

Cardiovascular Block

2020 CSIE: ACE – Syncope

Live Session Instructions	pages 1-2
Student Expectations	page 2
Learning Objectives	page 3
Clinical Approach Questions	pages 3-4
Further info for ACE Table	pages 4-7
ACE Table	pages 7-9
Practice Clinical Cases	pages 9-12
Selected Readings	pages 12-23
Quiz	pages 24-26

Overview of the ACE live session and instructions to Facilitators

Introductions (**Start at 8:30 am: 5-10 minutes**)

- Start the live session by introducing yourself; check timely attendance of each student
- Meet the students and let them introduce themselves
- If you are familiar with the students, take a few minutes to check in on them and how they are doing
- Let the pre-designated student/students connect the computer/laptop to the AV system to log on and bring up the google doc forms to be used in the session

Clinical Approach Questions (**Start no later than 8:40 am: 15 minutes**)

- Start with the **Clinical Approach Questions** document on the screen:
 - Ask each clinical approach question and elicit a response from the students (as a large group)
 - If students are not volunteering or speaking up, go around the room and call on them or use the student roster provided in your folder
 - If students are not giving the expected/correct answers, guide them further
 - Ensure that you fill in gaps, if students are missing important points, or necessary information relating to the learning objectives

ACE Table (**Start no later than 9:00 am: 40 minutes**)

- Next direct the students to bring up the **ACE Table** document on the screen:
 - Students will need to work together to fill in the table (via google docs)
 - They can access the electronic “Resources folder” at any time
 - Divide your large group into smaller subgroups of 2-3 students
 - Assign each smaller subgroup of 2-3 students, a diagnosis and let them fill in the missing information- *allow about 10-15 minutes*
 - Circulate or check on them periodically and guide them as needed
 - After the table is filled, let each subgroup present what they worked on- *allow about 25-30 minutes*

- o Let each student present if possible
- o Discuss or challenge them if you see gaps in the information provided or if there are erroneous information
- o Once completed, the students are allowed to keep this table for their educational purposes

Practice Clinical Cases (Start no later than 9:40 am: 20-25 minutes)- *please note that as we use Zoom, you will have till 10:00 am*

- Next, direct the students to the **Practice Clinical Cases** document on the screen:
 - o As a large group, work to discuss the practice clinical cases
 - o Rotate through the students to elicit responses to the questions in each practice clinical case and direct the discussion as needed
 - o Reveal the “working diagnosis” for each clinical case after all the questions have been answered
 - o *Alternatively* if you have ample time, divide your large group into smaller subgroups and give each subgroup a clinical case to work on (10 minutes) and let each subgroup present (15-20 minutes)

Quiz and feedback (Start no later than 10:00 am: 15-20 minutes)- *please note that as we use Zoom, you will likely start the quiz around 10:25. Please conclude session by no later than 10:40-10:45!*

- Ensure that all students log into eCampus and access the Quiz under the course/CSIE ACE folder/Quiz
- Access the **brown envelope in your maroon folder** and **read out loud the quiz password**
- If a student experiences technical issues, access the brown folder for a backup copy
- The quiz will take *10 minutes* to complete
 - o During the quiz, please access the one page **student evaluation form** (in the brown envelope) and evaluate each student using the given grading rubric
 - o When all students are done with the quiz, briefly go over the quiz questions/answers (*5-10 minutes*)
- Conclude the session and release the students by 10:20 am
- Leave the completed student evaluation form in the brown envelope within the maroon folder

PLEASE ENSURE THAT ALL FILLED-IN STUDENT EVALUATIONS ARE IN THE BROWN ENVELOPE BEFORE YOU LEAVE THE ROOM

Quick Overview of Student Expectations and Assessment:

Student Expectations

- Students are expected to arrive on time, in professional dress, white coat and badge.
- Students are expected to actively participate, show professional behavior such as appropriate listening skills and refraining from disrupting the session (please see the [student evaluation](#) rubric for more details).
- Students are expected to have prepared for the live session by reading the [preparation guide](#) and [assigned reading material](#) to prepare them for the live session.

ACE Assessment

- This CSIE ACE comprises of 3% of the overall block grade in the following way:
 - o 1.5% for the individual quiz given at the end of the session
 - o 1.5% for the student evaluation form (provided by you as the facilitator)

Syncope Learning Objectives:

1. Name the organ systems commonly associated with syncope.
2. Develop a clinical approach to the chief complaint of syncope.
3. Develop a differential diagnosis for syncope based on history, physical exam findings, and diagnostic tests.
4. Identify common versus major life threatening causes of syncope.
5. Differentiate between different causes of syncope (e.g. orthostatic, situational, psychogenic syncope etc.) given key clinical features.
6. Identify appropriate diagnostic testing to further evaluate syncope.
7. Identify vasovagal (neurocardiogenic) syncope as the most common cause of syncope.
8. Identify the clinical presentation of hypertrophic cardiomyopathy (structural heart disease), and recognize it as the most important predictor of total mortality and sudden death in patients with syncope.
9. Utilize the DAAD acronym to identify important causes of orthostatic syncope.
10. Differentiate syncope from seizures.

CC: Syncope

Clinical Approach & Questions:

Approach to the Patient with Syncope

Definitions:

Syncope – transient, self-limited loss of consciousness due to acute global impairment of cerebral blood flow.

Seizure – paroxysmal event due to excessive or synchronous neuronal activity in the brain with various manifestations ranging from dramatic convulsive activity to experiential phenomena not readily discernible by an observer.

1. What organ systems are commonly associated with syncope?

Cardiac, Vascular, Neuro-cardiogenic, Nervous/Psych, Other

2. Within these systems, what is your differential diagnosis for syncope within each?

Cardiac (*Electrical- rare vs structural*)

- Electrical (arrhythmias)
 - Severe brady-arrhythmias, heart block, ventricular fibrillation, ventricular tachycardia
- Structural

- Heart failure, severe valvular diseases, cardiomyopathies of any sort (e.g. hypertrophic obstructive cardiomyopathy- HOCM), myocarditis
- Other
 - Ischemia, myocardial infarction (MI), pulmonary embolus (PE)

Vascular

Orthostatic hypotension due to GI volume loss, heat illness, vascular anomalies, or hemorrhage

Neuro-cardiogenic (most common cause of syncope)

Vasovagal syncope, situational syncope, carotid sinus syncope

Nervous/Psych

Seizure, stroke, transient ischemic attack (TIA), hyperventilation, conversion disorder, narcolepsy, substances (TCA, cocaine, alcohol, marijuana, opiates, anaphylaxis from food allergies)

Other

Endocrine such as hypoglycemia

Acidosis

Electrolyte derangement

Medication effects

Toxin exposure such as carbon monoxide toxicity

3. What are some conditions that can cause syncope in children versus adults?

- Younger: Neuro-cardiogenic e.g. Vasovagal (event + prodrome), orthostatic hypotension, toxic exposure, dehydration
- Older: Orthostatic hypotension due to medications, reflex/neuro-cardiogenic syncope (particularly carotid sinus syndrome), hypoglycemia, and cardiac arrhythmias.

4. What are some *life-threatening* causes of syncope?

While most causes of syncope are benign and self-limiting, some critical diagnoses to consider in syncope are:

- Cardiovascular causes – fatal dysrhythmias (e.g. ventricular fibrillation), ischemia (e.g. myocardial infarction), and structural (e.g. thoracic aortic dissection, hypertrophic cardiomyopathy, critical aortic stenosis)
- Severe hypovolemia or hemorrhage (e.g. leaking aortic aneurysm, GI hemorrhage, subarachnoid hemorrhage (SAH), and ruptured ectopic pregnancy)
- Medication overdose or toxin exposure
- Pulmonary embolus- saddle embolus
- Anaphylaxis
- Acidosis
- Electrolyte derangement (e.g. severe hyponatremia in young healthy women)

Further information for the ACE table and discussion

What HPI questions would you ask the patient? (Pain, Onset, Duration, Timing, Location, Quality, Severity, Modifiers, Associated symptoms, Context)

- **Pain:** was there chest pain? Other kind of pain such as headache?
 - Chest pain may suggest acute coronary syndrome or pulmonary embolism. Headache may link to neurologic causes such as subarachnoid hemorrhage.
- **Onset:** Was it a sudden onset of syncope or were there symptoms leading into the syncope?
 - While not specific, abrupt onset may be seen with more serious causes, whereas slower onset particularly with a prodrome are commonly seen with benign causes.
- **Duration:** How long was the loss of consciousness (LOC), was there immediate recovery, or prolonged LOC with also prolonged confusion after event?
 - For benign causes of syncope, LOC is generally brief, when LOC is more prolonged consider seizures as etiology.
- **Timing:** Has this happened before, frequency?
 - Long term and non-progressive episodes are likely more benign.
- **Modifying factors:** Did exercise cause the event? Does standing up make it worse or sitting/lying help with symptoms?
 - **Exertional syncope** raises concerns for **dysrhythmias and structural heart disease**. Orthostatic syncope follows standing up from supine or sitting and is often of benign etiology. *Syncope while supine suggests an arrhythmia.*
- **Associated signs and symptoms:** Were there symptoms of nausea, pallor, sweating, palpitations, daytime sleepiness with vivid hallucinations while falling asleep as well as sleep paralysis, post ictal symptoms, generalized anxiety, chest pain, shortness of breath?
 - Diaphoresis and light-headedness are nonspecific. Tongue biting and incontinence of urine/stool suggest seizure. Syncope preceded by **emotional events, micturition, eating, bowel movements, emesis, and manipulation** of the neck stimulating the carotid sinus suggest a **neuro-cardiogenic response** (situational/carotid sinus syndrome).
- **Context:** What were the events leading into the event? (jogging, stress, standing, sitting, severe diarrhea, heavy periods, recent viral illness, diabetic)
 - See modifying factors above. Identify risk factors for hypovolemia or hemorrhage. Medications and toxic exposures may predispose to syncope. Hypoglycemia symptoms may mimic those of syncope.
 - Occurring during intense exertion suggests possible cardiac cause (e.g. hypertrophic cardiomyopathy, long QT syndrome.)

What other pertinent elements of the patient's history would you focus on?

- **Past Medical History**
 - *Congestive heart failure* is a determinant of mortality in the setting of any type of syncope, other risks include prior coronary artery or cerebrovascular disease, known cardiomyopathy, diabetes, hypertension, and other chronic diseases
 - For adolescents ask about eating disorders, diuretic or laxative abuse, inhalant abuse
 - For older patients ask about Parkinson disease, multiple sclerosis, and other degenerative conditions
- **Medications** – Medications associated with syncope include: QT interval prolonging agents, beta-blockers, insulin, and oral hypoglycemics

- **Family History-** History of sudden death, especially younger than 45-50 years, suggests cardiac syncope (e.g. Brugada syndrome)

What pertinent exam findings would you look for? (Vitals, General, HEENT, Lungs, Cardiac, Abdomen, Musculoskeletal, Neuro-Psych)

System	Finding	Significance
Vital signs	Pulse rate and rhythm	Tachycardia, bradycardia or other dysrhythmias
	Respiratory rate and depth	Tachypnea suggests hypoxia, hyperventilation, or pulmonary embolus
	Blood pressure**; systolic blood pressure < 90 mmHg	Shock may decrease cerebral perfusion; hypovolemia or medication may lead to orthostatic changes
	Temperature	Fever from sepsis may cause volume depletion and orthostasis
Skin	Color, diaphoresis	Pallor due to anemia and blood loss
HEENT	Tenderness and deformity	Signs of trauma, tongue biting suggest seizure
	Papilledema	Increased intracranial pressure, head injury
	Breath odor	Ketones from ketoacidosis
Neck	Bruits	Presence of cerebrovascular disease
	Jugular venous distention (JVD)	Right sided heart failure from left sided heart failure of any cause, tamponade, pulmonary embolism
Lungs	Breath sounds, crackles, wheezes	Infection, left sided heart failure
Heart	Murmur	Aortic stenosis, Hypertrophic cardiomyopathy
	Rub	Pericarditis, tamponade
Abdomen	Pulsatile mass	Abdominal aortic aneurysm; heart failure can lead to pulsatile liver
Rectum	Stool for gross blood or melena	Anemia, GI bleed
Pelvis	Uterine bleeding, adnexal tenderness	Anemia, ectopic pregnancy, hypovolemia
Extremities	Pulse inequality in upper extremities	Subclavian steal, thoracic aortic dissection
Neurologic	Mental status, focal neurologic findings, reflexes	Seizure, stroke, or other primary neurologic disease

Rosen's Emergency Medicine, Chapter 15, 135-141

**** Orthostatic blood pressure measurements consist of pulse and blood pressure after five minutes in a supine position, followed by repeat measurements after standing for 3-5 minutes. A positive result for orthostatic hypotension is defined as a drop in systolic BP of 20 mmHg, a pulse increase of 20 bmp, or recurrent syncope.**

What laboratory values and/or studies would you order to narrow your differential diagnosis? (depending on the clinical data, not all will be ordered on each patient)

Study	Indication (Concern for:)
Electrocardiogram (ECG) - recommended in most cases	Cardiac dysrhythmia, ischemia, cardiomyopathy
Pulse Oximetry – recommended in all cases	Hypoxia from any cause

Orthostatic vital signs- recommended in most cases	Orthostatic hypotension
Complete Blood Count (CBC) (note: routine labs without indications are not advised)	Anemia
Electrolytes, serum	Metabolic abnormality – hyponatremia, hyper or hypokalemia
Glucose	Hypoglycemia
D-dimer, serum	Pulmonary embolism
Cardiac proteins (e.g. cTnI, cTnT)	Myocardial infarction
Beta-HCG (pregnancy test)	Pregnancy
Toxicologic screen	Drug related syncope
Arterial blood gas	Acid base disturbance
Chest x-ray	Thoracic aortic dissection
Cranial CT or MRI	New onset or focal seizure, trauma, intracranial hemorrhage
Echocardiogram	Cardiac outflow obstruction, tamponade, thoracic dissection, valvular disease
CT pulmonary angiogram	Pulmonary embolism
Abdominal Ultrasound (US) or CT	Abdominal aortic aneurysm
Pelvic US	Ectopic pregnancy
Holter or loop ECG	Dysrhythmia
Exercise stress test	MI
Electrophysiologic study	Dysrhythmia
Carotid US	Stroke, TIA
Head-up tilt table test	Orthostatic hypotension
EEG	Seizure
Chest CT	Aortic dissection
Brain Natriuretic Peptide (BNP)	Heart failure or cardiomyopathy of any sort

Rosen’s Emergency Medicine, Chapter 15, 135-141

ACE Table

Chief Complaint – Syncope (there are 7 entities to be filled by students, see yellow highlights)

Diagnosis	System involved & category	History	Physical Exam Finding	Labs	Radiology/Procedure
1. Vasovagal Syncope	Neuro-cardiogenic	-Negative cardiac history (no palpitations, no SOB, no swelling, able to sleep lying flat, no problem exercising) -Prodromal symptoms (nausea or dizziness), stress, fear, noxious stimuli, heat exposure	-Normal VS (transient hypotension, bradycardia at time of syncopal event) - No murmurs	Labs normal	ECG normal

2. Situational Syncope	Neuro-cardiogenic	Occurs during or immediately after micturition, defecation, swallowing, or cough	Normal VS, normal Orthostatic BP, No murmurs	Labs normal	ECG normal
3. Carotid Sinus Syncope	Neuro-cardiogenic	More common in older men, unexplained fall, syncope with head rotation or pressure on carotid sinus (shaving, tight collar)	Auscultation for carotid artery bruit prior to consideration of carotid sinus massage <i>*facilitators emphasize to students*</i>	Labs normal	ECG normal, carotid massage (ventricular pause > 3 sec or decrease SBP > or = 50 mm Hg) (Avoid with bruit or h/o TIA or stroke)
4. Dysrhythmias: Long QT or Brugada syndrome, Bradyarrhythmias, Tachyarrhythmias such as V-tach	Cardiac-electrical	Abrupt, palpitations may precede, family history of sudden death	Most often normal, heart rate abnormalities, congenital deafness, skeletal abnormalities, immune dysfunction with some forms of Long QT	Electrolytes, Genetic testing (channelopathies)	Abnormal ECG – QT > 500 ms (long QT), ST elevates with RSR' in anterior leads (Brugada) Holter monitor or Zio patch for longer monitoring, Echo to look for structural abn, Exercise stress testing, EP testing
5. Hypertrophic cardiomyopathy	Cardiac-structural (Note: This category has highest total mortality and risk of sudden death)	Often asymptomatic, dyspnea/chest pain during or after exercise, family history of sudden death	Systolic murmur that intensifies from squatting to standing or during Valsalva maneuver (HOCM), Ventricular tachyarrhythmia occurs quite often with HCM	Labs normal	Echocardiogram is the diagnostic test of choice (hypertrophy of any sort; outflow obstruction is not necessary). ECG: high R+S waves; R at aVL > 11. ECG abnormalities do not correlate with severity or pattern of hypertrophy.
6. Valvular, Vascular, and Ischemic Heart Disease: AS, PS. Also MI, PE, Pulm HTN, Aortic dissection	Cardiac-structural	Often asymptomatic, dyspnea/chest pain during or after exercise, family history of sudden death	gallops, heaves, murmurs consistent with specific valvular disease, signs of CHF (exertional dyspnea, lung congestion, edema)	Troponin –if concern for MI BNP if concern for heart failure D-Dimer if concern for PE	Abnormal ECG Abnormal Echocardiogram CT of chest if concern for PE
7. Orthostatic Syncope (DAAD): <u>D</u> rugs <u>A</u> utonomic <u>A</u> lcohol <u>D</u> ehydration	Orthostatic	Prolonged sitting/standing up	Abnormal Orthostatic Blood Pressures/Heart rate (lying, sitting, standing); Look for signs of anemia and dehydration	CBC and Ferritin if concerned for anemia CMP, UDS, U/A	Normal ECG Tilt testing
8. TIA, CVA	Neural	Sudden onset of numbness, confusion, trouble speaking or walking, severe headache	Focal neuro deficit Think FAST <u>F</u> ace – facial droop <u>A</u> rm – unilateral weakness	Stroke work up	Normal ECG, CT of Head

			Speech – slurred Time – emergent care		
9. Psychogenic syncope	Psych	Often multiple times per day, vague symptoms, signs of stress or anxiety disorder	Normal PE	Normal as part of screening for above	Normal ECG May be induced during tilt test but HR and BP remain normal
10. Seizure disorder	Neural	Aura, sleepiness, confused and/or loss of memory afterwards; Tonic-Clonic Movements that start WITH LOC (vs hypoxic myoclonus which can occur with syncope), loss of continence, post-ictal recovery period	Tongue injury, Todd’s paralysis	CBC, CMP, Ca, Mg, U/A, UDS	Normal ECG Abnormal EEG, Consider MRI Brain

ACE- Syncope PRACTICE CLINICAL CASES (3 cases)

Case 1

CC: “I passed out”

Lori is a 19-year-old female college student, who two days ago experienced a sudden loss of consciousness while walking from her dorm to go to class.

1. What is your differential diagnosis?
Vasovagal, orthostatic hypotension, toxic exposure, dehydration
2. What is common in this age group?
Same as above
3. What other pertinent HPI questions would you ask the patient?
 - Sudden onset of dizziness, nausea and pallor just prior to loss of consciousness.
 - She reports holding onto her friend and was laid to the ground due to loss of consciousness.
 - Her friend who witnessed the event states that she was unconscious for 20 seconds and was then aroused.
 - She states that she was confused and “out of it” but she was able to answer questions and follow commands.
 - By the time EMS arrived she was fully conscious.
4. What pertinent ROS questions would you ask the patient?
 - Feeling well but lots of school related stress
 - Previously healthy
 - No medications
 - Denies being pregnant
 - Denies drug use/abuse

- No family history of sudden early death
5. What pertinent exam findings would you look for or expect for this patient?
 Normal physical exam including orthostatic blood pressures
 6. What other questions would you ask? Family history of sudden death
 7. What studies would you order?
 ECG normal, urine pregnancy negative, no other test performed, serum electrolytes
 8. Would the patient need close observation in the hospital?
 No need for observation or further testing at this time. Encourage hydration, adequate rest, stress reduction, and avoid excessive caffeine.

Diagnosis: Vasovagal syncope (reveal to students after they answer the above questions)

Case 2

CC: “Collapsed during football practice”

Sean is a 15-year-old boy who presents to the ER after collapsing during football practice. His trainer ran up to him and noticed that he was unresponsive and in agonal breathing. He checked his pulse and noted that he was in cardiac arrest.

1. What is your differential diagnosis?
 Sudden onset during exertion – make cardiac etiology very likely – cardiac dysrhythmia or structural disorder such as hypertrophic cardiomyopathy (HCM). (Less likely will be dehydration, vasovagal etc. given that he is in cardiac arrest).
2. What is common in this age group?
 Benign etiologies such as vasovagal, orthostatic hypotension, toxic exposure, dehydration more common in this age range
3. What other pertinent HPI questions would you ask the patient?
 - Sean was in his usual state of health when he collapsed during scrimmage at football practice.
 - His trainer ran up to him and noticed that he was unresponsive and in agonal breathing. He checked his pulse and noted that he was in cardiac arrest.
 - CPR was immediately performed and the school’s AED was applied. 2 minutes of CPR was performed and one shock was delivered by the AED.
 - EMS arrived and did notice a faint pulse but he was still unresponsive. He was immediately intubated, CPR continued, and brought to the ER.
 - In ER, Pediatric advanced life support provided including successful cardioversion for recurrent ventricular tachycardia
 - Transferred to PICU for continued care
4. What pertinent ROS questions would you ask the patient?
 - Previously healthy with no prior syncope
 - No medications or drug use/abuse
 - Birth father died at age of 32 - reason unknown, was not involved with Sean growing up
5. What pertinent exam findings would you look for or expect for this patient?
 - Sedated on ventilator

- Vitals RR 18, HR 120, BP 92/50 , on low dose phenylephrine drip (this is the vasoconstrictor to be used in this condition)
 - Bilateral crackles to lungs
 - Cool extremities
6. What studies would you order?
- ECG – after treatment of v-tach, shows signs of left atrial and ventricular hypertrophy (very tall R waves and steep S waves), also short PR/delta wave noted concerning for WPW (note: there is association with HCM and WPW)
 - Echocardiogram - LV hypertrophy, left atrial enlargement, small ventricular chamber size
7. Would the patient need close observation in the hospital?
 Yes, patient is in critical condition.
8. What other tasks would you do?
- Genetics testing
 - Family counseling
 - Negative genetic test does not mean there is not a mutation; rather, it means it is a mutation that has not been identified. A negative genetics test would mean all first-degree family members need serial echocardiograms to detect development of dysfunction and hypertrophy. Diastolic dysfunction occurs before the onset of hypertrophy

Diagnosis: Hypertrophic Cardiomyopathy (reveal to students after they answer the above questions)

Case 3

CC: “I passed out”

Gregg Holmes is a 77-year-old man who presented to the ER with acute onset of syncope.

1. What is your differential diagnosis?
 Orthostatic hypotension due to medications, reflex/neuro-cardiogenic syncope (particularly carotid sinus syndrome), hypoglycemia, and cardiac arrhythmias or other cardiac disease.
2. What is common in this age group?
 Same as above
3. What other pertinent HPI questions would you ask the patient?
 - The patient is a poor historian and does not remember the surrounding events of his syncope but his daughter provided most of the history.
 - The patient was in the kitchen, making his morning coffee and his daughter was in the other room when she heard a crash when his coffee pot broke.
 - She rushed into the kitchen and found her father on the floor with complete loss of consciousness.
 - 911 was called and when EMS arrived he had been unconscious for five minutes.
4. What pertinent ROS questions would you ask the patient?
 - Past medical history significant for insulin-dependent diabetes
 - Did not eat breakfast after insulin shot
5. What pertinent exam findings would you look for or expect for this patient?
 - Vitals: Afebrile, age appropriate vitals

- General: Now alert and oriented
 - Lungs: Clear
 - CV: strong pulses, no JVD, bruits, no murmurs
 - Neurologic – non-focal
6. What studies would you order?
 Glucose level – 20 when checked by EMS, IV placed and given glucose bolus with return of consciousness, current glucose 190
 ECG - normal
7. Would the patient need close observation in the hospital?
 Depends on social situation, resources, and follow up. Reasonable to observe to monitor for recurrent hypoglycemia, adjust insulin if needed, and provide diabetes education as needed.

Diagnosis: Hypoglycemia (reveal to students after they answer the above questions)

Selected Reading Resources for facilitators

Syncope

Syncope may be caused by a variety of cardiovascular diseases. It is the transient loss of consciousness due to inadequate cerebral blood flow. In the patient presenting with syncope as a primary complaint, one must try to *differentiate true cardiac causes from neurologic issues such as seizure and metabolic causes such as hypoglycemia*. Determination of the timing of the syncopal event and associated symptoms is very helpful in determining the etiology. **True cardiac syncope is typically very sudden, with no prodromal symptoms.** It is typically caused by an abrupt drop in cardiac output, which may be due to tachyarrhythmias such as ventricular tachycardia or fibrillation, bradyarrhythmias such as complete heart block, severe valvular heart disease such as aortic or mitral stenosis, or obstruction of flow due to left ventricular outflow tract (LVOT) obstruction. True cardiac syncope often has no accompanying aura. In situations such as aortic stenosis or LVOT obstruction, *syncope typically occurs with exertion*. Patients usually regain consciousness rather quickly with true cardiac syncope.

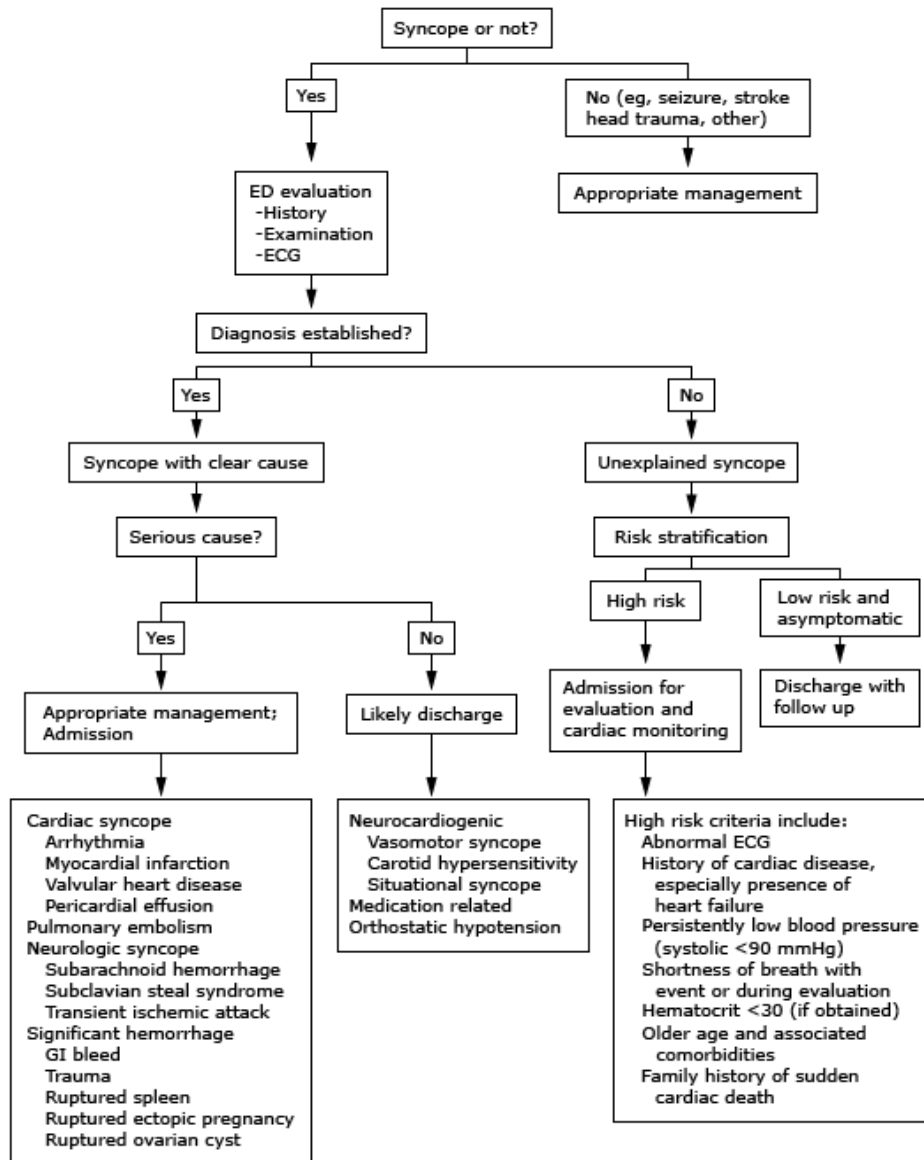
Neurocardiogenic syncope involves an abnormal reflexive response to a change in position. When one rises from a prone or seated position to a standing position, the peripheral vasculature usually constricts and the HR increases to maintain cerebral perfusion. **With neurocardiogenic syncope, the peripheral vasculature abnormally dilates or the HR slows or both. This leads to a reduction in cerebral perfusion and syncope. A similar mechanism is responsible for carotid sinus syncope and syncope associated with micturition and cough.** The patient usually describes a **gradual onset of symptoms** such as **flushing, dizziness, diaphoresis, and nausea before losing consciousness**, which lasts seconds. When these patients wake, they are often **pale and have a lower HR**. In the patient with **syncope due to seizures, a prodromal aura is typically present** before loss of consciousness occurs. Patients regain consciousness much more slowly and at times are incontinent, complain of headache and fatigue, and have a postictal confusional state. *Syncope due to stroke is rare, because there must be significant bilateral carotid disease or disease of the vertebrobasilar system causing brainstem ischemia. Neurologic deficits accompany the physical examination findings in these patients.*

The history is very important in determining the cause of a syncopal episode. This was previously studied by Calkins and colleagues, who found that men older than 54 years of age who had no prodromal symptoms were more likely to have

an arrhythmic cause of their episodes. **However, those with prodromal symptoms such as nausea, diaphoresis, dizziness, and visual disturbances before passing out were more likely to have neurocardiogenic syncope.** Many inherited disorders such as long-QT syndrome and other arrhythmias, hypertrophic cardiomyopathy with LVOT obstruction, and familial dilated cardiomyopathy lead to states conducive to syncope. For this reason, a very detailed family history is necessary.

Source: *Book Chapter: Evaluation of the Patient with Cardiovascular Disease*, [Andreoli and Carpenter's Cecil Essentials of Medicine](#), 3, 22-36, James Kleczka and Ivor J. Benjamin

Algorithm representing the emergency department approach to an adult patient with syncope



UpToDate®

Common causes of syncope

Neurocardiogenic syncope
Micturion
Defecation
Cough mediated
Deglutition
Glossopharyngeal nerve
Situational
Carotid sinus hypersensitivity
Head turning
Circumferential neck compression (neck tie)
Shaving
Orthostatic syncope
Volume loss
Autonomic dysfunction
Deconditioning, prolonged bed rest
Medication related syncope
Vaso active medications
Alpha and beta blockers, calcium channel blockers, nitrates, antihypertensive medications, diuretics, erectile dysfunction medications
Medications affecting conduction
Antiarrhythmics, calcium channel and beta blockers, digoxin,
Medications affecting the QT interval
Antiarrhythmics, antiemetics, antipsychotics/depressants
Diuretics

UpToDate®

Major life threatening causes of syncope

Cardiovascular syncope
Arrhythmia
Ventricular tachycardia
Long QT syndrome
Brugada syndrome
Bradycardia: Mobitz type II or 3rd degree heart block
Significant sinus pause >3 seconds
Ischemia
Acute coronary syndrome, myocardial infarction
Structural Abnormalities
Valvular heart disease: aortic stenosis, mitral stenosis
Cardiomyopathy (ischemic, dilated, hypertrophic)
Atrial myxoma
Cardiac tamponade
Aortic dissection
Significant hemorrhage
Trauma with significant blood loss
Gastrointestinal bleeding
Tissue rupture: aortic aneurysm, spleen, ovarian cyst, ectopic pregnancy, retroperitoneal hemorrhage
Pulmonary embolism
Saddle embolus resulting in outflow tract obstruction or severe hypoxia
Subarachnoid hemorrhage

UpToDate®

First Consult

Evaluation of syncope

Revised: April 14, 2013

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Key points

- Syncope is transient loss of consciousness and postural tone due to temporary insufficient blood flow to the brain, with spontaneous recovery that is not dependent on any electrical or chemical therapy
- Identify cardiac or cerebrovascular abnormalities as potentially life-threatening causes of syncope. Also, differentiate syncope from causes of nontraumatic transient loss of consciousness that do not involve transient cerebral hypoperfusion and require specific treatment, such as [epilepsy](#), metabolic disorders, intoxication, and vertebrobasilar [transient ischemic attack](#) (TIA)
- Initial evaluation consisting of a detailed history, physical examination, basic laboratory testing, and 12-lead electrocardiography (ECG) aids in the diagnosis of syncope and helps identify its cause in approximately 25% to 50% of patients
- Further evaluation may include cardiac testing, such as echocardiography, Holter monitoring, and intracardiac electrophysiologic studies; neurologic testing, such as electroencephalography (EEG); and special tests, such as head-up tilt-table (HUTT) testing, to evaluate neurally mediated syncope
- Treatment is directed to the specific underlying cause of the syncope
- In patients with reflex-mediated syncope, the prognosis is benign

Background

Description

- Syncope is transient loss of consciousness and postural tone due to temporary insufficient blood flow to the brain, with spontaneous recovery that is not dependent on any electrical or chemical therapy. This definition is more focused than that previously used, because it includes the cause of the transient insufficient blood flow to the brain
- Patients with syncope account for 1% of hospital admissions and 3% of emergency department visits
- Syncope can be classified as reflex or neurally mediated, cardiac, neurovascular, and orthostatic
- Nonsyncopal causes of transient loss of consciousness include [epilepsy](#), metabolic disorders, and intoxication

- Risk is related to the underlying disease rather than to syncope itself. Structural heart disease and rhythm disturbances are associated with an increased risk of sudden cardiac death. Orthostatic hypotension due to comorbidities is associated with a two-fold increased risk of death

Etiologies and causes of a transient loss of consciousness

- Reflex (neurally mediated) syncope; most common type in all age groups
- Cardiac syncope attributable to an arrhythmia or a structural cardiopulmonary etiology; second most common type
- Neurovascular syncope, which includes [TIA](#) due to vertebrobasilar insufficiency and subclavian steal syndrome; rare, but important to identify

[Orthostatic hypotension](#), which occurs more frequently in older patients than younger patients

- Nonsyncopal causes of transient loss of consciousness

Reflex (neurally mediated) syncope

Vasovagal response

Description:

- Vasovagal (also known as 'neurocardiogenic') syncope results from an abnormal vasodepressor reflex, causing a decrease in blood pressure and decreased blood flow to the brain. The decrease in blood pressure is secondary to vasodilation and/or [bradycardia](#), leading to dizziness or fainting
- Usually a physiologic response; in itself not life-threatening

Clinical features:

- Symptoms vary and often occur while standing or occasionally while sitting, but never while lying down
- Feeling of warmth and sweating before blacking out
- Nausea and, rarely, vomiting can precede the episodes, which last for less than a minute
- Some patients experience mild twitching while they are unresponsive
- Patients are aware of their surroundings and who and where they are soon after regaining consciousness
- Dizziness and weakness for 24 hours may be present

Carotid sinus hypersensitivity

Description:

- [Carotid sinus hypersensitivity](#) is syncope precipitated by stimulation of the carotid sinus which causes an exaggerated baroreceptor-mediated reflex involving the nerve of Hering and the medulla
- Also known as Charcot-Weiss-Baker syndrome, Weiss-Baker syndrome, and carotid sinus syndrome

Clinical features:

- Occurs during shaving, wearing tight collars, or turning the head
- Hypersensitivity to [carotid sinus massage](#), defined as a sinus pause longer than 3 seconds and a decrease in systolic blood pressure of at least 50 mm Hg when pressure is gently applied for 5 to 10 seconds over the carotid pulsation just below the angle of the jaw, near the carotid bifurcation. Massage should be applied in both the supine and upright positions, and to the right and left sides in turn (not simultaneously)

Situational syncope

Description:

- Syncope coinciding with micturition, coughing, swallowing, gastrointestinal stimulation, or defecation

Clinical features:

- By history, syncope occurs during or immediately after activity
- May be mild, such as a single episode, or recurrent

Cardiac arrhythmias

Arrhythmias

Description:

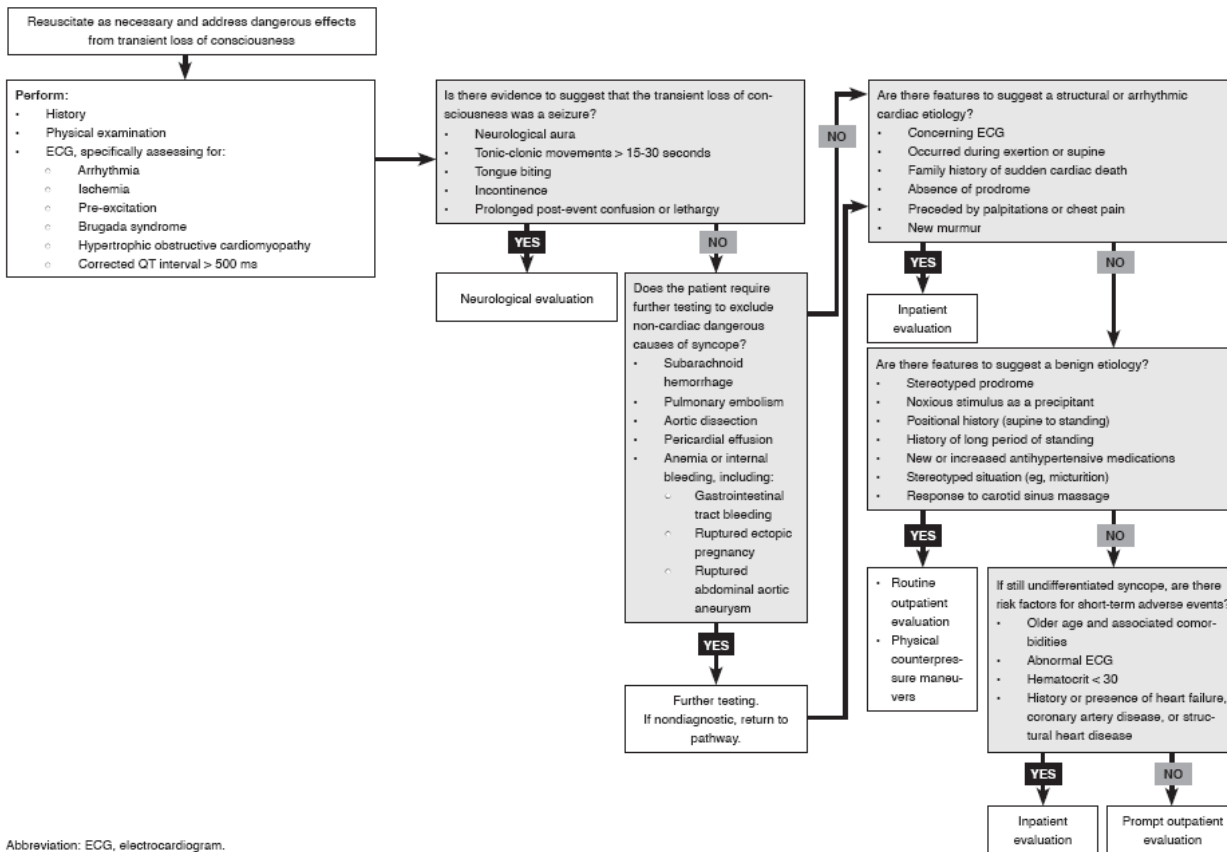
- Most common cause of cardiac syncope
- Common arrhythmias that may result in syncope include:
 - Bradyarrhythmias
 - Sinus node dysfunction/ [bradycardia](#)

- High-degree atrioventricular block with slow ventricular rate (intrinsic, ischemic, drug-induced, reflex-mediated)
- o Tachyarrhythmias
 - [Ventricular tachycardia](#), including ventricular fibrillation, often in the setting of structural heart disease ([cardiomyopathy](#)), [long QT syndrome](#), or drug-induced proarrhythmias, is the most common tachyarrhythmia to cause syncope
 - Paroxysmal supraventricular tachycardia, including atrioventricular nodal reentry tachycardia or orthodromic atrioventricular reentrant tachycardia, can cause syncope, although often with less severe symptoms (*eg*, palpitations, dyspnea, and lightheadedness)
 - [Atrial fibrillation](#) or [flutter](#) (often upon conversion to sinus rhythm or occasionally immediately after onset of tachycardia) can cause syncope, although often with less severe symptoms (*eg*, palpitations, dyspnea, and lightheadedness)
- o Atrial fibrillation with antegrade conduction in patients with [Wolff-Parkinson-White syndrome](#) may result in ventricular fibrillation and death
- Less common but potentially fatal causes include [long QT syndrome](#), idiopathic ventricular fibrillation, Brugada syndrome, short QT syndrome, and catecholaminergic polymorphic ventricular tachycardia

Clinical features:

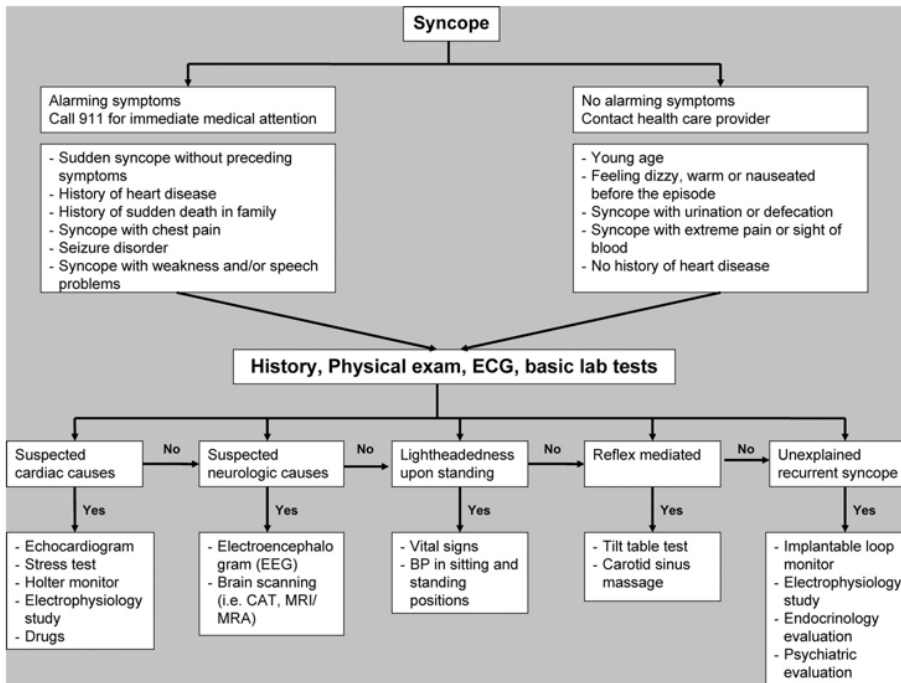
- Fluttering sensation in the chest
- Racing or slow heartbeat
- Chest pain, shortness of breath
- Lightheadedness/dizziness
- Fainting (syncope) or near fainting

Clinical Pathway For Syncope



Abbreviation: ECG, electrocardiogram.

Algorithm for evaluation of syncope and suggested diagnostic workup.

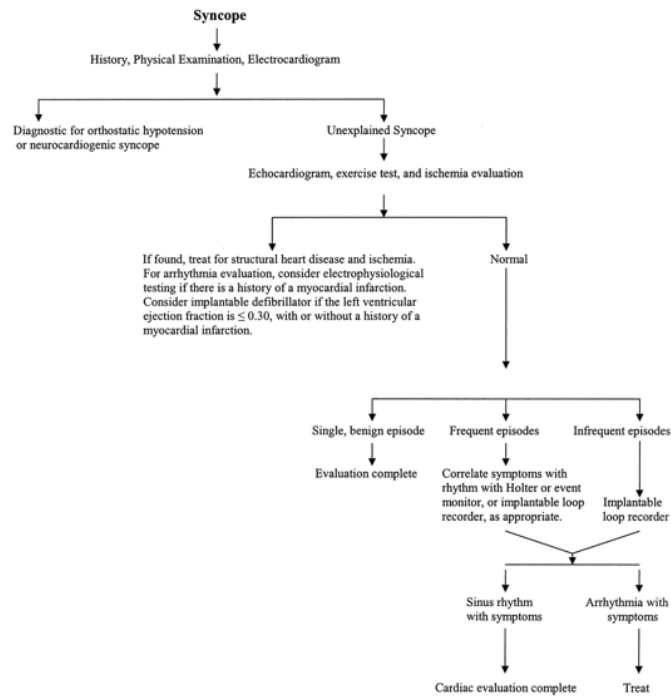


Gunjan J. Shukla, and Peter J. Zimetbaum *Circulation*. 2006;113:e715-e717



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Figure 1. Flow chart for the diagnostic approach to the patient with syncope.



S. Adam Strickberger et al. *Circulation*. 2006;113:316-327



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ACE Syncope Quiz Questions:

1. Beyond the neurally mediated and cardiac causes of syncope what other organ system/category is commonly associated with syncope?
 - a. Musculoskeletal
 - b. Renal
 - c. Genitourinary
 - d. Vascular
 - e. Immune

2. An otherwise healthy man experiences syncope while voiding. He recovers quickly and has a normal exam. His ECG is normal. What is his most likely diagnosis?
 - a. Psychogenic syncope
 - b. Situational syncope
 - c. Orthostatic syncope
 - d. Hypertrophic cardiomyopathy
 - e. Long QT syndrome

3. Which of the following entities would be considered a life-threatening cause of syncope?
 - a. Vasovagal syncope
 - b. Ventricular tachycardia
 - c. Carotid sinus hypersensitivity
 - d. Situational syncope
 - e. Orthostatic syncope

4. Which of the following statements regarding diagnostic testing for syncope is true?
 - a. Patients without focal neurologic deficits should undergo brain imaging
 - b. Carotid massage testing is contraindicated with a carotid bruit
 - c. EEG should routinely be used to differentiate seizures from syncope
 - d. Echocardiography is useful to diagnose orthostatic hypotension
 - e. Genetic testing is available for the evaluation of vasovagal syncope

5. True or False. Structural heart disease is the most important predictor of total mortality and sudden death in patients with syncope.
 - a. True
 - b. False

6. Which of the following is an important cause of orthostatic hypotension?
 - a. Drugs
 - b. Autonomic Insufficiency

- c. Alcohol
- d. Dehydration
- e. All of the above

7. A 20-year-old premed college student is volunteering in the ER, when he suddenly passes out. He relates that he was assisting in suturing a laceration, when he felt nauseous, sweaty and lost consciousness. He regained consciousness after a few seconds, but the ER nurse noted that he was pale, clammy, mildly hypotensive and bradycardic. Repeat vitals after 5 minutes were completely within the normal expected ranges. The student was fully conscious and resumed his normal activities within the hour. The most likely diagnosis in this patient represents:

- a. A condition with underlying significant electrical, structural and electrocardiogram abnormalities
- b. A condition with significant morbidity and mortality
- c. The most common cause of syncope
- d. A major life threatening cause of syncope
- e. A cause of syncope that warrants a thorough workup consisting of ECG, EEG, CBC, complete metabolic panel (CMP), and urine drug screen

8. Tonic-clonic movements that start with loss of consciousness best describes:

- a. Syncope
- b. Seizure

9. A 15-year-old boy has syncope during a basketball game. Exam shows a heart murmur that increases in intensity during Valsalva maneuver. A structural cardiac cause for his syncope is suspected. What is the most likely cause of his syncope?

- a. Psychogenic syncope
- b. Situational Syncope
- c. Carotid sinus syndrome
- d. Hypertrophic cardiomyopathy
- e. Cerebral vascular accident

10. A 61-year-old man with a history of unexplained fall presents for evaluation. His physical exam and ECG are normal. Upon Carotid massage testing he has a drop in systolic blood pressure of 60 mm Hg. What is the likely cause of his syncope?

- a. Vasovagal syncope
- b. Situational Syncope
- c. Transient ischemic attack
- d. Orthostatic hypotension
- e. Carotid sinus syndrome

Answers:

- 1. D (Objective 1, 2)
- 2. B (Objective 3, 5)
- 3. B (Objective 4)
- 4. B (Objective 6, 2)
- 5. A (Objective 8)
- 6. E (Objective 9)

7. C (Objective 7)
8. B (Objective 10)
9. D (Objective 8)
10. E (Objective 4)

ACE Syncope Learning Objectives:

1. Name the organ systems commonly associated with syncope.
2. Develop a clinical approach to the chief complaint of syncope.
3. Develop a differential diagnosis for syncope based on history, physical exam findings, and diagnostic tests.
4. Identify common versus major life threatening causes of syncope.
5. Differentiate between different causes of syncope (e.g. orthostatic, situational, psychogenic syncope etc.) given key clinical features.
6. Identify appropriate diagnostic testing to further evaluate syncope.
7. Identify vasovagal (neurocardiogenic) syncope as the most common cause of syncope.
8. Identify the clinical presentation of hypertrophic cardiomyopathy (structural heart disease), and recognize it as the most important predictor of total mortality and sudden death in patients with syncope.
9. Utilize the DAAD acronym to identify important causes of orthostatic syncope.
10. Differentiate syncope from seizures.