

## CONTINUING MEDICAL EDUCATION

### SYNCOPE

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#### INTRODUCTION

- Syncope is a common clinical problem that affects approximately one-third of individuals during their lifetime with a 30% recurrence rate.
- Most syncopal episodes are benign and self limiting, but they can be the presenting symptom of organic heart disease and account for significant injuries in 35% of patients.

#### Definition

Syncope (GR: syn = with, Kopto = interrupt) is classically defined as rapid transient loss of consciousness (LOC) and postural tone followed by complete spontaneous recovery. Presyncope or near-syncope refers to the condition in which syncope is imminent but without LOC. (1)

#### Causes

Syncope results from many potential causes of cerebral hypoperfusion (Table 1) that can be synthesized into five groups (2):

- Neurally mediated (reflex) syndrome of which vasovagal syncope (common faint) is the commonest cause and carotid sinus syndrome is the second common.
- Orthostatic hypotension
- Cardiac arrhythmias
- Structural cardiopulmonary disease
- Cerebrovascular causes

In general, cardiac causes of syncope are associated with increased mortality compared to noncardiac causes in which the prognosis is normal and survival is assured.

Syncope should be differentiated from other causes of LOC and/or postural tone (syncope mimics) these include seizures, transient ischemic attack (TIAs) strokes, drop attacks, hypoglycemia and psychogenic syncope. Features that help distinguish syncope from seizures are given in table 2.

TIAs may resemble syncope in being transient, self-limited, but they commonly last longer, are associated with transient localizing neurological signs and symptoms and in carotid TIAs are not associated with LOC.

Drop attacks are poorly defined conditions of unknown cause in which abrupt loss of postural tone occurs but consciousness is generally preserved.

**Table 1.** Causes of syncope.

#### Neurally mediated (reflex) syndromes

- Vasovagal syncope (common faint)
- Carotid sinus syndrome
- Situational syncope (hemorrhage, cough, sneeze, micturition, swallow, defecation, visceral pain, exercise)
- Glossopharyngeal and trigeminal neuralgias

#### Orthostatic hypotension

- Autonomic failure: primary, secondary (e.g. diabetic neuropathy, postural orthostatic tachycardia syndrome)
- Drugs and alcohol
- Volume depletion: hemorrhage, diarrhea, Addison disease

#### Cardiac arrhythmias

- Sinus node dysfunction
- Atrioventricular conduction disease
- Supraventricular and ventricular tachycardias
- Inherited syndromes (e.g. long QT, Brugada, hypertrophic cardiomyopathy)
- Device malfunction (pacemaker, cardioverter defibrillator)

#### Structural cardiopulmonary disease

- Valvular disease
- Acute myocardial infarction or ischemia
- Obstructive cardiomyopathy
- Acute aortic dissection
- Cardiac tamponade
- Pulmonary embolism or hypertension
- Atrial myxoma

#### Cerebrovascular disease

- Vascular steal syndromes

#### Pathogenesis

- In healthy young to middle-aged individuals, the average cerebral blood flow (CBF) is 50 to 60 ml/min/100g brain tissue. Normally cerebral autoregulation maintains the average CBF over a wide range of blood pressures, thus maintaining adequate cerebral O<sub>2</sub> requirement (3.0-3.5 ml O<sub>2</sub>/100 g brain tissue/min).
- An abrupt reduction in CBF for 6 to 8 seconds is enough to reduce blood flow to the reticular activating system (RAS) resulting in complete LOC. Lesser periods of reduced CBF result in presyncope or near syncope.

**Table 2.** Distinguishing seizure form syncope.

Clinical finding	Seizure likely	Syncope likely
Before the event	Aura (olfactory, gustatory, visual)	Nausea, vomiting, palpitation, lightheadedness
During the event	Abrupt onset Prolonged Tonic-clonic movements coinciding with LOC Automatism (lip smacking, frothing at the mouth) Blue face Tongue biting Bowel and bladder incompetence	Less abrupt onset (> 5s) Short Tonic clonic movements after onset of LOC (15s) Bradycardia Hypotension Dilated pupils
After the event	Prolonged confusion Aching muscles Soft-tissue injury	Short residual symptoms but oriented Prolonged fatigue Nausea, vomiting, pallor

- Vasovagal syncope historically has been attributed to activation of C fibers in the posterior and inferior walls of the left ventricle during vigorous contraction of a relatively empty ventricle (Bezold-Jarish reflex). This initiates a reflex mediated sympathetic withdrawal leaving the heightened parasympathetic activity relatively unopposed. Withdrawal of sympathetic activation causes peripheral arterial and arteriolar vasodilatation and hypotension and the parasympathetic predominance causes bradycardia. This concept has been recently challenged by the observation of a vasovagal-type response in cardiac transplant recipients who presumably would not have intact afferent and efferent innervations capable of propagating the vasovagal reflex. Alternative theories have been proposed involving various neurohormonal and neuroendocrine peptides and the ubiquitous nitric oxide. There also appears to be a genetic predisposition to vasovagal susceptibility.
- The carotid sinus syndrome has been attributed to alterations in the carotid baroreceptors. Recent work has focused as well on alterations in the mechanoreceptors and proprioceptive receptors in the sternocleidomastoid muscle causing cardioinhibitory and vasodepressor responses.
- Most of the conditions that lead to situational syncope result in a cardioinhibitory response (sinus bradycardia, sinus arrest and AV block). Syncope due to violent paroxysms of cough may as well be related to decreased cardiac output as a consequence of markedly increased intrathoracic pressure.
- Orthostatic hypotension is particularly common in the elderly who are frequently sedentary leading to attenuation of their postural reflexes. Marked drop in blood pressure may occur postprandially with blood volume sequestration in the splanchnic bed and abdomen. Iatrogenic factors such as excessive diuresis or excessive use of antihypertensive agents are also important contributors.

- Cardiac arrhythmias (tachycardia or bradycardia) can lead to hypotension particularly in the setting of significant left ventricular dysfunction. Additionally, these conditions can also recruit vasodepressor-type reflexes akin to vasovagal responses.
- Structural heart disease tends to be associated with syncope most often through susceptibility to cardiac arrhythmias in this setting or as a result of iatrogenic factors (principally drugs). However, occasionally haemodynamic disturbance may also be responsible for symptomatic hypotension. Acute myocardial ischemia or infarction and acute aortic dissection are probably the most important of these. In this setting, the basis of the faint is multifactorial, in part dependant on neural reflex effects leading to inadequate peripheral vascular compensatory response.

**Clinical features**

The cardinal clinical features associated with specific types of syncope are summarized in (Table 3) (3).

**Table 3.** Clinical features associated with specific types of syncope

<p><b>Neurally mediated syncope</b></p> <ul style="list-style-type: none"> <li>• Absence of cardiac disease</li> <li>• Long history of syncope</li> <li>• Following unpleasant sight, sound, smell or pain</li> <li>• After prolonged standing or being in crowded or hot places</li> <li>• Nausea and vomiting</li> <li>• During a meal or in the absorptive state</li> <li>• With head rotation, pressure on carotid sinus (shaving, tight collars, tumors)</li> <li>• After exertion</li> <li>• During unconsciousness, myoclonic jerking may occur</li> <li>• Vasovagal episodes tend to occur in clusters with periods of quiescence in between and there is a high rate of spontaneous resolution in the long term.</li> </ul>
<p><b>Orthostatic hypotension</b></p> <ul style="list-style-type: none"> <li>• After standing up</li> <li>• After a meal</li> <li>• After exertion</li> <li>• With beginning to take medication or changing the dosage</li> <li>• Presence of autonomic neuropathy or parkinsonism</li> </ul>
<p><b>Cardiac syncope</b></p> <ul style="list-style-type: none"> <li>• Presence of severe structural heart disease</li> <li>• During exertion or while supine</li> <li>• Preceded by palpitations or accompanied by chest pain</li> <li>• Family history of sudden death</li> </ul>
<p><b>Cerebrovascular syncope</b></p> <ul style="list-style-type: none"> <li>• With arm exercise</li> <li>• Difference in blood pressure or pulse between the two arms</li> </ul>

**Diagnostic strategy**

The most important and fruitful elements of the evaluation are detailed history and careful physical examination. The diagnostic yield of these elements ranges from 40% to 75% (4). Impressions obtained from history and physical examination are critical in further triaging patients for appropriate subsequent evaluation and management. A practical strategy for syncope evaluation is depicted in figure 1. The first step is differentiation of those individuals

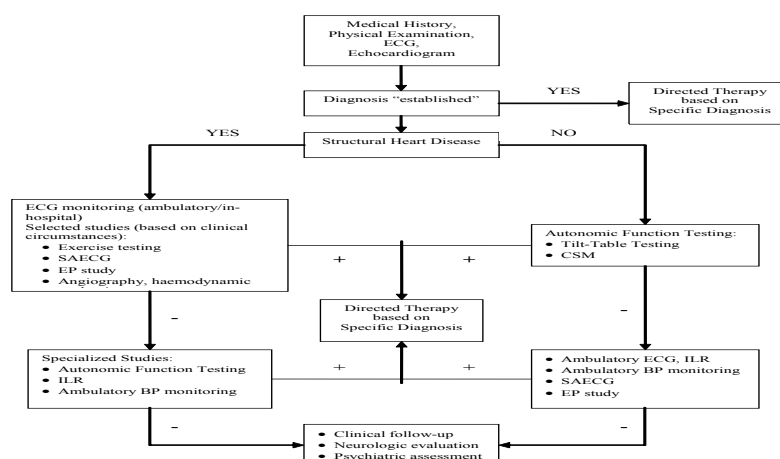
with normal cardiovascular status from those with evident cardiovascular disease. Usually physical examination and echocardiographic assessment are sufficient for this purpose, although exercise testing should be undertaken if syncope occurred with exercise or if ischemic heart disease is suspected. In the former group, if no other systemic problem has been identified, tilt-table testing should be undertaken. In the latter group, a functional assessment of the structural heart disease and evaluation of susceptibility to arrhythmias are appropriate at an early stage. Tilt-table testing should follow if the diagnosis remains in doubt. In only a few instances should special neurological studies be selected as an initial step.

**History**

- Specific detailed questioning regarding the presence or absence of structural heart disease and family history can quickly delineate high-risk from low-risk patients.
- The presence of prodromal symptoms such as nausea or diaphoresis usually heralds the onset of vasovagal syncope. In contrast, a lack of prodrome or the presence of previous myocardial infarction or heart failure points to a more malignant cause of syncope.
- The episode of unconsciousness with vasovagal syncope varies but may last 3 to 4 minutes. Prolonged episodes of unconsciousness such as 7 to 10 minutes or more are unlikely to be due to vasovagal or arrhythmic etiologies and instead suggest neurological processes.
- Injuries are uncommon with vasovagal syncope. Severe injuries and automobile accidents are suggestive of serious arrhythmic aetiology.
- Patients with vasovagal syncope may have post-syncope symptoms that can last from hours to a day or so including weakness, nausea, fatigue and tendency to recurrent syncope. Sudden LOC followed by fairly quick resumption of consciousness suggests tachyarrhythmias.
- Situational syncope during phlebotomy, prolonged standing, in a restaurant, in a dentist's office or following alcohol ingestion are almost universally vasovagal or neurocardiogenic.

**Physical examination**

- For patients with vasovagal syncope, the general and cardiovascular examination will generally be entirely normal.
- The presence of evidence of structural heart disease should suggest cardiac syncope. The finding of atrial fibrillation may point to sick sinus syndrome.
- Gross neurological evaluation showing evidence of lateralization or focal neurological deficit suggests either cardiac embolic phenomena or carotid atherosclerotic disease.
- Carotid sinus massage can be performed safely at the bedside but is contra-indicated in the presence of carotid bruit, known carotid stenosis, TIAs or CVAs. During ECG monitoring, sequential bilateral gentle carotid massage can be performed for 5 to 10 seconds with the patient in a supine, slightly elevated head position. Positive response consists of cardioinhibitory responses (asystole or AV block) > 3 seconds. Patients with carotid sinus syndrome frequently show an abrupt prolonged cardioinhibitory response with LOC. A vasodepressor response is assessed separately by arterial blood pressure monitoring while preventing bradycardia using a temporary dual-chamber pacing system. In absence of symptom reproduction, the demonstration of a pause of ≥ 5 s and/or a systolic blood pressure fall of ≥ 50 mmHg is probably supportive of a presumptive diagnosis of carotid sinus syndrome. If the findings with the supine patient are non-diagnostic, the massage should be repeated with the patient in a head-up position on a tilt-table.
- Orthostatic hypotension is defined as a systolic blood pressure decline of > 20 to 30 mmHg or a diastolic blood pressure decline of > 10 mmHg. These can be elicited either immediately on assuming an upright position from supine baseline or can occur more gradually at 1 to 3 minutes. Marked abrupt orthostatic hypotension is particularly prominent in multisystem atrophy ( Shy-Dragger syndrome) or in primary autonomic failure (Bradbury-Eggleston syndrome).



**Figure 1.** A proposed strategy for the syncope evaluation.

CSM = carotid sinus massage, EP study = electrophysiological study, ILR = implantable loop recorder, SAECG = signal averaged electrocardiography

## Specific testing

### Electrocardiographic recordings

The 12-lead ECG is usually too brief to exclude an arrhythmic cause. However, findings such as ventricular pre-excitation or QT abnormality suggest a potential mechanism. Exercise testing is usually of limited utility in the evaluation of syncope unless the events are clearly exercise related.

### Ambulatory electrocardiographic monitoring (5)

- Holter monitoring in the absence of structural heart disease is frequently unrewarding. Newer systems utilizing event recording technology may be more helpful to disclose intermittent episodes of brady or tachyarrhythmias. Ambulatory blood pressure monitoring devices are frequently used for patients suspected of having intermittent orthostasis. Future ambulatory and implantable ECG event recorders currently in development may also allow simultaneous blood pressure recording.
- Implantable loop recorders (ILRs) placed subcutaneously have the capability of long-term (18 to 20 months) continuous rhythm monitoring in patients with infrequent episodes of syncope that may have an arrhythmic cause. The potential disadvantage of this "wait-and-watch" approach is the uncertain risk of increased morbidity and mortality for the patient from waiting for another event to occur. Implantable loop recorders should not be used in high risk patients with syncope and severe left ventricular dysfunction.
- Recently, a new wireless ambulatory monitoring system called the Cardionet has been introduced. The patient wears a wireless two-channel/ lead ECG harness that transmits to a PDA (personal digital assistant)-size device that communicates constantly with a central monitoring center via the cellular telephone network. The device utilizes arrhythmia detection algorithms to determine arrhythmia severity. Feedback to the ordering physician can be immediate if certain "panic" arrhythmia criteria are met. Ideally it is aimed at the low-risk patient that is, one without structural heart disease in whom syncope or periodic palpitation are suspected to be benign arrhythmias such as sick sinus syndrome, SVT or atrial fibrillation.

### Tilt-table testing (6)

Currently head-up tilt protocol consists of at least 30 minutes of 70 degrees-tilt. Following an initial drug-free tilt test, intravenous administration of the  $\beta$ -agonist isoproterenol can increase the yield but may sacrifice the specificity.

### Established indications include:

- Recurrent syncope that is presumed (but not conclusively known) to be vasovagal.
- Recurrent syncope after exclusion of structural heart disease.

- Single syncopal episode with injury or a motor vehicle accident.
- Single syncopal episode in a high risk setting.
- Syncope of another established cause whose treatment might be affected by vasovagal syncope

The vasovagal response induced during tilt-table testing is classified into three subtypes;

- Mixed response manifested by co-existing bradycardia and hypotension.
- Cardioinhibitory response manifested by persistent bradycardia or prolonged pauses and an absence of significant hypotension when bradycardia is prevented by pacing or atropine.
- Vasodepressor response manifested by significant hypotension in the absence of bradycardia.

Tilt-table testing is contraindicated in patients with critical obstructive cardiac disease (e.g. critical proximal coronary stenosis, critical mitral stenosis or severe LV outflow tract obstruction) or critical cerebrovascular stenosis.

Although tilt-table testing may help establish a confident diagnosis, such testing is no longer advocated for predicting treatment efficacy.

### Electrophysiological study (EPS) (7).

Current clinical practice reserves EPS for a few selected indications:

- oWhen the initial evaluation suggest an arrhythmic cause of syncope.
- oTo evaluate the exact nature of an arrhythmia that has already been identified as the cause of syncope.
- oIn patients with high risk occupations in whom every effort to exclude a cardiac cause of syncope is warranted.

The following assessment should be made:

- oSinus node function.
- oAtrioventricular and His-Purkinje system conduction.
- oInducibility of supraventricular and ventricular arrhythmias.

An arrhythmic cause of unexplained syncope disclosed by an EPS in patients without underlying heart disease or any abnormalities on ECG is uncommon. On the other hand in patients with low ejection fraction, a negative EPS does not predict low mortality or low risk of sudden death. Prophylactic implantable cardioverter-defibrillator (ICD) is now indicated for patients with ejection fraction < 35 % with either ischemic or nonischemic cause and class II or III functional capacity. After patients in this population experience syncope, EPS is no longer necessary.

Noninvasive techniques to identify syncopal patients in whom ventricular tachycardia risk is high such as signal-averaged ECG (SAECG), heart rate variability and microvolt T-wave alternans remain in various stages of evaluation. As a rule, the positive predictive values of these techniques have been low. A high negative predictive value may help exclude certain patients from further ventricular tachycardia evaluation.

## Neurological studies

Electroencephalography and head CT and MRI should be restricted to situations in which clinical observations suggest organic nervous disease.

## Treatment

### *Vasovagal syncope*

- Reassurance as to the general eventual favourable prognosis of the condition with a high rate of spontaneous resolution in the long term.
- Avoidance of precipitating factors including sympathomimetic drugs (cold remedies, caffeine, tobacco), dehydration and alcohol.
- Expansion of salt and fluid intake.
- Patients should be instructed to recognize the premonitory symptoms of the attack plus a few techniques to avoid syncope such as immediately assuming a supine position with elevated and moving legs to increase venous return. Repetitive coughing may abort the faint.
- In highly motivated patients with recurrent vasovagal symptoms, exposure to progressively prolonged periods of still upright posture (tilt training) may be beneficial.
- Beta-blockers are frequently prescribed particularly when the patient has sinus tachycardia or postural orthostatic tachycardia preceding the vasovagal response as observed on a tilt-table test. They probably act by their negative inotropic and chronotropic effects to decrease the LV contraction and mechanoreceptor C-fiber activation. They may also leave the ambient  $\alpha$ -receptor mediated vasoconstriction unopposed and may exert a CNS effect by central serotonin- blocking activity. However, several trials have not shown any significant improvement with beta-blockers compared to placebo and they may suppress intrinsic escape cardiac pacemaker activity.
- Patients with significant hypovolemia (determined by radioiodine) benefit from high-salt diet and hydrofluorocortisone.
- If there is failure of vasoconstriction during upright posture, a vasoconstricting  $\alpha$ -agonist (midodrine) is given.
- Marked venous pooling encountered in sedentary patients is helped by support stockings, physical exercise and reconditioning of leg muscles.
- Young patients are frequently hypervagal (detected by power spectrum analysis) and may benefit from anticholinergic therapy (e.g. transdermal scopolamine).
- Serotonin reuptake inhibitors may be useful in some patients.
- The role of permanent pacemaker implantation for refractory cases and those with prolonged asystole on tilt-table testing is still controversial (8). At present, it is used as a last resort since the cardioinhibitory component of the vasovagal response occurs late after profound hypotension has developed. Several pacemaker designs have been evaluated. One design allows pacing at high rates (> 90 to 100 bpm) when it detects a rapidly falling

heart rate (rate-drop algorithm). It is hoped that rapid pacing would preserve cardiac output and maintain consciousness.

With regard to pharmacological therapies, none (with the exception of midodrine) have proved particularly effective in controlled trials. However, each may have benefit in individual patients.

### Carotid sinus syndrome

- DDD permanent pacemaker is used particularly in conditions with typical cardioinhibitory responses.
- Vasodepressor reflex is helped by high-salt diet and volume expanders plus elimination of potential offending medications (e.g. diuretics).

### Cardiac arrhythmias

- Specific treatment of individual arrhythmias.
- Patients with EF < 35% from any cause should receive implantable cardioverter-defibrillator (ICD) for primary prevention. Although ICD treatment may reduce mortality, syncope can still occur because of the time taken to diagnose the arrhythmia and charge the capacitor (9).

### Structural cardiopulmonary disease

Treatment is primarily directed at amelioration of the specific structural lesion or its consequences.

### Driving and syncope

- Patients with syncope should refrain from driving until a definitive diagnosis is established and successful treatment assured.
- Patients with vasovagal syncope should avoid driving for 3 months following apparently successful therapy.
- Patients with ventricular tachycardia who receive ICD should not drive for a probational period of about 6 to 7 months because LOC can occur quickly during ventricular tachycardia before ICD can terminate the attack. Commercial driving is best avoided by patients with ICDs.

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## QUESTIONS

- An 83 year-old man comes to see you complaining of three episodes of abrupt loss of consciousness in the last year. His PMH is negative. His internist recently performed a stress echo test and Holter monitoring that were normal. His ECG reveals “trifascicular block”. What is the next step?
  - a. Perform EPS, and if negative, implant a Reveal device.
  - b. Perform EPS, and if negative, implant an ICD.
  - c. Consider EPS for VT, and if negative, recommend a pacemaker.
  - d. Schedule a tilt test.

Answer is c: The presence of trifascicular or bifascicular block on ECG suggests that the underlying etiology of syncope may be intermittent heart block, Mobitz type II, third-degree heart block, so-called Stokes-Adams block. Given a normal stress echo test and normal LV function, electrophysiologic testing will likely be negative for ventricular tachycardia. Based on current ACC/AHA guidelines, when no other cause for syncope is found, ICD implantation is indicated for syncope that has not been demonstrated to be due to AV block.

- A 57 year-old man with dilated cardiomyopathy presents to the emergency department with a facial laceration. He reports that he was urinating during the night and suddenly lost consciousness, falling and sustaining the injury. He felt fine before and after the event. He has an EF of 25% secondary to probable viral myocarditis. His ECG reveals IVCD and occasional multifocal PVCs. What is the next step?
  - a. Discharge from the emergency department with a 48-hour Holter monitor.

- b. Admit, perform EPS, and if negative, implant a Reveal device.
- c. Schedule an outpatient tilt test.
- d. Admit, perform EPS, and if negative, offer an ICD.

Answer is d: Syncope in a patient with dilated cardiomyopathy is a very poor prognostic sign. Electrophysiologic testing has a low negative predictive value and therefore cannot be wholly relied on to screen patients who need a defibrillator, implantation of a defibrillator remains a Class 2B in the presence of “advanced structural heart disease” denoting severe ischemic or nonischemic cardiomyopathy. In addition, based on the ejection fraction alone, the patient qualifies for ICD implantation according to the recent Definite and SCD-Heft data.

- A 21 year-old female college student presents to her local emergency department because she fainted twice earlier that day. She reports that the first episode occurred while she was in the shower. It was preceded by nausea with diaphoresis, followed by sudden loss of consciousness. After she awoke, she felt very nauseated, diaphoretic, and vomited. She tried to stand but fainted again. She is otherwise healthy but has had the “flu” for 3 days. You are asked to consult. Her PMH, ECG, physical exam and labs are normal. She had previously fainted once, while donating blood at a blood drive. What tests should you order?
  - a. Tilt test.
  - b. Holter monitoring.
  - c. Stress echo test.
  - d. None of the above.

Answer is d: Patients who present with a typical vasovagal episode with a classic prodrome and sequelae, who are otherwise healthy, probably require no other diagnostic testing or therapy, as the most likely etiology is vasovagal syncope. Tilt table testing is indicated only if the syncope becomes recurrent, or after single episodes of syncope with atypical features or for a high-risk patient.

- Treatment of vasovagal syncope usually involves avoiding of offending stimuli, dehydration and prolonged standing; improving or decreasing venous pooling; and a high-salt and high-fluid diet. For recurrent episodes, pharmacologic therapy is often employed. Initial pharmacologic therapy consists of all of the following except:
  - a. Beta-blockers.
  - b. Disopyramide.
  - c. Serotonin reuptake inhibitors.
  - d. Florinef.

Answer is b: Disopyramide (Norpace) is a Type 1A sodium channel antiarrhythmic medication. It was proposed to be effective for vasovagal syncope based on its negative inotropic and anticholinergic effects. However, in a very well designed

study, using disopyramide loading and repeat tilt table tests, no efficacy was found. In addition, there is genuine concern for proarrhythmia in using the antiarrhythmic agents for a relatively benign disorder. Therefore, disopyramide may have a role for some patients, but it should not be used as initial therapy.

- Carotid sinus syncope is probably the second most common cause of neurally mediated syncope. It results from hypersensitivity of the carotid reflex and causes marked bradycardia and, frequently, concomitant hypotension. All of the following are true regarding the features and treatment of carotid hypersensitivity except:
  - a. The finding of carotid sinus hypersensitivity is extremely specific for the presence of carotid sinus hypersensitivity and syndrome, and mandates pacemaker implantation.
  - b. Pacemaker implantation has been shown to significantly reduce the number of syncopal spells.

- c. Patients with recurrent unexplained falls or injuries should be considered to have neurally mediated syncopal etiology such as carotid sinus hypersensitivity and be tested.  
either with carotid sinus massage testing or tilt table testing.
- d. Carotid sinus hypersensitivity syndrome may result from abnormal proprioception and baroreceptor responses, in the carotid artery and surrounding sternocleidomastoid.

Answer is a: Although the finding of carotid sinus hypersensitivity in a patient with recurrent syncope is highly suggestive, without the presence of the clinical syndrome the finding is relatively nonspecific. Carotid sinus hypersensitivity has been shown to be prevalent in patients with coronary disease and other forms of atherosclerotic disease as well. The sine qua non for carotid sinus syndrome is demonstration of carotid sinus hypersensitivity during carotid sinus massage, and a clinical scenario consistent with syncope resulting from direct stimulation of the carotid sinus baro and vagal reflex.