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The assessment of blackouts

Although most transient episodes of loss of consciousness are benign, they may signal serious cardiac or brain pathology or be a symptom of an illness not primarily involving the cardiovascular or central nervous systems.

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Transient loss of consciousness or a 'blackout' is a common symptom and one that can be extremely alarming to onlookers. Although there are numerous causes, most are accounted for by syncope and epileptic seizures. These two conditions have very different implications, and so it is important that they are correctly diagnosed.

While a typical simple syncopal attack or faint is easy to distinguish from a generalised tonic–clonic seizure, their differentiation may be more challenging when syncope produces convulsive movements or when seizures do not.

Mechanisms

Although the clinical manifestations of syncope and seizures may be similar, their pathophysiology could scarcely be more different. Syncope refers to loss of consciousness resulting from a fall in global cerebral blood flow, whereas seizures are due to a sudden excessive discharge of cortical neurones. The cerebral cortex in syncope is inactive due to inadequate blood flow, whereas in seizures, it is overactive and has (secondarily) increased blood flow.

The abnormal cerebral activity of a seizure may begin in a localised area of cerebral cortex, producing a focal (partial) seizure, or it may begin in both hemispheres simultaneously, producing a primary generalised seizure. When focal seizures are associated with impairment of consciousness, they are referred to as complex partial seizures. Simple partial seizures refer to those in which consciousness remains intact. During a focal seizure, the abnormal cerebral activity may spread to the opposite hemisphere, resulting in a secondarily generalised tonic–clonic seizure.

Causes and provoking factors

Both syncope and seizures have many causes and provoking factors, as shown in Tables 1 and 2.

The most common type of syncope is vasovagal (Figure) and affects people with no other detectable cardiovascular disorder. The patient experiences bradycardia and/or hypotension after a variety of stimuli or in particular situations (Table 1). Intrinsic cardiac disease can also cause syncope, which is often the initial manifestation and in people with cardiac arrhythmias, may be the only

IN SUMMARY

- **Most blackouts are caused by syncope or seizure.**
- **Syncope and seizure can usually be confidently distinguished by the history.**
- **Description by a witness to the event is an essential part of the history.**
- **Syncope is often misdiagnosed as seizure when it is accompanied by convulsive movements.**
- **Syncope is usually benign and does not need investigation, but when certain features are present, cardiac disorders, especially arrhythmias, should be excluded.**
- **Seizures have a wide range of causes and affected patients require neurological referral.**

Table 1. Syncope: examples of causes and provoking factors

Hot shower or bath
Sleep deprivation
Fasting
Pregnancy
Standing immobile
Sudden standing
Seeing blood or surgery
Venepuncture
Trauma, often minor
Pain
Alcohol use
Menstruation
Cough
Micturition
Diarrhoea
Vomiting
Emotional events (weddings, funerals, hospitals, horror movies)
Hot, stuffy environment
Anaemia
Any systemic illness, especially infections
Carotid hypersensitivity
Cardiac disorders – arrhythmia, cardiac failure, obstruction
Hypotension – resulting from hypovolaemia (bleeding, dehydration), drugs (especially antihypertensives), sepsis

Table 2. Seizures: examples of causes and provoking factors*

Genetic factors
Focal brain lesions
Stroke
Tumour
Abscess
Vascular malformations
Congenital developmental lesions
Trauma
Nonfocal brain disorders
Infection (meningitis, encephalitis)
Alzheimer's disease
Systemic disorders/factors
Medication/drug use or withdrawal, especially from alcohol and benzodiazepines
Metabolic disorders (low sodium, glucose or calcium levels or renal failure)
Provoking factors in susceptible individuals
Sleep deprivation
Fever in infants (febrile seizures)

* The cause may be present at the time of presentation or may have occurred months or years earlier – for example, patients with meningitis may experience seizures during the acute infection or experience their first attack some years later.

manifestation other than sudden death.

Epileptic seizures may accompany acute disorders, such as metabolic derangements, intoxication, infection or drug withdrawal, or may take the form of a chronic disorder (epilepsy). Epilepsies may have a genetic basis (with patients presenting with primary generalised seizures) or result from almost any form of cerebral pathology (causing focal seizures). In either case, the seizure may be the first and only manifestation of the underlying disorder.

Diagnosis of syncope and seizures

History

Since doctors do not usually witness the transient events of loss of consciousness, the diagnosis relies on the history. As the patient is unconscious during the episode, it is essential to try to speak to a witness. Telephone calls to any available witnesses are well worth the effort. The history can be logically and usefully divided according to the temporal sequence of events:

- the circumstances
- the prodrome
- the period of unconsciousness
- the period after the event.

Circumstances

Except in patients with the rare reflex epilepsies, seizures are apparently spontaneous, occurring at any time and in any situation, including sleep. In contrast, syncope, particularly vasovagal syncope, characteristically occurs in particular circumstances, such as those listed in Table 1. Often, the coincidence of several provoking factors causes syncope. For example, syncope associated with gastroenteritis may result from a combination of dehydration, fever, abdominal pain and the person getting out of bed to rush to the bathroom. Syncope due to cardiac arrhythmias, however, is often unprovoked, whereas syncope due to structural cardiac disease may be provoked by exertion.

Prodrome

Before syncope, patients usually feel lightheaded or faint and may feel that they need to sit down or their legs will give way. This is often described as 'dizziness', and it is important to distinguish this from vertigo, which is also referred to as 'dizziness', but which implies dysfunction of the central or peripheral vestibular system. Before syncope, vision may dim or fade and sounds may appear distant. Patients may feel hot. They may remember the start of the fall.

The symptoms immediately preceding the loss of consciousness of a focal seizure (the 'aura') vary greatly and depend on the site of seizure origin within the brain. For example:

- seizures arising in the occipital cortex may begin with visual hallucinations of colours, dots, shapes, flickering or visual distortion
- those arising in the motor cortex characteristically produce focal muscle twitching
- seizures that arise in sensory areas may begin with focal paraesthesia
- seizures that begin in the temporal lobe can produce a wide variety of symptoms, including déjà vu, a rising epigastric sensation or, very often, a

sensation that the patient finds difficult to put into words.

During both syncope and focal seizures, premonitory symptoms may at times occur in isolation rather than proceeding to loss of consciousness. In syncope, this occurs when the reduction in cerebral perfusion is insufficient to produce loss of consciousness, whereas in seizures, preservation of consciousness is due to failure of the seizure activity to spread to contralateral cortex.

Primary generalised seizures begin without warning because both cerebral hemispheres are involved simultaneously. However, patients with focal seizures evolving to secondarily generalised tonic–clonic seizures may not remember the aura, and if focal manifestations do not occur, it may be impossible to know if the seizure was a primary generalised seizure or secondarily generalised (i.e. beginning as a focal seizure). This distinction is important in guiding investigations and therapy and determining prognosis.

Table 3. Historical features useful in differentiating convulsive syncope from seizure

Feature	Convulsive syncope	Epileptic seizure
Occurs in specific circumstances	Usually	Rarely
Premonitory symptoms – Present – Content	 Almost always Lightheaded, faint	 Common but can be absent Specific or indescribable
Occurs while sitting or lying	Rarely	Common
Duration of amnesia	Seconds; patients usually recall waking where they fell	Minutes; often patients' first recollection is ambulance officers or hospital
Scream or moan at onset	No	Common
Complexion	Extreme pallor	Cyanosis
Sweating	Clammy	Mild, if any
Duration of convulsion	Less than 30 seconds	1-2 minutes
Tongue biting	Rare	Common
Postconvulsive appearance	Dead	Alive
Confusion after the event	None or lasting less than 30 seconds	Lasting more than 2 minutes

The period of unconsciousness

During syncope, there is usually marked pallor and witnesses may believe that the patient has died. There may be profuse sweating. Most syncopal attacks last only a few seconds. Some form of convulsive movement (either tonic or clonic) is very common and may lead to an erroneous diagnosis of seizure. When these convulsive movements are prominent, the term convulsive syncope may be used. Such convulsions do not have an epileptic basis and probably arise from the brain stem rather than the cerebral cortex, which is electrically silent during syncope. Antiepileptic medication is therefore not helpful. Convulsive syncope is sometimes assumed to be an epileptic seizure triggered by cerebral ischaemia, but this is an extremely rare phenomenon.

Generalised tonic–clonic seizures usually begin with a cry or moan, followed by stiffening of all limbs, with superimposed jerking. There is cyanosis, dribbling of saliva, which is sometimes frothy, and often tongue biting. The usual duration is about one minute but is usually significantly overestimated by witnesses.

Incontinence, while more common in seizures, occurs sufficiently often with syncope to be an unhelpful distinguishing feature.

Table 3 lists a number of features that

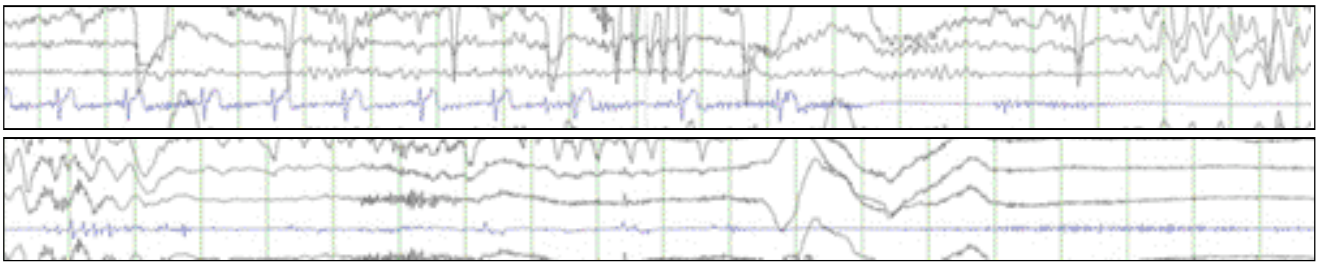


Figure. ECG and EEG during vasovagal syncope in a 26-year old man incorrectly diagnosed as having epilepsy. This episode was provoked by venepuncture. The upper box represents the first 20 seconds of the episode, the lower box represents the next 20 seconds of the episode. The ECG (blue trace) shows sinus bradycardia, then asystole. The EEG (black traces) shows a burst of slow waves as the patient loses consciousness, then flattening. Vertical lines mark 1 second intervals.

are useful in distinguishing convulsive syncope from generalised tonic–clonic seizures.

The period after the event

Following syncope, there is a rapid return to full consciousness, with no more than a few seconds of confusion, if any. If directly asked, patients often report hallucinations or feel they have been dreaming as they wake.

Immediately after a generalised tonic–clonic seizure, patients often experience snoring respiration and unconsciousness, followed by confusion for several minutes or more. Patients feel exhausted and will often sleep.

Examination

Patients with syncope require a careful cardiovascular examination, including measurement of lying and standing blood pressures. The aim is to identify postural hypotension, abnormalities of heart rate or rhythm and structural heart disease. Further examination will depend on the presence of noncardiovascular symptoms, such as abdominal pain, vomiting, diarrhoea, cough, urinary symptoms or fever.

Patients with epileptic seizures require a general neurological examination. Non-focal neurological signs, such as delirium, may point to an acute systemic illness or intracranial infection, whereas focal neurological signs raise the suspicion of a focal neurological lesion. However, the neurological examination in most patients presenting with seizures is normal. As with syncope, the presence of non-neurological symptoms, such as weight loss, haemoptysis or fever, will dictate which other systems need to be examined.

Investigation

Differentiating seizures from syncope

Few investigations will reliably differentiate syncope from seizure. One approach in patients with uncertain episodes is to conduct the relevant investigations for both syncope and seizures, as described below. This approach will be useful only if an abnormality is found that is specific to one of these conditions – for example:

- complete heart block or a long QT-interval seen on an ECG
- generalised spike-wave activity on an EEG.

Unfortunately, although epileptiform abnormalities on EEGs are rarely seen in people without epilepsy, about half of those with epilepsy do not show such abnormalities on their routine EEG. Thus, a normal EEG does not provide strong evidence against a diagnosis of seizure, just as a normal ECG does not exclude syncope.

Tilt table testing may provoke a syncopal attack, but because this procedure can provoke syncope in subjects with no

history of this condition, a positive study is not diagnostic unless the provoked episode is identical to the presenting episode.

Another approach is to try to record a blackout with video, audio, EEG and ECG recording. This is expensive, labour-intensive and limited to video-EEG monitoring units in tertiary epilepsy centres. To capture an episode with reasonable certainty during a five-day recording, the frequency of episodes will need to be at least once every five days. If the episodes are predictable, recording can be scheduled accordingly. If the episodes can be provoked, this can be done during video-EEG monitoring.

Fortunately, most blackouts can be correctly diagnosed as syncope or seizures simply by taking a detailed history and speaking to a witness.

Investigations for all patients

Routine tests for all patients who have experienced a blackout include full blood counts and measurements of serum electrolytes, urea, creatinine, calcium and glucose concentrations.

Investigations for syncope

As well as routine blood tests, a resting ECG should be performed in patients with syncope. If syncope occurred in a situation typically associated with vasovagal syncope, further investigation (other than might be dictated by additional symptoms) is rarely needed. When syncope occurred for no apparent reason, with exertion, or while seated or supine, or if there is evidence of cardiac disease on history, examination or ECG, the patient should be referred to a cardiologist. Investigations will then usually include:

- echocardiography
- exercise stress test
- sometimes, electrophysiological studies.

Holter monitoring may be useful, but a negative study does not exclude a cardiac arrhythmia unless one of the patient's typical attacks occurred during the recording. Implantable loop recorders, which can record ECG data for many months, are sometimes used to capture an elusive arrhythmia. Production of syncope with tilt table testing is often interpreted as evidence for vasovagal syncope, but, as mentioned above, can occur in subjects with no history of syncope or in those with other more serious causes of syncope.

Investigations for seizure

Patients who have had seizures require neurological evaluation, including an EEG, in addition to the routine blood tests (as for all patients who have had blackouts). Unless the EEG shows generalised spike-wave discharges, indicating that the seizure was a primary generalised seizure, neuroimaging should be performed, preferably with MRI.

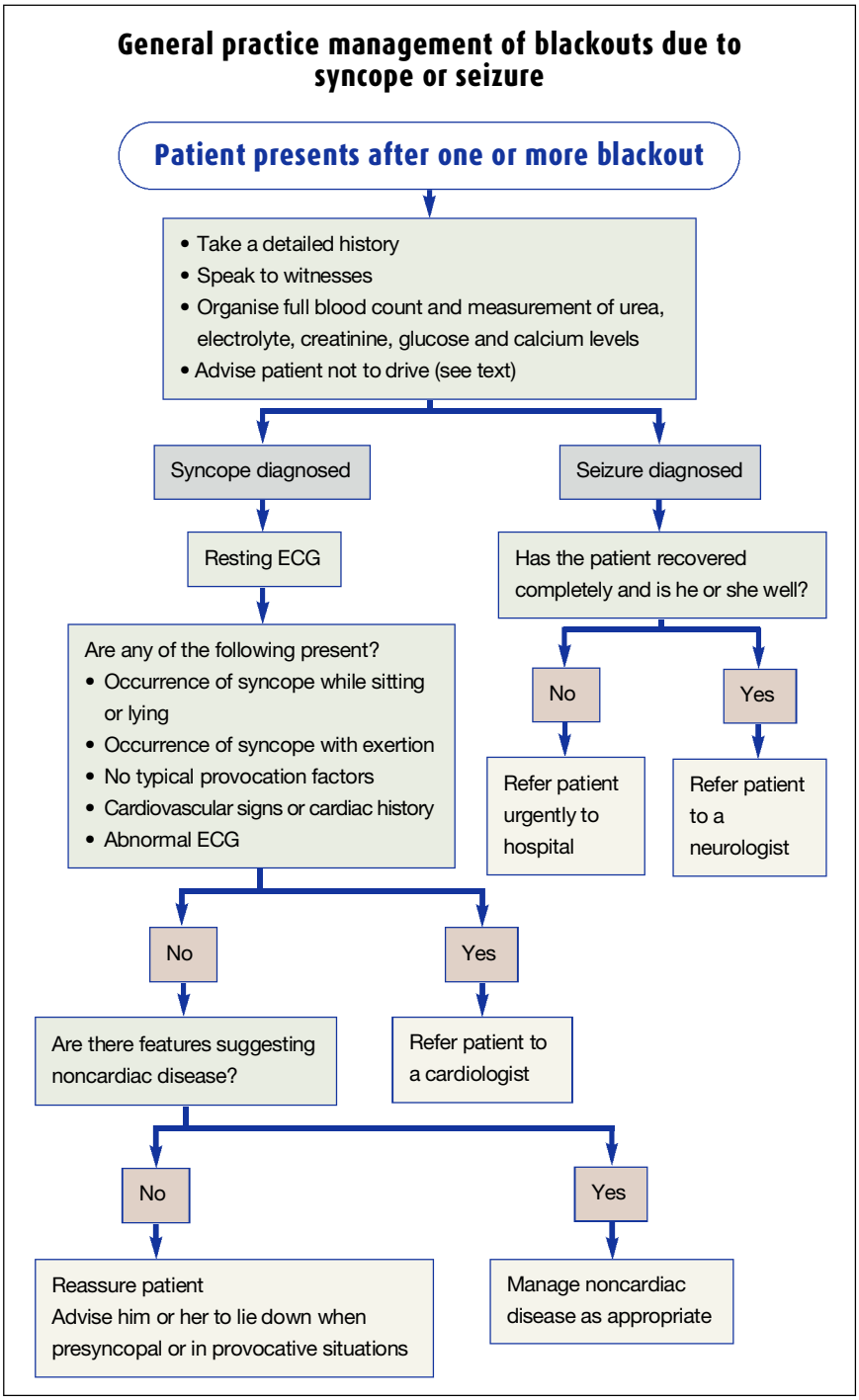
continued

When to refer

Most patients with syncope do not require referral to a cardiologist; however, all patients with seizures should be referred to a neurologist (see the flowchart below).

Driving

Patients with blackouts of any cause should be advised to stop driving. The duration of the nondriving period depends on the situation. The national standards published in *Assessing Fitness to Drive 2003*,[1] which all Australian State and Territory driver licensing authorities endorse, are inconsistent, with a nondriving period for patients with syncope varying from two to six months.



Conclusion

A careful history, including an interview of a witness, will lead to an accurate diagnosis of syncope or seizure in most patients who have had a blackout. Investigations are less useful in making the diagnosis but are essential in selected cases to detect the cause of the syncope or seizure, which may require treatment in its own right. MT

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Further reading

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