

CHAPTER 8

Episodic Loss of Consciousness

DEFINITION AND INCIDENCE

Syncope is the cause of 3% of emergency room visits and 6% of general medical hospital admissions. The term “faintness” includes the sensations of lightheadedness, dizziness, and weakness. Syncope literally means “a cutting short.” It is defined as a sudden, brief loss of consciousness and postural tone. It may develop suddenly without warning or be preceded by presyncopal faintness. At onset, the patient is in standing or sitting position, but syncope of cardiac origin may be exception. There is immediate spontaneous recovery after the unconsciousness. There is not postepisode confusion, although patients can briefly appear dazed. Syncope can be experienced by normal subjects when transient reversible decreased cerebral blood flow or metabolism occurs. The majority of attacks are benign due to altered reflexes affecting cardiac rate and vascular tone and less commonly due to severe cardiac disturbance; however, trauma can result from falls due to syncope, especially in elderly patients. Syncope occurs in 7% of elderly patients and recurs in 30%. Recurrent syncope may lead to traumatic injuries; syncope occurring while driving may lead to serious accidents. The mechanism of syncope and patient outcome are closely linked. In some patients, no cause is delineated despite complete medical cardiac and neurological evaluation. If the cause is cardiac (arrhythmias, valvular, cardiomyopathy), sudden death can occur in 20% of patients. Syncope should be taken seriously in elderly patients because survival rate is reduced even if attack is not of cardiac origin.

HISTORY

The patient and observers should be carefully questioned regarding:

- Precipitating factors: emotional stress, fasting state, exercise, cough, swallowing, urination, defecation, position (prolonged standing, occurrence of episode immediately on assuming standing position). Syncope associated with physical exertion suggests cardiac disease. Syncope precipitated by moving or manipulating the neck suggests carotid sinus syndrome.
- Warning symptoms: palpitations, chest pain, headache, visual blurring, dizziness, light headedness, paresthesias. Abrupt onset of syncope without warning symptoms suggests cardiac arrhythmia.
- Level of consciousness during the episode: twilight state, unconsciousness, aware of surroundings but unable to respond.
- Motor dysfunction: loss of muscle tone, tonic spasm, paralysis, myoclonic jerks.
- Evidence of injury as consequence of the episode.
- Sphincter incontinence may occur in syncope but is more common with seizure.

- Postepisode (postictal) neurologic abnormalities: confusion, amnesia, focal neurologic deficit, myalgias, headache. These suggest seizure, not syncope.
- Duration of episode is rarely in excess of 30 to 60 seconds in syncope; seizures last several minutes.
- Autonomic disturbances: skin pallor, diaphoresis, flushed or cyanotic appearance, heart rate or rhythm disturbances suggest neurocardiogenic syncope.
- Detailed medication history: vasodilators (nitroglycerin) antihypertensives, phenothiazines, cardiac antiarrhythmic agents, dopaminergic medication, calcium channel blocking agents, tricyclic antidepressants – all may precipitate syncope.

In syncopal attacks there is usually a brief warning including sensation of lightheadedness, objects moving relative to the patient, visual dimming or blurring, spots in front of the eyes, nausea, ringing in ears, sweating, coldness, skin pallor, or weakness. If the patient is standing or sitting, symptoms can be aborted by lying down. During syncopal attack, certain patients are not arousable for several minutes, whereas others awaken immediately because recumbent position restores cerebral perfusion. Certain patients are completely unresponsive; other appear dazed and unable to respond to verbal stimuli. Recovery of consciousness is usually rapid, but brief amnesia can remain. The patient is usually able to describe presyncopal symptoms, but is an unreliable observer for actual episode details. As the patient recovers, these changes occur: pulse rate becomes strong and regular, blood pressure normalizes, skin pallor recedes, and respiratory pattern normalizes. As the patient is in recumbent (horizontal) position, gravity allows blood flow to the brain to normalize and recovery occurs rapidly.

EXAMINATION

Attention is directed toward five specific areas:

- Blood pressure and pulse are monitored in both supine and standing positions; there should not be drop in blood pressure exceeding 10 to 20 mmHg in systolic or diastolic component; greater drop is postural orthostatic hypotension. Measure blood pressure in both arms; unequal pressure suggests subclavian steal syndrome. Listen for bruits in carotid, supraclavicular (subclavian artery), supraorbital, and temporal regions.
- Gentle carotid massage is performed with electrocardiographic (ECG) monitoring to exclude carotid sinus hyperactivity. Apply pressure to each carotid artery separately for less than 5 seconds while monitoring blood pressure and EKG. Cardiac pause lasting longer than 3 seconds or systolic blood pressure drop of more than 30 mmHg with syncopal symptoms is diagnostic of carotid sinus syncope. Do not apply carotid pressure if patient has carotid bruit, is known to have carotid stenosis, or has suffered prior stroke.
- Carotid artery is palpated and auscultated for bruits. Cardiac auscultation is performed to ascertain evidence of conditions causing left-ventricular outflow obstruction (aortic stenosis, hypertrophic subaortic stenosis, obstructive cardiomyopathy).
- The patient is hyperventilated for 3 minutes or to point of giddiness to reproduce syncope.

- Neurologic examination is performed to exclude disease causing muscle weakness and wasting.

PATHOPHYSIOLOGY

Transient disruption of cerebral blood flow for 8 to 10 seconds results in loss of consciousness. If systolic blood pressure falls below 70 mmHg or to mean pressure of 30 to 40 mmHg, syncope can result. Several pathophysiologic disturbances that decrease cerebral blood flow and metabolism cause syncope (Box 8-1). Three-quarters of blood volume is within venous system; impaired venous return reduces cardiac output unless systemic arterial vasoconstriction cardiovascular reflexes and pumping of blood by lower extremity muscular activity occur. If this compensatory mechanism fails, hypotension and cerebral hypoperfusion occur and syncope results.

Box 8-1

1. Cardiac abnormalities leading to decreased stroke volume and inadequate cardiac output (arrhythmias, valvular obstructive lesions, cardiomyopathies)
 2. Peripheral circulatory impairment caused by decreased blood volume, increased vasodilatation, decreased venomotor tone
 3. Cerebral blood flow impairment (hyperventilation, pulmonary embolism)
 4. Central neural inhibition (neurocardiogenic syncope, carotid sinus hypersensitivity)
 5. Insufficient brain energy metabolism (hypoglycemia, anemia, hypoxia)
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CLINICAL SYNDROMES

Cardiac Disorders

Cardiovascular conditions (cardiac arrhythmias) are important causes of syncope. In these cases reduced cerebral perfusion results from decreased cardiac output and peripheral vasodilatation. Cardiac arrhythmias causing syncope occur in patients with heart disease but rarely occur without prior cardiac abnormality. Ventricular tachycardia is the most common and serious arrhythmia; this can cause syncope and sudden unexplained cardiac death. Ventricular tachycardia can be precipitated by a metabolic abnormality, such as hypokalemia or hypomagnesemia, or certain drug effects, such as antiarrhythmic medication or tricyclic antidepressants. Syncope caused by heart block (Stokes Adam attacks) can occur abruptly without warning or occur when patient is recumbent as contrasted with other types of syncope, which usually occur when the patient is standing. In other cardiac syncopal episodes, palpitations or chest discomfort precede syncope. Carefully review the EKG for prolonged Q-T interval, short P-R interval and wide QRS complex (Wolff-Parkinson-White syndrome). Left-ventricular outflow obstruction and myocardial disease can result in impaired cardiac output; in such patients, syncope commonly develops during physical exertion. If cardiac abnormality is suspected, full cardiac evaluation should be carried out by the cardiologist, not piece-meal by the neurologist.

Peripheral Circulatory Impairment

Syncopal attacks from peripheral circulatory impairment characteristically occur when the patient has been motionless for prolonged time. Orthostatic syncope occurs most commonly in those patients who have the conditions described in Box 8-2.

Box 8-2

1. Severe muscle wasting of any cause
 2. Varicose veins causing peripheral blood pooling and impaired venous return
 3. Prolonged bed rest impairing venomotor tone
 4. Medication (antihypertensive, phenothiazines, diuretics, nitrates)
 5. Postsympathectomy state
 6. Adrenal cortical insufficiency resulting in hypovolemia
 7. Primary orthostatic hypotension
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For standing to occur without cerebral hypoperfusion, cardiovascular reflexes to maintain constant arterial pressure are initiated and this acts to counteract the effect of gravity. In orthostatic syncope, tachycardia reflexively develops in response to decreased blood pressure. In primary orthostatic hypotension, blood pressure rapidly decreases when the patient stands; compensatory tachycardia does not occur because of impaired autonomic reflexes. Vasodepressor syncope is common in elderly patients who have impaired vasomotor reflexes and are frequently taking multiple medications which may lead to postural hypotension. Neurologically mediated orthostatic hypotension may occur in patients with multiple system atrophy (Parkinsonism, cerebellar disease) and peripheral neuropathies. Postural hypotension may occur due to physical deconditioning after patients have suffered prolonged systemic illness in which they are recumbent and muscle wasting has occurred. Orthostatic hypotension is fall of at least 20-mmHg systolic pressure and 10 mmHg diastolic within 3 minutes of standing. Treatment of volume depletion, blood loss, and hypotensive medication effect are easily treated.

Cerebral Blood Flow Impairment

Hyperventilation results in decreased carbon dioxide tension, this stimulates cerebral vasoconstriction. Syncope results from two mechanisms: anxiety causes release of epinephrine, and hypocapnia causes cerebral vasoconstriction and peripheral vasodilation. Prodromal symptoms of hyperventilatory syncope include paresthesias and numbness (hands, feet, perioral), headaches, dry mouth, breathlessness, and chest pain. Hyperventilatory attacks are provoked by emotional stress. These episodes can occur when the patient is sitting or recumbent; symptomatology is not relieved by recumbency. The diagnosis is established by reproducing the symptoms by hyperventilation. Panic attacks can also cause syncope and are not provoked by hyperventilation, and are usually accompanied by other cardiac (palpitations, chest tightness), respiratory, gastrointestinal, autonomic symptoms and agoraphobia.

Carotid Sinus Hypersensitivity

This can be of two types. The first is cardioinhibitory, causing bradycardia as a result of atrioventricular block. This is the most common form (70%). Symptoms are reproduced by slight carotid digital compression producing sinus pause of longer 3 seconds. This condition is treated by atropine. The second is vasodepressor type, in which hypotension can occur with or without bradycardia. Symptoms are induced by gentle carotid massage. This is blocked by vasopressor agents such as epinephrine. Pure vasodepressor syncope is uncommon (5%). The mixed pattern can include cardioinhibitory and vasodepressor types (25%).

Neurocardiogenic Syncope

Other terms for this disorder include “vasovagal syncope,” “vasodepressor syncope,” or “neurally mediated cardiac syncope.” This is commonest cause of syncope. It occurs due to enhanced sympathetic tone and reduced venous return. Patients initially report presyncopal symptoms including dizziness, lightheadedness, visual blurring, and generalized weakness. It is believed that this occurs with the following sequence of events. Initially, there is peripheral blood venous pooling, which results in decreased venous return and decreased left-ventricular blood volume. This *should* trigger compensatory cardiovascular reflexes (baroreceptor stimulation in the aortic arch and carotid sinus), resulting in tachycardia and enhanced peripheral vascular resistance, which increase blood pressure and cardiac output. If this occurs, cerebral blood flow is maintained and syncope does not occur. This mechanism is impaired in patients with neurocardiogenic syncope. In this disorder, decreased left-ventricular volume causes increased left-ventricular contractility to result in myocardial sympathetic C fiber stimulation. The C fiber stimulation activates medullary vasomotor center with enhanced vagal tone and peripheral vasodilation; this results in hypotension and asystole. This is consequence of inhibition of efferent sympathetic tone (resulting in hypotension) and increased efferent parasympathetic tone (resulting in asystole). Myocardial C fiber sympathetic stimulation can be antagonized with beta-adrenergic receptor blocking agents (propranolol, metoprolol) or with theophylline and disopyramide; however, exact mechanism of these drugs is not clearly established. Serotonin may affect blood pressure regulation and the selective serotonin reuptake inhibitors may be effective therapy by altering sympathetic tone. Although it would seem logical that beta-adrenergic blocking agents would worsen the situation because they cause heart rate slowing and reduced cardiac output, these drugs are effective in neurocardiogenic syncope because they inhibit myocardial C fiber sympathetic stimulation. Neurocardiogenic syncope is most common in young patients and is uncommon in patients with impaired cardiac function, possibly because of myocardial C fibers being affected by cardiac disease. If these fibers are impaired, they cannot trigger the effect leading to syncope. Neurocardiogenic syncopal attacks are recurrent and can be precipitated by multiple stimuli such that patients can faint in varied emotional or physical situations.

Reflex Syncope

This results from peripheral vascular blood pooling and decreased venous return. There is reduced peripheral vascular resistance; normal compensatory increase in cardiac output does not occur. This usually occurs when patient is standing or, less commonly, in sitting position.

Reflex syncope is precipitated by response to emotional discomfort, painful stimulation, anxiety, or fear. In situational syncope there is mediation through stimulation of central medullary vagal center; this leads to bradycardia and hypotension. Examples of precipitating factors for reflex syncope include urination, defecation, coughing, swallowing, sneezing, weight-lifting, diving, trumpet playing, and other activities stimulating Valsalva maneuver.

Insufficient Energy Substrates

If systemic concentration of glucose or oxygen is decreased, brain metabolism can be impaired. If compensatory mechanisms (cerebral vasodilation) are not adequate to maintain brain function, syncope results. Hypoglycemia can result from endocrine (pancreatic, adrenal, and pituitary) or hepatic (impaired glycogen storage) disease. It occurs in diabetics taking insulin or can be reactive, occurring 2 to 5 hours after meals. Initial symptoms include headache, diaphoresis, pallor, tremor or shakiness, sense of hunger, abdominal cramps, mental irritability, and weakness. If this condition is not corrected, confusion and disorientation develop; more severe hypoglycemia results in loss of consciousness, sometimes with abnormal neurologic findings (decerebration, clonus, Babinski signs). Attacks are aborted by glucose administration. Following attacks patient frequently complains of severe headache caused by cerebral vasodilation. Diagnosis of hypoglycemia is established *only* when blood sugar is lower than 40 mg/dl and there are accompanying sympathetic hyperactivity features (unless these are suppressed by adrenergic blocking medication). In patients with pheochromocytoma, increased sympathetic activity (diaphoresis, hypertension, palpitations, tremor anxiety) are common. In patients with carcinoid syndrome or mastocytosis, flushing and gastrointestinal symptoms (nausea, vomiting, diarrhea) may be prominent with accompanying loss of consciousness.

Hypoxia (anoxia) causes syncope because of inadequate brain oxygen. Anoxia can occur because of cardiac or respiratory disorders or during high-altitude exposure. It occurs in anemic patients – consider the possibility of acute hemorrhage, especially from gastrointestinal system or in those with hemoglobinopathies impairing oxygen transport. Syncope caused by anoxia occurs during physical exertion and is usually preceded by presyncopal symptoms. In anoxic syncope brief *random* myoclonic jerking movements can occur (convulsive syncope). Hypoxia can cause cerebral vasodilation; this can result in headache, unusual for syncope but common in seizures. The brief duration of anoxic syncopal disorder and lack of postictal phase should allow differentiation from generalized tonic-clonic convulsion.

DIFFERENTIAL DIAGNOSIS

Epilepsy

The characteristic clinical features helping to differentiate fits from faints are listed (Table 8-1). In grand mal seizures, patients experience prodrome consisting of vague and uneasy feelings that warn of impending episode. These are feelings that patient has learned accompany the seizure and they must be differentiated from feelings such as lightheadedness, nausea, pallor, sweating, and weakness that patients experience before fainting. Falls with injury occur as the patient is in rigid (tonic) phase; however, this injury is rare in syncope because patient falls limply or has

adequate warning to avoid falling. In syncope, patients can bite their tongues; however, tongue maceration occurs only in seizures. Incontinence occurs more frequently in seizures than syncope. Random, diffuse, myoclonic jerks are common in syncope caused by cerebral hypoxia. Following syncope, patient may be dazed for several minutes, but disorientation – as seen commonly following seizures – does not occur. Convulsive syncope is defined as syncope with convulsive features of which tonic extensor spasms and random myoclonic jerks are most common. Rhythmic clonic jerking does not occur in convulsive syncope. The EEG shows diffuse slowing but does not show spike discharges in convulsive syncope. This condition represents primary syncope disorder and *not* indicated. It has been reported that 33% of patients initially diagnosed as having epileptic seizures were later diagnosed as having syncope. If there is any doubt, use of the tilt-table test with ECG, blood pressure, and EEG monitoring should settle the issue.

Cerebrovascular Disease

Cerebrovascular disease (stroke) by itself, rarely causes syncope. The vertebro-basilar system supplies the upper brain stem centers for consciousness and vascular lesions involving this region may lead to syncope or more likely “drop attacks.” Patients with severe bilateral carotid stenosis may develop syncope when they assume standing position and with exercise. If cerebrovascular disease is cause of syncope expect other focal neurological signs of stroke.

With subarachnoid hemorrhage (SAH) caused by ruptured aneurysm, there can be sudden transient loss of consciousness. In SAH, there is usually severe headache; and the patient does not usually fully regain consciousness and may have residual neurologic deficit. In migraine, autonomic instability leading to syncope is quite common. When loss of consciousness occurs in migraine, it can be difficult to differentiate syncope from seizure because both conditions can be associated with migraine. Transient ischemic attacks, which are caused by carotid artery ischemia, do *not* cause syncope. Drop attacks caused by vertebrobasilar insufficiency with ischemia involving ventral pons cause patients to fall to the ground, but these attacks are not associated with loss of consciousness. Drop attacks usually occur in middle-aged or elderly patients. Because these vascular episodes occur without warning and are brief in duration, the patient may not actually be able to report accurately if consciousness has been lost; therefore, these can be confused with syncopal attacks.

DIAGNOSTIC ASSESSMENT OF SYNCOPED PATIENT

Diagnostic studies (in addition to attempting to provoke the episode) that are indicated in evaluating patients with syncope include:

- Complete blood count
- Fasting blood glucose content and 5-hour glucose tolerance test
- Plasma cortisol and thyroid hormone levels
- Serum electrolytes, creatinine, liver function studies

- Electrocardiogram
- 24-hour Holter cardiac monitoring
- Echocardiogram
- Alcohol and drug screen
- Cardiac enzymes (to exclude myocardial infarction)
- Catecholamine screen for pheochromocytoma (24-hour urine sample for metanephrines and vanillylmandelic acid)
- 24-hour urine sample for 5-hydroxy-indole-acetic acid for carcinoid syndrome
- Pregnancy test
- Tilt table test
- Invasive cardiac electrophysiologic monitoring as part of cardiac work-up ordered by the cardiologist.

This list represents complete battery from which needed tests should be selected depending on clinical status as determined by the history and examination clues to the syncope pathophysiology. For example, not all studies would be necessary for teenage patient who faints on one occasion while standing motionless for prolonged period in church. Cardiologist should be consulted to analyze possible role of arrhythmias, myocardial or valvular dysfunction and cardiologist should determine the need for electro-physiological (noninvasive or invasive) testing, especially if syncope is recurrent. Because major risk in syncopal patients is presence of serious cardiac arrhythmia, ECG and Holter monitoring are essential parts of baseline evaluation. Invasive electrophysiological cardiac testing is warranted in patients when cardiac causes are suspected. This is becoming more routine because of several instances of sudden and unexplained deaths of famous healthy athletes. In women of child-bearing age, always initially perform a pregnancy test. Syncope can occur in early stages of pregnancy probably related to increased estrogen effect or also in later stages because of the compressive effect of the fetus on venous return.

In syncopal patients in whom diagnosis cannot be established by clinical history or listed diagnostic tests, tilt table test should be performed. The patient is positioned on tilt table that has a footboard for weight bearing. The table is tilted 60 to 80 degrees from horizontal position for 15 to 60 minutes with ECG, EEG, and blood pressure being continuously monitored. If syncope occurs, test is immediately discontinued. If syncope does not occur, patient is placed in horizontal position and isoproterenol is infused intravenously (1 to 2 mg/min) to increase heart rate by 20% over baseline, and tilt is repeated. Do not use isoproterenol if coronary artery disease or hypertrophic cardiomyopathy is suspected. Positive response consists of reproduction of symptoms, usually accompanied by hypotension and bradycardia. Positive tilt table tests confirm diagnosis of neurocardiogenic syncope, this form of syncope responds to appropriate treatment with beta-adrenergic or SSRI medication. Further neurodiagnostic and cardiologic tests are not necessary in these patients with positive tilt table responses. Certain normal patients without syncope develop hypotension and bradycardia on tilt table; however, positive tilt table response is usually accepted as diagnostic for neurocardiogenic syncope. It is positive in 50% of patients with unexplained syncope with 10% false positives.

Because intracranial lesions are rarely responsible for syncope, neuroimaging studies (CT/MRI) are not usually necessary; *however*, they are frequently performed with very low

yield. For example, if the patient has adrenal insufficiency, documented by laboratory studies such that a pituitary lesion is suspected. CT/MRI would be warranted as well as abdominal imaging studies for a more likely adrenal lesion. In a patient who has suffered episodic loss of consciousness, clinical evaluation is usually adequate to differentiate syncope from seizure; however, an EEG is routinely performed. This is because in certain young patients with classical syncopal attacks there is EEG evidence of generalized (nonfocal) bursts of abnormal waves such as spikes or slowing. This condition would be classified as generalized seizure disorder. These would almost never be associated with structural brain lesions, and neurologic examination would be expected to be normal; therefore, CT/MRI would not be necessary. These patients may clinically respond to anticonvulsants. Because 10% of normal persons without brain disease have nonspecific EEG abnormality, and therefore 10% of syncopal patients have nonspecific EEG abnormality, clinical history and EEG pattern should be carefully analyzed before considering treatment with anticonvulsants.

TREATMENT

Treatment of syncopal disorders requires understanding of underlying pathologic mechanism. If patient is observed during episode, physician should place patient in recumbent position, monitor vital signs, and, if necessary, secure patent airway. If facilities are available, intravenous line should be established and blood specimen obtained to determine fasting glucose, hemoglobin, and electrolyte values. If hypoglycemia is suspected and patient does not have rapid and immediate improvement when placed in recumbent position, glucose-containing substances (complemented by thiamine) should be administered. Oral intake is dangerous until patient is alert because aspiration can occur. Inhalation of ammonia salts is frequently used, but this is not necessary. The patient should remain recumbent until steady and fully alert. The episode can recur if the patient stands up rapidly before cardiovascular reflexes have stabilized or before the underlying disorder has been corrected.

In young patients, vasovagal responses and hyperventilation are the most common causes of syncope; these attacks are triggered by emotional factors, and, if possible, these should be avoided. In patients with carotid sinus hypersensitivity rapid neck movements are to be avoided. In postural hypotension the patient should be instructed to stand up slowly, and when sitting or standing for prolonged duration the patient should massage the calves. Use of support stockings in patients with impaired venomotor tone decreases the effect of peripheral venous pooling. Avoiding drugs that cause peripheral vasodilation is necessary. Salt and fluid loading may prevent vascular volume depletion. Treatment of neurocardiogenic syncope with beta-adrenergic blocking agents is warranted to decrease myocardial contraction and block central serotonin receptors (also accomplished with SSRI). Disopyramide blocks vagal nerve effects. Midodrine is an alpha-adrenergic agonist which may be effective by causing arteriolar vasoconstriction. Fludrocortisone is mineralocorticoid which increases arteriolar vasoconstrictive effects and increases blood volume and is effective but potential hypokalemia must be carefully managed. If syncopal attacks are preceded by presyncopal symptoms, the patient should assume a sitting or recumbent position as soon as these symptoms occur. Treatment of cardiac-mediated syncope depends on the underlying cardiac disorder.

SUMMARY

Paroxysmal and completely reversible episodes of loss of awareness and consciousness are common medical disturbances. The most common mechanism of these episodes is transient reduction in cerebral blood flow and metabolism. This episode is called syncope. The multiple causes of syncope are reviewed. Most are benign; however, certain cardiac etiologies must be recognized and treated to prevent sudden unexpected death. The most common neurologic cause of reversible loss of consciousness is a seizure. Based upon carefully obtained history of the episodes, it is usually possible to differentiate syncope (faint) from seizure (fit). EEG use can be helpful in certain selected cases, especially if epilepsy is suspected, and use of tilt table examination is important in provoking a characteristic episode, especially if syncope is suspected. Differentiation of syncope from seizure is important because antiepileptic medication is only warranted in treating epilepsy and would not be beneficial for other conditions causing transient loss of consciousness.

SUGGESTEDS READINGS

Evaluation

Day SC, Cook EF, and Funkenstein H: Evaluation and outcome of emergency room patients with transient loss of consciousness, *AM J Med* 73:15, 1982.

Dohrmann ML and Cheitlin MD: Cardiogenic syncope: seizure versus syncope, *Neurol Clin North Am* 4:549, 1986.

Grubb BP, Gerard G, and Roush K: Differentiation of convulsive syncope and epilepsy with head-up tilt testing, *Ann Intern Med* 115:871, 1991.

Grubb BP, Olshansky S: *Syncope: Mechanisms and management*, Futura Pub., Armonk, NY, 1998.

Kapoor WN: Diagnostic evaluation of syncope, *AM J Med* 90:91, 1991.

Linzer M. Diagnosing Syncope. *Ann Int Medicine* 126:969, 1997 and 127: 76, 1997.

Manolis AS, Linzer M, and Salem D: Syncope: current diagnostic evaluation and management, *Ann Intern Med* 112:850, 1990.

Noble RJ: The patient with syncope, *JAMA* 237:1372, 1977.

Shillingford JP: Syncope, *Am Journal Cardiol* 26:609, 1970.

Etiologies

Akhtar M, Jazayeri M, and Sra J: Cardiovascular causes of syncope, *Postgraduate Medicine* 90:87, 1991.

Engel GL: *Fainting*, Springfield, Ill, 1962, Charles C Thomas.

Lin JTY, Ziegler DK, Lai CW, and Bayer W: Convulsive syncope in blood donors, *Ann Neurol* 11:525, 1982.

Sra JS, Jazayeri MR, and Avitall B: Comparison of cardiac pacing with drug therapy in the treatment of neurocardiogenic (vasovagal) syncope with bradycardia or asystole, *N Engl J Med* 328:1085, 1993.

Young WF and Maddox DE: Spells: in search of a cause. *Mayo Clinic Proceedings* 70:757, 1995.

TABLE 8-1. Differentiating Features of Seizures and Syncope

Finding	Seizure	Syncope
Occurrence in sleep	+	-
Premonitory symptoms	+	+
Aura	+	-
Ictus		
Position	Opisthotonic	Limp (hypotonic)
Movement	Rhythmical jerking	Irregular myoclonic
Tongue biting	+	-
Incontinence	+	Can rarely occur
Postictal state		
Duration	Can be prolonged	Brief
Confusion	+	-
Focal deficit	+	-
Amnesia	+	-
EEG during episode	Spikes and slow waves	Diffuse slowing
Clinical pattern	Variable	Stereotyped
Autonomic disturbance		
Skin color	Flushed, cyanotic	Pallor
Perspiration	Hot and wet	Cold sweat
Headache	+	-
Myalgia	+	-

*+, can occur; -, not present