Blackout and collapse

Nicola Cooper

Abstract

'Collapse?cause' is a common acute medical presentation, and there are many different causes. This article focuses on syncope, which is caused by transient global cerebral hypoperfusion. There are four main subtypes of syncope, with neurally mediated and orthostatic syncope being by far the most common. Cardiac arrhythmias account for only 20% of all syncope. The initial evaluation is extremely important and leads to a diagnosis in around half of cases. Only some patients need tests. The tests to request depend on the history, whether the person has structural heart disease or not, and whether the syncope is frequent or severe. The evaluation of syncope is often unstructured and varies considerably among doctors. The European Society of Cardiology guidelines on syncope are the standard for syncope specialists and are outlined here.

Keywords driving; eyewitness account; initial evaluation; neurally mediated; structural heart disease; syncope

The terms 'blackout' and 'collapse'

'Blackout' and 'collapse' are not diagnostic terms; they are how patients present to an acute medical unit or family practitioner. 'Collapse?cause' is a commonly used term in UK medical practice and refers to one or more episodes of transient loss of consciousness before a thorough evaluation has been made.

There are many different causes of transient loss of consciousness in which a patient 'blacks out' and is apparently back to normal again by the time he or she consults the doctor. Figure 1 shows the main causes of transient loss of consciousness in adults.

Causes of transient loss of consciousness

As Figure 1 shows, different acute illnesses can present with transient loss of consciousness. The most common is infection, but acute coronary syndromes, bleeding, dissection of the aorta and pulmonary embolism can also present this way. In older people, in whom atypical presentations are more common, transient loss of consciousness may be the only presenting complaint, but there will always be clues in the history, physical examination and initial test results that an acute illness is present. If this is the case, treat the underlying illness, not the 'collapse'.

After acute illness has been excluded, the main differential diagnosis of transient loss of consciousness is syncope or seizure.

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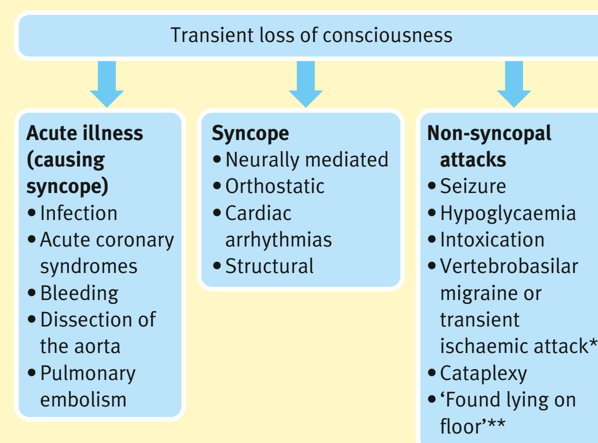
Main causes of transient loss of consciousness in adults



* Please note that transient ischaemic attacks do not cause syncope without focal neurological symptoms.
** 'Found lying on floor' is a common presentation in older people in which the precipitating event can be acute illness, syncope, seizure or fall – but the patient is unable to get up from the floor and may not have a clear memory of events.

Figure 1

However, Figure 1 also shows the other causes of non-syncopal attacks that should be considered. In most cases, these can be ruled out by the history, physical examination and initial test results.

The purpose of this article is to focus on syncope, which is one of the most common reasons for blackout or collapse presenting to acute medical services.

Syncope

The word 'syncope' is derived from the Greek words 'syn' (meaning 'with') and 'kopto' (meaning 'I cut' or 'I interrupt'). Syncope is always the result of transient global cerebral hypoperfusion. Syncope is characterized by:

- relatively rapid onset
- loss of consciousness
- loss of voluntary muscle tone, usually leading to a fall
- spontaneous, complete and prompt recovery[1] in most cases.

Syncope is a symptom, not a diagnosis. Figure 2 illustrates that there are four main types of syncope and several different underlying causes.

As the European Society of Cardiology (ESC) guidelines on syncope point out, 'Patients are asymptomatic at the time of the evaluation, and the opportunity to capture a spontaneous event during a diagnostic test is rare. Diagnostic evaluation has focused on physiological states that could cause loss of consciousness…in other words, the causal relationship between a diagnostic abnormality and syncope in a given patient is presumptive. Uncertainty is further compounded by the fact that there is a great deal of variation in how physicians take a history and perform a physical examination, the types of tests requested and how they are interpreted. These issues make the diagnostic evaluation of syncope inordinately difficult'.[2]

Despite this, following some simple guidelines can make the evaluation of syncope much easier, and these are described

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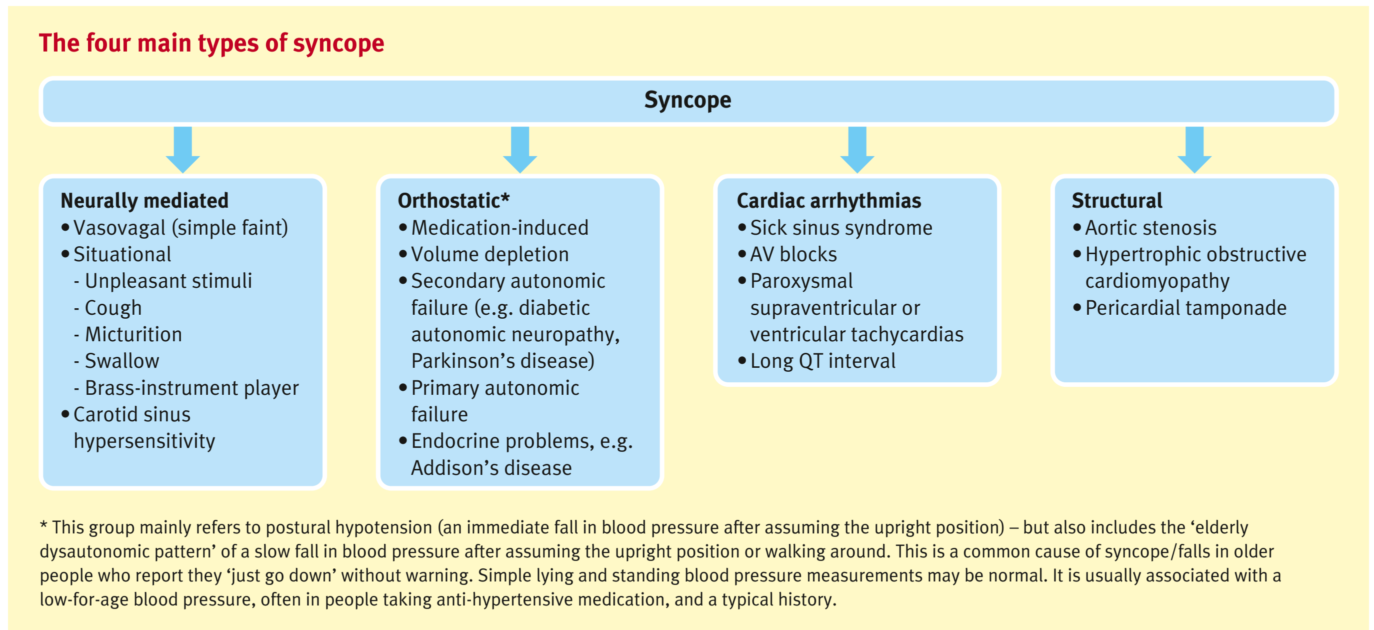

Figure 2

below. In a nutshell, syncope is due to either a low blood pressure problem or a heart problem.

Prevalence and impact on lifestyle

Syncope is one of the most common presentations to acute medical services in the UK. In the Framingham Study, 10.5% of subjects reported at least one syncopal episode during the 17-year period of the study. However, the prevalence varied with age and increased considerably over the age of 70 years.[3]

Syncope accounts for up to 5% of emergency department visits.[4] There are certain sub-groups of the population who are more likely to experience it – 25% of military recruits[5] and 23% of elderly people[6] are two commonly cited examples. One study found that 39% of medical students experienced syncope, with a prevalence in females twice as high as in males.[7]

Neurally mediated syncope is by far the most common type, accounting for around 50% of all syncope. Cardiac arrhythmias account for around 20% of all syncope. The prevalence of orthostatic syncope among the syncope population depends very much on age. It is uncommon in young people but accounts for at least one-third of syncope over the age of 70 years. Structural problems only account for around 3% of syncope. A significant minority of syncope cases (around 20%) remain unexplained after a thorough evaluation, and this is associated with a good prognosis.

Even though most syncope is considered benign, it can still impact considerably on people's lives. Some young people suffer from severe vasovagal syncope, which renders them incapable of work. Older people with syncope are more likely to injure themselves when they fall and reduce their 'life space' as a result of loss of confidence. There is a marked negative relationship between the frequency of syncopal attacks and perception of health. This is not helped by the fact that many patients are evaluated in an unstructured way, often by inexperienced doctors, and given limited information about their condition.

Initial evaluation

Syncope is diagnosed purely on the basis of history – from the patient and an eyewitness whenever possible. Table 1 shows the important questions that should be asked and Table 2 outlines the key features that differentiate syncope from seizures. Once a decision has been made that the event was syncopal, several studies suggest that the history and physical examination alone identify a potential cause of syncope in nearly half of patients.[2] Only some patients need further investigation.

In syncope, the initial evaluation consists of:

- history (including from an eyewitness)
- physical examination (focusing on the heart and neurological system)
- lying and standing blood pressure
- 12-lead electrocardiogram (ECG).

If the patient has had syncope, a key question is whether he or she has heart disease or not. The presence of structural heart disease is the most important predictor of a cardiac cause of syncope. On the other hand, the absence of structural heart disease rules out a cardiac cause of syncope in 97% of cases.[8]

After the initial evaluation, the cause of syncope may be obvious, in which case no tests are needed. If the cause is suspected, then tests may be needed to confirm the particular disorder. If the cause is unexplained, then the next steps depend on how severe and how frequent the attacks are and whether the patient has heart disease.

Figure 3 shows the ESC guidelines for unexplained syncope. Structural heart disease in this flow chart refers to:

- previous myocardial infarction
- clinically significant murmur (e.g. aortic stenosis)
- abnormal ECG
- in young people, a family history of sudden cardiac death.

An abnormal ECG refers to a prolonged QT interval, previous myocardial infarction, bradycardia (< 40 beats/minute) other than during sleep, tachyarrhythmias and blocks, rather than non-specific ST changes.

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Important questions to ask in syncope

Questions about before the attack

- Activity (what the person was doing at the time in detail)
- Position (lying, sitting or standing)
- Any provoking factors (e.g. unpleasant stimuli, warm environment, micturition)
- Prodromal symptoms

According to the Driver and Vehicle Licensing Agency UK (DVLA), the three Ps are strongly suggestive of vasovagal syncope: upright Posture, Provoking factors and a typical Prodrome of feeling warm, nauseated, light-headed, with blurred vision before losing consciousness

Questions about during the attack (from an eyewitness)

- How the person fell (floppy or rigid)
- What colour he or she was (white or blue)
- Whether he or she was allowed to lie flat or someone held him upright
- The presence of any jerking movements and their duration
- Any injuries (e.g. head injury) or incontinence

Questions about after the attack

- What the person was like when he or she came round
- How long it took to recover

'Teasing out' questions

- Any previous collapses and their circumstances
- Any symptoms of orthostatic problems (e.g. inability to stand for a long time without feeling 'queer' or dizziness when standing up quickly)
- Any palpitations

Table 1

Pitfalls in elderly people

There are a number of differences in elderly people, which can make the initial evaluation difficult. It is common for elderly people to experience vasovagal or orthostatic syncope without any prodrome whatsoever. This is because of an impaired sympathetic response to a falling blood pressure. On further questioning, elderly people may admit to feeling 'queer' briefly beforehand, but may not be able to give any more information than this. As described in Figure 2, the 'elderly dysautonomic pattern' is also common.

Elderly people commonly experience vasovagal or orthostatic syncope while sitting, something that rarely occurs in young people (except in situational syncope). Post-prandial hypotension also occurs. An elderly person may appear to 'slump to one side' if syncope occurs while sitting – this does not indicate a focal weakness, but reflects the loss of muscle tone that occurs during syncope.

Elderly people can appear post-ictal after an episode of syncope, particularly if they were not allowed to lie flat straight away. This reflects their inability to compensate for a period of cerebral hypoperfusion in the same way as younger people. This can lead to diagnostic confusion with seizures, which are also more common in the elderly population.

Carotid sinus hypersensitivity occurs almost exclusively in people over the age of 40 years. A history of syncope associated with head turning is rare. Most commonly, carotid sinus hypersensitivity causes syncope or falls without warning. Retrograde amnesia occurs in one-third of patients, so they may not even remember losing consciousness. In at least 30% of cases, a positive response to carotid sinus massage is only present in the upright position.[9]

Finally, heart disease is common in elderly people, yet non-cardiac causes of syncope are still more common. It is normal to find more than one potential cause of syncope after evaluating an elderly person.

Investigations

In Figure 3, cardiac evaluation includes:

- echocardiography
- stress testing
- prolonged ECG monitoring, or
- electrophysiology studies.

Patients with suspected cardiac syncope should be referred to a cardiologist for further investigation. If the cardiac evaluation is negative, then tests for neurally mediated syncope should be carried out.

Neurally mediated syncope evaluation consists of tilt testing, and carotid sinus massage in older patients. Tilt testing aims to reproduce the vasovagal sub-type of syncope[10] but is also useful for demonstrating orthostatic syncope. There are very few contraindications: a recent myocardial infarction or stroke, and a known tight stenosis anywhere. The false-positive rate is around 10%, and symptoms are more likely to be reproduced during a period when the patient is symptomatic. Various tilt testing protocols exist.[11,12] The patient lies flat for 10 minutes and is attached to a cardiac and non-invasive beat-to-beat blood pressure monitor. The patient is then tilted 60–70° head-up and observed for 30 minutes for symptoms and signs of syncope. If the patient remains asymptomatic, various methods may be used to increase orthostatic stress, e.g. the application of lower body negative pressure, and the patient is observed for a further 20 minutes.

A tilt test is considered positive when the patient's symptoms are reproduced and accompanied by hypotension, bradycardia or both, especially early during the test. There are different classifications of positive responses to tilt testing, and the same patient may demonstrate different responses at different times. More details about tilt testing can be found in the further resources.

Carotid sinus massage (CSM) is indicated in patients over the age of 40 years with syncope or unexplained falls in which the history, clinical examination and relevant tests have not clearly identified a cause of the symptoms. The main potential complication of CSM is transient ischaemic attack, which occurs around 1 in 1000 times or, more rarely, stroke.[9]

Implantable loop recorder

In situations where initial cardiac or neurally mediated investigations are negative, an implantable loop recorder (ILR) may be used. It is similar to a pacemaker box, and is inserted for a period of up to 18 months. When syncope occurs, the device can be retrospectively activated by the patient and interrogated to demonstrate the heart rhythm during an attack. An ILR is considered in recurrent unexplained or high risk syncope, i.e.:

- very frequent episodes affecting quality of life
- recurrent and unpredictable episodes, putting the patient at risk of trauma

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Key features which differentiate syncope from seizures (note: the overall picture is more important than any single feature)

Syncope more likely	Seizure more likely
• (Upright posture) • (Provoking factor present) • (Typical prodrome) • Patient goes 'white' • Loss of consciousness is brief • Brief jerking movements may occur after the patient has lost consciousness. • Quick recovery if allowed to lie flat • Fatigue afterwards is common (The features in brackets only refer to some kinds of syncope) Incontinence of urine can occur due to loss of muscle tone with a full bladder Significant injury is less likely in syncope but does occur in 5% of cases If the patient is not allowed to lie flat, he or she may go rigid and appear to have a 'seizure' due to cerebral anoxia	• Aura (e.g. funny smell) • Cyanosis • Rigidity which coincides with loss of consciousness, tonic–clonic movements lasting at least 30 seconds • Automatisms, tongue-biting • Prolonged confusion, headache or drowsiness afterwards* • At night in bed • Faecal incontinence

*If a person sustains a head injury during syncope, these features may be present due to concussion. Elderly people may appear 'post-ictal' after syncope because of their inability to cope with an episode of cerebral hypoperfusion compared with younger people.

Table 2

- occurring during 'high-risk' activity, e.g. operating machinery, driving, sports.

In one study, this was only around 5% of all syncope patients; 40% of these had structural heart disease and 60% did not.[13] The patients with structural heart disease were more likely to have a cardiac arrhythmia as a cause of their syncope, but the diagnosis remained unexplained in 43% of total cases.

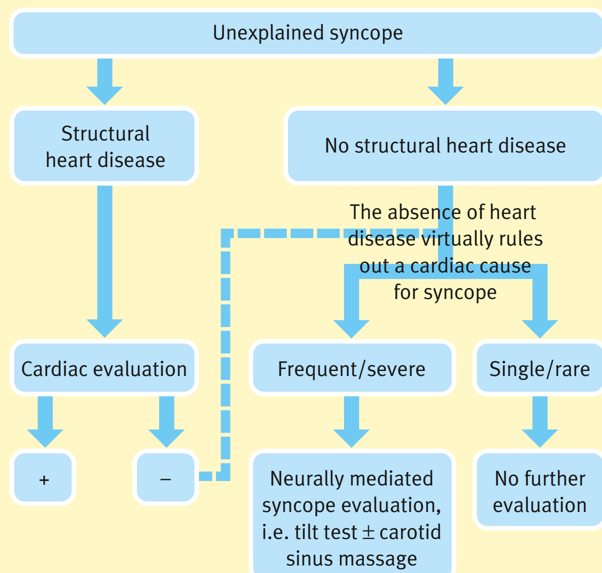

Figure 3

The mechanism of syncope is considered likely to be due to a primary cardiac arrhythmia when sudden onset atrio ventricular block or bradycardia or tachyarrhythmias are detected at the time of a syncopal attack. However, the ILR is also useful in picking up likely neurally mediated syncope, in which brady- or tachyarrhythmias are gradual and progressive in onset and termination at the time of a syncopal attack.

Pitfalls in the investigation of syncope

In clinical practice, the most common pitfalls in the investigation of syncope are:

- not spending enough time on the history, including from an eyewitness
- not clearly differentiating patients with and without structural heart disease
- doing unnecessary tests (e.g. 24-hour Holter monitor in people with normal hearts)
- doing a computed tomography (CT) brain scan – this is not a test for syncope
- getting side-tracked by incidental findings, especially in older people.

Management

The management of syncope depends on the underlying cause. The ESC guidelines state that patients with syncope and structural heart disease who present to acute medical services should be admitted and referred to a cardiologist. Similarly, if there is a suspicion of cardiac syncope or if syncope occurred during exercise, the patient should be admitted for cardiac evaluation.

Patients with normal hearts and no acute illness do not require admission to hospital, unless there is a severe injury. They should have a thorough initial evaluation, followed by

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Syncope and driving in the UK (2007)

Disorder	Group 1 licence (car or motorcycle)	Group 2 licence (bus, lorry) and taxi drivers
Vasovagal and situational syncope	No restrictions	No restrictions
Cough syncope	Driving must cease until liability to attacks has been controlled	Driving must cease and the person must be free of syncope for 5 years
Unexplained syncope and low risk of re-occurrence (i.e. no abnormality on cardiovascular and neurological examination and normal ECG)	Can drive 4 weeks after the event	Can drive 3 months after the event
Unexplained syncope and high risk of re-occurrence (i.e. abnormal ECG, structural heart disease, syncope causing injury, occurring at the wheel or whilst sitting or lying, more than one episode in the last 6 months)	Can drive 4 weeks after the event if the cause has been identified and treated If no cause identified, cannot drive for 6 months	Can drive after 3 months if the cause has been identified and treated If no cause identified, then licence revoked for 1 year
Loss of consciousness with seizure markers (i.e. strong clinical suspicion of a seizure but no evidence)	Cannot drive for 1 year	Cannot drive for 5 years
Loss of consciousness with no clinical pointers whatsoever (after evaluation by a specialist)	Cannot drive for 6 months	Cannot drive for 1 year

ECG, electrocardiogram. In the UK, fitness to drive is decided by the Driver and Vehicle Licensing Agency, which is a government agency. Patients are advised that 'by law' they may not drive for a certain time. In other countries, fitness to drive is decided by doctors. The European Society of Cardiology offers guidance on driving with syncope for doctors working in these countries.

Table 3

treatment or referral to a syncope outpatient clinic if their attacks are frequent or severe.

The management of neurally mediated syncope usually consists of stopping any exacerbating medication, general measures and medication to increase blood pressure in certain patients. Only a small proportion of patients require a dual-chamber pacemaker (e.g. cardio-inhibitory carotid sinus hypersensitivity). More details on the management of syncope can be found in the ESC guidelines and further resources.

Advice about driving

Finally, advice about driving is often forgotten. Table 3 shows the current UK guidelines on syncope and driving. ◆

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