APPROACH TO
McGill Journal of Medicine

Syncope

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Publication Date
June 1, 2025

MJM 2025 | 22 (1) 1043
https://doi.org/10.26443/mjm.v22i1.1043

McGill Journal of Medicine

www.mjm.mcgill.ca

ABSTRACT

Syncope, the transient and abrupt loss of consciousness with spontaneous recovery, is estimated to make up 1% of all emergency department presentations in Canada. (1) Syncopal cases are enigmatic as patients frequently present themselves as fully recovered from the episode, even though its etiology can still be present and life-threatening. It is up to the clinician to conduct a strong history, physical exam and the appropriate follow-up investigations to confirm a diagnosis. This Approach To article aims to provide an overview and step-wise approach to diagnosing and managing a syncopal presentation.

KEYWORDS
Syncope, TLOC, Cardiac, Orthostatic, Reflex-mediated, Vaso-vagal

1 | QUESTION

A 20-year-old college student presents to the ER for a recent episode of loss-of-consciousness. He had a stressful morning preparing for finals week and grabbed a latte at his local coffee shop before heading to school with his best friend. While standing in line, he began feeling sweaty and nauseous, followed by the sensation of "the world blacking out" before him. He woke up surrounded by people and immediately wondered why they crowded around him. This event has never happened to him before. His friend denies witnessing any tongue biting, incontinence, movement, or injury to the head. The event lasted seconds, and the patient could tell where he was upon waking.

Upon further questioning, he is otherwise healthy, has had no previous surgeries, and does not take medication. Both his parents have high blood pressure, and

Abbreviations: TLOC, transient loss of consciousness.

his maternal grandmother died from a heart attack at age 76. He drinks alcohol occasionally with friends on weekends, does not smoke, and denies consuming illicit drugs.

In the ER, his vital signs are as follows: BP: 127/76, HR: 87, RR: 14, SPO2: 99%, T: 36.4. After 5 minutes in the supine position, his blood pressure was 130/85 mmHg. Immediately after, in the sitting position, his blood pressure was 128/80 and 127/79 when asked to stand upright. His physical exam is unremarkable. What is the next best step?

A. No further testing is needed; reassure the patient and send them home.
B. Order an ECG.
C. Order an EEG.
D. Order a chest X-ray.
E. Order an echocardiogram.

The answer will be addressed at the end of the article, but first, let us explore the approach to syncope.

2 | INITIAL APPROACH

Syncope is a transient, self-limited event with an inability to maintain tone due to acute global cerebral hypoperfusion and is a classification that is part of the umbrella term: transient loss of consciousness (TLOC). (2) The diagnostic approach of syncope specifically, focuses on excluding whether the patient's presentation is due to the other causes of TLOC including, intoxications, metabolic disorders (hypoxemia, hypercapnia and hypoglycemia), psychogenic (subdivided into Psychogenic non-epileptic seizures and psychogenic pseudosyncope) and neurologic mimicker without generalized cerebral hypoperfusion (seizures, cataplexy, intracerebral/subarachnoid hemorrhage, cerebrovascular disorders (transient ischemic attacks and subclavian steal syndrome)) (3,4). Syncope itself can be divided into two main categories: cardiac and noncardiac causes. The latter can be further subdivided into two: reflex syncope (also called neurally mediated syncope) and orthostatic hypotension (4). Now, let's learn how using a solid history, physical exam, and investigations could help narrow the differential diagnosis.

2.1 | History and Physical Exam

Individuals will often present with a primary complaint of "fainting," "blacking out," or "passing out" (5). To obtain an adequate history, one must gather a past medical history, family history, and a history of present illness that includes a review of symptoms and clinical information about the event's evolution. Questions should include what happened before (what the patient was doing or seeing before the event, history of head trauma, associated prodrome, associated symptoms present), during (were there any movements during the event, urinary or fecal incontinence, tongue biting), and after (absence of confusion vs. a prolonged postictal state) the event. Corroboration from witnesses will likewise become vital in diagnosis. With this information, one can start to piece together the cause of syncope (Figure 1).

Cardiac syncope is secondary to an underlying rhythmic, structural, contractile, or cardiopulmonary etiology leading to decreased cardiac output and subsequent cerebral hypoperfusion (7). This presentation is associated with increased morbidity and mortality rates, which require additional workup and hospital admission (8). Cardiac-related syncope is more likely to occur in older male patients with a prior history of cardiac disease. Before the event, patients may have symptoms associated with exertion and report feeling short of breath or experiencing palpitations and/or chest pain. Often, they present without any prodrome, such as a sudden fall (9). As with non-cardiac syncope, the episode will most often have an absence of movement or associated symptoms during the event and complete recovery afterward. It is important to rule out symptoms associated with life-threatening causes, detailed in Figure 1 (i.e., pleuritic pain can be suggestive of a pulmonary embolism) (10). Clinical tools such as the Canadian Syncope Risk Score (CSRC), which predict 30-day serious outcomes not evident during initial ED evaluations of syncope, can be

used to further aid clinicians when dealing with this presentation (11).

Noncardiac syncope is divided into reflex-mediated and orthostatic (12). Orthostatic syncope is due to postural changes with insufficient counter-regulation (requires a postural decrease in BP 20/10 mm Hg). It can be secondary to medication effects (i.e., vasodilators, diuretics, phenothiazine, antidepressants), volume depletion (i.e., internal, or external hemorrhage, diarrhea, vomiting), or neurogenic disorders that can be primary (i.e., Parkinson's disease, and pure autonomic failure) or secondary (i.e., diabetes, amyloidosis) (3,9). Patients typically complain of light-headedness, nausea, and dizziness before the episode. Symptoms on history that are important to rule out are back or abdominal pain, and can be suggestive of a leaking abdominal aortic aneurysm or ruptured ectopic pregnancy. Reflex-mediated syncope, the most common type of syncope, is divided into vasovagal, situational, or secondary to carotid sinus activation (12,13). In this case and beyond linking TLOC to a specific event, patients are likely to have a strong prodrome associated with nausea, pallor, diaphoresis, hyperventilation, and impairment of the senses. Patients often complain of visual blackness and faintness of hearing before the episode occurs (14).

A detailed physical exam provides critical information about the origin of syncope. Orthostatic vitals can help confirm or rule out syncope of orthostatic etiology (14). Firstly, blood pressure and heart rate are taken in a lying position, followed by a sitting position, an immediate standing position, and lastly, taken after 3 minutes of standing. A carotid artery massage with a pause in heart rate 3 seconds or a fall in systolic BP 50 mm Hg can confirm carotid sinus hypersensitivity (6). Furthermore, conducting a detailed cardio-respiratory exam can further assess the underlying cause. Auscultating heart murmurs, extra heart sounds, or signs of respiratory compromise can save a patient from a life-threatening cause of syncope.

2.2 | Initial Investigation

Once a detailed history and physical exam are taken, the Canadian Cardiovascular Society (CCS) recommends a 12-lead ECG to rule in or out a cardiac event, which has implications on prognosis and management (12). ECG findings that must be ruled out are sinus node dysfunction (sinus arrest or block, bradycardia/tachycardia syndrome), atrioventricular conduction system disease (Mobitz type II, high-grade and complete AV block), and supraventricular and ventricular tachycardia (3,4). Suggestive findings that should be ruled out include PR in-

	Types		Underlying causes	Clinical features
Cardiac	Arrythmias		Bradycardia: Sinus node dysfunction Atrioventricular conduction system disease Tachycardia: Supraventricular tachycardia Ventricular tachycardia	Abrupt and unprovoked (most common presentation) Prodromal symptoms: palpitations
	Structural		Massive myocardial infarction Aortic stenosis Mitral valve prolapse Hypertrophic cardiomyopathy Cardiac tamponade	Personal or family history of coronary artery disease (CAD) Related to episodes of exertion Prodromal symptoms: palpitations, dyspnea, chest pain
	Cardiopulmonary		Pulmonary embolism Abdominal aortic aneurysm	
Non cardiac	Reflex mediated	Vasovagal	Prolonged standing Emotional stress Pain or injury	Prodromal symptoms: diaphoresis, dizziness, nausea Related to a precipitating factors (see causes)
		Situational	Cough Laughing gastrointestinal stimulation (swallowing, defecation)	Related to precipitating factors (see causes)
		Carotid sinus	Pressure on the carotid sinuses	Occurs during pressure ie (e.g., massage, shaving, tightening a necktie)
	Orthostatic		Volume depletion (hemorrhage, vomiting) Medications leading to vasodilation Prolonged bed rest Primary autonomic failure: diabetic neuropathy, Parkinson's disease	Related to precipitating factors (see causes): prolonged standing, abrupt changes in positions, dehydration Prodromal symptoms: lightheadedness, nausea, dizziness

FIGURE 1 Etiologies of cardiac and noncardiac causes of syncope (non-exhaustive list). Information taken from (3,6).

terval shortening with delta wave (i.e., Wolf-Parkinson-White syndrome) and prolonged QT interval (indicating a propensity for torsades des pointes). Prolonged monitoring may show a transient but recurring dysrhythmia. (10) Though cardiac monitoring is used in all patients with an acute presentation, its nature and extent (Holter or loop-event monitoring) depend on the frequency, severity of syncope, and suspicion of an arrhythmic etiology (10). Imaging (echocardiogram or stress testing) is conducted only with the clinical suspicion of ischemic, structural, or valvular heart disease. Regular stress testing should also be performed in patients with a high suspicion of syncope secondary to an ischemic cause. When standard imaging is inconclusive in patients with structural heart disease (congenital, inflammatory, or infiltrative heart disease), advanced cardiac imaging (CT, MRI) should be performed. After noninvasive testing, invasive electrophysiology studies (EPS) are only recommended in patients with an abnormal ECG or structural heart disease, which can have therapeutic benefits. Blood work is not recommended in the initial syncope workup unless there is suspicion for a specific condition, and can lead to a diagnosis (9, 12). Certain conditions may include myocardial infarction-troponins, pulmonary embolism-D-dimer, heart failure, NT-proBNP, CBC, and human chorionic gonadotropin testing in women of childbearing age. A hematocrit may explain orthostatic syncope (i.e., occult gastrointestinal bleeding), and a pregnancy test for potential childbearing-aged women is always warranted (i.e., red ectopic pregnancy). Electrolyte testing may show a decreased bicarbonate after a seizure, and hypomagnesemia can explain weakness or irritable myocardium (10).

The American College of Cardiology, American Heart Association, and Heart Rhythm Society guidelines agree with “Choosing Wisely” in discouraging neurologic testing in patients with syncope as a routine test (6,15,16). Brain imaging should only be performed when the intracranial disease is highly suspected as a contributing cause or if there is suspicion of head trauma from the syncopal episode. Without focal neurological findings, carotid artery imaging should not be performed (6). Utilizing the patient’s history, physical exam findings such as orthostatic vitals and carotid artery massage can confirm non-cardiac etiologies of syncope. Tilt table testing, when positive, can help distinguish syncope associated with myoclonic movements (involuntary twitching of muscle groups, known as convulsive syncope) from epilepsy in cases of diagnostic uncertainty (12). If non-cardiac causes are benign, no further workup is necessary for the ED if patient history, physical exam, and initial workup are negative for other life-threatening reasons (3).

Furthermore, it is crucial to consider that diagnostic workup can be done outside the emergency department. The European Society of Cardiology supports video monitoring to help with diagnostic dilemmas of syncope vs epilepsy. TLOC secondary to conversion syndromes when the initial workup is inconclusive (12).

3 | BEYOND THE INITIAL APPROACH

Shared decision-making to respect patients’ needs is a priority when approaching management and care. Below, we explain further management of selected causes of syncope:

3.1 | Vasovagal Syncope (VVS)

If recurrent VVS reduces one’s quality of life, multiple nonpharmacological approaches can aid in symptom management. These include education on the recognition of prodromes and lifestyle modifications such as avoidance/being aware of triggers and situations. Limited evidence supports increased water and salt intake. In younger patients with a sustained prodrome (> 1 minute), it is recommended to do counter-pressure maneuvers (leg-cross, limb/abdominal contract, squat). Lying down quickly is also encouraged with the onset of presyncope.

3.2 | Orthostatic Syncope

The CCS recommends education, reassurance, and adequate salt and water intake as a first-line therapy for patients if there are no contraindications. If non-pharmacologic measures are of no benefit, removal (vs dose adjustment) of any offending medications (such as vasoactive agents) is recommended if there are no clear indications for their use or are no suitable replacements. (3,6) With apparent persistent symptoms, patients are encouraged to try counter-pressure maneuvers (described earlier), compression garments, and head-up tilt sleeping. After nonpharmacological interventions are exhausted, first-line pharmacotherapy includes midodrine, fludrocortisone, or droxidopa (short-term use—not available in Canada) (12).

4 | THE GREAT MIMICKER – SEIZURE

Given the strong association between TLOC and seizures as a differential diagnosis, a non-exhaustive overview of critical clinical findings related to seizures is presented. It should be supplemented with additional resources that further discuss the topic.

To assess seizures as the underlying cause of TLOC, pointed questions about circumstances leading up to the event, the ictal behaviors, and the postictal state can help differentiate it from syncope (3). Risk factors such as infection, trauma, and triggers such as strong emotions, loud music, flashing lights, alcohol intoxication/withdrawal, and drug abuse can precipitate seizures (17). Patients often experience aura or motor symptoms before the event. During the episode, patients can have tongue biting, incontinence, and classic tonic-clonic movement lasting 2-3 minutes. An extended postictal state can likewise lead one to include seizure on the differential with patients experiencing confusion, suppressed alertness, aphasia, hemianopsia, and Todd paralysis (numbness or weakness of one part of the body) (17-19).

5 | ANSWER

B. Order an ECG.

This patient presents with a history suspicious of an episode of transient loss of consciousness, which could indicate a syncopal event. One should therefore aim to tease out whether the primary etiology is cardiac or non-cardiac. Therefore, after obtaining a history and physical exam in the emergency setting, evaluation with an ECG should be the next step to subcategorize the etiology and lend its way to prognosis and management (12).

The Final Outcome... Your 12-lead ECG has a regular rate and rhythm. Given your strong history and absence of physical exam findings, you feel confident that this episode is related to a vasovagal etiology. As such, you feel reassured to send him home, discuss common non-pharmacological approaches to decrease the incidence, and wish him luck on his exams.

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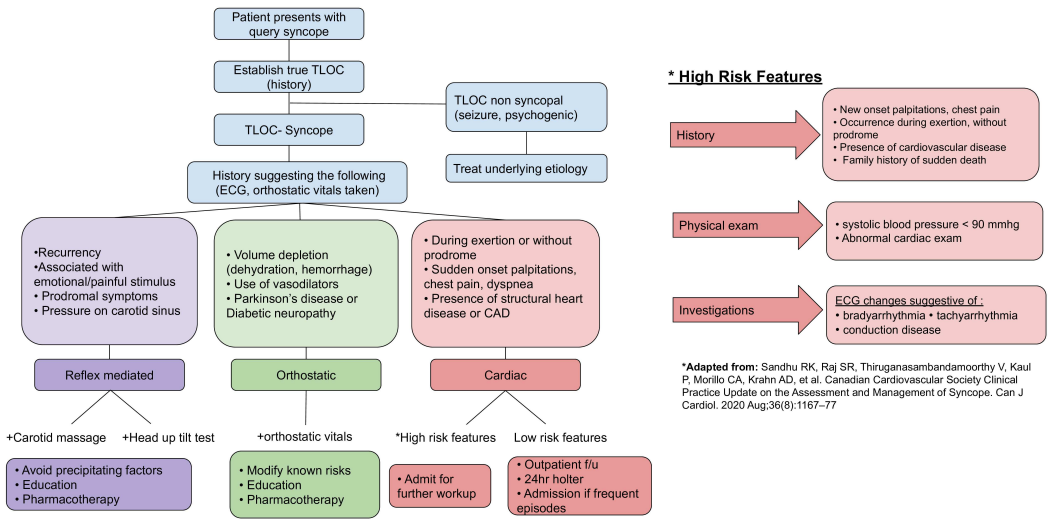

FLOWCHART 1 Flowchart summarizing an approach to syncope and initial management. Information taken from (3,6,12).