



## Center for Excellence in Healthcare Communication.



# SYNCOPE KEY RECOMMENDATIONS FOR PRACTICE

## COMMENTARY

### ETHICS DECLARATIONS

Competing interests:

-The authors declare no competing interests.

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-All authors contributed to the manuscript.

### ADDITIONAL INFORMATION

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## DEFINITION

Syncope is a transient loss of consciousness and postural tone followed by spontaneous recovery. If these characteristics are not fulfilled, you must rule out other causes of transient loss of consciousness such as seizures, hypoglycemia, stroke, trauma (1,2).

Recovery from syncope is characterized by immediate restoration in orientation and normal behavior, although it may be accompanied by fatigue. It is caused by a reduction and later restoration of the brain perfusion (1).

## DIFFERENTIAL DIAGNOSIS

In the evaluation of syncope, the history of the episode is critical; particular attention should be paid to any symptoms that occurred before loss of consciousness, for example prolonged exposure to sunlight, fasting and even negative emotions. The context within which syncope occurred, and any consequences (e.g., injury or postictal confusion) (2,3).

Important differential diagnosis for syncope includes

1. Seizure disorder: Seizures associated with aura, tonic-clonic activity, prolonged duration of unconsciousness, urinary and/or bowel incontinence, tongue biting and confusion after regaining consciousness. These differentiate syncope from seizures (3,4).
2. Hypoglycemia.
3. Panic attacks: Feeling impending doom, palpitations, air hunger and tingling of perioral region and tips of fingers.

### MNEMONIC: Causes of Syncope (“SYNCOPE”)

- Situational
- Vasovagal (The V looks like a Y. This is a type of neurally mediated syncope.)
- Neurogenic
- Cardiac
- Orthostatic hypotension
- Psychiatric
- Everything else (metabolic causes)

Causes of syncope can also be categorized according to etiology following the European Society of Cardiology Classification system (4,5).

## COMPLICATIONS

Patients can sustain injuries from fall due to syncope. These injuries can be worse if they were driving during the event (6).

## OVERVIEW

Syncope can be related to many causes. Some patients have a medical condition they may or may not know about that affects the nervous system or heart (7) alterations of blood

flow resulting in blood pressure drop when changing positions (e. g. going from lying down to standing).

Syncope should be differentiated from presyncope, dizziness, and vertigo—all of which do not result in loss of consciousness. Non-perfusion-related causes of alteration in consciousness such as seizures must be excluded (8, 9).

## PROGNOSIS

The overall prognosis of syncope depends on the underlying cause. Patients with syncope with structural heart disease and primary cardio electrical disease are at high risk of overall mortality and sudden cardiac death. Young patients with reflex syncope have an excellent prognosis (10).

Morbidity in patients with syncope is associated with recurrence of episodes and physical injury. In population studies, approximately a third of patients have recurrence of syncope in 3 years of follow up, and the rate of recurrence seems to be dependent on the number of previous episodes, but independent of gender, tilt test results, severity and presence of heart disease (8,9).

The predicted recurrence in 1 to 2 years for patients with 1 or 2 syncope episodes is 15% to 20% whereas for patients with 3 syncope episodes it is 36% to 45%. Young patients with psychiatric disease have high rates of recurrence of pseudosyncope (9).

## KEY RECOMMENDATIONS FOR PRACTICE: THE GENERAL PURPOSE OF ADMISSION:

- The examination should focus on initial vital signs; orthostatic blood pressure measurements; and vascular (pulses and carotid bruits), cardiac, pulmonary (evidence of congestive heart failure), abdominal, rectal, and skin/nail (anemia) signs. Any new focal neurologic findings suggest a primary central nervous system lesion.
- With the proper diagnosis and treatment, syncope can be managed and controlled. If you have had an episode of syncope, there is about a 30% chance you will have another episode. Your risk of another episode and how the condition affects you depends on several factors, including cause, age, underlying diseases, and medications.
- Orthostatic hypotension is treated with volume resuscitation and discontinuation of culprit drugs. Rarely, support stockings, salt tablets, fludrocortisone, and midodrine may be added if autonomic insufficiency is present and disabling.
- Psychiatric Evaluation: Consider with concurrent electroencephalography and video monitoring.
- Prevention: Medications, along with the use of alcohol or illicit drugs, should be reviewed carefully. Education about likely precipitants or triggers can help prevent recurrences.
- Presyncope is poorly studied, and the true incidence is unknown. A study of 881 patients presenting to the emergency department with presyncope showed a 30-



- day risk of serious outcomes of 5% and a mortality rate of 0.3%.
- The most common cause of syncope is vasovagal and is self-limiting. A primary care physician or hospitalist can manage this. Cardiology consultation is needed when cardiac etiology suspected. Neurology consultation is needed when cerebrovascular causes suspected.
  - Most of the patients with emergency room visits for syncope do not have a diagnosis at discharge. Even hospitalized patients leave the hospital with unclear etiology for their syncope. The majority of times the cause is benign but patients need close outpatient follow-up to make sure there is a cause identified and does not recur. This needs close follow-up with a primary care physician and cardiologist.
  - Evaluation for and treatment of any injuries sustained during a sudden fall require immediate attention.
  - The goal of treatment is to avoid recurrence. Treatment options include: Taking medications or making changes to medications you already take. Wearing support garments or compression stockings to improve blood circulation.
  - Admission to a hospital is necessary for syncope that may be secondary to coronary events, pulmonary embolization, stroke, unstable arrhythmias, and syncope-related injuries. Hospital admission is necessary for status epilepticus, need for detoxication, severe dehydration, or hypertensive crises, which may be part of the autonomic failure syndromes or a complication of the treatment given for syncope.
  - The best way to determine the need for hospitalization is to attempt to risk-stratify patients into low, moderate, and high-risk groups. low risk patients include those that are younger and have prodromal symptoms prior to the episode, not including chest pain or dyspnea. These patients do not require any hospitalization and don't require any specific follow-up or outpatient work-up unless symptoms are recurrent. Intermediate risk patients can be categorized based on the clinical suspicion for arrhythmia and the frequency of symptoms. High-risk patients include those with a history of heart disease (both structural and/or acute coronary syndrome), those with concerning ECG changes, advanced age, or prodromal symptoms such as syncope with exertion and new onset angina or dyspnea.
  - All patients presenting with syncope should have orthostatic vital signs and standard 12-lead electrocardiography.
  - Laboratory testing can be ordered based on history and examination to aid in diagnosis of syncope, but routine comprehensive testing in all patients with syncope is not necessary.
  - Patients with syncope and evidence of heart failure or structural heart disease should be admitted to the hospital for monitoring and evaluation.
  - Resting 12-lead electrocardiography can be beneficial in determining the cause.
  - In the evaluation of simple syncope and a normal neurologic evaluation, do not obtain brain imaging studies (computed tomography or magnetic resonance imaging).
  - The decision to perform an expedited and monitored evaluation of patients presenting with syncope is based on the likelihood of short-term adverse outcomes. Unnecessary admissions for patients meeting low-risk criteria result in high medical costs without improvements in morbidity and mortality, patient safety, or quality of life.
  - Clinical history suggestive of arrhythmic syncope (e.g., syncope during exercise, palpitations, or syncope without warning or prodrome).
  - Electrocardiographic history suggestive of arrhythmic syncope (e.g., bifascicular block, sinus bradycardia < 40 beats per minute in absence of sinoatrial block or medication use, QRS preexcitation, abnormal QT interval, ST segment elevation leads V1 through V3 [Brugada pattern], negative T wave in right precordial leads and epsilon wave [arrhythmogenic right ventricular dysplasia/cardiomyopathy]).
  - Transthoracic echocardiography may be beneficial if there is suspicion for structural heart disease.
  - Development of new ambulatory monitoring devices allow patients to receive prolonged cardiac monitoring in an outpatient setting and secondarily lower health care costs.
  - Types of ambulatory monitoring devices: Characteristics include duration that device is worn, whether the patient can trigger the device to record based on symptoms, whether the device records continuously or when a potential event is detected, whether the event strip is transmitted to the physician, and patient comfort/ease of performing their activities of daily living (ADLs). Patients that are intermediate risk may also require ambulatory monitoring but this can be guided by the clinical suspicion for an arrhythmia and the inpatient or outpatient resources available.
- Ambulatory monitoring: who to monitor
- \*Holter monitor: 24–48 hr
  - \*Event monitor: 30 days
  - \*Loop recorder: 30 days
  - \*Mobile cardiac telemetry system: 30 days
  - \*Long-term continuous rhythm recorders: 14 days
- Laboratory testing in the evaluation of syncope should be ordered as clinically indicated by the history and physical examination.
  - Psychiatric assessment is recommended in patients with frequent recurrent syncope who have multiple other somatic complaints and when initial evaluation raises concerns about stress, anxiety and possible other psychiatric disorders.
  - Rarely helpful; complete blood count, brain natriuretic peptide testing, fecal occult blood testing, human chorionic gonadotropin testing in women of childbearing age; troponin testing may be beneficial to identify cardiac etiology.
  - Carotid sinus massage: Diagnostic if ventricular pause is more than three seconds or if systolic blood pressure decreases by more than 50 mm Hg; contraindicated in patients with bruits or a history of transient ischemic attack/cerebrovascular accident within the past three months.
  - Sinus node abnormalities will typically lead to bradyarrhythmias. When sinus bradycardia is noted, the first intervention should assess iatrogenic causes, such as medications with bradycardia as an intended or unintended effect. If such medications are in use and



- unnecessary to the patient, they should be terminated. Otherwise, pacemakers should be the next step sought out in management. Implantable dual-chamber pacemakers are recommended to prevent future syncopal events. Implantation of a pacemaker should theoretically circumvent this outcome.
- ECG: Diagnostic yield is 3% to 5%, but can aid in diagnosing arrhythmia, ischemia, pulmonary embolus (increased pulmonary pressures or right ventricular enlargement), hypertrophic cardiomyopathy, and Brugada syndrome; findings suggestive of arrhythmia include bundle branch block, intraventricular conduction delay, sinus bradycardia (less than 50 beats per minute), prolonged QT interval, QRS preexcitation, Q waves.
  - Recurrent syncope with unremarkable initial evaluation; clinical or ECG features suggestive of arrhythmic syncope; unexplained falls: Holter monitoring for 24 to 48 hours, external loop recorders for four to six weeks, implantable loop recorders for up to three years; consider testing in patients with suspected epilepsy not responsive to therapy; implantable loop recorders have high diagnostic yield in recurrent unexplained syncope.
  - Echocardiography: Diagnostic in aortic stenosis, pericardial tamponade, obstructive cardiac tumors or thrombi, aortic dissection, hypertrophic cardiomyopathy, congenital anomalies of the coronary arteries, acute right ventricular strain (in pulmonary embolism).
  - Electrophysiology: Not recommended in patients without underlying heart disease; consider in high-risk patients with recurrent unexplained syncope.
  - Exercise testing: Inadequate blood pressure increase in younger patients suggests hypertrophic cardiomyopathy or ischemia; similar findings in older persons may suggest autonomic dysfunction; may unmask ECG changes of channelopathies.
  - Treatment of any correctable cardiac abnormality should be the first consideration (i.e., permanent pacemaker for patients with bradyarrhythmias, implantable cardiac defibrillators in patients with ventricular tachycardia, or valvular surgery for those with severe aortic stenosis).
  - Head-up tilt test: Used in patients with negative initial findings, normal cardiac structure, and no evidence of ischemia; contraindicated in patients with ischemic heart disease, uncontrolled hypertension, left ventricular outflow tract obstruction, or aortic stenosis.
  - Neurologic testing: Seizure can be confirmed with electroencephalography; cranial imaging studies as clinically indicated; 20% to 30% of seizures with transient loss of consciousness could be the result of syncope; tonic-clonic limb movements and muscle twitching may occur in cardiac and neurally mediated syncope.
  - Orthostatic blood pressure measurement: Diagnostic if systolic blood pressure is less than 90 mm Hg or decreases by 20 mm Hg or more (10 mm Hg or more in symptomatic patients); increase of 30 beats per minute suggests postural tachycardia syndrome; up to 40% of

asymptomatic adults older than 70 years and 23% of patients younger than 60 years will have positive orthostatic blood pressure measurements.

- Patients with frequent vasovagal syncope may benefit from a trial of midodrine,  $\beta$ -blockers, paroxetine, or disopyramide. In rare instances, patients with a significant cardioinhibitory component may benefit from permanent pacemaker placement. The most important component of treating vasovagal syncope is educating the patient about the condition, trigger avoidance, and reassurance.

Based on data from reference.



<b>HIGH RISK CRITERIA WHICH REQUIRE PROMPT HOSPITALIZATION OR INTENSIVE EVALUATION</b>
<i>Severe structural or coronary artery disease</i>
Heart failure or low left ventricular ejection fraction
Previous myocardial infarction
<i>Clinical or ECG features suggesting arrhythmic syncope</i>
Syncope during exertion or supine
Palpitation at the time of syncope
Family history of sudden cardiac death
Non-sustained ventricular tachycardia
Bifascicular block (LBBB or RBBB combined with left anterior or left posterior fascicular block)
Inadequate sinus bradycardia or sinoatrial block in absence of negative chronotropic medications or physical training
Pre-excited QRS complex
Brugada ECG pattern
ECG findings suggestive of arrhythmogenic right ventricular cardiomyopathy
<i>Important comorbidities</i>
Severe anemia
Electrolyte disturbance

LBBB: left bundle branch block; RBBB: right bundle branch block (11,12).

#### KEY RECOMMENDATIONS FOR PRACTICE: TREATMENT BY CAUSE OF SYNCOPE:

##### Vasovagal syncope:

- Avoid triggers, decongestants, cardiac stimulants, and vasodilators
- Normalize salt intake and maintain hydration using electrolyte-balanced fluids
- Use compression support stockings if venous pooling is present and teach physical counterpressure maneuvers
- Discontinue/reduce doses of medications that may cause orthostatic hypotension or dehydration
- Consider use of metoprolol in patients older than 40
- Consider midodrine in patients with refractory vasovagal syncope without hypertension or urinary retention
- Use fludrocortisone in pediatric population with severe vasovagal syncope
- Consider permanent cardiac pacemakers in patients with asystole during tilt table testing

##### Carotid sinus syndrome:

- Limit the use of tight collars or activities that trigger symptoms
- Consider cardiac pacing

##### Autonomic failure

- Postural education
- Blood volume expansion (8 to 10 gram of salt diet and fludrocortisone)
- Support garments
- Exercise for muscle tone
- Midodrine
- Noncardioselective beta blocker if no contraindications
- Avoid vasodilators
- If supine hypertension, elevate head-of-bed when supine; add hydralazine, nifedipine extended release or an angiotensin II receptor blocker

##### Orthostatic hypotension

- Educate patients about the importance of gradual changes in posture, especially in the morning and after meals
- Avoid dehydration, exercise in hot environment, prolonged standing, large meals, excessive alcohol intake, and medications that could exacerbate hypotension
- Raise the head of bed 6 to 8 inches to reduce renal perfusion and nocturnal diuresis
- Employ exercise training to improve muscle tone and physical counterpressure maneuvers
- Use compression support stockings or abdominal binders or both and recommend increase in daily salt intake
- Consider the use of midodrine in refractory cases in patient without hypotension and urinary retention

##### Postural tachycardia syndrome

- Regular, structured, exercise program
- Acute intravenous infusion of up to 2 L of normal saline in the acute decompensation
- Consider prescribing 2 to 3 L regular water consumption with 10 to 12 grams of salt daily



- Reasonable to use fludrocortisone, pyridostigmine, midodrine or low-dose propranolol in refractory cases
- In patients with prominent hyperadrenergic features, it is reasonable to use clonidine or alpha-methyl dopa
- Avoid vasodilators, diuretics, decongestants, and other cardiac stimulants

Based on data from reference.

### CONCLUSION:

The European Society of Cardiology - ESC guidelines recommend the creation of syncope units that can establish a rapid and accurate etiological diagnosis, thus reducing the costs of hospitalization and diagnostic exams. Most syncope units, unlike ours, operate within emergency department (EDs).

The authors present decision algorithms based on the European Society of Cardiology - ESC guidelines and their own four-year experience. In our syncope unit, an etiological diagnosis was obtained in 88% of patients and a diagnosis of cardiac syncope in 20%. These percentages were compared with those from other syncope units cited in the ESC guidelines, so as to gauge our results.

Historical factors that are strongly associated with cardiac syncope include a strong family history of sudden death or early myocardial infarction before 50 years of age, a history of heart disease (heart failure, myocardial infarction, valvular disease, and arrhythmias) and presenting symptoms suggestive of heart.

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ETIOLOGY OF SYNCOPE:

Five Classes of Syncope Defined by Their Associated Causes or Triggers (13).

CLASSIFICATION	DEFINITION	CAUSES
<b>Neurocardiogenic</b>	Inappropriate vasodilation ± bradycardia	Increases vagal tone (micturation, defecation); situational (prolonged standing); vagal nerve stimulation (shaving)
<b>Orthostatic</b>	Documented postural hypotension with symptoms	Drop in systolic blood pressure by ≥ 20 mmHg or tachycardia > 20 bpm; example: volume loss, dysfunction of autonomic nervous system, medication side effects
<b>Neurologic</b>	Least common, must return to baseline with no neurological deficits	Example: transient ischemic attack's, seizure, complex migraine, subclavian steal
<b>Cardiac</b>	Most dangerous form, can be life-threatening, multiple etiologies	Arrhythmias (tachy or brady), valvular heart disease, myocardial infarction, cardiac tamponade
<b>Unknown</b>	Unexplained despite thorough work-up	Rule out potential life-threatening causes

Classes of Syncope Defined by Their Associated Causes or Triggers (14).

EVALUATION - CAUSES	EVALUATION - TESTS TO DETERMINE CAUSES OF SYNCOPE INCLUDE:	DIFFERENTIAL DIAGNOSIS
<b>Cardiac Mechanical</b>	- Echocardiogram test. - Chest x-ray - D-dimer / CT Pulmonary Angiography - Cardiac Enzymes. Consider Stress Test if negative	Aortic Stenosis, Hypertrophic, Cardiomyopathy, Pulmonary Embolism, High blood pressure (hypertension), Stenosis, Aortic Dissection, Myocardial Infarction.
<b>Cardiac Electrical</b>	- EKG, Telemetry + Pulse Ox monitoring, - Pacer Interrogation - Discharge with rhythm monitor (e.g., ZioPatch or loop recorder).	(AV Block, Sick Sinus Syndrome, Arrhythmia, Long QT syndrome, Bradycardia, Ischemia, Pre-Excitation).
<b>Vasovagal</b>	- Carotid Massage	(Carotid sinus cardioinhibitory, vasodepressor central, Vasovagal cough, micturition defecation, post-prandial valsalva, sneeze).
<b>Orthostatic</b>	- Orthostatic Vital Signs - Review Electrolytes	(Dehydration, Diuretic drugs, Blood Loss, Autonomic insufficiency, Sympathetic nervous system blocker drugs, Adrenal Insufficiency, Vasodilator drugs, Idiopathic)
<b>Neurologic</b>	- Head CT - EEG - MRI/MRA - Cardiac Echo - Carotid Dopplers	Transient Ischemic Attack (TIA), Seizure, Takayatsu (Giant Cell) Arteritis, Intermittent Pressure Hydrocephalus, Subclavian Steal Syndrome, Vertebrobasilar insufficiency
<b>Metabolic</b>	- Blood sugar QAC/HS - Review CBC - Urine toxicology screen - Review medications (hypoglycemic dosing; sulfonylureas and insulin)	Hypoglycemia, Hypoxia, Shock, Hyperventilation, Anemia, Alcoholic.
<b>Psychiatric</b>	(Psychogenic / Conversion (self-concern, no self-injury).)	Panic disorders, depression, hysteria.

Based on data from reference.



<b>SYNCOPE CLINICAL MANAGEMENT IN THE EMERGENCY DEPARTMENT</b>
<b>HIGH-RISK PATIENTS ARE THOSE WHO HAVE AT LEAST ONE HIGH-RISK CHARACTERISTIC:</b>
<ul style="list-style-type: none"><li>• Syncope during exertion, in supine position, associated with new onset of chest discomfort, and palpitations before syncope;</li><li>• Family history of sudden death;</li><li>• Heart failure, aortic stenosis, left ventricular outflow tract disease, dilated cardiomyopathy, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, left ventricular ejection fraction &lt;35%, previously documented ventricular arrhythmia, coronary artery disease, congenital heart disease, previous myocardial infarction, pulmonary hypertension, previous implantable cardioverter-defibrillator implantation;</li><li>• Hemoglobin &lt;9 g/dl, lowest systolic blood pressure in the ED &lt;90 mm Hg, sinus bradycardia &lt;40 bpm; and</li><li>• New (or previously unknown) left bundle branch block, bifascicular block and first-degree atrioventricular (AV) block, Brugada ECG pattern, ECG changes consistent with acute ischemia, nonsinus rhythm (new), bifascicular block, prolonged QTc (&gt;450 ms).</li></ul>
<b>LOW-RISK PATIENTS ARE THOSE WITH ONE OR MORE LOW-RISK CHARACTERISTICS AND WITHOUT ANY HIGH-RISK CHARACTERISTICS. LOW-RISK CHARACTERISTICS INCLUDE:</b>
<ul style="list-style-type: none"><li>• Age &lt;40 years;</li><li>• Syncope occurring while in standing position, standing from supine/sitting position; nausea or vomiting before syncope; feeling of warmth before syncope; syncope triggered by painful or emotionally distressing stimulus or by cough, defecation, micturition; and</li><li>• Prolonged history (years) of syncope with the same characteristics of the current episode.</li></ul>

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Based on data from reference.





## IMPORTANT QUESTIONS TO ANSWER IN THE CLINICAL HISTORY

What were you doing at the time of the event?
What was the time of the day?
Were there any stressors like warm environment, prolonged standing, or fear?
Were you standing, sitting, lying down or exercising?
Did you have a fever?
Any alcohol involved?
Are you taking over-the-counter or prescribed medications? Any recent changes in medications or doses?
Symptoms at onset of event? Palpitations, nausea, chest pain, or shortness of breath?
Do you remember the event?
Did bystanders notice change of color, convulsions? How long did they report you were unconscious?
Did you bite your tongue during the event?
Was there urinary incontinence during the event?
Did you feel symptoms after you regained consciousness? Fatigue, nausea palpitations, pain, sweating, confusion?
Did symptoms improve by sitting down or by lying flat?
For women, are you pregnant?
Did you have any fluid loss (diarrhea, vomiting, bleeding, excess perspiration) preceding the event?
Have you lost weight? If yes, how?
Have you had recent surgeries or procedures requiring anesthesia?
Did you experience any injuries in relation to the event?
Have you experience similar events in the past?
Did you faint during childhood?
Is there family history of fainting? If yes, who and what was the diagnosis?
Do you have vertigo, or ear ringing?

Based on data from reference (14).