Syncope

Syncope is a transient loss of consciousness caused by transient global cerebral hypoperfusion characterised by rapid onset, short duration and spontaneous complete recovery.\(^1\) The term syncope excludes seizures, coma, shock or other states of altered consciousness.

Patients presenting with a history of blackouts, faints or collapse need careful evaluation to assess the precise nature of the problem. This is essential so as to assess both the risk of a serious underlying disorder and also the risk of recurrence and subsequent injury.\(^1\)

Aetiology\(^2\)

- Neurally mediated syncope (NMS) - also called reflex syncope:
  - Vasovagal syncope (common faint):
    - Emotional - eg, fear, severe pain, blood phobia, sudden, unexpected sight, sound or smell.
    - Orthostatic stress - eg, prolonged standing or when in crowded, hot places.
  - Situational syncope - eg, cough, sneeze, gastrointestinal stimulation (swallowing, defecation, visceral pain), micturition.
  - Carotid sinus hypersensitivity: occurs when rotating the head - eg, while shaving, especially if a collar is tight or in the presence of a neck tumour.
  - Glossopharyngeal neuralgia.

- Orthostatic hypotension (postural hypotension) - syncope occurs after standing up:
  - Autonomic failure:
    - Multiple system atrophy, Parkinson's disease, diabetic neuropathy, amyloidosis.
    - Medications - eg, antihypertensives.
    - Post-exercise.
    - Postprandial.
  - Hypovolaemia:
    - Haemorrhage.
    - Vomiting, diarrhoea and other causes of dehydration.
    - Addison's disease.

- Cardiac arrhythmias:
  - Sick sinus syndrome, atrioventricular (AV) conduction system disease.
  - Paroxysmal supraventricular tachycardia, ventricular tachycardia.
  - Inherited syndromes - eg, long QT syndrome, Brugada's syndrome.
  - Malfunction of a pacemaker or implantable cardioverter defibrillator (ICD).
  - Drug-induced arrhythmias.

- Structural cardiac or cardiopulmonary disease:
  - Obstructive cardiac valvar disease.
  - Acute coronary syndrome.
  - Hypertrophic obstructive cardiomyopathy.
  - Atrial myxoma.
  - Acute aortic dissection.
  - Pericardial disease or tamponade.
  - Pulmonary embolus or pulmonary hypertension.
Epidemiology

- Syncope is common in all age groups and affects 40% of people during their lifetime. However, only a minority of people with syncope will seek medical attention.\(^3\)
- Only 5% of adults in the community have a first syncope over the age of 40 years.
- NMS is by far the most common cause. The majority have experienced reflex-mediated episodes as teenagers and adolescents.\(^1\)
- The lifetime risk of syncope is 42% with a peak prevalence between age 10-30, mainly NMS.\(^4\)
- Cardiac causes, orthostatic and postprandial hypotension and the effects of medications are common causes in the elderly, in whom syncope increases with age; there is an annual incidence of almost 2% of individuals over the age of 80.\(^4\)
- A definite diagnosis for the cause of syncope in the elderly can be made in over 90% of patients.\(^1\)

Presentation\(^1\)

A thorough history and examination are essential. See also separate Cardiovascular History and Examination and Neurological History and Examination articles.

History\(^1\)

An accurate history, including from an eye-witness if available, is essential and often alone will lead to a correct diagnosis.\(^2\) Points to cover in the history include:

- Was loss of consciousness (LOC) complete?
- Was LOC temporary? How quickly did it come on and how long did it last?:
  - Syncope is usually brief, with complete loss of consciousness in NMS not lasting more than 20 seconds (but may occasionally be up to several minutes).
- Was there any warning: light-headedness, nausea, sweating, weakness or visual disturbance?:
  - Preceding nausea, sweating and blurred vision have been shown to be predictive of non-cardiac syncope in the elderly.\(^5\)
- Did it occur during exercise or while lying down? Were there any palpitations or was there accompanying chest pain? Was there any shortness of breath?:
  - If so, this suggests a cardiac cause.
  - Dyspnoea has been shown to be predictive of cardiac syncope in the elderly.\(^5\)
- Was recovery spontaneous and total? Were there any symptoms following recovery?:
  - Recovery from syncope is usually associated with almost immediate restoration of appropriate behaviour and orientation but there may be marked fatigue.
- Was there loss of postural tone?
- Was there a situational trigger?
- Has there been a recent change in medication?:
  - New medication or a change of dose causing orthostatic hypotension.
  - Syncope may be more likely to occur in the morning.
- Is there any family history of sudden death?
Examination
The following physical findings may indicate a likely underlying cause:

- **Syncope caused by orthostatic hypotension:**
  - There may be examination evidence of a drop in blood pressure (usually >20/10 mm Hg) within three minutes of standing, associated with syncope or presyncope.\[6\]

- **Cardiac syncope:**
  - Full cardiovascular examination may reveal a severe structural abnormality.

- **Cerebrovascular syncope:**
  - With arm exercise.
  - Differences in blood pressure or pulse in the two arms.

Differential diagnosis
Transient loss of consciousness is usually due to syncope. Other possible causes are:\[1\]

- Falls/trauma.
- Epilepsy.
- Narcolepsy, cataplexy.
- Drop attacks.
- Dizziness or vertigo without loss of consciousness.
- Alcohol/drug abuse.
- Transient ischaemic attacks/stroke.
- Psychogenic pseudosyncope.

See also separate Dizziness, Giddiness and Feeling Faint article.

**NB:** prolonged unconsciousness, witnessed abnormal behaviour before, during or after the event, confusion after the event, tongue biting, head turning or prolonged limb jerking, unusual posturing - all suggest a non-syncopal event and should prompt referral to a specialist in epilepsy to be seen within two weeks.\[7\]

**Psychogenic pseudosyncope**\[1\]

- Although psychological, pseudosyncope is usually involuntary.
- Most often occurs in younger adults or teenagers.
- There is often underlying anxiety - eg, poor performance at school.
- Attacks are often very frequent with many attacks occurring in a day and there is usually a lack of a recognisable trigger.
- Pseudosyncope usually lasts longer than syncope; patients may lie on the floor for many minutes.
- Symptoms are often vague; typical features of NMS, such as pallor and sweatiness, are absent.
- The eyes are usually open in epileptic seizures and syncope but are usually closed in functional transient loss of consciousness.
- Attacks may be induced on a tilt table but there is no change in heart rate or blood pressure and the ECG is unchanged during the syncope.

Investigations and assessment\[1\]
Investigations are guided by the history and examination. Initial tests in primary care include:\[7\]

- Orthostatic blood pressure measurement.
- ECG: there may be evidence of ischaemia or arrhythmias.
- FBC if anaemia or bleeding is suspected (acute anaemia will cause syncope but patients adapt in cases of chronic anaemia).
- Fasting blood glucose, if hypoglycaemia is a possibility.
In most cases, the initial assessment will lead to a definite, or at least a likely, diagnosis, which will clarify the selection of further investigations and management. However, syncope is often multifactorial, especially in older individuals.

**Risk stratification**

It is essential to assess the risk of major cardiovascular events or sudden cardiac death. The indications for urgent hospital assessment include:

- Severe structural or coronary artery disease - eg, heart failure, low left ventricular ejection fraction, previous myocardial infarction.
- Clinical or ECG features suggesting arrhythmic syncope:
  - Syncope during exercise or whilst supine.
  - Palpitations at the time of syncope.
  - Family history of sudden cardiac death.
  - Non-sustained ventricular tachycardia.
  - Bifascicular block (right bundle branch block and either left anterior or left posterior fascicular block).
  - Bradycardia with pulse heart rate below 50 or sinoatrial block in the absence of negative chronotropic drugs (eg, beta-blockers) or physical training.
  - QRS complex longer than 120 milliseconds.
  - Prolonged or short QT interval.
  - Right bundle branch block pattern with ST elevation in leads V1-V3 (Brugada pattern).
  - Features suggestive of arrhythmogenic right ventricular cardiomyopathy.
- Important comorbidities - eg, severe anaemia, electrolyte disturbance.

Referral is indicated if there is any suggestion of a serious underlying cause or if the episodes of syncope are frequent, have implications for driving, cause injuries or cannot be controlled by simple avoidance of precipitating factors. There are several risk scores to help identify those patients with syncope who are at high risk of adverse events but none of the scores is widely accepted:

- Osservatorio Epidemiologico sulla Sincope nel Lazio (OESIL) score.
- San Francisco Syncope Rule (SFSR): this is the simplest, and uses an abnormal ECG, heart failure, anaemia and systolic hypotension (below 90 mm Hg) to identify patients who require urgent action.
- European Guidelines in Syncope Study (EGSYS) score.

**Investigations in secondary care**

- NMS:
  - Carotid sinus massage, tilt testing, implantable loop recorder.
  - Carotid sinus massage should be avoided in patients with previous transient ischaemic attack, stroke within the preceding three months, or with a carotid bruit, except if carotid Doppler studies excluded significant stenosis.
- Cardiac syncope:
  - ECG ambulatory monitoring - eg, conventional ambulatory Holter monitoring, in-hospital monitoring, event recorders, external or implantable loop recorders, or remote (at home) telemetry. The gold standard for the diagnosis of cardiac syncope is when a correlation between the symptoms and a documented arrhythmia is recorded.
  - Adenosine triphosphate (ATP) test: rapid injection of a bolus of ATP (or adenosine) during ECG monitoring; the induction of AV block with ventricular asystole lasting over six seconds, or the induction of AV block lasting over ten seconds are considered abnormal.
  - Echocardiogram: to identify structural cardiac abnormalities and assess left ventricular function.
  - Transoesophageal echocardiography, CT and MRI may be performed in selected cases (eg, aortic dissection and haematoma, pulmonary embolism, cardiac masses, pericardial and myocardial diseases, congenital anomalies of coronary arteries).
  - Cardiac catheterisation and coronary angiography may be indicated for suspected cardiac ischaemia.
• Exercise testing:[1, 11]
  - For patients who have experienced episodes of syncope during or shortly after exertion.
  - Careful ECG and blood pressure monitoring should be performed during both the test and the recovery phase.
  - Syncope occurring during exercise may be due to cardiac causes; syncope occurring after exercise is almost invariably due to a reflex mechanism.

• 'Tilt testing' to invoke syncope:[12]
  - The patient lies flat on the table and is attached to an ECG and a beat-to-beat blood pressure monitor. After 10 minutes supine the table is tilted head up to 70° and the position maintained for 35 minutes. 400 micrograms sublingual GTN may be used if no symptoms have developed after 20 minutes. 50-60% of patients with unexplained syncope develop symptoms after about 20 minutes.
  - Pseudosyncope attacks may be induced on a tilt table but there is no change in heart rate or blood pressure and the ECG is unchanged during the syncope.

If the cause still remains unclear then repeat evaluation, including neurological investigations, and possible admission to hospital may be required.[2]

Management

NMS

• Most patients with NMS require only an explanation, reassurance and education regarding the nature of the problem and avoidance of triggering events - eg, avoiding prolonged standing in a hot environment or having a hot bath.
• They should be advised to take action at the first warning sign of collapse:
  - Lie down flat with the legs up on a chair or against a wall or sit down, ideally on the ground, with the head between the knees.
  - Squat down on the heels; this can be very effective and is less noticeable in public.
  - These techniques help move venous blood that has pooled in the limbs, aiding circulation to the brain.
  - When feeling better, advise them to get up carefully. If symptoms return, resume the position.

• Treatment may be desired if syncope is very frequent, unpredictable or could occur during high-risk activities such as driving. However, treatment options are limited:
  - Tilt training: prolonged periods of upright posture; requires good compliance, as several sessions are needed and deconditioning occurs quickly on stopping.[2]
  - Isometric counterpressure manoeuvres - eg, leg crossing or arm tensing, which can increase blood pressure enough to prevent syncope.
  - Medications: various medications have been used; however, a Cochrane review concluded that there was insufficient evidence to support their use.[13]
  - Cardiac pacing: the same Cochrane review concluded that there was also insufficient evidence to support the use of pacemakers in NMS.

Orthostatic hypotension[14]

• Stop any offending drugs.
• Avoid alcohol.
• Encourage a plentiful oral fluid intake: two large glasses of cold water prior to periods of increased orthostatic stress can be very effective.
• Raise the head of the bed.
• Wear support stockings to reduce pooling of vascular volume. An abdominal binder may also be used.
• Leg crossing and arm tensing.
• In some patients medication may be desired, although all may cause supine hypertension:
  - Low dose of fludrocortisone. May also cause hypokalaemia.
  - Midodrine, an alpha-1 adrenergic agonist which increases total peripheral resistance, is occasionally tried but there is insufficient high-quality evidence to support its use.[15]
Cardiac cause of syncope

- Treatment is aimed at the underlying cause - eg, anti-arrhythmic drugs, pacing, implantable cardiac defibrillators, correction or amelioration of structural disorders.
- Electrophysiological studies and ablation may also be required for arrhythmias.

Driving and syncope\(^{[16]}\)

- In the UK, following a single vasovagal syncope, driving is not restricted and the Driver and Vehicle Licensing Agency (DVLA) does not need to be informed. If recurrent, on each occasion it must be due to strong Provocation, associated with Prodromal symptoms and Posture, ie it is unlikely to occur while sitting or lying - the ‘3 Ps’.
- Greater restrictions apply if the situation is more complicated, such as cough syncope, or if diagnosis is less clear.
- If in doubt, contact the DVLA.

Complications\(^{[1]}\)

- Recurrent syncope has serious effects on quality of life. The impairment due to syncope is comparable with chronic illnesses such as chronic arthritis, recurrent moderate depressive disorders and end-stage kidney disease.
- Morbidity is particularly high in the elderly and includes loss of confidence, reduced mobility, depressive illness, fear of falling, fractures and subsequent institutionalisation.
- Female gender, a high level of comorbidity, the number of episodes of syncope and the presence of presyncope seem to be associated with poorer quality of life.
- Physical injury: soft tissue and bone injuries may occur. Syncope was found to be the cause of 21\% of road accidents involving loss of consciousness at the wheel, second only to epilepsy\(^{[11]}\).

Prognosis

- Prognosis varies according to the underlying cause. The all-cause mortality in subjects with reflex syncope is not higher than in the general population.\(^{[6]}\)
- Approximately 35\% of patients have recurrences of syncope at three years of follow-up.\(^{[11]}\)
- In young patients, syncope is a benign event.
- Isolated syncope (transient loss of consciousness in the absence of prior or concurrent neurological, coronary, or other cardiovascular disease) is not associated with any increased risk of transient ischaemic attack, stroke or myocardial infarction and is not associated with any excess of all-cause or cardiovascular mortality (including sudden death).
- Poor outcomes, including deaths, are largely related to the severity of the underlying disease rather than to syncope:
  - Structural heart disease and primary cardiac electrical disease are major risk factors for sudden cardiac death and overall mortality in patients with syncope.
  - Orthostatic hypotension is associated with a two-fold higher risk of death, owing to the severity of comorbidities compared with the general population.\(^{[1]}\)
- For non-cardiac causes of syncope, excluding children and adolescents, the presence of the following risk factors is associated with increased mortality at one year (presence of two or more factors is associated with over 16\% mortality):\(^{[17]}\)
  - Abnormal ECG (not sinus bradycardia or sinus tachycardia, or nonspecific ST/T-wave changes).
  - Age older than 45 years.
  - History of ventricular arrhythmias or congestive cardiac failure.

Further reading & references

- Transient loss of consciousness; NICE Quality Standard, October 2014
- Guidelines on Diagnosis and Management of Syncope; European Society of Cardiology (2009)
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7. Transient loss of consciousness (‘blackouts’) management in adults and young people; NICE Clinical Guideline (August 2010)
16. Assessing fitness to drive: guide for medical professionals; Driver and Vehicle Licensing Agency

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