



SYNCOPE

Syncope is defined as a transient loss of consciousness associated with loss of muscle tone. Subsequent recovery is normally spontaneous and complete. It is a common disorder affecting 30% of adults at some point in their lives and is responsible for up to 6% of attendances at Accident and Emergency Departments and 1% of hospital admissions. Syncope is particularly common in adolescence and early childhood and there is a second peak in people in their sixties and seventies. Patients may experience injuries related to an episode and these can adversely affect employment and personal freedom, for example by making them unfit to drive.

Syncope is a challenging puzzle, but fortunately most instances improve spontaneously. The diagnosis is dependent on a good clinical history, ideally supported by the account of a witness, careful physical examination paying particular attention to the blood pressure lying and standing and, where necessary, referral for further investigations. Treatment is dependent on identifying the underlying cause of these attacks.

Causes of syncope¹

Syncope is caused by transient loss of brain function because of inadequate blood flow. The latter is due to one of the following:

- Irregularities of heart rate and rhythm, eg heartblock, tachyarrhythmias
- Lesions in the heart which obstruct blood flow, eg aortic stenosis
- Marked falls in blood pressure (BP)

If a fall in BP is prolonged patients may experience other features including incontinence of urine or faeces and epileptic-like activity. The principal differential diagnoses are epileptic seizures or other brain disorders such as transient ischaemic attacks and hypoglycaemia.

In young people **neurocardiogenic (vasovagal) syncope** is the most common form. This is an exaggerated form of fainting which can be due to a marked drop in blood pressure.^{2,3} Sometimes the heart rate is slowed markedly, but more commonly these attacks are

accompanied by an increase in heart rate. Several explanations have been advanced. One is that there is an excessive pooling of blood in the veins in the lower limbs because of abnormalities in them. An alternative is that it is due to abnormal regulation of venous tone secondary to a central nervous abnormality.

Neurocardiogenic syncope can be situational in which episodes are triggered by passing urine, coughing, defecation or swallowing. It can also occur in circumstances which would often induce fainting: these include standing, fasting, injections, visits to the dentist, alcohol excess etc. In a small minority of people, syncopal episodes occur "out of the blue" without any warnings of faint feelings or dizziness beforehand. Such episodes are frequently associated with injury and if recurrent may necessitate insertion of a pacemaker.

Postural tachycardia syndrome manifests as an excessive increase in heart rate during standing, but with little reduction in blood pressure. People with this syndrome usually experience "near faints", fatigue, dizziness and are intolerant to exercise.

Orthostatic hypotension is a reduced ability to maintain blood pressure during standing. It tends to occur with advancing years, but can be aggravated by medication taken for high blood pressure, heart disease and depression. It can also be due to other psychotropic agents and levodopa, as well as being aggravated by alcohol.

Carotid sinus syndrome⁴ is a common cause in older people and can be aggravated by medications such as beta blockers: it will normally respond to their withdrawal.

Diagnosis

The diagnosis of syncope can be difficult because of its periodic and unpredictable nature. The ideal way is to determine what is happening to the heart rate and blood pressure during spontaneous episodes, but doctors often have to rely on the history, examination and abnormal laboratory tests in order to infer a diagnosis.

Patients with syncope use a variety of terms to describe their symptoms and these include dizziness, faintness, light-headedness, blackouts, head-rushing, hip-spinning and a sense of falling or flying. Those with neurocardiogenic syncope will typically describe a feeling of warmth associated with sweating and a sense of "greying out". Others describe problems with vision and odd sensations around the face or mouth, as well as pain in the neck and across the shoulders before blacking out. These symptoms reflect the gradual drop in blood pressure and over-activity of some nerves (and muscles) to compensate for the drop in blood pressure. Most of these episodes occur when people are standing and when an episode occurs they are unresponsive for less than a minute, but may complain of fatigue for many hours thereafter. In people who have Stokes Adams Attacks (syncope due to transient heart block) there is often very little warning and they collapse suddenly: witnesses may remark that the patient went very pale, but afterwards flushed profusely. By contrast, people who have tachyarrhythmias such as paroxysmal atrial fibrillation (Factfile 11/2000) may experience palpitations before the blackout.

These features in the history should be broadened to include questioning on the frequency of such episodes and triggering events. It is important also to determine whether or not there is a family history of such episodes and medications should be reviewed. Physical examination should include measurement of blood pressure, both lying and standing, and the heart must be examined for any valvular abnormalities, as well as its rate and rhythm.

Further investigations

Often a diagnosis can be made from the patient's history, examination and the manoeuvres previously outlined. If the examination or ECG suggests

underlying heart disease, patients may require electrophysiology studies. If neurocardiogenic syncope is suspected, patients can undergo a head up tilt⁵ on a special bed for a period of 40 minutes. Blood pressure and heart rate are monitored with the person in 70° head up position and medications may be given to trigger an attack. Carotid sinus syndrome can be reproduced by simple firm massage over the carotid sinus for a period of 5 seconds whilst recording heart rate and blood pressure.

If syncope remains unexplained after these procedures, longer-term (24-48 hours) external (Holter) monitoring will be undertaken in order to determine heart rate and rhythm. Alternatively, small devices known as loop recorders which are about the size of a pacemaker can be implanted under the skin of the chest wall.

Treatments

Simple conservative measures will cure most people. These include ensuring adequate fluid and salt intake and the avoidance of circumstances which cause or aggravate their symptoms. In addition they should be advised to sit or lie down when they experience warning symptoms. Venous support stockings can help if they fit well and it is important to reassess medications which may need either reduction in dosage or sometimes their withdrawal. In neurocardiogenic syncope additional measures include oral fludrocortisone to increase the volume of blood within the veins or midodrine to constrict the veins and make people less likely to lose consciousness when they stand. For those with tachyarrhythmias, betablockers or calcium channel blockers such as diltiazem or verapamil are helpful and ablation therapy may be necessary to destruct abnormal pathways conducting the fast heart rates (Factfile 11/2000 and 12/2000).

References

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