

Dizziness and loss of consciousness

Cardiovascular causes

BACKGROUND Dizziness and loss of consciousness are common clinical problems presenting in general practice.

OBJECTIVE This article aims to provide the practitioner with a pragmatic and logical approach to identifying the cardiovascular causes of dizziness and loss of consciousness.

DISCUSSION A range of disorders with varying pathology cause a transient loss of consciousness associated with postural collapse (syncope) by interruption of blood flow to the brain. Syncope and seizures are the only common causes of recurrent episodes of loss of consciousness. The vasovagal reaction or 'common faint' and postural hypotension are both common and benign causes of syncope. Syncope can also result from cardiac causes that include disorders of cardiac rhythm and mechanical obstruction to cardiac output. Cardiac causes of syncope are associated with much higher morbidity and mortality than postural hypotension or fainting. Specific treatment is available for the various cardiac causes of syncope and thus accurate diagnosis is imperative.

Loss of consciousness is a result of widespread interruption of cerebral cortical or brain stem function. Syncope, a transient loss of consciousness associated with postural collapse, is caused by cerebral hypoperfusion. Syncope is usually brief. If loss of consciousness is prolonged other causes must be considered including a seizure and sudden cardiac death. Therefore, a witnessed episode lasting more than a few seconds needs the urgent institution of appropriate first aid measures.

Recurrent episodes of loss of consciousness are nearly always caused by seizures or syncope. Distinguishing between these possibilities can be difficult. Features that suggest seizures include waking with a cut tongue, déjà vu or jamais vu, an association with emotional stress, head turning during, or confusion following the loss of consciousness. Features that suggest syncope include prodromal symptoms (presyncope) and loss of consciousness associated with prolonged sitting or standing.¹ Myoclonic jerks may be seen in syncope and misinterpreted as evidence of a seizure, a so-called 'convulsive syncope'.

Syncope

The causes of syncope may be classified into three groups by the underlying pathophysiology:

- neurally mediated reflex syncopal syndromes
- orthostatic syncope, and
- cardiac syncope (Table 1).

Presyncope

Syncope often has a prodrome, called presyncope. The symptoms of presyncope (or faintness) include lightheadedness, blurred vision, sweating, tinnitus, nausea and weakness. Presyncope is more common than syncope and its causes can be classified in the same way. Patients often complain of dizziness and it can be difficult to determine if they mean presyncope or vertigo. This distinction is important because vertigo is caused by inner ear or brain stem disease.

In reporting the frequency and prognosis of various causes of syncope we have relied heavily on a recent report from the Framingham Cohort² (Table 2). Because this study is community based,

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Table 1. Causes of syncope

Neurally mediated reflex syncopal syndromes

- Vasovagal faint (common faint)
 - situational faint
 - cough, sneeze
 - gastrointestinal stimulation (swallow, defaecation, visceral pain)
 - micturition (postmicturition)
 - postexercise
 - others (eg. brass instrument playing, weightlifting, postprandial)
 - glossopharyngeal and trigeminal neuralgia

Orthostatic

- Autonomic failure
 - primary autonomic failure syndromes (eg. pure autonomic failure, multiple system atrophy, Parkinson disease with autonomic failure)
 - secondary autonomic failure syndromes (eg. diabetic neuropathy, amyloid neuropathy)
 - drugs and alcohol
- Volume depletion
 - haemorrhage, diarrhoea, Addison disease

Cardiac syncope

- Arrhythmia
 - sinus node dysfunction (including bradycardia/tachycardia syndrome)
 - atrioventricular conduction system disease
 - paroxysmal supraventricular and ventricular tachycardia
- Structural heart disease
 - cardiac valvular disease
 - acute myocardial infarction/ischaemia
 - obstructive cardiomyopathy
 - atrial myxoma
 - acute aortic dissection
 - pericardial disease/tamponade
 - pulmonary embolus/pulmonary hypertension

we believe it is more likely to reflect patients seen in general practice than is the reported experience of a tertiary referral centre.

Neurally mediated reflex syncopal syndromes

There are many syndromes in this class. What they have in common is arterial and venous vasodilatation and relative or absolute bradycardia that is mediated by the inappropriate triggering of a reflex autonomic response.

Vasovagal episodes

The vasovagal (common) faint is the most frequently identified cause of syncope.² Noxious stimuli (emotional or physical) cause an increase in sympathetic activity. It is postulated that this increase in sympathetic activity causes cardiac hypercontractility and stimulation of cardiac stretch receptors. These receptors may act on the brainstem to reduce sympathetic drive and increase parasympathetic drive. The resulting peripheral vasodilatation and relative bradycardia causes progressive hypotension resulting in syncope unless the cycle is interrupted.

Tilt table testing is often part of the diagnostic pathway. The test is positive if symptoms are reproduced and syncope is observed. Data from the Framingham Study demonstrate that patients with vasovagal syncope, compared to those without, have no increased risk of death (of any cause), myocardial infarction, or death from coronary disease.³ Initial treatment is education about the process and advice to ‘go-to-ground’ when an impending event is recognised to reduce the risk of injury. General measures include avoidance of dehydration, prolonged fasting, prolonged upright posture, hot confining environments and known ‘triggers’, eg. venepuncture. Concurrent vasodilators may exacerbate vasovagal syncope.³

Table 2. Causes of first episode of syncope*

| Cause of first episode of syncope | Frequency % | Adjusted hazard ratio for death (95% CI) |
|-----------------------------------|-------------|--|
| Vasovagal | 21 | } 1.08 (0.88–1.34) |
| Orthostatic | 9 | |
| Cardiac | 10 | 2.01 (1.48–2.73) |
| Unknown | 37 | 1.32 (1.09–1.60) |

* Data from the Framingham Heart Study that studied a general population of 7814 participants for a mean of 17 years, demonstrated that 21.2% of first reports of syncope were vasovagal.²

If more aggressive treatment is required patients should be referred on. Further treatment may be guided by tilt table testing and implantable loop recording. Treatment options include β -blockers, volume expansion, vasoconstrictors such as midodrine, tilt table training and permanent pacing.

Orthostatic syncope

Orthostatic syncope is due to hypotension on assumption of upright posture and is responsible for 9% of first syncopal episodes.² This may be caused by:

- reduced intravascular volume, or
- inadequate vasoconstriction.

It is differentiated from neurally mediated reflex syncope by occurring soon after a change in posture and without bradycardia. It can be demonstrated by measuring blood pressure after resting supine for five minutes, and then at one and three minutes after standing. Orthostatic hypotension is defined as a decrease in the systolic blood pressure of ≥ 20 mmHg, or a reduction of systolic blood pressure to ≤ 90 mmHg on standing. The prognosis is generally good depending upon comorbidities and the underlying cause, the main risk being an elderly person sustaining an injury.

Reduced intravascular volume is commonly seen in patients treated with diuretics, with poorly controlled diabetes, following an alcoholic binge and acute haemorrhage.

Inadequate vasoconstriction is common in the elderly. It may be primary autonomic insufficiency but is more likely to be secondary. Common secondary causes include:

- prolonged bed rest
- peripheral neuropathy (eg. diabetic, alcoholic, amyloid), and
- drugs.

It is easy to recognise antihypertensives and vasodilators but many noncardiac drugs can also impair vasoconstriction, eg. amitriptyline, haloperidol, prazosin and L-dopa. In practice, successful treatment does not hinge on identifying the precise mechanism. Offending agents should be removed. Nonpharmacological measures such as increasing salt intake or wearing compression stockings should be tried. Pharmacological treatments are directed at increasing vascular volume (fludrocortisone) or increasing vascular tone (midodrine).

Cardiac syncope

Cardiac syncope is due to a sudden reduction in cardiac output. In the Framingham Heart Study 10% of first syncopal episodes were cardiac and these patients had double the risk of death from any cause.² The most important predictor of mortality is the severity of the underlying heart disease.³ Identification of the cause is important because specific therapy may be available. Cardiac syncope may have an arrhythmic or mechanical cause.

Rhythm disturbance

Either very slow or very fast heart rates can result in a marked decrease in cardiac output and reduce cerebral perfusion. Bradyarrhythmias result from disorders of the sinus node and/or atrioventricular conduction. Syncope may be precipitated by an ischaemic event or drugs that impair conduction, eg. beta blockers (including eye drops), digoxin, calcium channel blockers and anti-arrhythmic drugs. Treatment is withdrawal of the offending agent with permanent pacing often required.

Ventricular tachycardia is a frequent and serious cause of syncope that may progress to sudden cardiac death. Drug therapy alone may be sufficient, although some patients require an implantable pacemaker cardioverter defibrillator (ICD). Supraventricular tachyarrhythmias are an uncommon cause of syncope, usually at the onset or termination of the arrhythmia. Treatment options include drug therapy and possibly radio frequency ablation.

The diagnosis of an arrhythmic cause of syncope is often suggested by the baseline electrocardiogram (ECG) but is confirmed by symptom ECG correlation. If symptoms occur as frequently as once per week, a 24 hour Holter monitor may help in diagnosis. An external ECG event monitor such as King of Hearts[®] can monitor for a longer period of time, eg. two weeks. In difficult cases an implantable loop recorder is available. These are small devices that are implanted subcutaneously under local anaesthetic and last up to two years, thereby allowing symptom ECG correlation.

An invasive electrophysiology study can often predict the cause of syncope and in certain circumstances may be diagnostic. A normal study however, cannot completely exclude an arrhythmic cause.

Mechanical causes

Mechanical causes are in general obstructive lesions. It is important to identify symptomatic

aortic stenosis because of the poor prognosis without surgery. Other causes include hypertrophic cardiomyopathy, primary pulmonary hypertension, tetralogy of Fallot and left atrial myxoma.

In patients with a very recent onset of syncope, additional causes to consider are:

- myocardial infarction
- pulmonary embolism
- aortic dissection, and
- pericardial tamponade.

Mechanical causes are often suggested by history and examination findings and confirmed by standard investigations such as chest X-ray, ECG, ventilation perfusion scan (V/Q) and echocardiography. All the cardiac causes of syncope are serious and if a cardiac cause is suspected the patient should be referred to a specialist.

Unknown cause

In the Framingham Study 37% of first episodes of syncope were of an unknown cause.² This group has a worse outcome compared to patients without syncope, or with vasovagal syncope, with the adjusted hazard ratio for death 1.32 (95% CI: 1.09–1.60).² The adverse outcome may be due to unrecognised cardiac disease.

Driving

Driver syncope is an uncommon cause of motor vehicle accidents and specific guidelines have been developed.⁴ New Australian guidelines, ‘Assessing fitness to Drive’ are due to be released in 2003.⁵ For noncommercial drivers with neurally mediated reflex syncope, ie. vasovagal episodes driving is only restricted if symptoms are severe and uncontrolled. Specialist assessment is often required.

Conclusion

Dizziness and loss of consciousness are common clinical problems. If the initial assessment suggests presyncope or syncope as causing the symptoms, a detailed history, physical examination including postural blood pressure and 12 lead ECG will establish the diagnosis in many cases. Identifying cardiac disease is a priority because of its adverse prognosis. There are continuing advances in the diagnosis and treatment of syncope.

SUMMARY OF IMPORTANT POINTS

- Episodic loss of consciousness is usually due to seizure or syncope.
- A detailed history, physical examination including postural blood pressure, and 12 lead ECG will establish the diagnosis in many cases of syncope and presyncope.
- Identifying cardiac disease is a priority because the adverse prognosis can be improved by appropriate treatment.

Conflict of interest: none declared.

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