MSD MANUAL
Professional Version

Syncope

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Syncope is a sudden, brief loss of consciousness with loss of postural tone followed by spontaneous revival. The patient is motionless and limp and usually has cool extremities, a weak pulse, and shallow breathing. Sometimes brief involuntary muscle jerks occur, resembling a seizure.

Near-syncope is light-headedness and a sense of an impending faint without loss of consciousness. It is usually classified and discussed with syncope because the causes are the same.

Seizures can cause sudden loss of consciousness 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 second) seizure sometimes occurs with true syncope.

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

Pathophysiology of Syncope

Most syncope results from insufficient cerebral blood flow. Some cases involve adequate flow but with insufficient cerebral substrate (oxygen, 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.

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

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 medications, 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 cause loss of consciousness. However, basilar artery ischemia, due to transient ischemic attack, stroke, 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 central nervous system (CNS) requires oxygen and glucose to function. Even with normal cerebral blood flow, a significant deficit of either will cause loss of consciousness . In practice, hypoglycemia is the primary cause because hypoxia rarely develops in a manner causing abrupt loss of consciousness (other than in flying or diving incidents). Loss of consciousness due to hypoglycemia is seldom as abrupt as in syncope or seizures because warning symptoms occur (except in patients taking beta-blockers); however, the onset may be unclear to the examiner unless the event was witnessed.

Etiology of Syncope

Causes are usually classified by the mechanism (see table Some Causes of Syncope).

The **most common causes** are

- Vasovagal (neurocardiogenic)
- Idiopathic

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

TABLE

Some Causes of Syncope

Cause	Suggestive Findings	Diagnostic Approach*
Cardiac outflow or inflow obstruction		
Valvular disease: Aortic stenosis, mitral stenosis, tetralogy of Fallot, prosthetic valve dehiscence or thrombosis	Young or old patient Syncope often exertional; recovery prompt Heart murmur	Echocardiography
Hypertrophic cardiomyopathy (HCM), restrictive cardiomyopathy	Young or old patient Syncope often exertional; recovery prompt Heart murmur (in hypertrophic cardiomyopathy) S4 (in restrictive cardiomyopathy)	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	Sometimes D-dimer Sometimes POCUS or echocardiography CT angiography or nuclear scan
Cardiac arrhythmia		
	Syncope occurring without warning; recovery immediate on awakening	If ECG unclear, consider Holter monitor, event recorder, or occasionally an implantable loop

Bradyarrhythmias (eg, due to sinus node dysfunction, high-grade atrioventricular block, medications†)	May occur in any position Bradyarrhythmias more common in older adults Patient taking medications, especially antiarrhythmics or other medications that block AV nodal conduction in susceptible patients Structural heart disease	recorder (duration of monitoring determined by frequency of symptoms) Electrophysiologic testing if abnormalities detected or strongly suspected Serum electrolytes if clinical reason for abnormality (eg, diuretic use, vomiting, diarrhea)
Tachyarrhythmias, either supraventricular or ventricular (eg, due to ischemia, heart failure, myocardial disease, medications†, electrolyte abnormalities, arrhythmogenic right ventricular cardiomyopathy, long QT syndrome, Brugada syndrome, preexcitation)	Syncope occurring without warning; recovery immediate on awakening May occur in any position Patient taking medications, especially antiarrhythmics or other medications that prolong the QT interval Structural heart disease	If ECG unclear, consider Holter monitor or event recorder, or occasionally an implantable loop recorder (duration of monitoring determined by frequency of symptoms) Electrophysiologic testing if abnormalities detected or strongly suspected Serum electrolytes if clinical reason for abnormality (eg, diuretic use, vomiting, diarrhea)
Ventricular dysfunction		
Acute myocardial infarction, myocarditis, systolic or diastolic dysfunction, cardiomyopathy	Syncope a rare presenting symptom of myocardial infarction (most such patients are older), with arrhythmia or shock	Serum troponin ECG Echocardiography (traditional, POCUS, or both) Sometimes cardiac MRI
Pericardial tamponade or constriction	Jugular venous elevation; pulsus paradoxus > 10	Echocardiography (traditional, POCUS, or both) 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 usually prompt but not immediate (5 to 15 minutes) although can be prolonged in rare cases 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 minutes, but sometimes up to hours) Precipitant usually apparent	Clinical evaluation
Carotid sinus pressure	Warning symptoms (eg, dizziness, nausea, sweating); recovery prompt but not immediate (5 to 15 minutes, but sometimes up to hours) Precipitant usually apparent (eg, turning head, button collar, shaving)	Clinical evaluation
Swallowing (rare)	Warning symptoms (eg, dizziness, nausea, sweating); recovery prompt but not immediate (5 to 15 minutes, but sometimes up to hours) Precipitant usually apparent	Clinical evaluation
Medication		

Anaphylaxis	Medication administration, insect bite, allergy history, other signs of anaphylaxis (eg, rash, wheezing, hypotension)	Allergy testing
Orthostatic hypotension		
Medications†	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 examination	Clinical evaluation Sometimes tilt table testing
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	Complete blood count or hematocrit
Endocrine disorders (eg, adrenal insufficiency, hypothyroidism)	Symptoms developing within several minutes of assuming upright position Often associated symptoms of underlying endocrine disorder	Basic metabolic panel Morning cortisol measurement Thyroid stimulating hormone (TSH)
Cerebrovascular		
Basilar artery transient ischemic attack, or stroke	Sometimes cranial nerve deficits and ataxia	MRI or CT

Migraine	Aura with visual symptoms, photophobia; often unilateral, but may be bilateral	Clinical evaluation
Other		
Prolonged standing	Apparent by history; no other symptoms	Clinical evaluation
Pregnancy	Healthy female of childbearing age; no other symptoms Usually an early or unrecognized pregnancy	Urine pregnancy test
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.

† See table Some Medications That Cause Syncope.

AV = atrioventricular; BP = blood pressure; POCUS = point-of-care ultrasound; S4 = fourth

heart sound.

TABLE

Some Medications That Cause Syncope

Mechanism	Example
Bradyarrhythmia	Amiodarone, other rate-limiting drugs Beta-blockers Calcium channel blockers (not dihydropyridines) Digoxin
Tachyarrhythmia	Any medication that prolongs repolarization* (eg, some antiarrhythmics, some antipsychotics, some antidepressants, some antihistamines, some fluoroquinolones)
Orthostatic hypotension	Most antihypertensives (rarely beta-blockers) Antipsychotics (mainly phenothiazines) Doxorubicin Levodopa Diuretics (eg, loop diuretics such as furosemide; thiazide diuretics such as hydrochlorothiazide) Nitrates (with or without a phosphodiesterase inhibitor for erectile dysfunction) Quinidine Tricyclic antidepressants Vincristine

For more information, see Porta L, Lee MG, Hsu WT, Hsu TC, Tsai TY, Lee CC: Fluoroquinolone use and serious arrhythmias: A nationwide case-crossover study. *Resuscitation* 139:262–268, 2019. doi:10.1016/j.resuscitation.2019.04.030

Evaluation of Syncope

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. Guidelines for evaluation and management are available from the Canadian Cardiovascular Society (1) and the American College of Cardiology (2).

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 loss of consciousness, 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. Medications used should be identified (particularly antihypertensives, diuretics, vasodilators, and antiarrhythmics—see table Some Medications that Cause Syncope). 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 blood pressure are measured with the patient supine and after 3 minutes 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 (see figure Normal Jugular Vein Waves) while palpating the carotid or auscultating the heart may allow diagnosis of an arrhythmia if an ECG is not available. For example, cannon "a" waves occur when the atria contraction takes place against a closed tricuspid valve and indicate atrial-ventricular dissociation.

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

A neurologic examination is done to identify any focal abnormalities, which suggest a central nervous system 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, exertional syncope, or unexplained recurrent syncope or seizures

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 (see table Some Causes of Syncope) help suggest a cause in many 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 older patients after prolonged bed rest or in patients taking medications in certain classes) suggests orthostatic syncope.
- Syncope that occurs after standing for long periods without moving is usually due to venous pooling.

Dangerous causes are suggested by red flag findings.

- Syncope with exertion suggests cardiac outflow obstruction or exercise-induced arrhythmia. 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.
- Syncope while lying down also suggests an arrhythmia because vasovagal and orthostatic mechanisms do not cause syncope in the recumbent position.
- Syncope accompanied by injury during the episode increases the likelihood of a cardiac cause or seizure, and therefore the event is of greater concern. The warning signs and slower loss of consciousness that accompany benign vasovagal syncope somewhat reduce the likelihood of injury.

Loss of consciousness during a seizure or postictal confusion can sometimes be confused with syncope, but muscular jerking or convulsions that last more than a few seconds, incontinence, drooling, or tongue biting, if present, usually point to a seizure.

Testing

Testing typically is done:

- ECG
- Pulse oximetry
- Sometimes echocardiography
- Sometimes tilt table testing
- Blood tests only if clinically indicated
- Central nervous system imaging rarely indicated

In general, if syncope results in an injury or is recurrent (particularly within a brief period), more intensive evaluation is warranted. Cardiac and brain imaging are not done unless indicated by clinical findings (suspected cardiac etiology or neurologic deficits).

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, Brugada syndrome, pacemaker malfunction, myocardial ischemia, or myocardial infarction (3). If the diagnosis is questionable after this basic evaluation, measuring cardiac biomarkers and obtaining serial ECGs to rule out MI in older patients plus ECG monitoring for at least 24 hours 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 (which can record cardiac rhythm for longer periods) may be useful if warning symptoms precede syncope. A signal-averaged ECG may identify predisposition to ventricular arrhythmias in patients with ischemic heart disease, suspected arrhythmogenic right ventricular cardiomyopathy, or in patients during the post-myocardial infarction period. If syncopal episodes are infrequent (eg, < 1/month), an implantable loop recorder can be used for longer term recording. This device continuously records the rhythm and can be interrogated by an external machine that allows the cardiac rhythm to be printed.

Pulse oximetry should be done during or immediately after an episode to identify hypoxemia (which may indicate pulmonary embolism). If hypoxemia is present, CT angiography 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. Hematocrit is measured if anemia is suspected. Electrolytes are measured only if an abnormality is clinically suspected (eg, by symptoms or medication use). Serum troponin is measured if acute myocardial infarction is suspected.

Echocardiography (either point-of-care ultrasound [POCUS] or a traditional echocardiogram) is indicated for patients with clinically unexplained syncope, exercise-induced syncope, cardiac murmurs, or suspected intracardiac tumors (eg, those with positional syncope).

Stress testing (exercise or pharmacologic) is done when intermittent myocardial ischemia is suspected. It is often done for patients with exercise-induced symptoms. Exercise testing is less valuable unless physical activity precipitated syncope.

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.

Invasive electrophysiologic testing is considered if noninvasive testing does not identify arrhythmia in patients with any of the following:

- Unexplained recurrent syncope
- Unexplained red flag findings
- Ischemic cardiomyopathy, non-ischemic cardiomyopathy, and adult congenital disease, or unexplained syncope that does not otherwise meet criteria for an implantable cardioverter-defibrillator (ICD) used for primary prevention

An electrophysiologic study that is negative for an inducible abnormal rhythm defines a low-risk subgroup with a high rate of remission of syncope. The use of electrophysiologic testing is controversial in other patients.

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.

Evaluation references

1. Primary Writing Committee, Sandhu RK, Raj SR, et al: Canadian Cardiovascular Society Clinical Practice Update on the Assessment and Management of Syncope. *Can J Cardiol* 36(8):1167–1177, 2020. doi: 10.1016/j.cjca.2019.12.023
2. Writing Committee Members, Shen WK, Sheldon RS, et al: 2017 ACC/AHA/HRS guideline for the evaluation and management of patients with syncope: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Heart Rhythm* 14(8):e155–e217, 2017. doi:10.1016/j.hrthm.2017.03.004
3. Dovgalyuk J, Holstege C, Mattu A, Brady WJ: The electrocardiogram in the patient with syncope. *Am J Emerg Med* 25(6):688–701, 2007. doi:10.1016/j.ajem.2006.12.016

Treatment of Syncope

In witnessed syncope, pulses are checked immediately. If the patient is pulseless, CPR is begun. 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 than medications for patients who are not stable. 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. Driving and use of machinery should be prohibited until the cause is determined and treated.

Geriatrics Essentials: Syncope

The most common cause of syncope in older adults 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 older adults, syncope often has more than one cause. For example, the combination of taking several heart and blood pressure medications 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 central nervous system dysfunction, usually resulting 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 or longer of postrecovery symptoms.**
- **Syncope due to cardiac arrhythmias typically occurs abruptly and with quick recovery.**
- **Treatment is directed at the underlying disorder.**

- If a benign etiology is not clear, driving and use of machinery should be prohibited until the cause is determined and treated—the next manifestation of an unrecognized cardiac cause may be fatal.