

Syncope in Young Patients II: Presentation and Management of Specific Causes of Syncope

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This article, which is the second part of a 2-part series on syncope in young patients, will review the classifications, etiology, evaluation, and treatment of various types of syncope. The first part of the series, published in the April issue of *Hospital Physician*,¹ presented an approach to the evaluation of a patient with syncope. Case presentations will occasionally be used in this part of the series to illustrate certain clinical points. Particular attention will be paid to the varied manifestations and causes of cardiac syncope (**Table 1**), which generally denotes a poor prognosis and thus necessitates early recognition and aggressive treatment.

NEUROCARDIOGENIC SYNCOPE

Case 1 Presentation

A 19-year-old woman has a witnessed syncopal episode after standing for 2 hours at a crowded outdoor musical concert on a hot, humid August day. She regains consciousness shortly after falling to the ground and is taken by paramedics to a nearby hospital. On questioning, the patient reports having had 2 similar episodes since June; she says she typically feels nauseous and diaphoretic just prior to fainting. She has no history of previous cardiac problems.

Etiology of Neurocardiogenic Syncope

Neurocardiogenic syncope, also known as vasovagal syncope, is the most common cause of syncope in young persons and results from a maladaptive neurocardiovascular reaction to the assumption of an upright posture for prolonged periods. Paradoxical vasodilation (a vasodepressor response), combined with a bradycardic (ie, cardioinhibitory) response, leads to hypotension, cerebral hypoperfusion, and eventual syncope.²

Clinical Manifestations

Characterized by a prodrome of diaphoresis, nausea, vomiting, and dyspnea, neurocardiogenic syncope usually occurs when persons are in an upright position and resolves spontaneously once they are in a supine position. Aggravating factors that commonly trigger neurocardiogenic syncope include a postprandial state, exertion in a warm environment, a prolonged upright posture, use of diuretics, dehydration, and emotional or stressful situations. In contrast, syncope that occurs while a person is in a supine position or that is associated with a prolonged period of confusion after the syncopal event is less likely to be neurocardiogenic in origin.

Diagnosis and Treatment

In cases of neurocardiogenic syncope, a diagnosis is usually made based on the clinical picture. If necessary, a head-up tilt-table test can be used to confirm the diagnosis. Low-dose isoproterenol infusion has been shown to increase the sensitivity of this test but to result in a lower, although still acceptable, specificity (93%).³

Treatment typically includes avoidance of triggering factors, sufficient intake of fluids and salt, and (occasionally) the use of fludrocortisone. β -Blockers, anticholinergic agents, and selective serotonin reuptake inhibitors have also been administered to patients with neurocardiogenic syncope to counteract the maladaptive neurovascular response, but success of such treatments has varied. Cardiac pacing should be reserved for patients with documented prolonged symptomatic bradycardia.

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Table 1. Cardiac Causes of Syncope

Rhythm disturbances

Tachyarrhythmia

- Wolff-Parkinson-White syndrome
- Long QT syndrome
- Right ventricular outflow tract tachycardia
- Idiopathic (primary) ventricular tachycardia/fibrillation
- Right ventricular dysplasia
- Brugada's syndrome

Bradyarrhythmias

- Infectious causes of rhythm disturbances
- Rhythm disturbances caused by sarcoidosis

Coronary artery defects

Vascular flow defects

- Pulmonary hypertension
- Atrial septal defect
- Tetralogy of Fallot

Outflow obstruction

- Hypertrophic obstructive cardiomyopathy
- Mitral valve prolapse
- Left atrial myxoma
- Pulmonary stenosis
- Pulmonary embolism

Follow-up Discussion of Patient 1

Based on her history, patient 1's syncopal episode is diagnosed as neurocardiogenic syncope. She is encouraged to increase her fluid and salt intake.

CARDIAC SYNCOPE

Rhythm Disturbances

Case 2 Presentation. A healthy 20-year-old man has an episode of sudden cardiac death (SCD) while at work. He is resuscitated by paramedics and transferred to a nearby hospital. On admission, results of electrocardiography (ECG) show pre-excitation and a delta wave in the QRS complex.

Tachyarrhythmias. Tachyarrhythmias are characteristic of several clinical syndromes.

Wolff-Parkinson-White syndrome. Not typically associated with structural heart disease, Wolff-Parkinson-White (WPW) syndrome commonly causes supraventricular tachycardia. With subsequent rapid conduction via the accessory pathway, ventricular fibrillation can occur and result in syncope and SCD. WPW syndrome is also associated with atrial fibrillation accompanied by very

rapid ventricular arrhythmias that are triggered by preferential conduction of fibrillatory waves via the fast conducting accessory tract.

Emergency treatment of stable patients with WPW syndrome is intravenous administration of procainamide; unstable patients typically receive direct-current cardioversion. In cases of WPW syndrome, avoidance of nodal blocking agents (eg, calcium channel blockers, digitalis) when treating atrial fibrillation is key. Definitive treatment is radiofrequency ablation of the accessory pathway.⁴ Treatment of asymptomatic patients with WPW syndrome remains controversial.

Based on his clinical presentation and ECG results, patient 2's condition is diagnosed as WPW syndrome. Unfortunately, he is declared brain dead soon after his arrival at the hospital, and his organs are donated by his family.

Long QT syndrome. Long QT syndrome is caused by defects in the body's potassium and sodium channels that result in prolongation of action potentials and trigger ventricular arrhythmias.⁴ It can be either acquired or congenital. Romano-Ward syndrome, a congenital form, is an autosomal dominant disease, whereas Jervell and Lange-Nielsen syndrome, a more severe form, is an autosomal recessive disease associated with deafness. Long QT syndrome is sometimes a familial disorder and is also associated with a family history of SCD.

Typically, long QT syndrome is diagnosed on the basis of ECG evidence of a corrected QT interval of more than 0.44 seconds; a corrected QT interval of more than 0.50 seconds is associated with an increased risk for SCD. Long QT syndrome is sometimes associated with the presence of decreased heart rates and the absence of QT shortening with exercise.

Standard treatment of long QT syndrome often includes administration of β -blockers and phenytoin. Other treatment modalities include pacemaker placement, left stellate ganglionectomy (to prevent the catecholamine surge that occurs during the induction of ventricular arrhythmias), and placement of an implantable cardioverter defibrillator.⁵

Right ventricular outflow tract tachycardia. Right ventricular outflow tract (RVOT) tachycardia is the most common cause of idiopathic ventricular tachycardia and is associated with either nonsustained monomorphic ventricular tachycardia or exercise-induced sustained ventricular tachycardia. Although some patients may not be aware of their arrhythmia, others may experience palpitations, dizziness, and syncope. The prognosis of patients with RVOT tachycardia is usually benign, and SCD is rare.

Management of patients with RVOT tachycardia should first differentiate between this rhythm disturbance and the more aggressive disorder of right ventricular dysplasia. Whereas patients with RVOT tachycardia typically have normal results on resting ECG and on signal-averaged ECG, patients with right ventricular dysplasia usually have abnormal results on resting ECG and late potentials on signal-averaged ECG. The presence of polymorphic ventricular tachycardia similarly makes a diagnosis of RVOT tachycardia doubtful.⁶ Moreover, results of echocardiography are normal in 90% of patients with RVOT tachycardia, although newer modalities (eg, cine magnetic resonance imaging [MRI]) have detected subtle structural abnormalities in the right ventricle.

Given the generally benign nature of this syndrome, patients with RVOT tachycardia who have no (or only mild) symptoms should just be followed. More symptomatic patients should first be treated with administration of β -blockers or calcium-channel blockers (or both). If symptoms are unrelieved, radiofrequency catheter ablation is recommended and is curative in 86% of patients.

Idiopathic (primary) ventricular tachycardia/fibrillation. In most cases, idiopathic ventricular tachycardia/fibrillation is a diagnosis of exclusion in patients with ventricular tachycardia and fibrillation who have undergone a complete evaluation, including Holter monitoring, echocardiography, cardiac catheterization, and stress testing, but still have no definitive diagnosis. The presence of myocarditis, which accounts for 20% to 40% of SCD in young persons, should always be considered in patients with idiopathic ventricular tachycardia and fibrillation. Myocarditis most often results from infection with group B coxsackievirus and is commonly preceded by an influenza-like illness followed by signs of heart failure. Diagnosis of myocarditis is confirmed by echocardiography and endomyocardial biopsy. An electrophysiologic study may be needed to map a specific focus for the associated tachyarrhythmia and possibly to guide therapy.⁴

Management of myocarditis is supportive; rest should be recommended, as should avoidance of exertion. Other conditions in which idiopathic ventricular tachycardia/fibrillation occurs can be treated with either placement of an implantable cardioverter defibrillator or administration of antiarrhythmic drugs.⁵

Right ventricular dysplasia. Right ventricular dysplasia is an arrhythmogenic disorder caused by fibrofatty infiltration of the right ventricular myocardium. Sometimes familial in origin, right ventricular dysplasia often presents as exercise-induced palpitations, syncope, or

SCD. ECG results in patients with this disorder typically show right bundle branch block, with a QRS duration in lead V₁ of more than 0.11 seconds, an epsilon wave in leads V₁ through V₂, and T-wave inversion in the right precordial leads. Additionally, right ventricular dysplasia typically causes ventricular arrhythmias of left bundle branch block morphology.⁷

Diagnosis of right ventricular dysplasia is difficult, even with the use of ECG, 2-dimensional echocardiography, and angiography. Other diagnostic tools that have been used include cardiac MRI and endomyocardial biopsy. However, none of these techniques is sensitive enough to exclude the diagnosis. Leading risk factors include a family history of SCD, the presence of ECG abnormalities, and right ventricular structural disease.

The most effective treatment of right ventricular dysplasia is unclear. Administration of antiarrhythmic drugs may be useful. Alternative therapies include radiofrequency ablation of the right ventricular outflow tract and cardiac transplantation.⁴

Brugada's syndrome. Brugada's syndrome is a recently identified disorder affecting young patients with the presenting symptom of syncope (and sometimes SCD). Recent studies⁵ report a 34% incidence of arrhythmias in patients with Brugada's syndrome, and approximately two thirds of affected patients have syncope or aborted cardiac arrest as their initial symptom. Brugada's syndrome is considered a highly lethal disorder.

Typically, results of ECG in patients with Brugada's syndrome reveal a pattern of right bundle branch block with ST-segment elevation in the right precordial leads. Electrophysiologic study is also helpful in diagnosing Brugada's syndrome. Diagnostic challenge with administration of procainamide and disopyramide during electrophysiologic study causes ST-segment elevation in the same leads and may lead to induction of ventricular dysrhythmia.⁵ Electrophysiologic study is also helpful in diagnosing Brugada's syndrome.

Administration of β -blockers combined with other antiarrhythmic drugs was not found to be effective in decreasing the high mortality rate of Brugada's syndrome. Definitive treatment is placement of an implantable cardioverter defibrillator.⁶

Bradyarrhythmias. Bradyarrhythmias are a rare cause of syncope in young patients. They usually occur as secondary manifestations of systemic diseases.

Infectious causes of rhythm disturbances. Lyme disease can cause rhythm disturbances leading to syncope 1 to 2 months after the onset of infection. The most common conduction abnormality caused by the infection is atrioventricular nodal block (in 87% of cases⁸), followed by bundle branch and fascicular blocks. Diagnosis

requires maintaining a high index of suspicion in patients from endemic areas who have presenting symptoms of syncope and heart block. Administration of antibiotic agents may resolve the heart block, but some patients will need a temporary or permanent pacemaker.⁸

Chagas' disease is an infection caused by *Trypanosoma cruzi*. Very common in South Americans (with an incidence reported to be as high as 6%⁹), Chagas' disease usually causes cardiac problems during the chronic stage of the disease. More specifically, it can cause biventricular failure that may lead to ventricular arrhythmias, syncope, and SCD. Standard treatment usually involves administration of class III antiarrhythmic agents (especially amiodarone). In patients who are refractory to medical treatment, electrophysiologic study with intracardiac mapping is indicated, followed by either radiofrequency ablation or surgical excision of the infected myocardial wall.⁹

Rhythm disturbances caused by sarcoidosis. Although cardiac involvement is rare in sarcoidosis, occurring in only approximately 3% of cases,¹⁰ ventricular dysrhythmias, conduction defects, blocks, and syncope can occur with the disorder.

Coronary Artery Defects

Case 3 presentation. A 22-year-old man who is a marathon runner reports angina and dizziness while training for an upcoming event. He subsequently has a witnessed episode of syncope during the final days of training. Because of suspicious findings on 2-dimensional echocardiography, his cardiologist refers him for coronary angiography. Results of coronary angiography confirm an anomalous origin of the left main coronary artery. The patient undergoes corrective surgery. After a year of observation, he resumes his marathon training

Discussion. Exertional syncope, angina, and SCD are all characteristic symptoms of coronary artery defects. This type of anatomic defect is the second most common cause of SCD (19%¹¹) in young athletes. In the most common type of coronary artery defect, the left main coronary artery originates from the right sinus of Valsalva. Ischemia results from the exaggerated sharp angle between the aorta and the pulmonary trunk that occurs in the aberrant origin with exercise. Other common defects include having the right coronary artery originate from the left coronary sinus and having the left anterior descending artery originate from the pulmonary artery.¹¹

Evaluation of patients with suspected coronary artery defects should include exercise imaging, with specific attention paid to the aortic root and the origin

of the coronary arteries. Coronary angiography is often required to confirm the abnormal anatomy. The only definitive treatment is surgical correction.

Vascular Flow Defects

Pulmonary hypertension. Pulmonary hypertension occurs as a result of long-standing congenital heart disease that ultimately results in reversal of the shunt flow to a right-to-left direction (ie, Eisenmenger syndrome). Syncope and SCD can occur because of the abnormal blood flow or because of ventricular arrhythmias that are secondary to associated hemodynamic abnormalities. Both primary and secondary pulmonary hypertension can be complicated by syncope, presenting as effort-related syncope arising from inadequate cardiac output in response to systemic vasodilation; in a young patient who experiences syncope during or shortly after exertion but who does not have a cardiac murmur, primary pulmonary hypertension should be a strong contender in the differential diagnosis.

Optimal management of pulmonary hypertension is surgical correction of the hemodynamic abnormalities before development of shunt reversal. Some studies have alleged that patients with primary pulmonary hypertension can benefit from blade and balloon atrial septostomy, which creates an atrial level of communication; this atrial defect facilitates right-to-left shunting and preserves cardiac output during exacerbations of pulmonary hypertension.¹² Treatment of the associated arrhythmias with drugs, placement of an implanted cardioverter defibrillator, or radiofrequency ablation may also be necessary after correction of the hemodynamic abnormality (or if correction of the abnormality is contraindicated).⁴

Atrial septal defect. An atrial septal defect of the ostium secundum type can cause syncope when secondary pulmonary hypertension, a large right-to-left shunt, and Eisenmenger syndrome develop. Patients with a moderate-to-large defect and those with reversal of the shunt should not be allowed to participate in sports. Definitive treatment requires closing the defect, either by surgery or by a percutaneous catheter technique. Generally, patients can again participate in sports 6 months after their defect is corrected, provided they have been thoroughly evaluated with exercise echocardiography and are under supervision.¹³

Tetralogy of Fallot. Syncope in patients with tetralogy of Fallot occurs after development of Eisenmenger physiology, a large right-to-left shunt, and hypoxia. Syncope is usually associated both with exertion resulting from decreased systemic vascular resistance and preferential blood flow through the shunt to the left ventricle and

with hypoxia. Optimal treatment necessitates early recognition of the tetralogy and surgical correction, prior to development of pulmonary hypertension. Patients with established Eisenmenger syndrome and hypoxia should not be allowed to participate in sports.⁴

Outflow Obstruction

Case 4 presentation. A 22-year-old man has a witnessed syncopal episode while playing in a college basketball game. He has a known family history of SCD. After immediate transfer to the nearest hospital, he undergoes 2-dimensional echocardiography in the emergency department. Results show a left ventricular outlet obstruction that was worsened by prior administration of amyl nitrate.

Case 5 presentation. A healthy 20-year-old woman is found to have an apical diastolic murmur with a click during a routine physical examination. The patient then undergoes 2-dimensional echocardiography.

Hypertrophic obstructive cardiomyopathy. With a prevalence of 1 in 500 in the US population, hypertrophic obstructive cardiomyopathy (HOCM) is now considered more common than was previously recognized,⁴ especially in men and in African Americans. In another study, it was identified as the most common cause (36%) of SCD in athletes.¹⁴ HOCM causes syncope and SCD primarily by way of ventricular tachyarrhythmias and myocardial ischemia; bradyarrhythmias are less commonly involved. Persons at highest risk for the disorder include survivors of cardiac arrest, those with high-risk genotypes, those with a significant family history of SCD, those with massive hypertrophy (wall thickness > 35 mm), and those with sustained or nonsustained ventricular tachycardia.¹⁵ Interestingly, the presence of repolarization (ie, T-wave) alternans is predictive of increased risk for ventricular arrhythmia and decreased survival; conversely, results of electrophysiologic study are not good predictors of mortality in cases of HOCM.

Standard treatment of the disorder involves administration of β -blockers or calcium-channel blockers, although this therapy may not prevent sudden death. High-risk patients should undergo placement of an implantable cardioverter defibrillator, with optional administration of amiodarone. Surveillance of other family members of patients with HOCM is warranted.

Patient 4 receives a diagnosis of HOCM based on the results of his 2-dimensional echocardiography. Because of his personal and family history, he undergoes placement of an implantable cardioverter defibrillator.

Mitral valve prolapse. Mitral valve prolapse is a relatively common entity in the general population, with a

worldwide prevalence of 4% to 5%.⁶ Prolongation of the QT interval can result from delayed repolarization caused by abnormal papillary muscle stretching. Most patients with mitral valve prolapse have a benign course. However, patients with this disorder and significant mitral regurgitation are at increased risk for ventricular tachyarrhythmias that can lead to syncope and SCD; a proposed theory suggests that these ventricular arrhythmias result from endocardial friction lesions in 86% of cases.⁶ Although the prevalence of ventricular arrhythmias is increased in patients with mitral valve prolapse who have significant mitral regurgitation, this fact does not translate into an increased risk for SCD.⁶

Factors that are predictive of SCD in patients with mitral valve prolapse include a personal history of syncope (reported in 50% of patients with mitral valve prolapse who experience SCD⁶), a family history of SCD at young age, and abnormalities on ECG at rest, including a prolonged QT interval.

Asymptomatic patients with mitral valve prolapse require no treatment. Those with palpitation caused only by ventricular ectopy can be treated with administration of β -blockers. Those with tachyarrhythmia can be treated with administration of various antiarrhythmic drugs; however, there is no evidence that suppression of the arrhythmias prevents SCD. Also, studies of mitral valve repair or replacement have yielded mixed results. In high-risk patients, the presence of ventricular tachyarrhythmias should be documented, and placement of an implantable cardioverter defibrillator should be considered.⁶

Results of 2-dimensional echocardiography in patient 5 confirm a diagnosis of mitral valve prolapse; however, there is no evidence mitral regurgitation or left ventricular hypertrophy. On further questioning, she recalls no palpitations or syncopal events. Because she is asymptomatic, her physician decides not to treat her at this time.

Left atrial myxoma. Syncope in patients with left atrial myxoma is caused by obstruction of blood flow to the left ventricle and is usually related to postural changes.

Treatment is surgical removal of the myxoma.

Pulmonary stenosis. Many patients who have pulmonary stenosis survive to adulthood without experiencing any symptoms. However, as the pulmonary valve becomes more fibrotic with age, patients begin to experience symptoms of right ventricle failure, exertion-related dyspnea, and syncope. Diagnosis is easily made based on results of 2-dimensional echocardiography, with determination of the pressure gradient. Symptomatic patients with mild stenosis (ie, a pressure gradient < 25 mm Hg)

can undergo percutaneous valvuloplasty. Patients with moderate and severe stenosis require either valvotomy or valvuloplasty.

Pulmonary embolism. A diagnosis of pulmonary embolism should be considered in any young patient with paraplegia or a clotting disorder who experiences syncope. Usually, if the pulmonary embolism is large enough to cause syncope, the patient will most likely need thrombolytic therapy.

ORTHOSTATIC HYPOTENSION

Typical Presentation and Outcome

Orthostatic hypotension occurs when a susceptible person assumes an upright posture. Persons who experience orthostatic hypotension tend to have decreased arterial pressure, a feeling of light-headedness, and blurred vision. Total recovery from these signs and symptoms follows the assumption of a recumbent position. Orthostatic hypotension is usually aggravated by venous blood pooling and/or volume depletion. The disorder can be an adverse effect of various medications, most notably diuretics, antihypertensive agents, nitrates, tranquilizers, and antidepressants. Avoiding inciting factors and following simple protective measures when assuming an upright posture are usually recommended as standard therapy.

Postural Orthostatic Tachycardia Syndrome

Postural orthostatic tachycardia syndrome is a variant form of orthostatic hypotension that affects young persons. A pathologic disorder, it occurs when insufficient sympathetic activation during standing causes recurrent symptoms of light-headedness, fainting, and syncope. Recently, postural orthostatic tachycardia syndrome has been closely linked to chronic fatigue syndrome in young adults.¹⁶ Diagnosis of postural orthostatic tachycardia syndrome requires first documenting the presence of orthostatic hypotension, tachycardia, and other relevant symptoms on head-up tilt-table testing.

NEUROLOGIC CAUSES OF SYNCOPE

Seizures

An early, essential part of the diagnostic evaluation of patients with syncope of presumed neurologic cause is distinguishing between syncope and a seizure. Common features of seizures include sudden onset, lack of a prodrome, absence of pallor, injury from falling, convulsions, incontinence, prolonged unconsciousness, and postictal confusion. Temporal lobe seizures (ie, absence seizures) are the most difficult to differentiate from syncope. Interestingly, a seizure may

be the initial manifestation of an arrhythmia, especially long QT syndrome,¹⁷ or may occur secondary to prolonged cerebral hypoperfusion in cases of syncope.

Migraines

Syncope is often caused by the vasospasm that is part of the pathophysiology of migraines, especially when they affect the basal arterial system. More specifically, there is the premonitory aura of the migraine itself, followed by loss of consciousness for several minutes. The patient usually awakens with a severe occipital or lateralizing headache.

Transient Ischemic Attacks/-strokes

In young patients, transient ischemic attacks or strokes are generally unlikely to cause syncope. However, when syncope does occur as a result of these neurologic insults, it is more likely to be associated with vertebrobasilar insufficiency and a defect in the medullary arousal center. Although microemboli are most commonly involved in such syncopal episodes, vasculitis and systemic hypotension can also play a role.

Treatment of syncope caused by transient ischemic attacks or strokes should be directed at the cause of the cerebrovascular disease. For example, patients with thrombophilia and clotting disorders may need lifelong anticoagulation, whereas patients with vasculitis will often require immunosuppression with corticosteroids.

MISCELLANEOUS CAUSES OF SYNCOPE

Alcohol

Increased alcohol consumption has been suggested as a cause of syncopal episodes, most likely by aggravating orthostatic hypotension. Even short-term alcohol ingestion elicits hypotension during orthostatic stress because of impairment of vasoconstriction.¹⁸

The increased prevalence of alcohol abuse by young persons necessitates the exclusion of alcohol abuse in any young patient presenting with syncope.

Drugs

Cocaine abuse is known to cause cardiomyopathy, hypertension, aortic dissection, accelerated coronary artery disease, and SCD. Moreover, it has recently been reported that cocaine abuse can cause bradyarrhythmias that first present as syncope.¹⁹ Cocaine abuse has also been reported to cause ventricular arrhythmias and bundle branch blocks. Similarly, heroin and tricyclic antidepressants have been associated with heart block and SCD.²⁰

Hysterical Syncope

Hysterical syncope is seldom associated with injury and most often occurs in young adults in the presence of an audience. Although such syncopal episodes can be prolonged, persons experiencing them generally maintain a normal heart rate and blood pressure. Unlike syncope caused by cardiac and other causes, hysterical syncope is not relieved by assuming a supine position.

Psychogenic Factors

When psychogenic factors are responsible for syncope, the episode is typically characterized by rapid deep breathing, a feeling of chest tightness, palpitations, circumoral and hand numbness, and tingling. Symptoms are reproducible by inducing hyperventilation. Standard treatment is encouraging breathing through a bag or cupped hands and providing reassurance.

Eating Disorders

Both anorexia nervosa and bulimia have been associated with syncope and SCD. Patients with these disorders typically have electrolyte imbalances, bradycardia, and a prolonged QT interval on ECG.²⁰

CONCLUSION

This 2-part series has attempted to provide useful clinical information about the issue of syncope in young patients, first detailing an approach to patients with syncope and then outlining the most common causes of syncope in this population. Despite diagnostic challenges, key elements from the history, physical examination, and laboratory and other evaluations of patients with syncope can often differentiate cardiac and noncardiac causes. Therapy should be directed toward the specific causes of syncope. In the absence of available specific therapy, avoidance of situations in which injury can occur to the patient with syncope is advisable. **HP**

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