedure but has suboptimal reproducibility, sensitivity and specificity.[1,2] Importantly, a positive response only demonstrates a tendency for vasovagal response, also known as hypotensive susceptibility, and does not imply causality. Indeed, one study reported that around half of patients with cardiac syncope have a positive tilt test.[21] Thus, correlation with presenting symptoms is paramount to reduce false-positive rates and it is more appropriate in patients with suspected reflex syncope or orthostatic hypotension.[1,2] Indeed, in both the American and European guidelines the recommendations on tilt testing have been downgraded.

Exercise testing should be performed under close supervision in patients presenting with exertional presyncope or syncope. This stress test may unmask arrhythmias or ischaemia, however, postexertional syncope may also be observed which is most commonly reflex-mediated.[22]

Prolonged ECG monitoring should be employed when there is a high pretest probability of capturing an arrhythmia. The value lies in correlating the heart rhythm with the presence of symptoms. It can therefore be diagnostic but also confidently rule out arrhythmic aetiology if no abnormalities are observed during typical episodes. Monitors should be selected according to the frequency of syncope and the likelihood of recording the events.

The new generation of ambulatory monitor, such as the Zio™ XT patch, uses a water-resistant adhesive patch with a 1-lead ECG which reduces artefacts. After completing the 14 days of monitoring, patients send the device by post to the company where it is

Table 4

Low-risk and high-risk features (Adapted from the ESC syncope guidelines)[2]

Low-risk features	High-risk features
Syncope	**Syncope**
● Features suggestive of vasovagal syncope or orthostatic hypotension	● New onset of chest pain, dyspnoea, abdominal pain or headache ● Preceded by palpitation ● Supine or exertional
Past medical history	**Past medical history**
● Long history of similar syncope ● Structurally normal heart	● Severe structural heart disease or coronary artery disease
Physical examination	**Physical examination**
● Normal	● Unexplained systolic BP < 90 mmHg ● Persistent bradycardia (< 40 bpm) ● Undiagnosed systolic murmur ● Severe injury
ECG	**ECG**
● Normal	● Ischaemic changes ● Atrial fibrillation with slow ventricular response ● Mobitz type II, 3rd degree atrioventricular block ● Ventricular tachycardia ● Bundle branch block, intraventricular conduction delay, left ventricular hypertrophy ● QTc > 460 ms ● Brugada type I pattern

analysed, and a report generated. The Zio™ is smaller, more comfortable and has higher compliance and detection rates than the Holter.[23, 24]

Implantable cardiac monitors (ICM) are small injectable devices with a battery longevity of 3 to 5 years. Several studies demonstrate a higher diagnostic yield when compared with conventional measures and, although more expensive, they are cost effective.[25,26] An ICM is indicated in recurrent syncope of unknown origin or syncope with high-risk features and likely arrhythmic cause.

The role of electrophysiology studies (EPS) has diminished due to expanding non-invasive technology. A positive EPS can uncover an arrhythmic aetiology, but a negative test does not exclude it. Currently indications include unexplained syncope despite investigations in patients with prior myocardial infarction or bifascicular block on a 12-lead ECG.[2]

MANAGEMENT

The cornerstone of management in reflex syncope is reassurance and lifestyle modifications: avoidance of triggers, stopping hypotensive medications and increasing hydration and salt intake. Pharmacological treatments may be considered in selected cases. Permanent pacing is almost never indicated, the exception being profound cardioinhibitory reflex syncope with injury.

Drug-induced autonomic failure is the main culprit in orthostatic hypotension. A careful review of the medications with elimination of the offending drugs, education and adequate hydration and salt intake are all helpful in most cases.

Patients who present with no high-risk features, and have a clear or probable diagnosis of vasovagal syncope, orthostatic hypotension or situational syncope have an excellent prognosis and need reassurance.[2,27]

If following initial assessment cardiac syncope is suspected, patients should be referred for urgent specialist cardiovascular assessment or emergency department admission. NICE recommends referral within 24 hours in patients with T-LOC and any of the following features:[27]

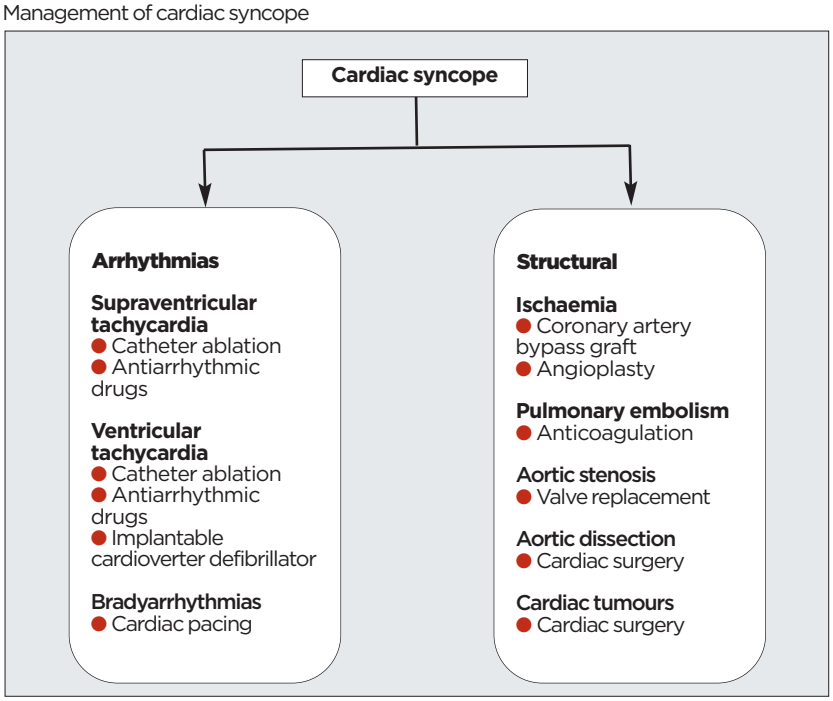
- Any ECG abnormality
- Heart failure
- Heart murmur or dyspnoea
- Syncope on exertion
- Family history of sudden cardiac death

The treatment of cardiac syncope is mainly directed at the underlying cardiac disease (see figure 2, below).

Potential reversible causes of arrhythmias (drugs, electrolyte imbalances) should be investigated in the first instance and treated if present.

Critical obstruction such as severe aortic stenosis or hypertrophic cardiomyopathy may be amenable to surgical intervention. Cardiac pacing is recommended for treatment of bradycardia-induced syncope resulting from significant conduction disease.

FIGURE 2
Management of cardiac syncope



Catheter ablation and drugs are potential treatment options for tachycardia-induced syncope.

Implantable cardioverter defibrillator therapy might be indicated in the case of ventricular tachycardia with structural or electrical heart disease and in the case of unexplained syncope (suspected as arrhythmic) in patients with high-risk conditions including severely depressed left ventricular systolic function (LVEF < 35%), hypertrophic cardiomyopathy, Brugada syndrome and long QT syndrome.

The goal of treatment of syncope secondary to structural heart disease is treatment of the underlying disease with reduction of the associated risk of death.

Specialist input is also warranted for recurrent traumatic syncope or frequent episodes which cannot be controlled by simple avoidance of triggers. If epilepsy is suspected after initial assessment patients should be reviewed by neurology within two weeks.

DRIVING

T-LOC while driving can cause injuries to the driver and others. In a large Danish cohort, the incidence of road traffic accidents almost doubled in patients with syncope.[28] Patients should be counselled regarding driving restrictions they may incur depending on the nature of their syncope and driving licence category. Patients have a duty to notify the DVLA. If the aetiology of syncope is unclear, they should not drive until specialist assessment is undertaken. Extensive recommendations are available on the DVLA website, (see useful information box, p20).

Competing interests: Professor Tim Betts has received speakers' honoraria from Pfizer, Boston Scientific and Abbott Medical. Dr Andre Briosa e Gala has no competing interests

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Useful information

Arrhythmia Alliance
www.heartrhythmcharity.org.uk

STARS (Syncope Trust And Reflex anoxic Seizures)
www.stars.org.uk

DVLA guidance for medical professionals
www.gov.uk/government/publications/assessing-fitness-to-drive-a-guide-for-medical-professionals

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