Syncope in adults: Clinical manifestations and initial diagnostic evaluation

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INTRODUCTION

Syncope is a clinical syndrome in which transient loss of consciousness (TLOC) is caused by a period of inadequate cerebral blood flow and oxygenation, most often the result of an abrupt drop of systemic blood pressure. Typically, the inadequate cerebral nutrient flow is of relatively brief duration, and, by definition, syncope is self-limited.

Unfortunately, the term "syncope" is often misapplied to encompass other forms of abrupt collapse such as seizures or accidents, which may or may not be accompanied by TLOC. Such broader, less-specific usage of the term "syncope" should be avoided, as imprecise usage impairs accurate diagnosis and undermines comparison of clinical study outcomes. [1]. (See 'Differential diagnosis' below.)

Issues relating to the clinical presentation and diagnosis of syncope in adults will be reviewed here. Related issues are discussed separately:

- (See "Syncope in adults: Epidemiology, pathogenesis, and etiologies".)
- (See "Reflex syncope in adults and adolescents: Clinical presentation and diagnostic evaluation".)
- (See "Approach to the adult patient with syncope in the emergency department".)
- (See "Syncope in adults: Management and prognosis".)

- (See "Syncope in adults: Risk assessment and additional diagnostic evaluation".)

DEFINITIONS

Patients may present with syncopal or presyncopal episodes, or both types of episodes on different occasions.

- Syncope is a transient, self-limited loss of consciousness caused by transient, self-terminating, inadequate nutrient flow to the brain. Syncopal episodes may or may not be preceded by prodromal symptoms, which are described below. (See 'Onset/prodrome' below.)

- Presyncope (or near-syncope) is a clinical manifestation that is suggestive of an impending syncope often occurring in conjunction with a similar set of prodromal symptoms (see 'Onset/prodrome' below) reflecting the same conditions that might lead to syncope in the same individual at other times.

APPROACH TO INITIAL EVALUATION

The initial evaluation of patients with transient loss of consciousness (TLOC), some with suspected true syncope, serves both diagnostic and prognostic purposes (algorithm 1). This evaluation enables the clinician to ascertain whether the episode was true syncope or another type of event, determine whether the affected patient should be admitted to the hospital or can be safely managed in the outpatient environment (ie, risk stratification) (table 1), and assess potential causes (table 2). Establishing the likely etiology guides appropriate diagnostic and treatment strategies to assess prognosis and prevent future events.

For nearly all patients, the initial evaluation of suspected syncope should include:

- Obtaining a comprehensive history, including information about the episode(s) and past medical history. (See 'Clinical features' below and 'Medical history' below.)

- Performance of a physical examination (which may include careful carotid sinus massage in older patients). (See 'Physical examination' below.)

- Review of an electrocardiogram (ECG) and any available rhythm strips.

- A transthoracic echocardiogram is performed if structural heart disease is suspected or cannot be otherwise excluded after the above evaluation is completed.

The efficacy of initial clinical evaluation has been summarized in professional society practice guidelines [2,3]. Overall, various reports suggest that an etiology for syncope may be obtained by an experienced clinician after thorough evaluation in 45 to 65 percent of patients.

CLINICAL FEATURES

Obtaining a detailed medical history is the first step in determining whether apparent transient loss of consciousness (TLOC) episodes are true syncope or the result of some other cause of collapse. If the history obtained is thorough, the story provided by the patient (and witnesses, if any) will often reveal the most likely cause(s) of syncope and will provide a means of focusing subsequent testing and treatment. Identifying any common threads with regard to associated symptoms, "downtime," or circumstances of onset can be helpful in suggesting an etiology.

However, history taking is highly dependent on the experience of and time available to the clinician and whether the patient and witnesses can convey an accurate account of events, which may be limited due to communication barriers (eg, language barrier or cognitive dysfunction) or patient discomfort or intoxication. Also, in stressful circumstances, time periods (ie, duration of the event) may be difficult to accurately estimate. Even under ideal circumstances, the sensitivity of the history taking alone has not been well established. Consequently, confirmatory testing (with focus based upon the clinical evaluation) may be required to establish a diagnosis.

Frequency and duration — The number, frequency, duration of episodes, and span of time (eg, days, months, or years) over which the patient has experienced presumed syncope events are important.

- **Number and frequency of episodes** – The frequency of episodes due to benign causes (such as vasovagal faint) is variable; many patients may experience only single or very rare episodes, but others will have multiple episodes over many years, leading to an impaired quality of life and predisposing to injury.

  Of note, the patient with multiple episodes occurring over a short period of time (ie, several days or weeks) is more likely to be suffering from a serious underlying disorder (eg, intermittent high-grade atrioventricular [AV] block, paroxysmal ventricular tachycardia [VT], etc) and requires aggressive evaluation. However, it is unusual for multiple syncope episodes to occur in a single day. In particular, while reflex vasovagal faints may recur immediately if the affected individual tries to stand up prematurely, it is unusual for multiple vasovagal events to occur on the same day.

Individuals having many episodes of apparent "syncope" each day and/or episodes lasting many minutes in duration may be suffering from nonsyncope psychogenic disorders ("psychogenic pseudosyncope" or "psychogenic pseudoseizures") which are considered to be conversion reactions rather than true syncope. (See 'Differential diagnosis' below.)

- **Span of time marked by episodes** – In general, the longer the period of time over which episodes have occurred (eg, many years versus very recent onset) and the younger the patient's age at onset (particularly when less than 35 years), the less likely that the cause of syncope is life-threatening, unless the individual has identifiable structural heart disease (assessed by clinical evaluation and testing) or a suspected channelopathy (which may be suggested by family history and ECG findings) [4].

Triggers and circumstances — A detailed history should assess circumstances at the time of syncope, as this information may identify possible triggers.

- **Triggers** – Reflex syncope may be triggered by emotional or orthostatic stress, fear, or intense pain, or by a warm and/or crowded environment. However, in many instances, it may not be possible to confidently identify the trigger. (See "Reflex syncope in adults and adolescents: Clinical presentation and diagnostic evaluation", section on 'Triggers'.)

  Situational syncope (a type of reflex syncope) occurs during or immediately after certain apparent triggers such as urination, defecation, coughing, swallowing, or after eating a meal [2,3]. (See "Reflex syncope in adults and adolescents: Clinical presentation and diagnostic evaluation", section on 'Situational syncope'.)

  Carotid sinus syndrome (CSS) is suggested by syncope occurring immediately following abrupt neck movements. Carotid sinus hypersensitivity triggered during carotid sinus massage (CSM) may suggest this susceptibility, but should not be relied on to establish the diagnosis of CSS unless other possible causes have been considered and excluded and CSM reproduces syncope symptoms. Carotid sinus syncope is usually a condition of older patients (generally >60 years of age with male predominance) or patients with prior head and neck surgery or irradiation. (See "Carotid sinus hypersensitivity and carotid sinus syndrome".)

- **Patient position** – Assessing the patient's position (ie, supine, sitting, or standing) at the time of syncope, along with any recent changes in position during the preceding few minutes prior to syncope, provides clues to the etiology.

  - **While in an unprotected position (eg, prolonged standing)** – Reflex syncope most commonly occurs when the patient is upright (standing or seated) and almost never

when supine. Syncope resulting from orthostatic hypotension is frequently associated with a change from a supine to erect posture, although several minutes may pass between the patient arising and the subsequent collapse.

- **While supine** – Syncope that occurs when the patient is supine or recumbent suggests a cardiac arrhythmia and is therefore worrisome. Even if the arrhythmia persists, the TLOC is often brief given the body's adaptive hemodynamic compensatory responses (eg, vascular constriction).

- **Relation to exercise** – The timing of syncope in relation to exercise is very important, as syncope during full-flight exercise may be indicative of a serious condition (eg, exercise-triggered tachyarrhythmia or hypotension), while syncope immediately after exercise tends to be more innocent and reflex in origin. Syncope that occurs during exertion suggests a potentially life-threatening etiology (eg, aortic stenosis, hypertrophic cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia or other channelopathy) and should be taken very seriously. On the other hand, syncope occurring soon after termination of exertion (eg, during cooling-off period) is more likely reflex in origin, similar to the vasovagal faint.

Onset/prodrome — Symptoms preceding syncope can point toward a specific cause (table 2) [2,3]. Most patients who experience syncope have a warning premonition period of at least a few seconds or longer prior to losing consciousness. This is particularly the case for reflex faints. On the other hand, some patients will suddenly lose consciousness without apparent warning or, due to retrograde amnesia, may not recollect a warning symptom.

Extended symptoms with a classic prodrome are more commonly associated with the vasovagal form of reflex syncope, while sudden onset of syncope with minimal or no prodrome is more common among patients with cardiac syncope. However, some patients with impaired memory may be amnestic following the event and unable to recall any prodrome, and may therefore report sudden TLOC without warning. (See "Reflex syncope in adults and adolescents: Clinical presentation and diagnostic evaluation".)

The following are classic prodromal (presyncopal) symptoms associated with imminent syncope and presyncope. These are particularly common in younger patients with the vasovagal form of reflex syncope but less so in older vasovagal fainters or those with syncope from other causes:

- Lightheadedness.

- Feeling unstable in the upright position.

- A feeling of being warm or cold/clammy.
- Sweating.
- Palpitations – The sudden onset of palpitations immediately followed by syncope suggests a cardiac arrhythmia, but palpitations due to sinus tachycardia may also precede reflex syncope.
- Nausea, vomiting, or nonspecific abdominal discomfort.
- Visual "blurring" occasionally proceeding to temporary darkening or "white-out" of vision.
- Diminution of hearing and/or occurrence of unusual sounds (particularly a "whooshing" noise).
- Pallor reported by onlookers.

Prodromal symptoms may be very disconcerting and are often described by patients as "nearly blacking out" or "nearly fainting." Distinguishing true presyncope from more nonspecific complaints such as "lightheadedness," "brain fog," or other conditions such as "vertigo" may be difficult but is important. (See 'Differential diagnosis' below.)

Witnessed signs — If the syncopal event was observed, the witness should be asked to provide as much information as possible in addition to the history obtained from the patient. A mobile phone video can be very helpful if the witness is capable of obtaining one. Witnesses should be asked to describe the following features:

- The manner in which the collapse occurred (eg, was there an abrupt fall with possibility of injury or was there purposeful avoidance of injury?).

  Loss of postural stability is inevitable with TLOC, and, consequently, syncope is generally associated with physical collapse. Physical collapse may cause injury due to a fall (such as may occur if the person is standing) or other type of accident (eg, if syncope occurs while driving or while in a high-risk environment such as working on a ladder or with machinery). The injury risk, of course, applies not only to the "fainter" but may affect others who are injured secondarily (eg, motor vehicle accident) [2,3].

- The appearance of the patient (eg, was the skin pale or clammy, or were the eyelids open or closed?).
- The duration of the loss of consciousness – True syncope of any etiology is usually brief since the loss of postural tone (and the subsequent gravitationally neutral position)

generally restores brain blood flow.

Longer periods of real or apparent loss of consciousness (especially if >5 minutes) suggest that the event is not syncope or is not syncope alone. A prolonged event could be syncope resulting in a fall complicated by head injury (concussion may prolong TLOC), a seizure, or psychogenic pseudosyncope (a collapse deemed secondary to a conversion reaction or dissociative episode [2,5]).

Estimates of the duration of a TLOC episode by patients or witnesses are generally imprecise. Determining the duration of spontaneous TLOC events is further complicated when retrograde amnesia occurs, which is more common in older patients with or without head trauma. Nonetheless, an estimate from witnesses should be sought and may be helpful from a diagnostic perspective.

- Alterations in the patient's breathing pattern.

- Any physical movements (eg, tonic-clonic or myoclonic movements, tongue biting, incontinence, etc). Did these movements begin before the collapse (favors seizure) or after (favors syncope)?

Clinical features after the event — Recovery from true syncope is usually complete, with episodes rarely lasting more than a minute. (See 'Frequency and duration' above.)

Symptoms following syncope reported by patients and findings observed by witnesses after the episode can help point toward a specific cause (table 2) [2,3]. Patients should be asked about experiencing any of the following during the event or shortly after recovery:

- Confusion
- Fatigue
- Injury
- Nausea
- Vomiting
- Feeling cold or clammy
- Palpitations
- Shortness of breath
- Chest pain
- Bladder or bowel incontinence

Persistence of nausea, pallor, and diaphoresis in addition to prolonged fatigue (lasting minutes to hours) after a syncope episode suggest a reflex event (particularly a vasovagal episode).

These findings are helpful in distinguishing reflex syncope from syncope due to an arrhythmia.

Neurologic changes or confusion during the recovery period may suggest a stroke or seizure. However, postevent confusion may also occur with syncope and may complicate accuracy of recall of the event, including time estimates.

MEDICAL HISTORY

Preexisting conditions — A variety of preexisting medical conditions can suggest but not definitively prove an etiology for the patient's transient loss of consciousness (TLOC)/collapse. Patients should be questioned about personal history of the following:

- Structural heart disease (eg, coronary artery disease with or without prior myocardial infarction, valvular heart disease, congenital heart disease, cardiomyopathies, prior cardiac surgery, etc). Among patients with structural heart disease, there is an increased risk of cardiac arrhythmias that may cause syncope. Also, some types of structural heart disease (eg, severe aortic stenosis) are associated with other mechanisms for syncope. (See "Syncope in adults: Epidemiology, pathogenesis, and etiologies".)
- Neurologic conditions (eg, seizure disorders, migraine headaches, Parkinson disease, autonomic failure, stroke, etc).
- Diabetes mellitus. Patients with diabetes mellitus may develop true syncope resulting from orthostatic hypotension (OH) secondary to autonomic neuropathy or exhibit an apparent "syncope" due to hypoglycemia.
- Intoxications (eg, alcohol, illicit drug use, or prescription narcotics).
- Psychiatric disorders may be associated with episodes of apparent TLOC secondary to hyperventilation, panic attacks, conversion reactions, or medications.

Medications — A variety of prescription and over-the-counter medications can predispose patients to syncope through a number of different mechanisms. Patients should be asked to provide a comprehensive list of prescription and over-the-counter medications, and should be queried specifically about the recent addition of any new agents or recent dose adjustments.

Some examples of mechanisms and potential offending medications include:

- **Hypovolemia** – Diuretics or excessively strict salt avoidance.
- **Electrolyte disturbances** (eg, hypokalemia) – Diuretics.

- **Hypotension (primarily OH)** – All classes of antihypertensive agents, but particularly vasodilators, may induce or worsen hypotension, particularly OH.

- **Bradyarrhythmias** – Numerous medications (table 3), including beta blockers and calcium channel blockers.

- **Torsades de pointes** (polymorphic VT with associated QT interval prolongation) – Drugs include antiarrhythmic agents, antiinfective drugs (eg, azole antifungals, fluoroquinolones, macrolides, etc), antipsychotic drugs, and antidepressants. (See "Acquired long QT syndrome: Definitions, pathophysiology, and causes".)

Family history — Important elements of the family history include the following:

- Sudden death, particularly if unexpected and/or at a young age (less than 40 years of age).

- Familial cardiomyopathy (eg, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy) or channelopathy (eg, long QT syndrome, short QT syndrome, Brugada syndrome, catecholaminergic polymorphic VT, familial conduction system disease).

- Familial predisposition to syncope. Familial associations have been observed for reflex syncope [6] and for the above cardiac conditions.

- Seizure disorders or migraine headaches. (See "Evaluation and management of the first seizure in adults", section on 'Family history' and "Pathophysiology, clinical manifestations, and diagnosis of migraine in adults", section on 'Genetic basis'.)

PHYSICAL EXAMINATION

A number of findings on physical examination can aid in the identification of some of the common causes of syncope, including abnormalities in vital signs, cardiovascular disturbances, and, less frequently, neurologic signs [2].

Vital signs

- **Orthostatic vital signs** – Pulse and blood pressure should be obtained with the patient supine, seated, and standing (the last immediately upon standing as well as after standing at least five minutes). These measurements may detect susceptibility to orthostatic hypotension (OH). A drop of ≥20 mmHg (>30 mmHg in hypertensive patients) in systolic pressure and/or a drop of ≥10 mmHg in diastolic pressure is considered diagnostic of OH.

However, such numbers should only be used as touchstones, and should not be considered definitive proof of the etiology of syncope unless it is consistent with the patient's clinical presentation, such as syncope triggered immediately (immediate form) or three to five minutes (delayed or classic form) after standing or occurring after prolonged upright posture in absence of other explanation. A noninvasive system for continuous beat-by-beat blood pressure and heart rate monitoring (eg, Nexfin) that avoids multiple inflations of a sphygmomanometer cuff is preferred. Conventional blood pressure cuff pressures may not be taken rapidly enough to avoid missing the transient blood pressure fall often associated with immediate OH during change of posture. (See "Mechanisms, causes, and evaluation of orthostatic hypotension", section on 'Diagnosis'.)

- **Heart rate and rhythm** – The heart rate may be slow or rapid due to a number of possible rhythm disturbances, or irregular due to atrial fibrillation, atrial flutter, or frequent ectopy. Irregularities suspected based on evaluation of the pulse should be confirmed by ECG. (See 'Electrocardiogram' below.)

- **Respiratory rate** – Hyperventilation with an elevated respiratory rate can be seen with pulmonary embolism or psychiatric causes of apparent transient loss of consciousness (TLOC; eg, anxiety, etc). (See "Hyperventilation syndrome in adults", section on 'Somatic symptoms'.)

Cardiovascular findings — Important cardiovascular findings on physical examination include differences in blood pressure in each arm (suggesting possible aortic dissection or, very rarely, aortic coarctation), pathologic cardiac murmurs (suggesting aortic stenosis, hypertrophic cardiomyopathy, myxoma, etc), and signs of pulmonary embolism (tachypnea and tachycardia being most common but, alone, lack specificity). (See "Auscultation of cardiac murmurs in adults" and "Physiologic and pharmacologic maneuvers in the differential diagnosis of heart murmurs and sounds".)

Careful carotid sinus massage with gentle initial pressure is performed in selected older patients (usually over age 40 years) with syncope of unknown etiology without contraindication, as discussed separately . (See "Carotid sinus hypersensitivity and carotid sinus syndrome", section on 'Diagnostic evaluation'.)

Neurologic findings — Most focal neurologic conditions do not cause true syncope. Exceptions are those with major autonomic dysfunction components, such as pure autonomic failure, Parkinson disease, or, occasionally, temporal lobe seizures that may trigger "ictal asystole." Signs of focal neurologic disease, such as hemiparesis, dysarthria, diplopia, vertigo, or signs of Parkinsonism, are suggestive of (but not diagnostic of) a neurologic cause of impairment of

consciousness, warranting a full neurologic evaluation. In general, however, while neurologic disease may be responsible for other forms of TLOC, such as generalized epilepsy, true syncope is not indicative of a primary neurologic condition. On the other hand, syncope or accidental falls with secondary head trauma leading to concussion may necessitate neurologic evaluation.

INITIAL TESTING

Electrocardiogram — An ECG should be obtained in all patients with suspected syncope [2,3]. The 12-lead ECG only rarely identifies a specific arrhythmic cause of syncope, although certain findings may be very helpful (table 1). In many instances, prolonged ambulatory ECG monitoring for weeks to months is necessary [7].

The 2018 European Society of Cardiology guidelines list the following as probable causes of arrhythmia-related syncope, but careful confirmative evaluation remains essential [3]:

- Persistent sinus bradycardia <40 beats per minute or sinus pauses >3 seconds in an awake patient. These findings are deemed suggestive of a bradycardic etiology, as it requires >8 to 10 seconds of hypotension to trigger syncope. (See "Sinus bradycardia".)
- Mobitz II second-degree AV block. (See "Second-degree atrioventricular block: Mobitz type II".)
- Third-degree (complete) AV block. (See "Third-degree (complete) atrioventricular block".)
- Alternating left and right bundle branch block. (See "Chronic bifascicular blocks", section on 'Definitions'.)
- VT or paroxysmal supraventricular tachycardia with rapid ventricular rate and/or in the setting of substantial left ventricular dysfunction. (See "Sustained monomorphic ventricular tachycardia: Clinical manifestations, diagnosis, and evaluation" and "Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation".)
- Nonsustained polymorphic VT with long or short QT interval. (See "Approach to sudden cardiac arrest in the absence of apparent structural heart disease" and "Acquired long QT syndrome: Clinical manifestations, diagnosis, and management" and "Congenital long QT syndrome: Epidemiology and clinical manifestations" and "Short QT syndrome" and "Catecholaminergic polymorphic ventricular tachycardia".)
- Pacemaker or implantable cardioverter-defibrillator malfunction with cardiac pauses. (See "Pacing system malfunction: Evaluation and management".)

In addition, a variety of abnormal ECG findings (table 2) may indicate the presence of heart disease and thereby provide a basis for proceeding with further testing [3]:

- Bifascicular block (defined as left or right bundle branch block combined with left anterior or left posterior fascicular block), especially with concomitant first-degree AV block. (See "Chronic bifascicular blocks".)
- Other intraventricular conduction abnormalities (QRS duration ≥0.12 seconds). (See "Basic approach to delayed intraventricular conduction".)
- Mobitz I second-degree AV block. (See "Second-degree atrioventricular block: Mobitz type I (Wenckebach block)".)
- Sinus bradycardia (≤40 beats per minute) or atrial fibrillation with a slow ventricular rate (≤40 beats per minute) in the absence of negatively chronotropic medications. (See "Sinus node dysfunction: Clinical manifestations, diagnosis, and evaluation".)
- Nonsustained VT. (See "Nonsustained ventricular tachycardia: Clinical manifestations, evaluation, and management".)
- Preexcited QRS complexes, suggesting Wolff-Parkinson-White syndrome. (See "Wolff-Parkinson-White syndrome: Anatomy, epidemiology, clinical manifestations, and diagnosis".)
- Long or short QT intervals. (See "Congenital long QT syndrome: Diagnosis" and "Short QT syndrome".)
- Early repolarization. (See "Early repolarization".)
- Right bundle branch block pattern with ST elevation in leads V1 to V3 (Brugada syndrome). (See "Brugada syndrome: Clinical presentation, diagnosis, and evaluation".)
- Negative T waves in right precordial leads or epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy. (See "Arrhythmogenic right ventricular cardiomyopathy: Anatomy, histology, and clinical manifestations".)
- Left ventricular hypertrophy, suggesting hypertrophic cardiomyopathy. (See "Hypertrophic cardiomyopathy: Clinical manifestations, diagnosis, and evaluation".)
- Presence of an epsilon wave in V1 and V2, suggesting arrhythmogenic right ventricular cardiomyopathy.

Echocardiography — When structural heart disease is known or is suspected based on the results of the history, physical examination, and ECG, a transthoracic echocardiogram should be performed to evaluate for structural heart disease [2]. This approach is consistent with professional society guidelines, which recommend echocardiography in patients with syncope when structural cardiac disease is suspected (table 2) [3].

In general, echocardiographic findings are used to identify the presence of structural heart disease, but they do not usually provide a specific causal diagnosis in syncope patients, as more than one potential diagnosis may be contributing. However, certain findings are highly suggestive of a cause of syncope, including left atrial myxoma, severe aortic valvular stenosis, hypertrophic cardiomyopathy with significant left ventricular outflow tract obstruction, marked pulmonary arterial hypertension, certain forms of congenital heart disease, such as abnormal aortic origin of a coronary artery, and pericardial tamponade [8]. The clinical features associated with the episode must be carefully considered, and other tests may be warranted to confirm the cause of syncope.

Additional diagnostic evaluation may include other cardiac imaging modalities (eg, to evaluate for cardiomyopathy); these additional tests should be individualized based on the suspected etiology of syncope. (See "Syncope in adults: Risk assessment and additional diagnostic evaluation" and "Determining the etiology and severity of heart failure or cardiomyopathy".)

DIFFERENTIAL DIAGNOSIS

Syncope should be distinguished from other causes of abrupt collapse which may or may not be accompanied by transient loss of consciousness (TLOC) (algorithm 1) [1]. Syncope is only one of the many causes of TLOC or apparent TLOC, including seizure disorders, traumatic brain injury (ie, concussion), intoxications, metabolic disturbances, mechanical falls, and conversion disorders (ie, psychogenic "pseudosyncope" or "pseudoseizures") [9-12]. Distinguishing these conditions from true syncope can be challenging, but is crucial to appropriate management and assessment of prognosis.

When caring for patients who present with TLOC/collapse, it is also important to consider causes that are not syncope (algorithm 1). Examples of nonsyncopal causes of TLOC or apparent TLOC include:

- Seizures. (See "Evaluation and management of the first seizure in adults".)
- Cardiac arrest, which requires resuscitation. (See "Overview of sudden cardiac arrest and sudden cardiac death" and "Adult basic life support (BLS) for health care providers" and

"Advanced cardiac life support (ACLS) in adults".)

- Sleep disturbances, including narcolepsy and cataplexy. (See "Clinical features and diagnosis of narcolepsy in adults".)
- Accidental falls or other incidents resulting in traumatic brain injury (ie, concussion).
- Intoxications and metabolic disturbances (including hypoglycemia).
- Some psychiatric conditions (eg, conversion reactions resulting in psychogenic pseudosyncope or pseudoseizures, with the latter termed "nonepileptic seizures" by some neurologists). (See "Nonepileptic paroxysmal disorders in adolescents and adults".)

ADDITIONAL EVALUATION

Additional diagnostic evaluation is individualized based on the suspected etiology of syncope (table 4 and table 2) [2]. The major causes of syncope and their evaluation are discussed separately. (See "Syncope in adults: Epidemiology, pathogenesis, and etiologies" and "Syncope in adults: Risk assessment and additional diagnostic evaluation".)

Of note, many patients have multiple comorbidities that can contribute to TLOC, and, consequently, there may be multiple plausible causes of syncope that require careful assessment. An observed abnormality should not be assumed to be the cause of collapse without first giving careful consideration to alternative diagnoses and interactions among various coexisting conditions. For example, orthostatic hypotension is common among older patients, but susceptibility to syncope may be the result of medications that the patient has been prescribed, an intercurrent illness, a previously unrecognized neurologic disease (eg, Parkinson disease), or even a previously unsuspected arrhythmia that undermines the individual's hemodynamic stability. Similarly, carotid sinus hypersensitivity (CSH) is a common finding in older patients, but only infrequently is CSH the cause of syncope (in which case the condition is termed carotid sinus syndrome). (See "Mechanisms, causes, and evaluation of orthostatic hypotension" and "Carotid sinus hypersensitivity and carotid sinus syndrome".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Syncope".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Syncope (fainting) (The Basics)")
- Beyond the Basics topic (see "Patient education: Syncope (fainting) (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Definition** – Syncope is a transient, self-limited loss of consciousness caused by transient, self-terminating, inadequate nutrient flow to the brain; episodes may or may not be preceded by prodromal symptoms. Presyncope (or near-syncope) is a manifestation of prodromal symptoms reflecting the same conditions that might lead to syncope in the same individual at other times. (See 'Definitions' above.)
- **Initial evaluation** – The initial evaluation of patients with transient loss of consciousness (TLOC) and suspected syncope should focus on differentiation of true syncope from other events (algorithm 1), risk stratification (table 1), and assessment of potential causes (table 2).

  The evaluation should generally include a comprehensive history (including information about events, preexisting conditions, medications, and family history), witness observations if available, a physical examination (which may include careful carotid sinus massage in older patients), and review of ECGs. Documentation of medications that the patient is taking is an important element of the initial evaluation. A transthoracic

echocardiogram is useful to evaluate for structural heart disease if this is suspected. (See 'Approach to initial evaluation' above.)

- **Clinical features** – The clinical features associated with a syncopal event may be diagnostic (table 1 and table 2). Key features of events include the frequency and duration of events, triggers and circumstances, presence and types of prodromal symptoms, witnessed signs during episodes, and features of recovery. (See 'Clinical features' above.)

  Classic prodromal symptoms associated with syncope and presyncope, particularly the vasovagal form of reflex syncope, include lightheadedness, a feeling of being warm or cold, sweating, palpitations, pallor, nausea, visual blurring, and diminution of hearing and/or occurrence of unusual (often "whooshing") sounds. (See 'Onset/prodrome' above.)

- **Physical examination** – A number of findings on physical examination can aid in the identification of some of the common causes of syncope, including abnormalities in the vital signs, orthostatic blood pressure changes, cardiovascular abnormalities, and, less frequently, neurologic signs. (See 'Physical examination' above.)

- **Electrocardiogram** – A 12-lead ECG should be obtained in all patients with suspected syncope, and an ambulatory ECG is warranted in many cases. The 12-lead ECG only rarely identifies a specific arrhythmic cause of syncope, although certain findings (eg, persistent bradycardia <40 beats per minute, high-grade atrioventricular (AV) block, ventricular or supraventricular tachycardia with rapid ventricular rate, pacemaker malfunction, etc) are considered diagnostic. (See 'Electrocardiogram' above.)

- **Differential diagnosis** – Distinguishing syncope from other conditions with or without TLOC requires careful diagnostic assessment (algorithm 1). Nonsyncopal causes of TLOC or apparent TLOC include accidental falls, cardiac arrest, seizures, sleep disturbances, intoxications, metabolic disorders, and some psychiatric conditions. (See 'Differential diagnosis' above.)

- **Additional testing** – Additional diagnostic evaluation is individualized based on the suspected etiology of syncope (table 2 and table 4). Many patients have multiple comorbidities that can contribute to TLOC, and thus multiple plausible causes of syncope may require careful assessment. (See 'Additional evaluation' above and "Syncope in adults: Epidemiology, pathogenesis, and etiologies" and "Syncope in adults: Risk assessment and additional diagnostic evaluation".)

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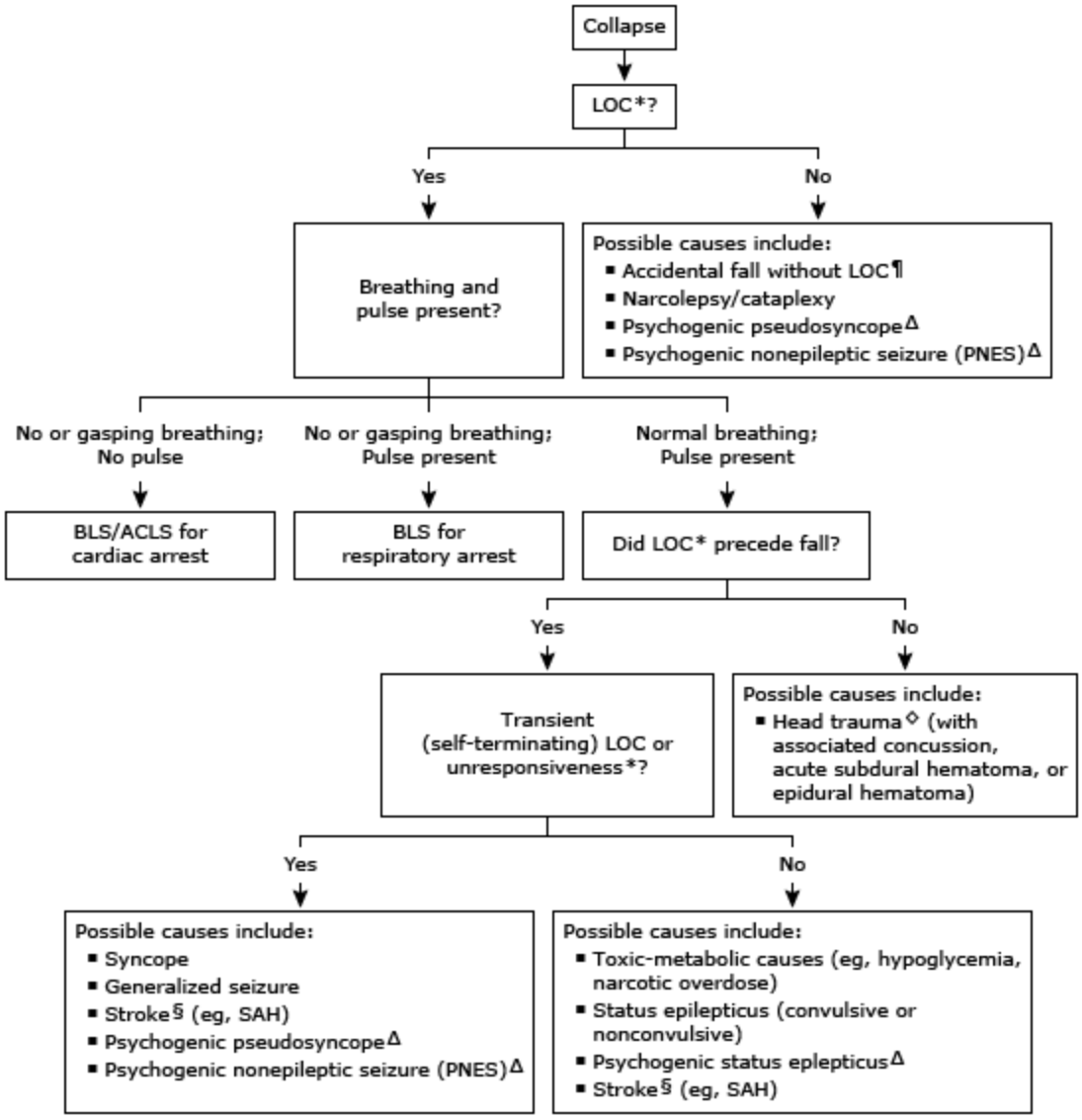
Topic 969 Version 46.0

GRAPHICS

Algorithm for syncope/collapse



This algorithm poses key questions about a collapse episode, including whether and when LOC occurred. However, in the absence of a credible witness, information about such episodes is often limited, as the affected individual may not have accurate recall of the event. Some causes have more than one possible type of presentation.

LOC: loss of consciousness; BLS: basic life support; ACLS: advanced cardiac life support; SAH: subarachnoid hemorrhage; TIA: transient ischemic attack.

* This includes actual LOC as well as apparent LOC.

¶ Accidental falls without LOC often have multiple causes, including gait, posture, or balance impairment, environmental hazard, vertigo, focal seizure, TIA, stroke, and presyncope.

Δ These conditions result in apparent transient LOC, although consciousness may be preserved.

◇ Other causes of collapse may cause secondary head trauma.

§ Most TIAs and strokes are not associated with LOC. An SAH may cause transient or prolonged LOC. A rare cause of LOC is a brainstem stroke.

Graphic 131146 Version 1.0

Clinical and electrocardiographic (ECG) features of patients with syncope at high risk of an arrhythmic cause

▪ Significant structural heart disease or CAD (including reduced LVEF, heart failure, CAD with prior MI, severe aortic or mitral stenosis, hypertrophic cardiomyopathy)
▪ Persistent sinus bradycardia <40 beats per minute or sinus pauses >3 seconds in an awake patient
▪ Third-degree (complete) AV block
▪ Mobitz II second-degree AV block
▪ Preexcited QRS complexes, suggesting Wolff-Parkinson-White syndrome
▪ Alternating left and right bundle branch block
▪ VT or paroxysmal supraventricular tachycardia with rapid ventricular rate
▪ Nonsustained polymorphic VT with long or short QT interval
▪ Long or short QT intervals
▪ Right bundle branch block pattern with ST elevation in leads V1 to V3 (Brugada syndrome)
▪ Negative T waves in right precordial leads and epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy
▪ Pacemaker or implantable cardioverter-defibrillator malfunction with cardiac pauses

CAD: coronary artery disease; LVEF: left ventricular ejection fraction; MI: myocardial infarction; AV: atrioventricular; VT: ventricular tachycardia.

Adapted from: Brignole M, Moya A, de Lange FJ, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J 2018; 39:1883.

Graphic 118883 Version 3.0

Clinical features of syncope that suggest a cause

Neurally mediated syncope:
Absence of heart disease
Long history of recurrent syncope
After sudden unexpected unpleasant sight, sound, smell, or pain
Prolonged standing or crowded, hot places
Nausea, vomiting associated with syncope
During a meal or postprandial
With head rotation or pressure on carotid sinus (as in tumors, shaving, tight collars)
After exertion
Syncope due to OH:
After standing up
Temporal relationship with start or changes of dose of vasodepressive drugs leading to hypotension
Prolonged standing, especially in crowded, hot places
Presence of autonomic neuropathy or Parkinsonism
Standing after exertion
Cardiovascular syncope:
Presence of definite structural heart disease
Family history of unexplained sudden death or channelopathy
During exertion or supine
Abnormal ECG
Sudden onset palpitation immediately followed by syncope
ECG findings suggesting arrhythmic syncope:
▪ Bifascicular block (defined as either LBBB or RBBB combined with left anterior or left posterior fascicular block)
▪ Other intraventricluar conduction abnormalities (QRS duration ≥0.12 s)
▪ Mobitz I second-degree AV block
▪ Asymptomatic inappropriate sinus bradycardia (<50 bpm), sinoatrial block or sinus pause ≥3 s in the absence of negatively chronotropic medications
▪ Nonsustained VT

▪ Preexcited QRS complexes
▪ Long or short QT intervals
▪ Early repolarization
▪ RBBB pattern with ST elevation in leads V1 to V3 (Brugada syndrome)
▪ Negative T waves in right precordial leads, epsilon waves and ventricular late potentials suggestiv of ARVC
▪ Q waves suggesting myocardial infarction

OH: orthostatic hypotension; ECG: electrocardiogram; LBBB: left bundle branch block; RBBB: right bundle branch block; AV: atrioventricular; bpm: beats per minute; VT: ventricular tachycardia; ARVC: arrhythmogenic right ventricular cardiomyopathy.

Reproduced with permission from: European Heart Rhythm Association (EHRA), Heart Failure Association (HFA), Heart Rhythm Society (HRS), et al. Guidelines for the diagnosis and management of syncope (version 2009): the Task Force for the Diagnosis and Management of Syncope of the European Society of Cardiology (ESC). Eur Heart J 2009; 30:2631. Copyright © 2009 Oxford University Press.

Graphic 66884 Version 10.0

Causes of bradycardia

Intrinsic	Extrinsic
Idiopathic degenerative disorder	Drugs
Ischemic heart disease	Antiarrhythmic agents
Chronic ischemia	Class IA - quinidine, procainamide
Acute myocardial infarction	Class IC - propafenone, flecainide
Hypertensive heart disease	Class II - β-blockers
Cardiomyopathy	Class III - sotalol, amiodarone, dronedarone
Trauma	Class IV - diltiazem, verapamil
Surgery for congenital heart disease	Cardiac glycosides
Heart transplant	Antihypertensive agents
Inflammation	Clonidine, reserpine, methyldopa
Collagen vascular disease	Antipsychotic agents
Rheumatic fever	Lithium, phenothiazines, amitriptyline
Pericarditis	Autonomically mediated
Infection	Vasovagal syncope (cardioinhibitory)
Viral myocarditis	Carotid sinus hypersensitivity
Lyme disease (Borrelia burgdorfer I)	Hypothyroidism
Neuromuscular disorder	Intracranial hypertension
Friedreich ataxia	Hypothermia
X-linked muscular dystrophy	Hyperkalemia
Familial disorder	Hypoxia
	Anorexia nervosa

Graphic 65521 Version 11.0

Major cardiovascular causes of syncope

Reflex-mediated*

- Vasovagal
  - Orthostatic vasovagal syncope: usually after prolonged standing, frequently in a warm environment, etc
  - Emotional vasovagal syncope: secondary to fear, pain, medical procedure, etc
  - Unknown trigger
- Situational
  - Micturition, defecation
  - Swallowing
  - Coughing/sneezing
- Carotid sinus syndrome

Orthostatic hypotension*

- Medication-related
  - Diuretics (eg, thiazide or loop diuretics)
  - Vasodilators (eg, dihydropyridine calcium channel blockers, nitrates, alpha blockers, etc)
  - Antidepressants (eg, tricyclic drugs, SSRIs, etc)
- Volume depletion
  - Hemorrhage
  - Gastrointestinal losses (ie, vomiting or diarrhea)
  - Diminished thirst drive (primarily in older patients)
- Autonomic failure
  - Primary: pure autonomic failure, Parkinson disease, multiple system atrophy, Lewy body dementia
  - Secondary: diabetes mellitus, amyloidosis, spinal cord injuries, autoimmune neuropathy (eg, Guillain-Barré), paraneoplastic neuropathy

Cardiac

- Tachyarrhythmias
  - Ventricular tachycardia
  - Supraventricular tachycardias
- Bradyarrhythmias (with inadequate ventricular response)
  - Sinus node dysfunction
  - Atrioventricular block
- Structural disease

- Severe aortic stenosis
- Hypertrophic cardiomyopathy
- Cardiac tamponade
- Prosthetic valve dysfunction
- Congenital coronary anomalies
- Cardiac masses and tumors (eg, atrial myxoma)

- Cardiopulmonary/vascular
  - Pulmonary embolus
  - Severe pulmonary hypertension
  - Aortic dissection

SSRI: selective serotonin reuptake inhibitor.

* Reflex-mediated syncope and syncope due to orthostatic hypotension are more likely to occur, or are more severe, when other factors may also be contributing, such as medication(s) causing low blood pressure, volume depletion, pulmonary diseases causing reduction in brain oxygen supply, alcohol use, and/or environmental factors (excessive heat or humidity).

Adapted from: Brignole M, Moya A, de Lange FJ, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J 2018; 39:1883.

Graphic 118175 Version 4.0