

An approach to the evaluation and management of syncope in adults

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Cohort and population based studies suggest that around 40% of the adult population has experienced a syncopal episode (usually described as a “faint” or “blackout”),^{1 2 w1 w2} with women more likely than men to report such an episode.^{w1-3} The incidence is higher with advancing age,^{2 w3} and this trend coincides with the increase in prescription of vasoactive drugs and increasing incidence of cardiac arrhythmia in the elderly population. Although comprehensive reviews and guidelines are available for specialists who care for patients referred with syncope,^{3 4 w4 w5} guidance for the non-specialist is sparse. This review provides guidance for generalists who encounter patients who have had transient loss of consciousness that may be syncopal in nature. It is based on cohort studies, randomised controlled trial evidence, and expert consensus guidelines.

“Collapse query cause”: did my patient have syncope?

Patients with transient loss of consciousness often present non-specifically with an episode of collapse. The most common cause of such a presentation is syncope—rapid onset loss of consciousness of short duration as a result of global cerebral hypoperfusion with loss of postural tone, which is followed by spontaneous and complete recovery.^{3 4} A syncopal episode typically lasts around 20-30 seconds and almost invariably less than five minutes, although more prolonged episodes are occasionally recorded.^{w6}

What can cause syncope?

Table 1 provides a detailed list of the causes of non-cardiac syncope, with commonly used synonyms for clarity, because the terminology used can be confusing and misleading. The most common causes of syncope in all age

SOURCES AND SELECTION CRITERIA

We searched Medline, PubMed, and Cochrane databases for English articles whose titles included the keywords “syncope”, “vasovagal syncope”, “orthostatic hypotension”, “tilt table test”, “cardiac arrhythmia”, or “sudden cardiac death”, with and without the limits “meta-analysis”, “review”, and “randomised controlled trial”. We also consulted recent international guidelines on syncope (including the specialist societies the European Society of Cardiology and the American Heart Association/American College of Cardiology Foundation), and reviewed our personal archives and relevant current textbooks.

groups are neurally mediated disorders (fig 1). These are benign conditions with no associated increase in mortality that are characterised by vasodilatation or vagally driven bradycardia or asystole, which cause profound systemic hypotension and consequent dizziness, presyncope, and syncope. Other causes include orthostatic hypotension, cardiac arrhythmias, ischaemia, structural heart disease, cardiopulmonary abnormalities including pulmonary embolism, and steal syndromes.³ The frequency with which these disorders cause syncope is age dependent—for example, cardiac causes, which are more serious, are rare in younger people but underlie a third of events in older patients (fig 1).⁵

What are the costs of syncopal episodes?

The event rate for a syncopal episode recorded in the general population is 1.9-4.3 times higher than in general practice populations and 13 times higher than the number of cases presenting to emergency departments, which suggests that not all people who experience an event present to medical practitioners.^{1 6 w7} Around 50% of middle aged and elderly patients with syncope present to medical services,² whereas the percentage is much lower (around 10%) in the teenage and young adult group.¹ Nevertheless, transient loss of consciousness accounts for around 1% of all emergency department attendances.⁶ Costs per diagnosis range from £611 (€693; \$956) in the UK⁷ to £1037 in Spain,⁸ whereas the annual healthcare costs of syncopal episodes to the United States have been estimated at \$2.4bn.⁹ Obviously costs are not limited to financial considerations. A Dutch study of 385 patients with transient loss of consciousness found statistically

SUMMARY POINTS

Syncope is common in all age groups, and it affects 40% of people during their lifetime
Few people with syncope seek medical attention
Neurally mediated syncope, which is benign, is the most common cause
Cardiac syncope as a result of arrhythmias or structural cardiopulmonary disease is more common with increasing age
Cardiac syncope is associated with increased mortality and must be excluded
Brain imaging, carotid Doppler ultrasound, electroencephalography, and chest radiography are not needed in patients with syncope

Table 1 | Classification and synonyms of non-cardiac syncope

Classification of syncope	Synonyms and suggested diagnoses
Neurally mediated syncope:*	Reflex syncope, neurocardiogenic syncope, neurocardiovascular syncope, neurogenic syncope
Vasovagal syncope	Simple faint, swoon, vasovagal attack, vasodepressor syncope, reflex anoxic seizures
Situational syncope	
Needle or blood phobia	
Respiratory (cough, sneeze) syncope	Cough syncope
Urinary (micturition) syncope	Micturition syncope, postmicturition syncope
Gastrointestinal (defecation, swallowing, visceral pain) syncope	Defecation syncope, deglutition syncope, swallow syncope, syncopal dysphagia
Postexertional syncope	Exercise syncope
Postprandial syncope	Postprandial hypotension
Laughter syncope	Laugh or laughter induced syncope, gelastic syncope
Valsalva induced (for example, weightlifting, brass instrument playing) syncope	Weightlifter's syncope; trumpet blower's syncope
Carotid sinus syndrome	Carotid sinus syncope
Carotid sinus hypersensitivity with syncope	
Orthostatic hypotension:	Postural hypotension
Primary autonomic failure	Pure autonomic failure, Bradbury-Eggleston syndrome, idiopathic orthostatic hypotension, multiple systems atrophy, Shy-Drager syndrome, Parkinson's plus syndromes
Secondary autonomic failure	Autonomic neuropathy (for example, diabetic, alcohol related, and amyloid)
Volume depletion	Diuretics, dehydration, haemorrhage, Addisonism

*All may have vasodepressor, cardioinhibitory, and mixed components.

significant impairment in quality of life on generic and disease specific measures equivalent to that seen in other long term conditions, including epilepsy^{w8} and rheumatoid arthritis.^{w9}

How should people who have had transient loss of consciousness be assessed?

The guiding principle of assessment is to differentiate syncope from other causes of transient loss of consciousness and the more benign causes of syncope from the potentially sinister. In practice, this means differentiating non-cardiac syncope from cardiac syncope. Cardiac syncope carries a high mortality in all age groups. The Framingham study cohort's age and sex adjusted hazard ratios for death over a mean follow-up of 8.6 years was 2.4 (95% confidence interval 1.78 to 3.26) for cardiac syncope compared with 1.17 (0.95 to 1.44) in the "vasovagal group," which included orthostatic hypotension.² Orthostatic hypotension has a lower although important effect on morbidity and mortality because of its underlying causes.¹⁰

Initial assessment

A detailed history and examination complemented by 12 lead electrocardiography and lying and standing blood pressure (box 1) is the mainstay of evaluating patients with syncope, and it can provide an initial diagnosis in 66% of cases, with a diagnostic accuracy of 88%.¹¹ In one study that compared 191 patients with suspected or certain heart disease with 146 patients with no heart disease, this approach alone excluded a cardiac cause of syncope in 97% of patients.¹² Figure 2 provides an outline of the evaluation of the patient with transient loss of consciousness, with more detailed algorithms readily available.^{w10} Table 2 provides more detail on the relevant components of the history.^{3 4 13 14 w4 w5 w11 w12} The history has the important dual role of helping with diagnosis

and with risk stratification in the emergency setting (see below).

What are the next steps?

In most cases the initial assessment will lead to a definitive or provisional diagnosis, which will inform the need for referral and further investigation. A confident diagnosis of vasovagal syncope or orthostatic hypotension can be made without further investigation if the history is suggestive and cardiac examination and electrocardiography are normal.^{3 4} A patient with orthostatic hypotension may need further diagnostic tests, which should be guided by clues in the systemic inquiry, physical examination, and routine laboratory tests. Patients with cardiac syncope and those with non-cardiac syncope in whom diagnostic and management difficulties exist should be referred to specialist services. A structured approach with strict adherence to guidelines and the presence of a dedicated syncope investigation unit reduce unnecessary investigations and improve the diagnostic yield from 36% to 98%.^{15 w13 w14}

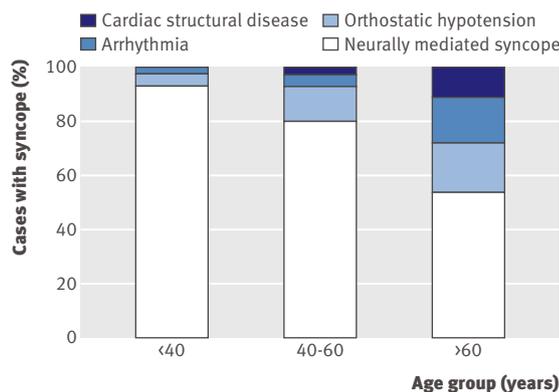


Fig 1 | Causes of syncope by age

Box 1 | How to obtain and interpret lying and standing blood pressure (active stand) measurements^{3,4}

Technique

Lie patient supine (not sitting) for 10 minutes
 Record baseline blood pressure and heart rate
 Stand patient, with help if needed
 Record blood pressure and heart rate at three minutes after standing (if a sphygmomanometer is used, it has been suggested that measurements be taken for each of the three minutes, but continuous non-invasive blood pressure measurement is more useful if frequent measurements are needed)
 Inquire about symptoms
 Measure blood pressure after five minutes of standing if postural orthostatic tachycardia syndrome is suspected
 Passive standing with a tilt table at 60-70° is an accepted alternative

Definition and diagnostic considerations

Orthostatic hypotension is defined by a drop in systolic blood pressure of 20 mm Hg or diastolic pressure of 10 mm Hg within three minutes of standing.

Orthostatic hypotension is considered clinically important if:

- The reduction in blood pressure is sustained at or beyond three minutes
- Original symptoms are reproduced during active or passive standing

Heart rate should rise with standing:

- Excessive rise in heart rate (≥ 30 beats/min) or to a rate of 120 beats/min or more is diagnostic of postural orthostatic tachycardia syndrome
- Lack of heart rate response suggests autonomic failure, rate limiting drugs, or chronotropic incompetence

What red flags should prompt referral for specialist investigation?

Urgent referral or hospital admission for investigation is needed for patients who have chest pain, breathlessness, a history of cardiac disease, family history of sudden death, signs of heart failure, or abnormalities on electrocardiography. The electrocardiography features that suggest cardiac arrhythmias include ventricular tachycardia, a widened QRS complex (>120 ms), sinus bradycardia (<50 beats/min), prolonged or excessively shortened corrected QT interval (>450 ms and <300 ms, respectively), T wave

inversion leads V1-V3, epsilon waves or ventricular late potentials (arrhythmogenic right ventricular dysplasia), and right bundle branch block with ST elevation and T wave inversion in V1-V3 (Brugada syndrome). Patients who report a history of syncope with no warning symptoms (Stokes-Adams attack), syncope during exercise, palpitations preceding syncope, and syncope in the supine position should be investigated by a specialist. People with frequent or injurious syncope or implications for driving also warrant specialist input. Prolonged unconsciousness, confusion after the event, or neurological signs and lateral tongue biting suggest a non-syncope event, and this should prompt neurological evaluation.^{w11}

What are the diagnostic pitfalls?

Misdiagnosing epilepsy

Myoclonic jerks and incontinence may occur during syncope and the episode may be mistaken for a seizure. Several observational studies of prospective and retrospective design have found a tendency to misdiagnose epilepsy in people with underlying syncope; this can have adverse long term consequences, including potentially avoidable deaths.^{14 w11 w12} The pointers for history taking provided in table 2 help differentiate “convulsive syncope” from seizure. Shaking that begins after the onset of loss of consciousness and aching muscles after the episode are more common after seizures than after syncopal episodes.

What syncope is not

Each component of the carefully worded definition of syncope helps differentiate alternative causes of falls, temporary immobility, or transient loss of consciousness from true syncope. The definition excludes, for example, transient ischaemic attack (transient neurological deficit lasting less than 24 hours, but no loss of consciousness); trauma induced loss of consciousness (not related to cerebral hypoperfusion); complex partial seizures (postural tone is maintained); and intoxications and hypoglycaemia (often prolonged, or do not resolve without intervention). Hyperventilation, which is associated with hypocapnia and cerebral vasoconstriction, may result in light headedness and reduced mental function along with characteristic digital and perioral paraesthesia, but it is only rarely associated with loss of consciousness in the absence of an accompanying vasovagal response.

Falls may result from syncope

The diagnosis of syncope may also be missed in people presenting with falls. Older people being evaluated for falls may have experienced brief syncopal episodes that resulted in loss of postural tone but may not have been aware of loss of consciousness.^{16 w15} People with sudden falls who cannot remember or explain the fall, and those with facial and head injuries, may benefit from investigation for syncope.^{17 w16}

How might a specialist further investigate a patient with syncope?

Carotid sinus massage and the head-up tilt table test
 In patients over 45 years with syncope but no suggestion of cardiac disease, five to 10 seconds of bilateral

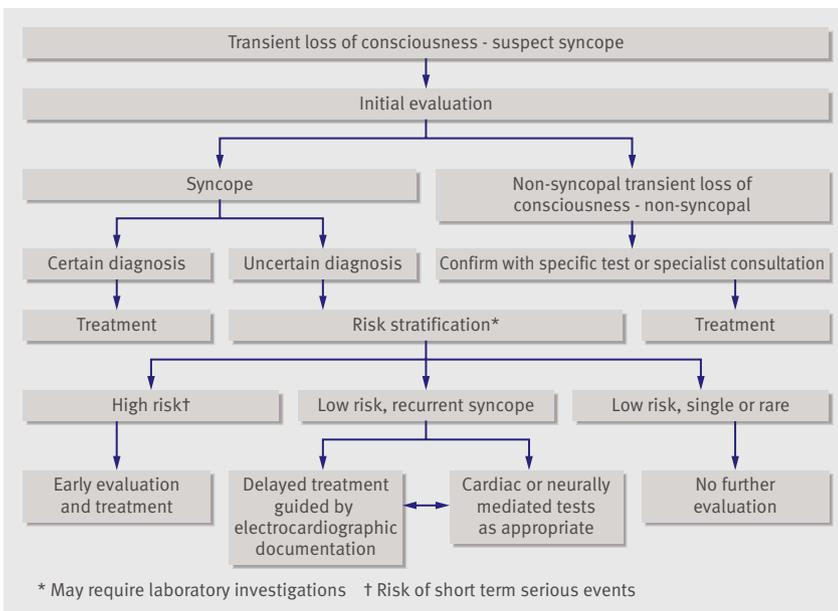


Fig 2 | Diagnostic flowchart in patients with transient loss of consciousness. Supplied, with thanks, by Wouter Weiling

Table 2 | History taking pointers in syncope

Features from history	Suggested diagnosis
The “5Ps”	
<i>Precipitants:</i>	
Warm or crowded environments, pain, emotional distress, fear, exercise, dehydration (as a result of drugs or illness), specific activities (coughing, laughing, micturition, eating)	Vasovagal syncope, orthostatic hypotension, situational syncope
Head movements, tight collars, shaving	Carotid sinus syndrome
During exercise, or no obvious precipitant	Arrhythmia, structural heart disease
<i>Prodrome:</i>	
Light headedness, dizziness, blurred vision	Vasovagal syncope, orthostatic hypotension
Nausea, sweating, abdominal pain	Vasovagal syncope
None	Vasovagal syncope in older people, cardiac syncope
Chest pain, shortness of breath, or no prodrome	Cardiac syncope
Déjà vu, jamais vu	Seizure
<i>Palpitations</i>	Arrhythmia
<i>Position:</i>	
Prolonged standing	Vasovagal syncope, orthostatic hypotension
Sudden changes in posture	Orthostatic hypotension
Supine	Arrhythmia, structural heart disease
<i>Post-event phenomena:</i>	
Nausea, vomiting, fatigue	Vasovagal syncope
Immediate complete recovery	Any cause, common in arrhythmia
Appearance	
Pallor, sweating	Syncope rather than seizure
“Blue”	Seizure
Abnormal movements:	
Minor twitching but floppy while unconscious (myoclonic jerks)	Syncope (any cause)
Rhythmic jerking preceded by rigidity or abnormal posturing	Seizure
Eyes:	
Open	Seizure or syncope
Closed	Pseudoseizure, psychogenic syncope
Mental state:	
Prolonged confusion, retrograde amnesia	Seizure
Transient disorientation	Common in neurally mediated syncope
Amnesia concerning loss of consciousness	Neurally mediated syncope in older people
Other:	
Incontinence	Non-specific, but unusual in syncope
Tongue biting	Seizure
Chronic medical problems:	
Pre-existing heart disease	Cardiac syncope
Diabetes, Parkinson’s disease, Parkinson plus syndromes, alcohol dependence, renal replacement therapy	Orthostatic hypotension
Hypertension	Drug related neurally mediated syncope, orthostatic hypotension
Family history of sudden cardiac death	
	Hereditary long and short QT syndromes, Brugada, arrhythmogenic right ventricular dysplasia, structural heart disease

sequential carotid sinus massage, right sided^{w17} then left, supine and erect (because a third of diagnoses will be made during upright massage^{w18}) is recommended by consensus guidelines early in the investigative pathway.³ Transient ischaemic attack and stroke occur as adverse events in about 1/1000 episodes of massage.⁴ Head-up tilt table testing may be useful in patients with atypical features, recurrent symptoms, injurious events, and driving or occupation related implications, or when a cardiac cause has been excluded.^{3 4}

Echocardiography

Echocardiography is used to determine any underlying structural heart disease when a cardiac cause of syncope is suspected from the initial assessment.^{3 w4 w5}

Ambulatory electrocardiography monitoring

The diagnosis of arrhythmic syncope depends on demonstrating a correlation between symptoms and a rhythm disturbance. An arrhythmic cause of syncope can be safely excluded if electrocardiography is normal during a characteristic episode.^{3 18} Investigation of all patients who experience syncope with ambulatory electrocardiography monitoring has a low diagnostic yield, unless symptoms are frequent. In a case-control study of a prospective versus historical cohort of patients presenting to emergency services with syncope, 24 hour Holter monitoring achieved only one diagnosis in 155 tests in patients with no evidence of heart disease compared with 18 diagnoses in 195 tests in those with established cardiac syncope.⁷ Prolonged external loop recording has a much higher diagnostic yield.¹⁸ In a case-control study, 44 of 78 patients achieved a symptom-rhythm correlation with extended loop recording versus 12 of 55 who had conventional Holter monitoring.¹⁸

Other investigations: what helps and what doesn’t

Depending on the clinical picture, implantable loop recording (with a battery life of up to 36 months),^{19 w19} exercise testing, electrophysiology studies, coronary angiography, and computed tomography pulmonary angiography may be indicated.^{3 w4 w5 w20} Brain imaging and electroencephalography, echocardiography in those with no evidence of structural heart disease, chest radiography, and carotid Doppler studies are not useful in syncope and should be avoided unless specific symptoms or findings indicate their use.^{3 w20}

What risk scores can help identify patients who need referral?

Several risk scores have been developed and validated in recent years in an attempt to identify which patients with syncope are at high risk of adverse events. These include the OESIL (Osservatorio Epidemiologico sulla Sincope nel Lazio) score,²⁰ SFSR (San Francisco syncope rule),²¹ and EGSYS (European Guidelines in Syncope Study) score.²² No score is widely accepted in emergency practice. The simplest, the SFSR, uses the presence of an abnormal electrocardiogram, heart failure, anaemia (haematocrit <30%), or systolic hypotension (<90 mm Hg) to identify patients who require urgent action. In the original validation study, the SFSR had 96% (92% to 100%) sensitivity and 62% (58% to 6%) specificity in identifying patients with short term adverse outcomes.²³ Later studies validating the SFSR in other populations have been less successful—a study that compared the SFSR with assessment by the emergency department doctor found that both identified high risk patients equally well.²¹

How should syncope be treated?

Patients with all forms of syncope require a full explanation of their diagnosis and management plan.

Non-cardiac syncope

Box 2 details the management advice for patients with vasovagal syncope, orthostatic hypotension, and vasodepressor carotid sinus syndrome (>50 mm Hg fall in systolic blood pressure during carotid sinus massage). It

comprises mainly reassurance, advice on how to abort an incipient attack, and advice on how to prevent attacks in the long term.^{24-27 w14 w22 w23} In most patients, these simple measures resolve symptoms or improve them greatly. Permanent pacing in vasovagal syncope is not recommended routinely because of contradictory evidence from clinical trials.²⁸ It is sometimes used, however, in patients over 40 with vasovagal syncope whose attacks have no warning symptoms (so that injury often occurs) and when prolonged asystole has been demonstrated during tilt testing and during a real life episode.³ Orthostatic hypotension is managed in much the same way as vasovagal syncope (box 2), with the addition of abdominal binders and raising the head of the bed during sleep.

Cardiac syncope

Treatment of cardiac syncope is dictated by the underlying cause.

Should patients with syncope drive?

In the United Kingdom, the Driver and Vehicle Licensing Agency (DVLA) offers detailed recommendations for patients with syncope (www.dft.gov.uk/dvla/medical/atag-lance.aspx).²⁹ Patients with unexplained syncope despite intensive investigation are asked to refrain from driving for six months. Those with vasovagal syncope and an adequate prodrome, characteristic provocative factors, and no previous supine or seated syncope have no restrictions. More stringent rules apply to cough syncope and other higher risk symptoms and diagnoses.

New research and unresolved matters

The physiology and pathophysiology of the neurally mediated syndromes is far from clear, although recent work

from our unit has implicated disordered cerebral autoregulation in the aetiology of carotid sinus syndrome.³⁰ There is debate on whether carotid sinus syndrome is a manifestation of ageing rather than a disease process, given its presence in up to 35% of community dwelling older people,³¹ and debate on its response to pacing in older people who fall.^{w24} Positive tests for orthostatic hypotension, carotid sinus hypersensitivity, or vasovagal syncope were seen in 77% of patients with Lewy body dementia and 51% of those with Alzheimer's disease, and the severity of the drop in blood pressure with carotid sinus massage correlated with the degree of deep white matter changes.³² Any causal or consequential associations between neurally mediated syncope and dementia have yet to be established.

The best way to investigate syncope remains to be established, and guidelines need to be streamlined for appropriate risk stratification. The potential of the implantable loop recorder as an early rather than a late investigation tool has yet to be clarified, although the ongoing ISSUE (International Study of Syncope of Unknown Etiology) 3 study may help. The adenosine test (20 mg intravenous adenosine with appropriate electrocardiography monitoring and recording of duration of heart block or asystole) has been used in several centres to identify indications for pacing in people with intermittent bradycardia and vasovagal syncope,^{33 w25} although debate continues on the test's usefulness in clinical practice.

Finally, potential drug treatment for vasovagal syncope and orthostatic hypotension is poorly researched. In particular, alternative drugs to midodrine³⁴ and fludrocortisone³⁵ would be useful for older patients with hypertension, in whom they are relatively contraindicated.

Box 2 | Treating vasovagal syncope, carotid sinus syndrome, and orthostatic hypotension

Vasovagal syncope, orthostatic hypotension, and vasodepressor carotid sinus syndrome

Measures that can help abort an incipient attack (must be done as soon as premonitory symptoms start):

- Sit, preferably on the floor with knees drawn up and head between the legs
- Squat, on haunches if able, or preferably
- Lie with legs raised
- Perform physical counterpressure manoeuvres²⁷ (these can be used to treat and prevent episodes in situations known to induce symptoms):
- Isometric exercises to raise systolic blood pressure
- Leg crossing and gluteal clenching (lower body muscle tensing)
- Sustained hand grip, forearm clenching

Longer term prevention of attacks

Ensure that fluid intake is adequate. In the absence of heart failure or other cause for caution, we routinely suggest 1.5-2 l of non-caffeinated fluid (preferably water) by lunchtime then "enough to keep the urine clear"²⁵

Use compression stockings (for example, Duomed grade II)

Refractory symptoms

Treatment with β blockers is no longer recommended for vasovagal syncope.²⁶ Patients should be referred to a specialist^{w14 w22}

Cardioinhibitory and mixed carotid sinus syndrome*

Insertion of a dual chamber permanent pacemaker²⁴

*Carotid sinus syndrome is defined as asystole for more than three seconds with (mixed) or without (cardioinhibitory) a 50 mm Hg fall in systolic blood pressure during carotid sinus massage with symptom reproduction

ADDITIONAL EDUCATIONAL RESOURCES FOR HEALTHCARE PROFESSIONALS AND PATIENTS

- European Society of Cardiology (www.escardio.org/guidelines-surveys/esc-guidelines/Pages/syncope.aspx)—Downloads available for full text, pocket guide, and PDA versions of 2009 syncope guidelines
- American Heart Association/American College of Cardiology Foundation (<http://content.onlinejacc.org/cgi/reprintframed/47/2/473>)—Scientific statement on the evaluation of syncope
- Driver and Vehicle Licensing Authority (www.dft.gov.uk/dvla/medical/atag-lance.aspx)—At a glance guide to the current medical standards of fitness to drive
- STARS (Syncope Trust and Reflex Anoxic Seizures) (www.stars.org.uk/)—Patient support group and professional resource with links to relevant websites. Blackout symptom checklist for patients, patient information, kids and teens pages (info@stars.org.uk)
- Arrhythmia Alliance: the Heart Rhythm Charity (www.hearrhythmcharity.org.uk/)—UK based cooperative venture between patient support groups and professional groups to raise awareness and help foster good practice in the detection and management of arrhythmia
- CRY (Cardiac Risk in the Young) (www.c-r-y.org.uk/index.htm)—UK based patient support group for younger patients and families at risk of arrhythmias and sudden cardiac death. Includes professional pages and links

TIPS FOR NON-SPECIALISTS

- Syncope is the most common cause of transient loss of consciousness
- Most cases of neurally mediated syncope can be treated effectively with lifestyle modification and medical reassurance
- Transient ischaemic attacks do not present with loss of consciousness
- Diagnosis can often be made after a clinical history, physical examination, 12 lead electrocardiogram, and lying or standing blood pressure measurements
- Twenty four hour ambulatory electrocardiography monitoring has a low yield and will probably diagnose arrhythmias only in patients who have daily symptoms
- Patients with suspected cardiac syncope or atypical neurally mediated syncope (particularly with injury or driving or occupational related implications) need referral to specialist services

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Endpiece

Beware of the expert

“Gentlemen, beware of the expert: by the time he is generally recognised as such, in my experience, he should usually be referred to in the past tense”.

Roger Altounyan (1922-1987)

Submitted by John H Turney, retired physician, Leeds

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