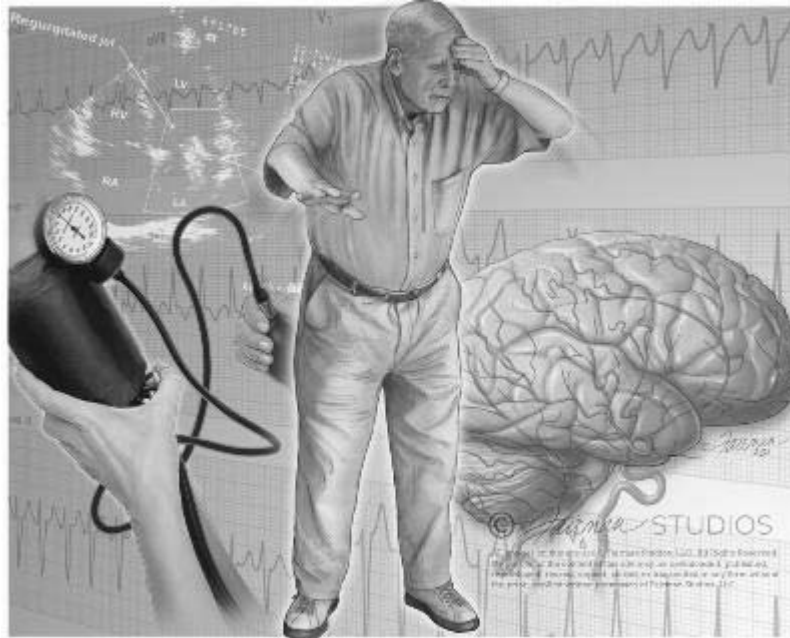


SYNCOPE



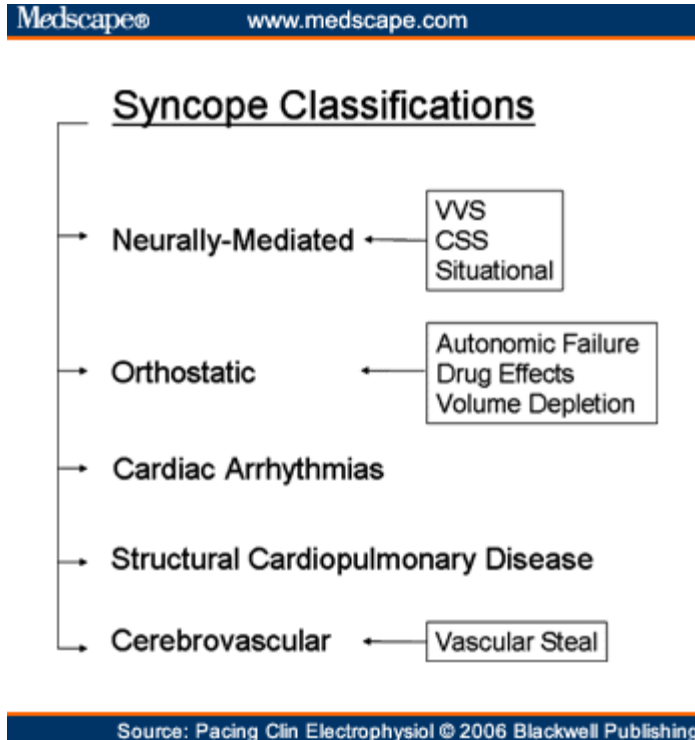
Syncope is a common medical condition for which 911 is called. As you will read below, syncope has many different causes. Some are life-threatening, and others are benign. Therefore it is important to be thoughtful in the assessment and treatment of such patients, and to be aware of the potential danger if a patient initially refuses medical care. Below are excerpts from an article about syncope that was published on the *Medscape* website.

Author – Rumm Morag, MD, FACEP, Member of Salem Emergency Physician Services, PC (SEPS), Salem Hospital. Coauthor – Barry E. Brenner, MD PhD, FACEP, Professor of Emergency Medicine, Professor of Internal Medicine, Program Director for Emergency Medicine, Case Medical Center, University Hospitals, Case Western Reserve University School of Medicine.

Background

Syncope is defined as a transient loss of consciousness with an inability to maintain postural tone that is followed by spontaneous recovery. Postural tone is the continuous and passive partial contraction of the specific muscles by motor neurons. Although most causes of syncope are benign, this symptom may lead to a life-threatening event in a small subset of patients. A syndrome rather than a diagnosis or distinct disease entity, syncope creates several assessment challenges. The presentation of syncope evokes a wide differential diagnosis, including such potentially life-threatening etiologies as acute coronary syndrome, malignant dysrhythmia (ventricular with high one-year mortality rate), aortic dissection, ectopic pregnancy, and pulmonary embolism, as well as neurologic emergencies, such as subarachnoid hemorrhage. The differential diagnosis also includes more benign entities, such as vasovagal events, orthostasis due to volume loss, autonomic disease, or medications; and situational syncope (e.g., cough syncope, micturition syncope (following urination), and defecation syncope). In some circumstances, syncope may mimic other disorders, such as seizure. The term syncope excludes seizures, coma, shock, or other states of altered consciousness. To further add to the diagnostic challenge, it can be difficult to elicit the history of the present illness, as patients frequently appear normal by the time EMS arrives. The event may also have been unwitnessed, and the patient may have little recall of symptoms leading up to the syncopal episode, or time to recovery, thus further complicating the clinical picture.

Syncope is a prevalent disorder, accounting for 1-3% of emergency department (ED) visits and as many as 6% of hospital admissions each year in the United States. As much as 50% of the population may experience a syncopal event during their lifetime. Although many etiologies for syncope are recognized, categorization into reflex (via nervous system), orthostatic, and cardiac (cardiovascular) may be helpful during the initial evaluation. Cardiac syncope is associated with increased mortality, whereas noncardiac syncope is not. Syncope may result in significant morbidity due to falls or accidents that occur as a result. In the United States alone, an estimated \$2 billion annually is spent on patients hospitalized with syncope.



A classification of the causes of syncope (some examples are indicated, but see text for additional details).
VVS = Vasovagal Syncope; CSS = Carotid Sinus Syndrome.

Pathophysiology

Syncope occurs due to cerebral hypoperfusion. Brain tissue depends on adequate blood flow to provide a constant supply of glucose, the primary metabolic source of energy. Brain tissue cannot store energy in the form of high-energy phosphates found elsewhere in the body; therefore, a cessation of cerebral perfusion lasting only 3-5 seconds can result in syncope.

Cerebral perfusion is maintained relatively constant by an intricate and complex feedback system involving cardiac output, systemic vascular resistance, arterial pressure, intravascular volume status, cerebrovascular resistance with intrinsic autoregulation, and metabolic regulation. A clinically significant defect in any one of these may cause syncope.

Cardiac output (CO) can be diminished secondary to mechanical outflow obstruction (such as aortic stenosis), pump failure (cardiogenic shock), hemodynamically significant arrhythmias (bradycardias or tachycardias), or conduction defects (heart blocks). Systemic vascular resistance (SVR) can drop secondary to vasomotor instability, autonomic failure (neurogenic shock), or vasodepressor/vasovagal response. Mean arterial pressure (MAP) decreases with all causes of hypovolemia (as in hemorrhage, sepsis, massive burns, etc.). Medications can affect any one of these parameters leading to a drop in blood pressure.

Other conditions can mimic syncope. A central nervous system event, such as a hemorrhage or an unwitnessed seizure, can present as syncope. Syncope can occur without reduction in cerebral blood flow in patients who have severe metabolic derangements (e.g., hypoglycemia, hyponatremia, hypoxemia, hypercarbia). A young person who resorts to extreme measures to lose weight may try crash diets, use of laxatives or cleanses, that may create dangerous changes in body electrolytes. In this instance, syncope may be an early sign of worsening metabolic changes.

Frequency

Framingham data demonstrate a first time occurrence rate of 6.2 cases per 1000 patient-years. Syncope reoccurs in 3% of affected individuals, and approximately 10% of affected individuals have a cardiac etiology.

Mortality/Morbidity

Data suggest that patients with cardiac syncope are more likely to experience a poor outcome. Patients who have a significant cardiac history and those who seem to have a cardiac syncope (because of associated chest pain, dyspnea, cardiac murmur, signs of congestive heart failure [CHF], or ECG abnormalities) should be considered to be at increased risk of sudden death. Syncope in a patient with poor baseline cardiac function is a poor prognosis irrespective of etiology. Patients with cardiac syncope appear to do worse than patients with noncardiac syncope.

Morbidity from syncope includes recurrent syncope, which occurs in 20% of patients within one year of the initial episode. Lacerations, extremity fractures, head injuries, and motor vehicle accidents can occur secondary to syncope.

Decision rules may assist the health care provider in identifying patients who are at risk. Martin, and colleagues, describes a risk stratification system that predicts an increased incidence of death at 1 year based on the presence of abnormal ECG findings, a history of ventricular arrhythmia, a history of CHF, and age older than 45 years. Sarasin, and colleagues, demonstrates a risk of arrhythmia that is proportional to the number of cardiac risk factors, including abnormal ECG findings, history of CHF, and age older than 65 years.

The San Francisco Syncope Rule identifies patients who are at immediate risk for serious outcomes within 7 days, with a 96% sensitivity based on the presence of abnormal ECG findings, a history of CHF, dyspnea, a hematocrit level of less than 30, and hypotension. The presence of these findings frequently results in hospital admission.

San Francisco Syncope Rule

Congestive heart failure history
Hematocrit < 30%
ECG abnormal (non-sinus rhythm, or new changes compared with old ECG)
Shortness of breath
Systolic blood pressure < 90 mm Hg at triage

Another study by Constantino and colleagues discovered that 6.1% of patients had severe outcomes within 10 days of syncope evaluation. It yielded risk factors associated with serious short-term outcomes. These risk

factors included abnormal ECG, history of CHF, age older than 65 years, male gender, history of chronic obstructive pulmonary disease (COPD), structural heart disease, presence of trauma, and lack of prior symptoms.

Serious Outcomes as Defined by the San Francisco Syncope Rule

Death
Myocardial infarction
Arrhythmia (on monitor and tied to the syncopal event)
Pulmonary embolism
Stroke
Subarachnoid hemorrhage
Significant hemorrhage (tied to syncope and requiring transfusion)
Any condition causing return to the ED <i>and</i> hospitalization for related event

Race, Sex, and Age

No significant differences regarding race are observed with respect to syncope risk. Larger prospective studies fail to show clinically significant differences between men and women.

National Hospital Ambulatory Medical Care Survey (NHAMCS) data show that syncope occurs in all age groups but is most common in adult populations. Noncardiac causes tend to be more common in young adults, whereas cardiac syncope becomes increasingly more frequent with advancing age.

Syncope is relatively uncommon in pediatric populations. Pediatric syncope warrants prompt detailed evaluation. About two-thirds of the time, sudden death in the young is due to a heart abnormality.

Advancing age is an independent risk factor for both syncope and death. Various studies suggest categorizing patients older than 45 years, 65 years, and 80 years as "higher risk." Advancing age correlates with increasing frequency of coronary artery and myocardial disease, arrhythmia, vasomotor instability, autonomic failure, polyneuropathy, and use of polypharmacy.

History

History of present illness and physical examination are the most specific and sensitive ways to evaluate syncope. The diagnosis is achieved with a thorough history and physical examination in 50-85% of patients. No single laboratory test has greater diagnostic efficacy!

A detailed account of the event must be obtained from the patient. The account must include the circumstances surrounding the episode:

- the precipitating factors,
- the activity the patient was involved with prior to the event, and
- the patient's position when it occurred.

Precipitant factors can include fatigue, sleep or food deprivation, warm ambient environment, alcohol consumption, pain, and strong emotions such as fear or apprehension.

Activity prior to syncope may give a clue as to the etiology of symptoms. Syncope may occur at rest, with change of posture, on exertion, after exertion, or with specific situations such as shaving (stimulation of nerve receptors), coughing, urination, or prolonged standing (venous pooling). Syncope occurring within two minutes of standing suggests orthostatic hypotension.

Assess whether the patient was standing, sitting, or lying down when the syncope occurred. Syncope while seated or lying is more likely to be cardiac.

The following questions are part of a thorough assessment:

- Was loss of consciousness complete and total?
- Was loss of consciousness rapid in onset and of short duration?
- Was recovery spontaneous, complete, and without subsequent symptoms or findings?
- Was postural tone lost?

Prior faintness, dizziness, or light-headedness occurs in 70% of patients experiencing true syncope. Other symptoms, such as vertigo, weakness, diaphoresis, epigastric discomfort, nausea, blurred or faded vision, pallor, or paresthesias, may also occur in the presyncopal period. Symptoms of nausea or diaphoresis prior to the event may suggest syncope rather than seizure when the episode was not witnessed, whereas an aura may suggest seizure.

Patients with true syncope do not remember actually falling to the ground. Presyncope, also called near-syncope) involves the same symptoms and pathophysiology but terminates prior to loss of consciousness and can occasionally include loss of postural tone. The duration of symptoms preceding a syncopal episode has been reported to be an average of 2.5 minutes in vasovagal syncope and an average of only 3 seconds in arrhythmia-related cardiac syncope.

Health care clinicians should specifically inquire as to red flag symptoms, such as exertional onset, chest pain, dyspnea, low back pain, palpitations, severe headache, focal neurologic deficits (such as hemiparesis or hemiplegia), diplopia (double vision), ataxia (lack of muscle coordination), or dysarthria (difficulty in speaking) prior to the syncopal event.

Patients should be asked to estimate the duration of their loss of consciousness. Patient estimates range from seconds up to 1 minute in most cases. To discriminate from seizures, patients should also be asked if they remember being confused about their surroundings after the event or whether they have oral trauma, incontinence, or myalgias (muscle pains).

A detailed account of the event must also be obtained from any available witnesses. Witnesses can assist by providing critical pieces to the puzzle and help differentiate among syncope, altered mental status, and seizure. Witnesses may be able to estimate the duration of unconsciousness and to assist in ascertaining whether the patient experienced confusion following the syncopal episode.

Postevent confusion is the most powerful tool for discriminating between syncope and seizure. A postictal phase suggests that a seizure has occurred. Postevent confusion has been described with syncope, but the confusion should not last more than 30 seconds. Seizure-like activity can occur with syncope if the patient is held in an upright posture, causing loss of blood flow to the brain.

A medication history must be obtained in all patients with syncope with special emphasis placed on cardiac and antihypertensive medications. Drugs commonly implicated in syncope include the following:

Drug-Related Causes or Contributors to Syncope

- Blood pressure-reducing agents (e.g. antihypertensive drugs, diuretics, nitrates)
- Agents affecting cardiac output (e.g. beta-blockers, digoxin, antiarrhythmics)
- Agents prolonging cardiac output (QT) interval (e.g. tricyclic antidepressants, phenothiazines, quinidine, amiodarone)
- Sensorium-altering agents (including alcohol, cocaine, analgesics with sedative properties)
- Electrolyte-altering agents (particularly diuretics)
- Certain classes of drugs as associated with an increased risk of syncope, including diuretics, calcium channel blockers, ACE inhibitors, ARBs, nitrates, antipsychotics, antihistamines, levodopa, narcotics and alcohol

ACE = angiotensin-converting enzyme, ARB = angiotensin II receptor blockers

Past Medical History

Inquiry must be made into any personal or familial past medical history of cardiac disease. Patients with a history of myocardial infarction (MI), arrhythmia, structural cardiac defects, cardiomyopathies, or CHF have a uniformly worse prognosis than other patient groups.

Remember to consider the broad differential diagnosis of syncope. Assess whether the patient has a history of seizure disorder, diabetes, stroke (CVA), deep venous thrombosis (DVT), or abdominal aortic aneurysm or if pregnancy is a possibility.

Physical

Always analyze the vital signs. Postural changes in blood pressure (BP) and heart rate may point toward an orthostatic cause of syncope but are generally unreliable. Tachycardia may be an indicator of pulmonary embolism, hypovolemia, tachyarrhythmia, or acute coronary syndrome. Bradycardia may point toward a vasodepressor cause of syncope, a cardiac conduction defect, or acute coronary syndrome.

A fingerstick **glucose level** should be evaluated in any patient with syncope. Hypoglycemia can produce a clinical picture identical to syncope, including the prodromal symptoms, absence of memory for the event, and spontaneous resolution.

A detailed **cardiopulmonary examination** is essential. Irregular rhythms, ectopy, bradyarrhythmias, and tachyarrhythmias should be detected. Auscultation of heart sounds may reveal murmurs indicating high-grade valvular defects. Search for objective evidence of CHF, including jugular venous distension, lung rales, hepatomegaly, and pitting-dependent edema. Examine the abdomen for the presence of a pulsatile abdominal mass.

A detailed **neurologic examination** assists in establishing a baseline as well as defining new or worsening deficits. Patients with syncope should have a normal baseline mental status. Confusion, abnormal behavior, headache, fatigue, and sleepiness must not be attributed to syncope; a toxic, metabolic, or CNS cause must be considered. The patient should have a detailed neurologic examination consisting of assessment of mental status, speech, and motor and sensory function.

The patient must be examined for **signs of trauma**. Trauma may be sustained secondary to syncope with resultant head injury, lacerations, and extremity fractures. Tongue trauma is thought to be more specific for seizures. Remember to consider head injury resulting in loss of consciousness, as opposed to syncope with resultant trauma, if the history or findings are unclear.

Orthostatic changes marked by a decrease in systolic blood pressure by 20 mm Hg, a decrease in diastolic blood pressure by 10 mm Hg, or an increase in heart rate by 20 beats per minute (bpm) with positional changes or systolic BP less than 90 mm Hg with the presence of symptoms may indicate postural hypotension.

Causes

Cardiac (cardiopulmonary) syncope may be due to vascular disease, cardiomyopathy, arrhythmia, or valvular dysfunction and predicts a worse short-term and long-term prognosis. Obtaining an initial ECG is mandatory if any of these causes are possible for the differential diagnosis. Low flow states, such as those associated with advanced cardiomyopathy, CHF, and valvular insufficiency, may result in hypotension and cause transient cerebral hypoperfusion. Often, these patients are on medications that reduce afterload (the pressure against the heart) which include drugs like nitroglycerin, ACE inhibitors (such as captopril, enalapril, lisinopril), or calcium channel blockers (such as diltiazem, or nifedipine) which may contribute to the cause of syncope.

Ventricular arrhythmias, such as ventricular tachycardia and torsade de pointes, tend to occur in older patients with known cardiac disease. These patients tend to have fewer recurrences and have a more sudden onset with few, if any, presyncopal symptoms. Associated chest pain or dyspnea may be present. This type of syncope is generally unrelated to posture and can occur during lying, sitting, or standing. Often, these arrhythmias are not revealed on the initial ECG but may be captured with prolonged monitoring.

Syncope can also result from an acute MI, acute aortic dissection, and pulmonary embolus. These conditions can have associated chest pain, neck pain, shoulder pain, dyspnea, epigastric pain, hypotension, alteration of mental status and can result in sudden death.

Reflex (neurally mediated) syncope may be due to vasovagal syncope, which is mediated by emotional distress such as fear or physical pain. Situational syncope describes syncope that occurs with a fixed event such as micturition (urination), swallowing, exercise induced, and carotid sinus syncope. These causes tend to be more benign and do not predict poor outcomes.

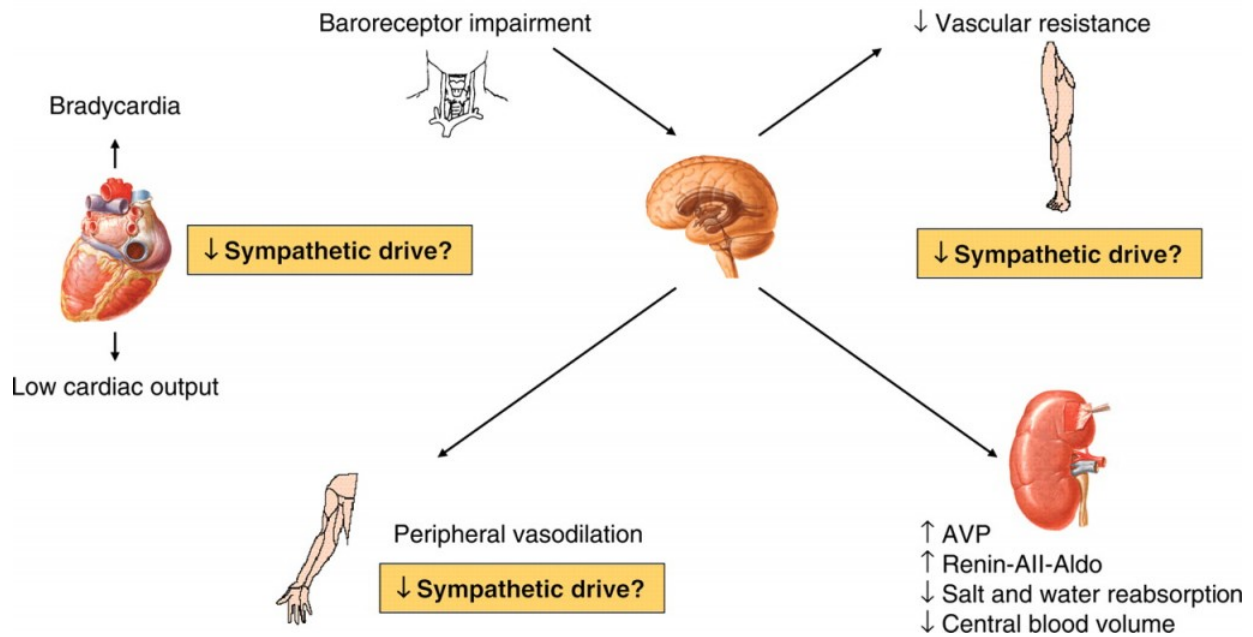
Vasovagal syncope is the most common type in young adults but can occur at any age. It usually occurs in a standing position and is precipitated by fear, emotional stress, or pain (e.g., after a needlestick). Autonomic symptoms are predominant. Classically, nausea, diaphoresis, fading or "graying out" of vision, epigastric discomfort, and light-headedness precede syncope by a few minutes. It is not life threatening and occurs sporadically.

Syncope due to orthostatic hypotension can occur through several mechanisms. Pure autonomic failure can be associated with Parkinson's disease or dementia. Secondary autonomic insufficiency can be due to diabetes, uremia, or spinal injury. Drugs such as alcohol cause orthostatic intolerance and medications such as vasodilators and antidepressants block orthostatic reflexes. Volume depletion due to blood loss, vomiting, diarrhea, poor oral intake, and diuretics also cause orthostatic syncope.

Dehydration and decreased intravascular volume contribute to orthostasis. Orthostatic syncope describes a causative relationship between orthostatic hypotension and syncope. In elderly patients, 45% of these cases are related to medications. Orthostasis is a common cause of syncope and tends to be recurrent. Bedside orthostatics cannot exclude this as an etiology; if suspected, patients should be referred to a primary care provider for outpatient tilt-table testing. This simple, inexpensive test is used to help identify the causes of syncope. The tilt table test involves placing a patient on a table with a foot-support, then tilting the table upward. The tilt-table

starts off in a horizontal position and is tilted by degrees to a completely vertical position. The patient's blood pressure, pulse, and symptoms are monitored throughout the test.

Overview of the alterations in vasovagal syncope



Vasovagal Syncope - Is decreased sympathetic drive the cause of your patient's syncope?

AVP = Arginine Vasopressin, also known ADH (antidiuretic hormone)
 Renin-AII-Aldo = Renin-Angiotensin II-Aldosterone

Prehospital Care

Prehospital management of syncope covers a wide spectrum of acute care and includes rapid assessment of airway, breathing, circulation, and neurologic status.

Treatment may require the following:

- Oxygen administration
- Advanced airway techniques
- Glucose administration
- Intravenous access
- Medication administration
- Cardioversion or cardiac pacing

Electrocardiography

Paramedics should obtain a standard 12-lead ECG in syncope. This is a level A recommendation by the 2007 American College of Emergency Physicians (ACEP) consensus guidelines for syncope. ECG is used in most every clinical decision rule for risk stratification.

Normal ECG findings are a good prognostic sign. Likewise, ECG can be diagnostic for acute MI or myocardial ischemia and can provide objective evidence of preexisting cardiac disease or dysrhythmia such as Wolff-Parkinson-White syndrome, Brugada syndrome, atrial flutter, or AV blocks.

Bradycardia, sinus pauses, nonsustained ventricular tachycardia and sustained ventricular tachycardia, and atrioventricular conduction defects occur with increasing frequency with age and are truly diagnostic only when they coincide with symptoms.

RMA

As always, patients have a right to refuse medical aid (RMA) if they display decisional capacity. Prehospital providers should have a high index of suspicion for a severe cause for the syncopal episode, and therefore will need to contact online medical control (“Telemetry”) for RMA approval.

Emergency Department Care

In patients brought to the ED with a presumptive diagnosis of syncope, appropriate initial interventions include intravenous access, oxygen administration, and cardiac monitoring. ECG and rapid blood glucose evaluation should be promptly performed.

Syncope may be the manifestation of an acute life-threatening process but is generally not emergent. Clinically ruling out certain processes is important. The treatment choice for syncope depends on the cause or precipitant of the syncope. Patients in whom a cause cannot be ascertained in the ED, especially if they have experienced significant trauma, warrant supportive care and monitoring.

Situational syncope treatment focuses on educating patients about the condition. For example, in carotid sinus syncope, patients should be instructed not to wear tight collars, to use a razor rather than electric shaver, and to maintain good hydration status; they should also be informed of the possibility of pacemaker placement in the future.

Orthostatic syncope treatment also focuses on educating the patient. Home discharge instructions often inform patients about avoiding postprandial (after meal) dips in blood pressure (from shunting of blood to the gut), teach them to elevate the head of their bed to prevent rapid blood pressure fluctuations on arising from bed, and emphasize the importance of assuming an upright posture slowly. Additional therapy may include thromboembolic disease (TED) stockings, mineralocorticoids (e.g., fludrocortisone for volume expansion), and other drugs such as midodrine (an alpha₁-agonist with vasopressor activity). Patients' medications must be reviewed carefully to eliminate drugs associated with hypotension. Some older adults intentionally limit fluid to decrease trips to the bathroom. Increased oral fluid intake is useful in decreasing frequency and severity of symptoms of presyncope and syncope in these patients.

Cardiac arrhythmic syncope is treated with antiarrhythmic drugs or pacemaker placement. Referral to a cardiologist or inpatient management may be warranted since this is more commonly associated with poor outcomes due to a high risk of sudden death. Cardiac mechanical syncope may be treated with beta-blockade to decrease outflow obstruction and myocardial workload. Valvular disease may require surgical correction. This, too, is associated with increased future morbidity and mortality.

In summary, syncope is a common clinical problem seen in the out-of-hospital setting with a variety of different causes. Detailed history taking is essential to piecing together the critical timing and setting of a syncopal episode, in particular to distinguish the syncopal episode from the postictal phase of a seizure. The history of present illness helps to distinguish the various forms of syncope. The primary purpose of the evaluation of the patient with syncope is to determine whether the patient is at increased risk for death. This involves, among other things, identifying patients with underlying heart disease, and then the identification of the cause of syncope in an

attempt to improve the quality of the patient's life and to prevent future injury to the patient or others. Syncope in older persons generally has more than one etiology, making the diagnosis more difficult.

References:

1. Syncope. *Medscape*. July 13, 2006
2. Syncope: Emergency Department Evaluation and Disposition. *Medscape*. July 13, 2007

Written by: **DR. BRADLEY KAUFMAN**
FDNY First Deputy Medical Director

CME JOURNAL 2014_J08-09: SYNCOPE

1. What is the primary energy source for the brain?
 - a. Water
 - b. Glucose
 - c. Protein
 - d. Lipids
2. Which of the following causes of a syncopal episode is most likely to have a poor outcome?
 - a. Vasovagal
 - b. Orthostatic
 - c. Neuro/Stroke
 - d. Cardiac
3. Which of the following is not an indication of a potential risk for a bad outcome?
 - a. Abnormal ECG
 - b. Respiratory rate of 16 bpm
 - c. No prior syncopal episode
 - d. Congestive heart failure history

4. Cardiac causes of syncope are more frequent with which of the following?
 - a. Advancing age
 - b. Exercise tolerance
 - c. Sedentary lifestyle
 - d. Stress level

5. Witnesses can aid the clinician in differentiating among syncope, altered mental status, and seizure.
 - a. True
 - b. False

6. Syncopal episodes typically cause the patient to have which of the following?
 - a. Postictal phase
 - b. Incontinence
 - c. Tongue biting
 - d. Loss of postural tone during the episode

7. The most common type of syncope in young adults is:
 - a. Dehydration
 - b. Trauma
 - c. Vasovagal
 - d. Cardiac

8. Normal ECG findings in a patient who had a syncopal episode are a good prognostic sign.
 - a. True
 - b. False

9. The history obtained from bystanders who witnessed a patient's syncopal event is usually unreliable.
 - a. True
 - b. False

10. EMTs and Paramedics should have a high index of suspicion for a bad outcome when a patient who has had a syncopal episode wants to RMA.
 - a. True
 - b. False

Journal CME Credit Answer Sheet

Based on the CME article, place your answers to the quiz on this answer sheet.
 Respondents with a minimum grade of **80%** will receive **1 hour** of Online/Journal CME.

Please submit this page **only once**, by one of the following methods:

- FAX to 718-999-0119 or
- MAIL to FDNY OMA, 9 MetroTech Center 4th flr, Brooklyn, NY 11201

Contact the Journal CME Coordinator at 718-999-2790:

- three months before REMAC expiration for a report of your CME hours.
- for all other inquiries.

Monthly receipts are not issued. You are strongly advised to keep a copy for your records.

Note: if your information is illegible, incorrect or omitted you **will not** receive CME credit.

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other

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 NY State / REMAC # or "n/a" (not applicable)

 Work Location

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Submit answer sheet by
 the last day of September 2014

August – September 2014 CME Quiz		
1.		Questions 1-10 for all providers
2.		
3.		
4.		
5.		
6.		
7.		
8.		
9.		
10.		

Citywide CME

Sessions are subject to change without notice. Please confirm through the listed contact.

Boro	Facility	Date	Time	Topic	Location	Host	Contact
BK	Kingsbrook	TBA	TBA	TBA: contact to inquire →	ED Conference Room	Dr Hew	Manny Delgado 718-363-6644
	LICH	TBA	TBA	TBA: contact to inquire →	Avram Conference Rooms	Dr Brandler	Aaron Scharf 718-780-1859
	Lutheran	4 th Wed	1730-1930	Call Review RSVP →	Contact for location →	Dr Chitnis	Dale Garcia 718-630-7230 dgarcia@lmcmc.com
MN	Mt Sinai Hosp	TBA	TBA	TBA: contact to inquire →	Contact for location →	TBA	eunice.wright@mountsinai.org
	NY Presbyterian	TBA	TBA	TBA: RSVP →	Weill Cornell Campus TBA	Dr Williams	ssamuels@nyp.org Ana Doulis 212-746-0885 x2
	NYU School of Medicine	TBA	TBA	TBA: contact to inquire →	Schwartz Lecture Hall 401 E 30 Street	TBA	Jessica Kovac 212-263-3293
QN	Elmhurst Hosp	1 st Wed	1300-1400	Call Review: Trauma Rounds	A1-22 Auditorium	TBA	Anju Galer, RN 718-334-5724 galer@nychhc.org
	Flushing Hosp	TBA	TBA	Call Review RSVP →	Contact for location →	Dr Crupi	kortiz@jhmc.org
	Mt Sinai Qns	last Tues	1800-2100	Lecture or Call Review	25-10 30 Ave, conf room	Dr Dean	Donna Smith-Jordan 718-267-4390
	NYH Queens	TBA	TBA	TBA: contact to inquire →	East bldg, courtyard flr	Dr Sample	Mary Ellen Zimmermann RN 718-670-2929
	Queens Hosp	2 nd Thurs 4 th Thurs	1615-1815	Call Review	Emergency Dept		718-883-3070
	St John's Episcopal	TBA	TBA	TBA: contact to inquire →	Board Room	TBA	Judith Brown 718-869-7223 jbrown@ehs.org
SI	RUMC	TBA	1400	TBA: contact to inquire →	MLB conf room	TBA	William Amaniera 718-818-1364
	SIUH North	TBA	TBA	TBA: contact to inquire →	Regina McGinn Center 475 Seaview Ave	TBA	Andrea Kleboe 718-226-7878
	SIUH South	TBA	TBA	TBA: call to inquire →	346 Seguine Ave	Dr Barbara	pbarbara.md@gmail.com 917-903-7475

2014 NYC REMAC Examination Schedule

updated 3/19/14

Month	Registration Deadline	Refresher exams*				Basic exams** Written & Scenario exams Sundays 09:30-16:00	NYS/DOH Written Exam***
		Written exam only CME letter required					
January	1/1/14	1/22 @10:00	1/22 @18:00	1/23 @18:00	1/26 @10:00	1/26/14	1/16/14
February	2/1/14	2/18 @18:00	2/20 @10:00	2/20 @18:00	2/23 @10:00	2/23/14	2/20/14
March	3/1/14	3/18 @18:00	3/20 @10:00	3/20 @18:00	3/22 @18:00	3/23/14	3/20/14
April	4/1/14	4/24 @10:00	4/24 @18:00	4/26 @18:00	4/28 @18:00	4/27/14	4/17/14
May	5/1/14	5/15 @18:00	5/19 @18:00	5/21 @10:00	5/21 @18:00	5/18/14	5/15/14
June	6/1/14	6/19 @18:00	6/23 @18:00	6/25 @10:00	6/25 @18:00	6/22/14	6/19/14
July	7/1/14	7/19 @18:00	7/23 @10:00	7/23 @18:00	7/24 @18:00	7/20/14	7/17/14
August	8/1/14	8/20 @10:00	8/20 @18:00	8/21 @18:00	8/23 @18:00	8/31/14	8/21/14
September	9/1/14	9/16 @18:00	9/18 @10:00	9/18 @18:00	9/20 @18:00	9/21/14	9/18/14
October	10/1/14	10/21 @18:00	10/23 @10:00	10/23 @18:00	10/25 @18:00	10/26/14	10/23/14
November	11/1/14	11/18 @18:00	11/20 @10:00	11/20 @18:00	11/22 @18:00	11/23/14	11/20/14
December	12/1/14	12/17 @10:00	12/17 @18:00	12/23 @18:00	12/27 @18:00	12/28/14	12/18/14

* The **REMAC Refresher Written examination** is offered for paramedics who meet CME requirements **and** whose REMAC certifications are either current or expired **less** than 30 days. To enroll, go to the REGISTER link under “News & Announcements” at nycremsco.org before the registration deadline above. Candidates may attend an exam no more than 6 months prior to expiration.

** **REMAC Basic Written & Scenario examination** is for initial certification, **or** inadequate CME, **or** certifications expired **more** than 30 days. Seating is limited. Registrations **must** be postmarked by the deadline above. Exam fee is \$100 by **money order**. Email Christopher.Swanson@fdny.nyc.gov for instructions.

*** **NYS/DOH exam dates** are listed for information purposes only. Scheduling is through your paramedic program or contact NYS DOH for more information.