Sorting out syncope

Syncope is a common and serious diagnostic dilemma. A structured and systematic approach to assessment and management will help ensure that serious and treatable causes are not overlooked.

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Syncope is a common medical problem, with an incidence of 6.2 cases per 1000 person-years.[1] It accounts for up to 3% of presentations to the emergency department and 6% of hospital admissions.[2] Often, however, syncope is poorly diagnosed and treated, which is due, in part, to the myriad of possible causes and the variety of investigations as well as management options that are available.

Syncope can be described as a transient loss of consciousness accompanied by loss of postural tone and followed by full and rapid recovery of neurological function. Rapid recovery is an important feature, as it assists in differentiating syncope from seizures, transient ischaemic attacks (TIAs), Ménière's disease, hypoglycaemia and other metabolic disturbances that typically cause a loss of consciousness associated with prolonged recovery. Myoclonic jerks (at times repetitive), incontinence and tongue biting can all be a part of genuine syncope ('convulsive syncope'), and in the absence of a postictal phase should not be attributed to epilepsy.[3]

Syncope is associated with impaired cerebral perfusion rather than cerebral ischaemia. The general mechanism is decreased cardiac output secondary to cardiac arrhythmias, outflow obstruction, hypovolaemia, orthostatic hypotension or reduced venous return. Reduced perfusion of the reticular activating system in the brainstem and cerebral cortex causes loss of consciousness and of postural tone, respectively, resulting in the body slumping into a more horizontal position with consequent reduction in the negative impact of gravity on cerebral perfusion – a 'faint'.

Aetiology

Although the possible causes for syncope are many and varied (see Table 1), the most frequent causes are vasovagal (21.2%), cardiac (9.5%) and orthostatic (9.4%); the cause is undetermined for 36.6% of cases after appropriate investigations.[1] The prognosis for patients with syncope of undetermined cause is generally good; otherwise, the prognosis reflects that of the underlying condition.[4]

IN SUMMARY

- **Syncope is not a diagnosis in and of itself.**
- **In one-third of syncopal cases a cause is not found, in one-third the cause is benign, and in one-third there is a serious underlying cause.**
- **A diagnosis can be made in 50% of patients on history, examination and ECG alone.**
- **The clinician should aim to exclude serious causes of syncope and treat other causes as appropriate.**
- **Management can often be achieved through noninterventional means.**
- **An holistic approach is required for elderly patients with syncope.**

Table 1. Important causes of syncope

Serious causes

Cardiac

- Arrhythmias: heart block, Stokes–Adams attack (transient third degree heart block), ventricular tachycardia, supraventricular tachycardia, atrial fibrillation with rapid ventricular response
- Poor cardiac output: ischaemic heart disease, heart failure, cardiomyopathy
- Valvular disease: aortic stenosis, hypertrophic obstructive cardiomyopathy, tricuspid stenosis
- Pulmonary embolism

Vascular

- Aortic dissection or leaking aneurysm
- Blood loss and hypovolaemia (e.g. gastrointestinal bleeding)
- Vasodilatation: medications, autonomic neuropathy from diabetes or Parkinson's disease

Nonserious causes

Vasovagal

- Emotion, stress, pain or noxious stimuli that cause reduced vasomotor tone or vagal bradycardia
- Micturition and defaecation syncope secondary to the parasympathetic drive associated with these activities
- Cough or sneeze

Carotid hypersensitivity syndrome

- Increased vagal output from pressure on the carotid sinus (e.g. due to wearing a tight shirt collar or to reaching up to hang out washing)

Heat

Poor cardiovascular reserve and vasodilatation

Psychiatric

Anxiety disorders and hyperventilation

A useful rule of thumb for syncope is as follows:

- in one-third of cases a cause is not found (and the prognosis is good)
- in one-third of cases the cause is benign (vasovagal), and
- in one-third of cases there is a serious underlying cause.

The objectives of the attending clinician should be to exclude serious causes and treat others as appropriate.

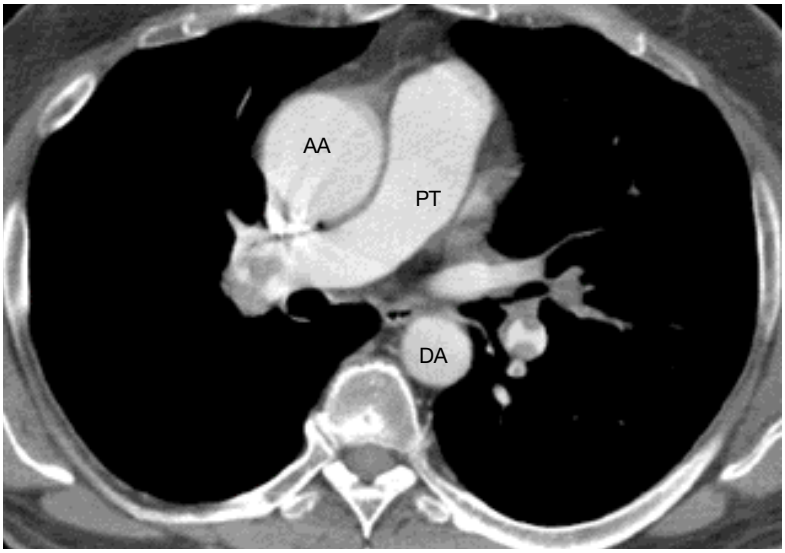

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Figure. A possible cause of syncope. CT pulmonary angiogram showing a bilateral proximal clot in a patient with extensive pulmonary emboli (AA=ascending aorta; DA=descending aorta; PT=pulmonary trunk).

Assessment

Taking a thorough history and performing a careful examination are the first steps in distinguishing between sinister and benign causes of syncope, and will assist in excluding other types of loss of consciousness.

History

It is essential to determine whether the patient is actually experiencing syncope, according to the description given above. The history should address the circumstances of the episode (or episodes) and presence of associated cardiovascular or respiratory symptoms (Table 1). It may be necessary to ask specifically what happened; a witness's account of this may be useful. Important aspects of the chronological history include:

- before the attack – warning, circumstances and abortive methods
- during the attack – loss of consciousness, movements, injury, cyanosis
- after the attack – confusion, drowsiness, amnesia.

Symptoms commonly encountered in presyncope include lightheadedness, weakness, giddiness, visual blurring and field narrowing, tinnitus, gastrointestinal symptoms, pallor or diaphoresis. Common features preceding a vasovagal syncope include salivation, diaphoresis or a rushing sound in the ears. It is worth asking whether the patient was supine or erect when the episode occurred – vasovagal syncope is unlikely if the patient was

Table 2. Key differential diagnoses for common syncopal signs

Sign	Diferential diagnoses
Duration	
Lasting seconds	Autonomic insufficiency, dysrhythmia
Lasting minutes	Hyperventilation, psychiatric disorders
Posture	
On rising	Autonomic insufficiency, hypovolaemia, low flow TIA
Recumbent	Dysrhythmia
Activity	
Exertional	Ischaemic dysrhythmia, ventricular tachycardia
Postexertional	Aortic stenosis, subaortic stenosis, bradyarrhythmia
Chest pain	Myocardial ischaemia, pulmonary embolus
Tachypnoea	Myocardial ischaemia, pulmonary embolus, hyperventilation
Palpitations	Tachyarrhythmia, anxiety
Parasthesiae	Hyperventilation

supine, and this position suggests cardiac syncope that may be associated with palpitations, chest pain or shortness of breath. However, cardiac or neurological causes of syncope may also occur without any warning to the patient. From a cardiac viewpoint, a sudden onset is more likely to be related to ventricular fibrillation or tachycardia, whereas a more gradual onset is more likely to be a supraventricular arrhythmia or to have vasovagal aetiology. Table 2 summarises some key differential aetiologies for common syncopal signs.

It is important to enquire about any fall that may have occurred in conjunction with the syncope. A subdural haemorrhage or other cerebral injury is all too commonly missed when it is secondary to a syncopal episode.

A review of the patient's current medications is important because drugs present a possibly reversible cause of syncope. It may be useful to ascertain how long the patient has been taking each medication and whether any changes have recently been made to the dosages.

Examination

The physical examination should focus on the cardiovascular, respiratory and neurological systems, looking for signs to support the historical features. It may be useful to see if syncope can be reproduced by the Valsalva manoeuvre or hyperventilation.[5] In the acute setting, the examination should initially focus on vital signs and consequences of trauma associated with the syncope.

Investigations

The yield from nondirected diagnostic testing for syncope is relatively low, and investigations are generally only necessary when a serious cause is suspected.[6] Overall, a diagnosis can be established in 45% of patients on the basis of the history and examination alone; an ECG improves this by 5%.[4] Potential syncopal investigations may include:

- ECG (for acute myocardial infarction, arrhythmia, long QT interval in young people)
- full blood count (for anaemia)
- urea, electrolytes and creatinine (for potassium abnormalities)
- arterial blood gases (if the patient is hypoxic or pulmonary embolus is likely)
- chest x-ray (for cardiomegaly and aortic arch dissection)
- CT or pulmonary angiography (if suspicion of pulmonary embolus is high) – see Figure
- Holter monitoring (for arrhythmia)
- echocardiography (for cardiac or valvular causes, especially if ECG is abnormal)
- tilt-table test (for autonomic dysfunction or vasovagal syncope).

Tilt-table testing for vasovagal syncope can be obtained from some large tertiary referral centres and captures 95% of patients who are vulnerable to fainting. It is useful in patients with syncope of unknown origin, although the specificity is only approximately 90%. Tilt-table testing is indicated where there is recurrent syncope and no evidence of structural heart disease, and it may be useful in ascertaining the efficacy of prophylactic medications. Tilt-table testing is not warranted for a patient with a single syncopal episode with clear vasovagal features, but it may have a role for differentiating convulsive syncope from true seizures.[7,8]

Patients with postural syncope but no blood pressure changes should have further vascular imaging, searching for significant vascular stenoses (particularly in the aortic and vertebrobasilar systems). Carotid stenosis is not associated with syncope.

The noninvasive transthoracic echocardiogram has a role in patients who have an abnormal ECG or cardiac findings on physical examination. Electrophysiological testing to determine the threshold for induction of atrial and ventricular dysrhythmias may be diagnostic and potentially therapeutic with radioablation or placement of a permanent automatic implanted defibrillator.[9] Electroencephalography is of little diagnostic value in unselected cases, but may be of use when seizure cannot be excluded.[5]

Management

Management options depend on whether a cause is identified and the nature of that cause. It can be helpful to categorise

patients as high risk for treatable or sinister causes if they are over 70 years of age or have a past history of cardiovascular disease or recurrent syncope (more than one episode in the last week) – this can help to ensure they are more aggressively investigated and managed.

The following points may be useful in managing certain types of syncope.

- Patients should be educated about avoiding triggering events, aversion manoeuvres and recognising presyncopal symptoms.
- Increasing salt and fluid intake may be helpful in alleviating orthostatic symptoms, particularly on waking. Tight stockings may also help with this.
- There should be a review and possible adjustment of current medications (particularly cardiac medications), which may have actions or interactions that alter salt and fluid balance.
- Patients who hyperventilate may respond to rebreathing into a paper bag to normalise arterial pCO_2.
- Recurrent ventricular fibrillation responds poorly to medications, and treatment needs to be tailored to the individual with electrophysiological studies. Implantation of an automatic defibrillator has been shown to enhance survival and is well tolerated. Cardiac pacing may be appropriate in patients with cardioinhibitory vasovagal or mixed carotid sinus syndrome.[10,11]
- Patients with anxiety-based syncope can be reassured that the condition is benign and should be taught avoidance strategies and anxiety management.
- Medication manipulation may alleviate autonomic insufficiency, even if caused by an underlying neurodegenerative disorder. First line drug therapy involves beta blockers and alfa agonists, but discontinuation or alteration of antihypertensive agents may be required.
- Patients with serious or complex causes for their syncope should be referred to the relevant specialist.
- Medications such as beta blockers, alfa agonists, fludrocortisone (Florinef) and selective serotonin reuptake inhibitors may be useful in the treatment of syncope.[12] However, these agents should be used judiciously and generally after specialist review.

Syncope in the elderly

Transient loss of consciousness in the elderly often presents a more difficult diagnostic challenge than that in younger patients. The incidence of syncope increases with age;[14] in general, disease and medication use dominate the causes for syncope in elderly patients, but physiological and pathophysiological changes make them more susceptible to syncope by altering ability to sense and respond to volume deficiency and hypotension. Elderly people are often predisposed to volume deficiency through reduced access to fluids (immobility), modified diets (for example, if using thickened fluids for impaired swallowing) and altered cognition (which is associated with reduced food and fluid intake).

In younger patients, a single factor may be identified as causing syncope, but in the elderly – particularly the frail elderly – a combination of factors may summate to present as syncope (e.g. mild hypovolaemia, cardiac medications and disease combined with a urinary tract infection). Management is, therefore, likely to require a multifactorial approach (i.e. a package of care), rather than a single intervention.

Prognosis

The prognosis for patients with syncope generally reflects the prognosis for the underlying cause. Vasovagal syncope seems to have a benign prognosis, but patients with syncope from any other cause are at increased risk of death.[1] The one-year mortality for cardiac syncope approaches 30%.

Predictors of an adverse outcome in syncope include a history of ventricular arrhythmias, an abnormal ECG in the emergency department, age over 45 years, and a history of congestive cardiac failure. Patients with such features may require admission to hospital.[13]

Conclusion

Syncope is a common medical problem that has many possible causes. A structured and systematic approach to diagnosis and management will help to ensure that serious and treatable causes are not overlooked. Often no cause is found, but the reader is cautioned against adopting the attitude of 'What you don't know won't hurt you'. Patients with syncope that escapes causal attribution should be followed carefully until serious causes can be excluded. MT

A list of references is available on request to the editorial office.

DECLARATION OF INTEREST: None.

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References

1. Soteriades ES, Evans JC, Larson MG, et al. Incidence and prognosis of syncope. N Engl J Med 2002; 347: 878-885.
2. Day SC, Cook EF, Funkenstein H, Goldman L. Evaluation and outcome of emergency room patients with transient loss of consciousness. Am J Med 1982; 73: 15-23.
3. Kempster PA, Balla JI. A clinical study of convulsive syncope. Clin Exp Neurol 1986; 22: 53-55.
4. Linzer M, Yang EH, Estes NA 3rd, Wang P, Vorperian VR, Kapoor WN. Diagnosing syncope. Part 2: unexplained syncope. Clinical Efficacy Assessment Project of the American College of Physicians. Ann Intern Med 1997; 127: 76-86.
5. Hoefnagels WA, Padberg GW, Overweg J, Roos RA, van Dijk JG, Kamphuisen HA. Syncope or seizure? The diagnostic value of the EEG and hyperventilation test in transient loss of consciousness. J Neurol Neurosurg Psychiatry 1991; 54: 953-956.
6. Eagle K, Black H. The impact of diagnostic tests in evaluating patients with syncope. Yale J Biol Med 1983; 56: 1-8.
7. Benditt DG, Ferguson DW, Grubb BP, et al. Tilt table testing for assessing syncope. American College of Cardiology. J Am Coll Cardiol 1996; 28: 263-275.
8. Fitzpatrick AP, Theodorakis G, Vardas P, Sutton R. Methodology of head-up tilt table testing in patients with unexplained syncope. J Am Coll Cardiol 1991; 17: 125-130.
9. Moss AJ, Hall WJ, Cannom DS, et al. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. Multicenter Automatic Defibrillator Implantation Trial Investigators. N Engl J Med 1996; 335: 1933-1940.
10. Chen-Scarabelli C, Scarabelli TM. Neurocardiogenic syncope. BMJ 2004; 329: 336-341.
11. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. N Engl J Med 1997; 337: 1576-1583.
12. Brignole M, Alboni P, Benditt D, et al; Task Force on Syncope, European Society of Cardiology. Guidelines on management (diagnosis and treatment) of syncope. Eur Heart J 2001; 22: 1256-1306.
13. Martin TP, Hanusa BH, Kapoor WN. Risk stratification of patients with syncope. Ann Emerg Med 1997; 29: 459-466.
14. Kapoor W, Snustad D, Peterson J, Wieand HS, Cha R, Karpf M. Syncope in the elderly. Am J Med 1986; 80: 419-428.