Syncope in older people

Syncope in the elderly population is a complex and often ill understood entity. Therefore, its diagnosis and management can prove to be challenging. This challenge is of particular relevance for the general practitioner, who is frequently called upon to initiate management of patients in the community presenting with “collapse; query cause”. The National Institute for Health and Clinical Excellence has recently published guidelines on the management of transient loss of consciousness (T-LOC).\(^1\) This article is a review of the epidemiology, aetiology, pathophysiology and diagnostic pitfalls of syncope in the elderly.

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Almost 40% of the adult population will have experienced a syncopal episode in their lifetime.\(^2,3\) According to the Framingham study, the incidence of syncope is 6.2 per 1000 person years with a cumulative incidence of 6% over 10 years.\(^2\) Syncope accounts for 3–5% of all accident and emergency presentations and 1–6% of all hospital admissions.\(^4,5\) It entails a large financial burden to the NHS, with diagnosis costing from £611 per patient in the UK.\(^6\) In the US, the annual estimate of expenditure incurred in syncope management is approximately $2 billion.\(^7\)

**Definition**

The term “transient loss of consciousness” (T-LOC) incorporates all conditions presenting with self limiting loss of consciousness irrespective of aetiology. T-LOC can broadly be classified as being traumatic or non traumatic. Traumatic T-LOC or concussion has a clear history of preceding injury and does not usually cause problems with diagnosis. Non-traumatic T-LOC can be due to syncope, epileptic seizures, psychogenic pseudosyncope, and other less common conditions.

Syncope is defined as a sudden and rapid T-LOC due to global cerebral hypoperfusion with loss of postural tone, followed by spontaneous and complete recovery.\(^2,3\) It is the commonest cause of T-LOC. The main feature differentiating syncope from the other causes of loss of consciousness is therefore its underlying mechanism of reversible global reduction in cerebral blood flow. A syncopal event characteristically presents as loss of consciousness for 20–30 seconds and almost invariably less than five minutes, although there are occasional recorded episodes of longer duration.

Syncope is often confused with other conditions presenting with true or apparent loss of consciousness. For example, epilepsy, drug intoxication, metabolic disorders, and posterior circulation transient ischemic attack (TIA) can all masquerade as syncope. They also present with a true loss of consciousness but they have different underlying aetiologies from syncope. Other conditions, such as falls and carotid circulation TIA, can present with collapse without true loss of consciousness. A careful history and clinical examination will usually help to differentiate true syncope from other diagnoses.

**Initial assessment**

A comprehensive history, detailed physical examination, and 12-lead electrocardiogram (ECG) are vital in the diagnostic evaluation of syncope, and it can offer a possible diagnosis in 66% of cases with 88% precision.\(^8\) The initial assessment should be targeted at answering three crucial questions.\(^9\)
1. Is it syncope?
2. What is the probable cause of the episode?
3. Are there any features indicating a high risk of cardiovascular events?

In 23–50% of cases, preliminary assessment is sufficient to provide clues to a possible cause.10,11 The new National Institute for Health and Clinical Excellence (NICE)1 guidance for T-LOC highlight several useful pointers in the history of T-LOC to aid choice of investigations and management options.

**NICE recommends** ascertaining the circumstances in which the event occurred. If LOC was related to exertion, it is vital to elicit whether the syncopal event occurred during exertion (suggesting a cardiac cause) or soon after cessation of exercise (probable vasovagal episode). Other factors important to consider are:

- The individual’s posture immediately before the LOC as syncope occurring supine or sitting would suggest a cardiac cause
- The presence or absence of prodromal symptoms, such as sweating, flushing, nausea, dizziness or lightheadedness, the presence of which would suggest neurally-mediated syncope
- The presence of any tongue biting (suggesting epilepsy) and other associated injuries, with details of site and extent
- The duration of LOC from onset to recovery (duration of LOC with syncope tends to be brief)
- The presence of any confusion during recovery and unilateral weakness, which would suggest the presence of epilepsy

- Whether the patient has a history of LOC; if they do have a history of event, the number and frequency should be elicited.

It is imperative to record any past history of cardiac disease and family history of cardiac disease or sudden cardiac death. For instance, the presence of pallor and sweating suggests vasovagal syncope and eyes shut but resist passive opening may indicate psychogenic syncope.

A collateral account of the history, if available, should always be sought. In particular, the witness/witnesses should be asked about the patient's appearance (eg, whether they were pale, sweaty, or their eyes were open or shut, and whether limb movements were present) and the character and duration of the LOC as these factors can provide vital clues to the diagnosis.

All patients presenting with history of syncope should have vital signs recorded, including pulse rate, respiratory rate, temperature, oxygen saturations and these should be repeated if required. Lying and standing blood pressure should be recorded, especially if orthostatic hypotension is considered a possibility. Blood glucose should be tested if hypoglycaemia is suspected, and other relevant tests such as a sepsis screen, renal function tests, full blood count or clotting should be done if sepsis, dehydration, anaemia or bleeding have been identified as possible underlying causes from the detailed history.

A 12-lead ECG should be performed following a detailed history and clinical examination. Important abnormalities to be established from the ECG are listed in Box 1. The presence of an abnormal ECG, history of heart disease,12 and other “red flag” symptoms indicate a high risk of life-threatening cardiac cause. If a life-threatening cardiac cause is identified as a possibility, NICE advises that patients should be referred for urgent specialist assessment within 24 hours.1 The “red flag” features include syncope during exertion,

### Box 1: “Red flags” on ECG

- Conduction abnormalities such as any degree of heart block, left or right bundle branch block (LBBB or RBBB)
- Long/short QT interval (corrected QT >450 ms or < 350 ms)
- ST segment/ T wave abnormalities
- Persistent bradycardia
- Ventricular arrhythmias including ventricular ectopic beats
- Sustained atrial arrhythmias
- Paced rhythm
- Ventricular pre-excitation
- Brugada syndrome (congenital arrhythmogenic heart disease with RBBB and ST elevation in leads V1-V3).
ongoing shortness of breath, lack of prodromal symptoms, family history of sudden cardiac death, signs/symptoms of heart failure, and the presence of heart murmurs.

**Investigations**

Indiscriminate use of 24-hour Holter monitoring has a low diagnostic yield and should be avoided. In older individuals, rhythm abnormalities of uncertain significance are frequently detected during cardiac monitoring. As a result, this test should only be considered diagnostic if a rhythm abnormality is detected in the presence of symptoms. The use of ambulatory cardiac monitoring should therefore be tailored to the individual's history and frequency of syncopal episodes. 24-hour ECG monitoring is only likely to be diagnostic in individuals with daily symptoms. More prolonged cardiac monitoring, either in the form of external loop recorders or implantable loop recorders, should be considered if symptoms are less frequent. The cost effectiveness of implantable loop recorders has been endorsed by the recent NICE T-LOC guidelines.

Echocardiography should be performed in patients with suspected structural heart disease. This will include the presence of ECG abnormalities, audible cardiac murmurs, family history of sudden death, palpitations and heart failure. Therefore, cardiac imaging has an important role in risk stratification.

Syncope occurring during exercise should raise the suspicion of cardiac syncope. For these patients, NICE recommends urgent exercise testing within seven days. Exceptions to this include conditions where exercise testing may be contraindicated as in suspected aortic stenosis or hypertrophic cardiomyopathy. The results of exercise testing will provide a basis for arranging further investigations as appropriate. These patients should be advised to abstain from carrying out strenuous exercise pending further tests.

Head-up Tilt-Table Testing (HUTT) is the diagnostic test for vasovagal syncope. This is performed using a tilt-table with a footplate usually during non-invasive continuous blood pressure and ECG monitoring. The patient is tilted at 60–70 degrees tilt-angle for 20 to 40 minutes using a number of published protocols. The provocation agents glyceryl trinitrate, isoproterenol or lower body negative pressure are now routinely administered to enhance the sensitivity of the test. HUTT is no longer recommended for individuals with isolated syncopal episodes with classical prodrome and brief periods of loss of consciousness. It should, however, be considered in those with recurrent symptoms, injurious events and atypical features. Older patients are more likely to require HUTT as they are less likely to present with classical symptoms.

Carotid sinus hypersensitivity (CSH) is characterised by exaggerated response to carotid sinus massage (CSM). It is diagnosed when there is asystole lasting >3 seconds (cardioinhibitory CSH), significant hypotension with a fall in systolic blood pressure >50 mmHg (vasodepressor CSH) or a combination of the two in response to CSM. CSM should be performed in a specialist centre with continuous ECG and blood pressure monitoring. The investigation should be avoided in those with recent strokes and known carotid disease due to possibility of neurological complications.

Other tests including routine brain imaging, electroencephalogram (EEG) and carotid Doppler, have a limited role in syncope and are not recommended unless there are other indications for doing so.

**Diagnosis**

**Non-cardiac syncope**

Neurally mediated or reflex syncope is by far the most frequent cause of syncopal events. Vasovagal syncope (VVS), carotid sinus hypersensitivity (CSH) and situational syncope are all classified as neurally-mediated syncope

Vasovagal syncope is the commonest cause of syncope. It can be diagnosed based on history alone. Additional investigations are not required if initial assessment does not suggest any other diagnoses. Typically history should demonstrate presence of the three “P’s”: prodromal symptoms, such as sweating, flushing, and lightheadedness; posture with history of prolonged standing preceding the episode or previous incidences of possible syncope being aborted by sitting or lying down; and provoking factors such as severe pain.

Situational syncope is diagnosed if there is definite
history of syncope being provoked consistently by specific factors such as micturition, defaecation, coughing, swallowing or laughing, and there is no suggestion from history or initial assessment indicating another cause.

Carotid sinus syndrome (CSS) is diagnosed in individuals with CSH in the presence of symptoms of syncope, drop attacks or unexplained falls. It is rare before the age of 40 years and increases in prevalence with age, with a positivity rate of over 40% in symptomatic individuals over the age of 90 years. Drop attacks and unexplained falls occur in individuals with CSH as a result of amnesia for loss of consciousness. Asymptomatic CSH is also known to occur, and the underlying mechanisms converting asymptomatic CSH to symptomatic CSS remains unknown.

The definition of orthostatic hypotension (OH) has been revised in the most recent syncope guidelines from the European Society of Cardiology (ESC). The diagnosis of classical orthostatic hypotension, a drop in blood pressure of at least 2 mmHg systolic or 10 mmHg diastolic within three minutes of standing still remains. However, definitions for “transient OH” (a brief reduction in blood pressure of 40 mmHg or greater with standing) and “delayed OH” (a slow progressive decline in systolic blood pressure on assuming the erect posture) have been introduced. OH presenting acutely may be an indication of systemic upset due to sepsis, gastrointestinal bleeding or dehydration, while OH of more insidious onset is commonly drug-induced. Individuals with autonomic failure due to diabetes mellitus, Parkinson’s disease or multi-system atrophy may also present with OH.

**Cardiac syncope**

While cardiac syncope is uncommon in the younger age group, it can be devastating in cases of sudden cardiac death associated with inherited cardiac conditions. The presence of cardiac syncope rises sharply with age. As cardiac syncope is associated with increased mortality, the investigative strategy for individuals with syncope should be to rule out any possibility of cardiac syncope first and foremost.

Cardiac conditions associated with syncope can be divided into rhythm abnormalities and structural abnormalities. Abnormalities of cardiac rhythm include bradycardia due to sinoatrial or atrioventricular disorders, atrial fibrillation, supraventricular tachycardia and ventricular tachycardia. Structural abnormalities include myocardial ischaemia, aortic stenosis, hypertrophic cardiomyopathy and pulmonary emboli. As mentioned above, any suspicion of cardiac syncope should prompt immediate referral to cardiologists.

**Pitfalls in diagnosis**

A major diagnostic pitfall is distinguishing epilepsy from true syncopal attacks. A comprehensive history can provide vital clues to help distinguish between the two, as twitching episodes and incontinence can sometimes occur in syncope as well as in epilepsy.

Patients with history of looking pale and sweaty with transient twitching after T-LOC are more likely to have had a syncopal episode. But, a history of patient appearing “blue” associated with rhythmic jerking of the body, which may be prolonged, and unusual posturing and stiffening indicates a probable epileptic seizure. Typically, jerking movements in epileptic seizures coincide with onset of LOC and there may be history of aching muscles following recovery of consciousness. Tongue biting occurs more often in epilepsy but is usually on the side of the tongue as opposed to the tip of the tongue in syncope.

Urinary incontinence can occur in both.

Other indicators of possible epilepsy are confusion after the episode and typical prodromal déjà vu or jamais vu (the experience of being unfamiliar with a person or situation that is actually very familiar; associated with certain types of epilepsy), or the presence of unusual stereotypical behaviour. If there is history of prodromal symptoms that have sometimes been terminated on sitting or lying down, it suggests a diagnosis other than epilepsy. While the presence of retrograde or prolonged antegrade amnesia tends to indicate epilepsy, amnesia for loss of consciousness can be present in individuals with syncope.

Some patients may present with persistent episodes of LOC that do not appear to fit any pattern. One should consider a possible diagnosis of psychogenic non-epileptic seizures (PNES) or psychogenic pseudosyncope, particularly if symptoms vary in nature and duration and tend to be bizarre and manifold with occasional atypical prolonged episodes. These patients may benefit from specialist
psychological assessment.

**Treatment**

The management of cardiac syncope is guided by the underlying aetiology, which is beyond the scope of this paper. The treatment of non-cardiac syncope should be directed at addressing the underlying cause and aim to improve quality of life and reduce if not prevent the number and frequency of physical injuries resulting from syncopal events.

**Conservative measures**

Patients with reflex syncope and orthostatic hypotension should receive information about the underlying mechanism of their symptoms and reassured of a potentially good prognosis. Many patients will respond to preliminary measures including medication review (common culprits include vasodilators, diuretics) and conservative advice relating to lifestyle changes such as avoidance of precipitating factors (standing in queues, hot crowded places, excess alcohol), and ensuring optimal hydration. Early recognition of warning symptoms and subsequently taking abortive measures such as sitting or lying down immediately are often helpful.

Other management options available are isometric hand exercises such as hand grip, arm tensing and leg crossing,25,26 tilt training which involves either repeated HUTT or home orthostatic training and compression hosiery.28 While salt supplementation is often advocated in younger adults, we do not recommend this approach in older individuals.30

**Pharmacological therapy**

Pharmacological therapy may be appropriate in patients who have on-going symptoms refractory to conservative management. Most commonly used medications include fludrocortisone (mineralocorticoid)30,31 and midodrine (α-agonist vasoconstrictor). Midodrine is currently not available as a licensed medication in the UK, and in the absence of any other readily available alternative, it is currently prescribed on a named-patient basis, and its use is generally restricted to specialist centres.32,33 These agents are, however, often poorly tolerated by older patients.30

**Pacemakers**

Permanent pacemaker implantation may be effective in alleviating or reducing severity of symptoms in patients with cardioinhibitory and mixed forms of CSS presenting with syncope.34 Two large randomised, controlled trials evaluating the value of cardiac pacing in patients with vasovagal syncope, however, have been negative.35 Cardiac pacing is, therefore, not recommended for patients with bradycardic responses during HUTT. The most recent ESC guidelines have now recommended that these individuals should first be evaluated with an implantable loop recorder to detect any spontaneously occurring asystolic or bradycardic events.9

**Driving implications**

The Driver and Vehicle Licensing Agency (DVLA) imposes no driving restrictions for individuals with isolated episodes of “simple faint” not occurring while sitting or lying down, with definite provocations and prodromes. All patients with syncope awaiting specialist assessment should be advised not to drive. The DVLA provides clear recommendations on driving regulations for patients with syncope within the UK (http://bit.ly/a2HTeC).

**Prognosis**

Syncope in general is associated with increased morbidity and mortality, particularly in the elderly population. In one study, individuals aged 65 years and over with syncope had an increased mortality rate of 23.9% compared with 2.3% in those with syncope under 65 years.35 Mortality rates are also higher for people with cardiac syncope as opposed to those with non-cardiac causes.36,37 Syncope usually leads to falls, sometimes with resultant fractures, with the associated morbidities of loss of confidence, fear of falling38 and increased rates of institutionalisation.39

**Conclusion**

Syncope in the elderly is a frequently encountered problem in general practice and a common presentation to the A&E department, often resulting in hospital admissions.2,4,5 A structured approach to diagnosis and management will help prevent unnecessary and inappropriate use of resources.40 High-risk individuals requiring urgent specialist intervention should be identified using established “red flag” features. While the recently published NICE T-LOC guidelines have established clear timeframes for referral and strengthened the current ESC syncope guidelines with economic analyses, there remains much more to be done to develop cost effective pathways for this condition, which is becoming increasingly common
in a rapidly ageing population.

Conflict of interest: none


13. Farwell DJ, Sulke AN. Does the use of a syncope diagnostic protocol improve the investigation and management of syncope? Heart 2004; 90: 52–8


