



## Chapter 12 – Syncope

**NOTE: CONTENT CONTAINED IN THIS DOCUMENT IS TAKEN FROM ROSEN'S EMERGENCY MEDICINE 9th Ed.**

*Italicized text is quoted directly from Rosen's.*

### Key Concepts:

- 1. The pathophysiology of syncope is dysfunction of both cerebral hemispheres or the brainstem (reticular activating system), usually from acute hypoperfusion. Reduced blood flow may be regional (cerebral vasoconstriction) or systemic (hypotension). Loss of consciousness results in loss of postural tone, with the resulting syncopal episode.*
- 2. Presyncope (near-syncope) or lightheadedness are less severe symptoms and may be considered on a continuum with syncope and share causes, mechanisms, and outcomes.*
- 3. The potential causes of syncope are protean. The first differential diagnostic consideration is to distinguish syncope from other causes of an apparent sudden loss of consciousness, especially seizure and uncommon disorders such as cataplexy.*
- 4. Most cases of syncope arise from benign causes, so the history is largely focused on identifying those cases caused by serious pathology. The past medical history, particularly cardiovascular disease and heart failure, is a key factor in determining future risk of morbidity and mortality.*
- 5. The physical examination of syncope focuses primarily on the elements affecting the cardiovascular and neurologic systems.*
- 6. The chief diagnostic adjunct in evaluating syncope is the 12-lead ECG. It should be obtained on nearly all patients. Studies suggest an overall diagnostic yield of 2% to 9%.*
- 7. Routine hematologic, chemistry, urine, and imaging studies have limited usefulness in the evaluation of syncope and are generally not indicated unless directed by specific factors in the history or physical examination.*
- 8. Disposition of syncope patients can be informed through identification of factors suggesting increased risk of short-term mortality. Predication rules and scoring systems have not yet been validated or shown to be superior to physician gestalt and should not be used alone.*
- 9. Hospitalization is required for patients with chest pain, unexplained shortness of breath, history of congestive heart failure, significant valvular disease, or serious ECG findings. Admission is recommended for patients with factors indicating high-risk of short-term mortality.*



10. *Men younger than 45 years and women younger than 55 years and without worrisome symptoms, signs, or electrocardiographic findings are generally at very low risk for adverse outcome and can often be treated on an outpatient.*

### **Core Questions:**

1. List 10 life-threatening causes of syncope
2. List 10 medications that can precipitate syncope
3. What are the red flags on history and physical exam in syncope?
4. What are markers of increased short-term risk in syncope patients? (box)
5. What are 5 ECG findings to look for in the syncopal patient?
6. List five indications for admission and inpatient evaluation for the patient with syncope?

### **Wisecracks:**

1. What is the significance of a patient presenting with syncope vs. near syncope?  
(<https://rebelem.com/30-day-outcomes-in-syncope-vs-near-syncope/>)
2. What is the utility of orthostatic vital signs?
3. What degree of cerebral hypoperfusion is needed to cause unconsciousness?

### **Rosen's in Perspective**

Syncope is a “sudden transient loss of consciousness with a loss of postural tone,” typically with an immediate return to baseline afterwards. This is a common ED presentation. Causes are typically benign, however there are some killers that can hide in these presentations. History, physical and ECG are your key diagnostic modalities.

The final common pathway resulting in syncope is bilateral cortical dysfunction and/or brainstem dysfunction (esp reticular activating system, secondary to hypoperfusion. Loss of consciousness causes the loss of postural tone and bam, syncope. Less severe hypoperfusion can cause feelings of presyncope, which we consider to be on the same continuum of disease.

There are 3 major classifications of syncope: vasovagal, orthostatic hypotension, and cardiovascular. Other general causes and mimics include seizures, hypoglycemia, toxins, metabolic derangements, hyperventilation, psychiatric causes, and some primary neurologic conditions.

### **Core Questions:**



## [1] List 10 life-threatening causes of syncope

### See Box 12.3.

In general - Cardiac - Neuro - Vascular - Toxic/Metabolic - will cover your critical diagnoses  
Rosens breaks it up into causes that involve cerebral hypoperfusion vs. those that don't.

Causes of Cerebral hypoperfusion include

- Cardiac
  - Outflow obstruction (PE, valvular heart dz)
  - Reduced CO (tachy and bradydysrhythmias, also include hereditary channelopathies, long QT, etc)
  - Pump dysfunction - Dissection, MI, Cardiomyopathy
- Neurocardiogenic
  - Vasovagal
  - Situational (carotid sinus sensitivity, post exercise, GI (straining, vomiting, etc), post micturition)
- Orthostatic
  - Volume depletion (poor intake, hemorrhage)
  - Autonomic dysfunction
  - Drug induced
- Focal CNS hypoperfusion
  - Hyperventilation
  - Subclavian Steal (retrograde flow in vertebral artery or internal thoracic artery due to narrowing/occlusion of subclavian artery, which occurs at the expense of the vertebrobasilar circulation)
  - SAH
  - Basilar artery migraine (now called migraine with brainstem aura. a rare form of migraine with aura. signs and symptoms seem to originate from the brainstem, with no evidence of weakness),

Causes of CNS dysfunction and syncope with normal perfusion:

- Hypoglycemia
- Hypoxia, asphyxiation
- Seizure
- Narcolepsy
- Psychogenic (anxiety, conversion d/o, PNES)
- **Tox**
  - Drugs
  - CO
  - Other agents



Now that we have gone over how to structure a ddx - here are the killers (Box 12.3, Rosen's 9th Ed.)

1. Myocardial infarction
2. Dysrhythmias
3. Thoracic aortic dissection
4. Critical aortic stenosis
5. Hypertrophic cardiomyopathy
6. Pericardial tamponade
7. Abdominal aortic aneurysm
8. Massive pulmonary embolism
9. Subarachnoid hemorrhage
10. Stroke (cerebrovascular accident)
11. Toxic-metabolic derangements
12. Severe hypovolemia or hemorrhage
13. Ruptured ectopic pregnancy
14. Sepsis

**[2] List 10 medications that can precipitate syncope**

Haha, just kidding. There are way more. See box 12.2

1. Cardiovascular agents
2. Beta blockers
3. Vasodilators—beta blockers, calcium channel blockers, nitrates, hydralazine, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, phenothiazines, phosphodiesterase inhibitors
4. Diuretics
5. Central antihypertensives (eg, clonidine, methyldopa)
6. Other antihypertensives (eg, guanethidine)
7. QT-prolonging agents (eg, amiodarone, disopyramide, flecainide, procainamide, quinidine, sotalol)
8. Other antidysrhythmics
9. Psychoactive agents
10. Anticonvulsants (eg, carbamazepine, phenytoin)
11. Antiparkinsonian agents
12. Central nervous system depressants (eg, barbiturates, benzodiazepines)
13. Monoamine oxidase inhibitors
14. Antidepressants
15. Narcotic analgesics
16. Sedating and nonsedating antihistamines



17. Cholinesterase inhibitors (eg, donepezil, tacrine, galantamine)
18. Drugs with other mechanisms
19. Drugs of abuse (eg, cannabis, cocaine, alcohol, heroin)
20. Digitalis
21. Insulin and oral hypoglycemics
22. Neuropathic agents (vincristine)
23. Nonsteroidal antiinflammatory drugs
24. Bromocriptine

### **[3] What are the red flags on history and physical exam in syncope?**

#### **History**

- Event:
  - Preceded by chest pain/Headache/abdo pain
  - Sudden onset with no warning/prodrome. Conversely, look for that vasovagal prodrome or inciting event (eg postmicturition)
  - Exertional syncope
  - GI bleeding
  - Associated PV bleeding or cramping in female of childbearing age
  - Dyspnea, hemoptysis
  - Features that would suggest a mimic (eg postictal phase, tongue bite)
  - Fevers, chills, other infectious symptoms
- PMHx/Meds:
  - VTE risk factors
  - Meds that can precipitate syncope (also look at recent changes - esp. Diuretics, beta blockers, antihypertensives)

#### **Exam**

Vitals - pay close attention (HR too fast, slow, hypoxia, hypotension etc)

General Appearance

Neuro - LOC, lateralizing deficits

Cardiac/Resp - murmurs, pulses (unequal in dissection, steal), volume status

Abdominal/Rectal/GU (if concern for bleeding, tenderness etc)

### **[4] What are 5 ECG findings to look for in the syncopal patient?**

1. Dysrhythmias
2. Pre-excitation
3. Shortened PR
4. Prolonged QTc
5. ST Elevation (regional and diffuse)



6. Brugada pattern (RBBB in association with ST elevation in V1-V3)
7. Right ventricular strain pattern
8. Electrical alternans

**[5] What are markers of increased short-term risk in syncope patients? See Box 12.4**

The following list is adapted from Box 12.4 in Rosen's 9th Edition.

1. Age >65 years
2. Male gender
3. History of CHF
4. History of CVD or serious dysrhythmia
5. History of structural heart disease
6. Family history of early (<50 years) sudden death
7. Syncope without prodrome
8. Exertional syncope
9. Dyspnea or shortness of breath
10. Syncope during supine position
11. Hypotension - systolic BP <90 mmHg
12. Abnormal EKG
13. Anemia with HCT <30% or hemoglobin <90 g/L

**[6] List five indications for admission and inpatient evaluation for the patient with syncope?**

According to Rosen's 9th Edition, the following are indications for admission and inpatient evaluation of patients with syncope:

1. Presence of chest pain
2. Dyspnea or SOB that is unexplained
3. History of CHF
4. History of significant valvular disease
5. Patients with electrocardiographic evidence of ventricular dysrhythmias, ischemia, significant QT prolongation, or new bundle branch block

Potentially consider prolonged monitoring for patients with:

1. Age > 65 years
2. Pre-existing cardiovascular or congenital heart disease
3. Family history of sudden death
4. Serious comorbidities (e.g., diabetes mellitus)
5. Exertional syncope



## Wisecracks:

### [1] What is the significance of a patient presenting with syncope vs. near syncope?

This is a question we have all asked ourselves at one time or another. We so often have patients who complain about a vague history of lightheadedness on review of systems, and for many emergency clinicians, complaints of presyncope are often seen as being less worrisome. However, some new research suggests that presyncope is JUST AS SIGNIFICANT as complaints of syncope.

Study: Bastani A et al. Comparison of 30-Day Serious Adverse Clinical Events for Elderly Patients Presenting to the Emergency Department with Near-Syncope Versus Syncope. Ann Emerg Med 2018. PMID: [30529112](https://pubmed.ncbi.nlm.nih.gov/30529112/)

Design: Prospective observational study

Outcomes:

- Primary Outcome
  - Incidence of 30-day death or serious clinical events

Results:

- Syncope Group
  - 18.2% mortality or serious clinical events
- Presyncope Group
  - 18.7% mortality or serious clinical events

Clinical Predictors of Serious Outcomes

- Dyspnea
- Abnormal EKG
- History of Dysrhythmia
- Physician Risk Assessment (i.e., gestalt)

For more nuanced FOAMED evaluation of this article, check out REBEL EM's post here:

<https://rebelem.com/30-day-outcomes-in-syncope-vs-near-syncope/>

### [2] What is the utility of orthostatic vital signs?



We all know that orthostatic vitals to assess a patient's fluid status are not necessarily useful. However, according to Rosen's 9th Edition, orthostatic vitals could add valuable evidence to your diagnostic work up.

New evidence, however, suggests this is not the case. In a critical appraisal by Schaffer et al. published in the Journal of Emergency Medicine in 2018 (<https://www.ncbi.nlm.nih.gov/pubmed/30316621>) show that orthostatic changes in the emergency department do not reliably diagnose or exclude orthostatic syncope. Additionally, orthostatic vitals do not help to exclude the existence of a potentially serious or life threatening cause of a patient's syncope.

### **[3] What degree of cerebral hypoperfusion is needed to cause unconsciousness?**

Quick little tidbit to help you impress your attending on your next ED shift:

- Hypoperfusion resulting in a reduction of cerebral blood flow by  $>/35\%$  will reliably results in unconsciousness
- This hypoperfusion can be the result of changes in CO, SVR, blood volume, regional systemic vascular resistance