Syncope

the same.

Seizures can cause sudden LOC but are not considered syncope. However, seizures must be considered in patients presenting for apparent syncope because history may be unclear or unavailable, and some seizures do not cause **tonic-clonic convulsions**. Furthermore, a brief (<5 sec) seizure sometimes occurs with true syncope.

Diagnosis depends on a careful history, eyewitness accounts, or fortuitous examination during the event.

Pathophysiology

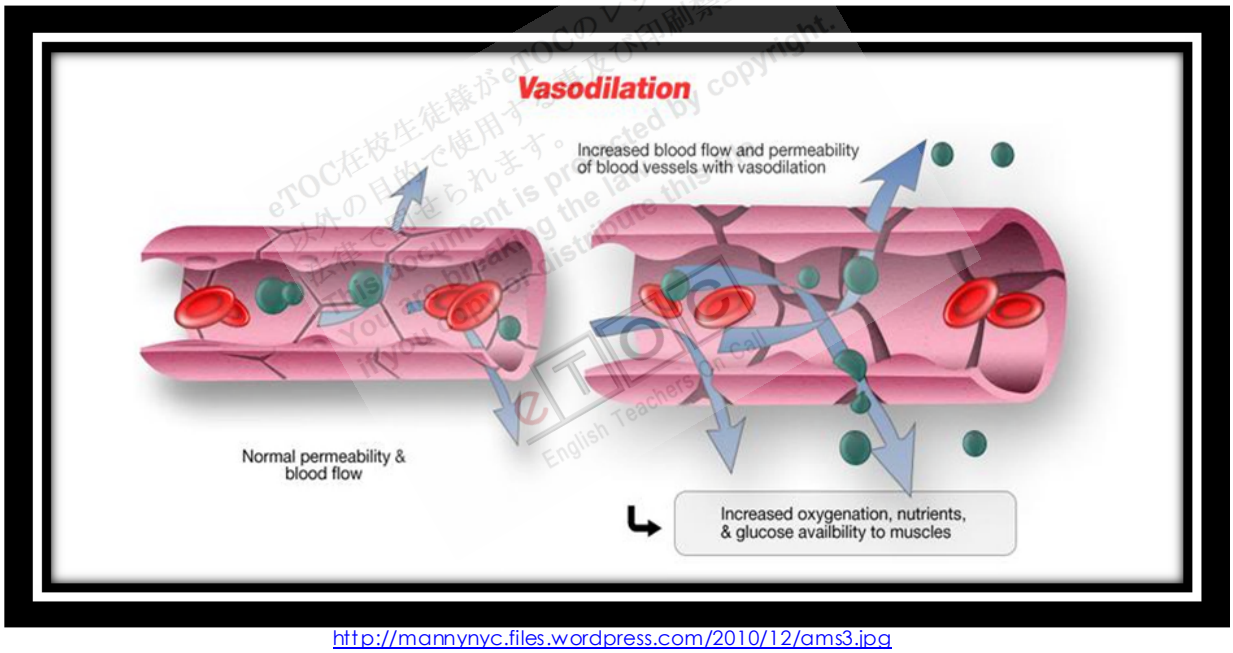
Most syncope results from insufficient cerebral blood flow. Some cases involve adequate flow but with insufficient cerebral substrate (O_2, glucose, or both).

Insufficient cerebral blood flow: Most deficiencies in cerebral blood flow result from decreased cardiac output (CO).

Decreased CO can be caused by

- Cardiac disorders that obstruct outflow
- Cardiac disorders of systolic dysfunction
- Cardiac disorders of diastolic dysfunction
- Arrhythmias (too fast or too slow)
- Conditions that decrease venous return

Outflow obstruction can be exacerbated by exercise, **vasodilation**, and **hypovolemia** (particularly in **aortic stenosis** and **hypertrophic cardiomyopathy**), which may precipitate syncope.



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Arrhythmias cause syncope when the heart rate is too fast to allow adequate ventricular filling (eg > 150 to 180 beats/min) or too slow to provide adequate output (eg, < 30 to 35 beats/min).

Venous return can be decreased by hemorrhage, increased **intrathoracic pressure**, increased vagal tone (which can also decrease heart rate), and loss of sympathetic tone (eg, from drugs, carotid sinus pressure, autonomic

dysfunction). Syncope involving these mechanisms (except for **hemorrhage**) is often termed **vasovagal** or **neurocardiogenic** and is common and **benign**.

Orthostatic hypotension, a common benign cause of syncope, results from failure of normal mechanisms (eg, sinus tachycardia, vasoconstriction, or both) to compensate for the temporary decrease in venous return that occurs with standing.

Cerebrovascular disorders (eg, strokes, transient ischemic attacks) rarely cause syncope because most of them do not involve the **centrencephalic structures** that must be affected to produce LOC. However, basilar artery ischemia, due to transient ischemic attack or migraine, may cause syncope. Rarely, patients with severe cervical arthritis or **spondylosis** develop **vertebrobasilar** insufficiency with syncope when the head is moved in certain positions.

Insufficient cerebral substrate: The CNS requires O_2 and glucose to function. Even with normal cerebral blood flow, a significant deficit of either will cause LOC. In practice, **hypoglycemia** is the primary cause because hypoxia rarely develops in a manner causing abrupt LOC (other than in flying or diving incidents). LOC due to hypoglycemia is seldom as abrupt as in syncope or seizures because warning symptoms occur (except in patients takingβ-blockers); however, the onset may be unclear to the examiner unless the event was witnessed.

Etiology

Causes are usually classified by the mechanism.

The **most common causes** are

- **Vasovagal**
- **Idiopathic**

Many cases never have a firm diagnosis but lead to no apparent harm. A smaller number of cases have a serious cause, usually cardiac.

Table 7

Some Causes of Syncope

Cause	Suggestive Findings	Diagnostic Approach*

Cardiac outflow or inflow obstruction		
Valvular disease: **Aortic** or **mitral stenosis**, **tetralogy of Fallot**, **prosthetic valve dehiscence** or **thrombosis**	Young or old patient Syncope often exertional; recovery prompt Heart murmur	Echocardiography
Hypertrophic cardiomyopathy, **restrictive cardiomyopathy**, **tamponade**, **myocardial rupture**	Young or old patient Syncope often exertional; recovery prompt Heart murmur	Echocardiography
Cardiac tumors or **thrombi**	Syncope may be positional Usually a murmur (possibly variable) Peripheral embolic phenomena	Echocardiography
Pulmonary embolism, **amniotic fluid embolism**, or, rarely, **air embolism**	Usually from large embolus, accompanied by dyspnea, tachycardia, or tachypnea Often risk factors for pulmonary embolism	D-Dimer CT angiography or nuclear scan
Cardiac arrhythmia		
Bradyarrhythmias (eg, from sick sinus syndrome, high-	Syncope occurring without warning; recovery	If ECG unclear, consider Holter monitor or event

grade atrioventricular block, drugs†)	immediate on awakening May occur in any position Bradyarrhythmias more common in the elderly Patient taking drugs, especially antiarrhythmics or other cardiac drugs Structural heart disease	recorder **Electrophysiologic testing** if abnormalities detected or high suspicion Serum electrolytes if clinical reason for abnormality (eg, diuretic use, vomiting, diarrhea)
Tachyarrhythmias, either **supraventricular** or **ventricular** (eg, from **ischemia**, heart failure, **myocardial disease**, drugs†, electrolyte abnormalities, **arrhythmogenic** RV **dysplasia**, long QT syndrome, **Brugada syndrome**, preexcitation)	Syncope occurring without warning; recovery immediate on awakening May occur in any position Patient taking drugs, especially antiarrhythmics or other cardiac drugs Structural heart disease	If ECG unclear, consider Holter monitor or event recorder Electrophysiologic testing if abnormalities detected or high suspicion Serum electrolytes if clinical reason for abnormality (eg, diuretic use, vomiting, diarrhea)
Ventricular dysfunction		
Acute MI, **myocarditis**, systolic or diastolic dysfunction, **cardiomyopathy**	Syncope a rare presenting symptom of MI (most such patients are	Serum troponin ECG Echocardiography Sometimes cardiac

	elderly), with arrhythmia or shock	MRI
Pericardial tamponade or **constriction**	Jugular venous elevation; pulsus paradoxus > 10	Echocardiography Sometimes CT
Vasovagal (neurocardiogenic)		
Increased **intrathoracic pressure** (eg, tension **pneumothorax**, cough, straining to urinate or defecate, Valsalva maneuver)	Warning symptoms (eg, dizziness, nausea, sweating); recovery prompt but not immediate (5 to 15 min or longer) Precipitant usually apparent	Clinical evaluation
Strong emotion (eg, pain, fear, sight of blood)	Warning symptoms (eg, dizziness, nausea, sweating); recovery prompt but not immediate (5 to 15 min) Precipitant usually apparent	Clinical evaluation
Carotid sinus pressure	Warning symptoms (eg, dizziness, nausea, sweating); recovery prompt but not immediate (5 to	Clinical evaluation

	15 min) Precipitant usually apparent	
Swallowing	Warning symptoms (eg, dizziness, nausea, sweating); recovery prompt but not immediate (5 to 15 min) Precipitant usually apparent	Clinical evaluation
Anaphylaxis	Drug administration, insect bite, allergy history	Allergy testing
Orthostatic hypotension		
Drugs†	Symptoms developing within several minutes of assuming upright position Drop in BP with standing during examination	Clinical evaluation Sometimes tilt table testing
Autonomic dysfunction	Symptoms developing within several minutes of assuming upright position Drop in BP with standing during	Clinical evaluation Sometimes tilt table testing

	examination	
Deconditioning caused by prolonged bed rest	Symptoms developing within several minutes of assuming upright position Drop in BP with standing during examination	Clinical evaluation Sometimes tilt table testing
Anemia	Chronic fatigue, sometimes dark stools, heavy menses	CBC
Cerebrovascular		
Basilar artery transient ischemic attack or stroke	Sometimes cranial nerve deficits and ataxia	CT or MRI
Migraine	Aura with visual symptoms, photophobia; unilateral	Clinical evaluation
Other		
Prolonged standing	Apparent by history; no other symptoms	Clinical evaluation
Pregnancy	Healthy woman of childbearing age; no other symptoms Usually an early or unrecognized	Urine pregnancy test

	pregnancy	
Hyperventilation	Often **tingling** around mouth or on fingers prior to syncope Usually in context of an emotional situation	Clinical evaluation
Hypoglycemia	Altered mental status until treated, onset seldom abrupt, sweating, piloerection Usually history of diabetes or insulinoma	Fingerstick glucose Response to glucose infusion
Psychiatric disorders	Not true syncope (patient may be partially or inconsistently responsive during events) Normal examination Often history of psychiatric disorder	Clinical evaluation

□ *ECG and pulse oximetry are done for all.

Table 8

Some Drug Causes of Syncope	
Mechanism	**Example**

Bradyarrhythmia	Amiodarone, other rate-limiting drugs β-blockers Ca channel blockers (not dihydropyridines) Digoxin
Tachyarrhythmia	Any antiarrhythmic drug Quinidine
Orthostatic hypotension	Most antihypertensives (rarely β-blockers) Antipsychotics (mainly phenothiazines) Doxorubicin Levodopa Loop diuretics Nitrates (with or without a phosphodiesterase inhibitor for erectile dysfunction) Quinidine Tricyclic antidepressants Vincristine

Evaluation

Evaluation should be done as soon as possible after the event. The more remote the syncopal event, the more difficult the diagnosis. Information from witnesses is quite helpful and best obtained as soon as possible.

History: **History of present illness** should ascertain events leading up to the syncope, including the patient's activity (eg, exercising, arguing, in a potentially emotional situation), position (eg, lying or standing), and, if standing, for how long. Important associated symptoms immediately before or after the event include whether there was a sense of impending LOC, nausea, sweating, blurred or tunnel vision, tingling of lips or fingertips, chest pain, or palpitations. Length of time recovering should also be ascertained. Witnesses, if any, should be sought and asked to describe events, particularly the presence and duration of any seizure activity.

Review of systems should ask about any areas of pain or injury, episodes of dizziness or near-syncope upon arising, and episodes of palpitations or chest pain with exertion. Patients should be asked about symptoms suggesting possible causes, including bloody or tarry stools, heavy menses (anemia); vomiting, diarrhea, or excess urination (dehydration or electrolyte abnormalities); and risk factors for pulmonary embolism (recent surgery or immobilization, known cancer, previous clots or **hypercoagulable** state).

Past medical history should ask about previous syncopal events, known cardiovascular disease, and known seizure disorders. Drugs used should be identified (particularly **antihypertensives**, diuretics, vasodilators, and antiarrhythmic. Family history should note presence at a young age of heart disease or sudden death in any family member.

Physical examination: Vital signs are essential. Heart rate and BP are measured with the patient supine and after 2 min of standing. Pulse is palpated for irregularity.

General examination notes patient's mental status, including any confusion or hesitancy suggesting a postictal state and any signs of injury (eg, bruising, swelling, tenderness, tongue bite).

The heart is auscultated for murmurs; if present, any change in the murmur with a Valsalva maneuver, standing, or squatting is noted.

Careful evaluation of the jugular venous waves while palpating the carotid or auscultating the heart may allow diagnosis of an arrhythmia if an ECG is not available.

Some clinicians carefully apply unilateral carotid sinus pressure during ECG monitoring with the patient supine to detect bradycardia or heart block, suggesting carotid sinus hypersensitivity. Carotid sinus pressure should not be applied if a carotid bruit is present.

Abdomen is palpated for tenderness, and a rectal examination is done to check for gross or **occult blood**.

A full neurologic examination is done to identify any focal abnormalities, which suggest a CNS cause (eg, seizure disorder).

Red flags: Certain findings suggest a more serious etiology:

- Syncope during exertion
- Multiple recurrences within a short time
- Heart murmur or other findings suggesting structural heart disease (eg, chest pain)
- Older age
- Significant injury during syncope
- Family history of sudden unexpected death

Interpretation of findings: Although the cause is often benign, it is important to identify the occasional life-threatening cause (eg, tachyarrhythmia, heart block) because sudden death is a risk. Clinical findings help suggest a cause in 40 to 50% of cases. A few generalizations are useful.

Benign causes often lead to syncope. Syncope precipitated by unpleasant physical or emotional stimuli (eg, pain, fright), usually occurring in the upright position and often preceded by **vagally mediated warning symptoms** (eg, nausea, weakness, yawning, apprehension, blurred vision, diaphoresis), suggests vasovagal syncope.

Syncope that occurs most often when assuming an upright position (particularly in elderly patients after prolonged bed rest or in patients taking drugs in certain classes) suggests orthostatic syncope. Syncope that occurs after standing for long periods without moving is usually due to venous pooling.

LOC that is abrupt in onset; is associated with **muscular jerking** or convulsions, incontinence, or tongue biting; and is followed by postictal confusion or **somnolence** suggests a seizure.

Red flag findings suggest a **dangerous cause.**

Syncope with exertion suggests cardiac outflow obstruction. Such patients sometimes also have chest pain, palpitations, or both. Cardiac findings may help identify a cause. A harsh, late-peaking, basal murmur radiating to the carotid arteries suggests aortic stenosis; a systolic murmur that increases with the Valsalva maneuver and disappears with squatting suggests hypertrophic cardiomyopathy.

Syncope that begins and ends suddenly and spontaneously is typical of cardiac causes, most commonly an arrhythmia. Because vasovagal and orthostatic mechanisms do not cause syncope in the recumbent position, syncope while lying down also suggests an arrhythmia.

If the patient is injured during the episode of syncope, the likelihood of a cardiac cause or seizure is somewhat greater, and therefore the event is of greater concern. The warning signs and slower LOC that accompany benign vasovagal syncope somewhat reduce the likelihood of injury.

Testing: Testing typically is done.

- ECG
- **Pulse oximetry**
- Sometimes echocardiography
- Sometimes tilt table testing
- Blood tests only if clinically indicated
- CNS imaging rarely indicated

In general, if syncope results in an injury or is recurrent (particularly within a brief period), more intensive evaluation is warranted.

Patients with suspected arrhythmia, myocarditis, or ischemia should be evaluated as inpatients. Others may be evaluated as outpatients.

ECG is done for all patients. The ECG may reveal arrhythmia, a conduction abnormality, **ventricular hypertrophy**, **pre-excitation**, **QT prolongation**, **pacemaker malfunction**, **myocardial ischemia**, or MI. If there are no clinical clues, measuring cardiac markers and obtaining serial ECGs to rule out MI in older patients plus ECG monitoring for at least 24 h are prudent. Any detected arrhythmia must be associated with altered consciousness in order to be implicated as the cause, but most patients do not experience syncope during monitoring. On the other hand, the presence of symptoms in the absence of rhythm disturbance helps rule out a cardiac cause. An event recorder may be useful if warning symptoms precede syncope. A signal-averaged ECG may identify predisposition to ventricular arrhythmias in patients with ischemic heart disease or in post-MI patients.

Pulse oximetry should be done during or immediately after an episode to identify hypoxemia (which may indicate pulmonary embolism). If hypoxemia is present, CT or a lung scan is indicated to rule out pulmonary embolism.

Laboratory tests are done based on clinical suspicion; reflexively obtained laboratory panels are of little use. However, all females of childbearing age should have a pregnancy test. Hct is measured if anemia is suspected. Electrolytes are measured only if an abnormality is clinically suspected (eg, by symptoms or drug use). Serum troponin is measured if acute MI is suspected.

Echocardiography is indicated for patients with exercise-induced syncope, cardiac murmurs, or suspected intracardiac tumors (eg, those with positional syncope).

http://www3.gehealthcare.com/en/Products/Categories/Ultrasound/Vivid/~/media/Images/Product/Product-Categories/Ultrasound/Vivid/Vivid-E9/VE9_clinician_patient_ultrasound.ashx

Tilt table testing may be done if history and physical examination indicate vasodepressor or other reflex-induced syncope. It is also used to evaluate exercise-induced syncope if echocardiography or exercise stress testing is negative.

Stress testing (exercise or pharmacologic) is done when intermittent myocardial ischemia is suspected. It is often done for patients with exercise-induced symptoms.

Invasive electrophysiologic testing is considered if noninvasive testing does not identify arrhythmia in patients with unexplained recurrent syncope; a negative response defines a low-risk subgroup with a high rate of remission of syncope. The use of **electrophysiologic testing** is **controversial** in other patients. Exercise testing is less valuable unless physical activity precipitated syncope.

EEG is warranted if a seizure disorder is suspected.

CT and **MRI** of the head and brain are indicated only if signs and symptoms suggest a focal CNS disorder.

Treatment

In witnessed syncope, pulses are checked immediately. If the patient is pulseless, CPR is begun and cardiac **resuscitation** is done. If pulses are present, severe bradycardia is treated with atropine or **external transthoracic pacing**. Isoproterenol can be used to maintain adequate heart rate while a temporary pacemaker is placed.

Tachyarrhythmias are treated; a direct-current synchronized shock is quicker and safer for unstable patients. Inadequate venous return is treated by keeping the patient supine, raising the legs, and giving IV normal saline. **Tamponade** is relieved by **pericardiocentesis**. Tension pneumothorax requires insertion of a **pleural cannula** and drainage. **Anaphylaxis** is treated with parenteral epinephrine.

Placing the patient in a horizontal position with legs elevated typically ends the syncopal episode if life-threatening disorders are ruled out. If the patient sits upright too rapidly, syncope may recur; propping the patient upright or transporting the patient in an upright position may prolong cerebral hypoperfusion and prevent recovery.

Specific treatment depends on the cause and its pathophysiology.

Geriatrics Essentials

The most common cause of syncope in the elderly is postural hypotension due to a combination of factors. Factors include rigid, noncompliant arteries, reduced skeletal muscle pumping of venous return due to physical inactivity, and degeneration of the **sinoatrial node** and conduction system due to progressive structural heart disease.

In the elderly, syncope often has more than one cause. For example, the combination of taking several heart and BP drugs and standing in a hot church during a long or emotional service may lead to syncope even though no single factor might cause syncope.

Key Points

- Syncope results from global CNS dysfunction, usually from insufficient cerebral blood flow.
- Most syncope results from benign causes.
- Some less common causes involve cardiac arrhythmia or outflow obstruction and are serious or potentially fatal.
- Vasovagal syncope usually has an apparent trigger, warning symptoms, and a few minutes of post-recovery symptoms.
- Syncope from cardiac arrhythmias typically occurs abruptly and with quick recovery.
- Seizures have a prolonged (eg, hours) recovery period.
- If a benign etiology is not clear, driving and use of machinery should be prohibited until the etiology is determined and treated—the next manifestation of an unrecognized cardiac cause may be fatal.

Reference: http://www.merckmanuals.com