

Syncope in Young Patients I: An Approach to the Patient with Syncope

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Syncope is among the most common causes of emergency department and outpatient clinic visits in the United States, accounting for 5% of such visits and 1% to 3% of all hospital admissions.¹ Despite a benign outcome in most cases, syncopal events can cause significant distress to patients and their families, especially when the patient is young and otherwise healthy.

This article, which is the first part of a 2-part series, will review the necessary steps to determine the cause of syncope, especially in young persons. The importance of history taking, physical examination, and laboratory and other diagnostic testing will be stressed. Moreover, the implications of syncope in the specific population of children and young adults who are athletes or otherwise participate in sports will be discussed, as will the related issue of sudden cardiac death (SCD).

INITIAL CLASSIFICATION

Persons who experience syncope should have a basic work-up before undergoing more complicated, low-yield diagnostic studies. The key initial step should be to classify the syncopal episode as cardiac or noncardiac.¹ This general classification (**Figure 1**) aids in triaging the patient, determining the necessary investigations to perform and treatment to provide, and defining the general prognosis. Patients whose syncope has a cardiac etiology tend to have a relatively poor prognosis, compared to patients whose syncope is noncardiac in origin.

TAKING THE HISTORY

Determining the history surrounding the syncopal episode is of paramount importance (**Table 1**); despite a low specificity, the history can provide a general direction to the evaluation and may even yield a diagnosis. Special emphasis should be given to previous similar episodes. Likewise, a history of pre-existing cardiac disease is an important predictor of arrhythmic syncope and mortality. In contrast, specific symptoms, although

helpful in discovering many noncardiac causes of syncope, are not useful in risk-stratifying the mortality of patients whose syncope cannot be classified by either history or physical examination findings.² However, the details of the syncopal event, in particular its relation to exertion, are of extreme importance in identifying the risk for SCD (especially in athletes) and can usually limit the differential diagnosis to fewer diseases. Similarly, a family history of hereditary cardiac problems or SCD can be crucial in defining the risk for the patient. Finally, obtaining a social history detailing any drug and/or alcohol abuse is vital, as is obtaining any travel history and learning of any exposure to infections.

PHYSICAL EXAMINATION

The physical examination should be directed toward the discovery of possible causes of the syncopal event (**Table 2**). Again, special attention should be paid to excluding noncardiac causes of syncope. In the evaluation of orthostatic blood pressure, for example, the finding of tachycardia (ie, a heart rate > 100 bpm) and the occurrence of symptoms of dizziness and syncope on assuming an upright position are more specific than is a decrease in blood pressure in identifying patients with orthostatic hypotension or with dehydration (which can aggravate syncope because of volume depletion).³ Signs of long-standing cardiac disease can also help in the diagnosis. Notably, normal results on neurologic examination do not exclude transient ischemic attacks or seizure disorders.

LABORATORY AND OTHER DIAGNOSTIC TESTING

Following history taking and physical examination,

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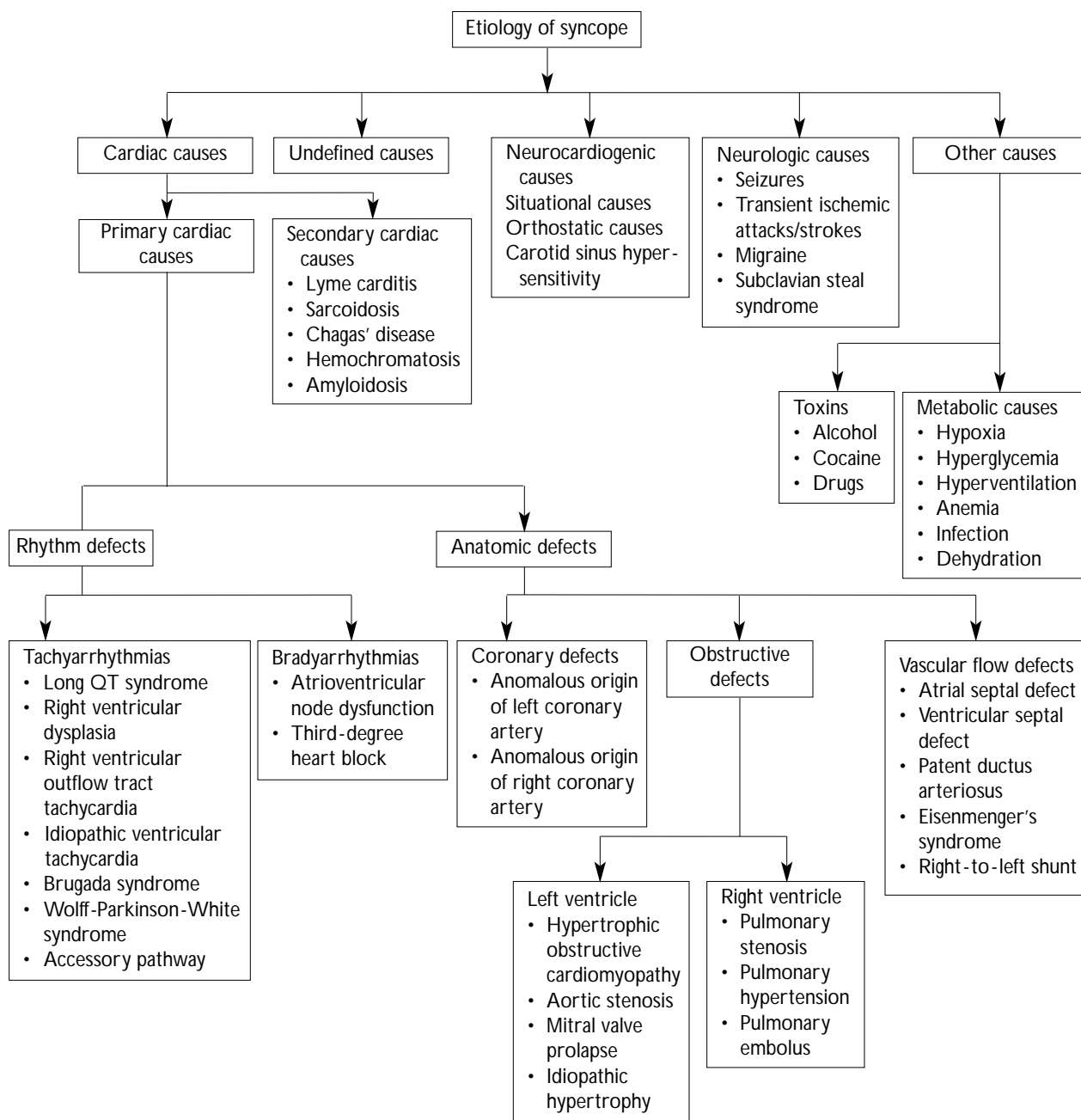


Figure 1. Algorithm illustrating the etiology of syncope.

the patient should undergo some basic laboratory testing (Table 3) to aid in classifying the cause of syncope. Of course, this basic testing is more beneficial in excluding some obvious causes of syncope (eg, metabolic, toxic, hematologic diseases) than in arriving at a definitive diagnosis of a rare or unusual cause. Similarly, baseline electrocardiography (ECG) should be performed to guide the diagnosis to a specific entity (Table 4). In fact,

it is reasonable to place patients on ECG monitoring while in the emergency room until a disposition is clear.

EVALUATION WHEN A CARDIAC CAUSE OF SYNCOPE IS SUSPECTED

If the clinical scenario and basic evaluation suggest a cardiac cause, the patient should be admitted to a telemetry bed for further investigation. The work-up of a

Table 1. History Taking in Patients with Syncope

Key Elements	Significance
Medical history	
Previous similar episodes	Multiple episodes are more likely to be noncardiac in origin and to have a good prognosis.
History of cardiac problems in childhood	Pulmonary hypertension and Eisenmenger's syndrome are more likely in the presence of long standing congenital cardiac diseases.
History of infection, malnutrition, or dehydration	Orthostatic hypotension can occur and commonly present as syncope.
Details concerning past menstrual period	Pregnancy should be ruled out in any young female patient.
History or symptoms of endocrinopathy*	Endocrine conditions are generally easy to diagnose and treat.
History of a tick bite	Lyme disease can cause variable degrees of heart block and present as syncope.
Description of syncopal episode(s)	
Episodes of exertion-related syncope	Such history usually suggests a cardiac origin and a worse prognosis.
Postexertional onset	Postexertional syncope has a worse prognosis.
Onset immediately on standing	This finding suggests orthostatic hypotension.
Typical onset with standing, preceded by a prodrome of warmth, diaphoresis, and lightheadedness	These elements strongly suggest neurocardiogenic syncope.
Sudden onset, no relation to posture, and no prodrome	These elements strongly suggest cardiac arrhythmia.
History or symptoms suggestive of seizure	Absence seizures can present as syncope in young persons.
Relation to postural changes	This finding suggests possible subclavian steal syndrome or left atrial myxoma.
Invariably orthostatic in nature	This finding excludes the presence of postural orthostatic tachycardia syndrome.
Association with headaches	This finding suggests migraines or seizures.
Family history of sudden death	This finding can suggest the potential presence of conditions such as hypertrophic obstructive cardiomyopathy, coronary artery anomalies, right ventricular dysplasia, mitral valve prolapse, Marfan syndrome and long QT syndrome.
Social history	
History of alcohol abuse	Alcohol potentiates neurocardiogenic syncope.
History of drug abuse	Cocaine causes tachyarrhythmias and bradyarrhythmias.

*For example, diabetes mellitus, diabetic ketoacidosis, and adrenal or thyroid dysfunction.

patient with presumed cardiac syncope is shown in **Figure 2**. The distinction between the investigations required to discover arrhythmic as opposed to anatomic causes of syncope is arbitrary, because patients with anatomic defects frequently need to undergo further studies to exclude secondary arrhythmia, and vice versa. The value of ECG monitoring is either to confirm arrhythmia as the cause of syncope (if it occurs in conjunction with the symptoms) or to exclude it (if the symptoms occur in absence of any arrhythmia).⁴ Patients

with inconclusive results on ECG and Holter monitoring can undergo electrophysiologic tests. If all previous investigations fail to establish a definitive cause of the syncopal event but the history is suggestive of arrhythmic syncope, the patient can be discharged with either an ambulatory Holter monitor or an ambulatory continuous-loop monitor. However, both modalities have limited sensitivity for confirming the cause of syncope.⁵

If a patient's symptoms are suggestive of structural heart disease but the initial evaluation for arrhythmias

Table 2. Steps in Physical Examination of Patients with Syncope

Finding	Significance
Vital signs	
Pulse	Whether the pulse is regular or irregular and whether there is bradycardia or tachycardia can be significant.
Blood pressure	Whether the patient is hypotensive, is dehydrated, or has an infection should be determined.
Orthostasis	The occurrence of syncope and tachycardia on standing up are a sensitive sign for the presence of orthostasis and volume depletion.
Neck examination findings	
Hypersensitive carotid sinus	This finding indicates carotid sinus hypersensitivity.
Carotid artery bruits with dissimilar blood pressures in each arm	These findings suggest aortic dissection or subclavian steal syndrome.
Pulmonary examination findings	
Cyanosis, clubbing, oxygen desaturation	These findings suggest the presence of pulmonary hypertension and Eisenmenger's syndrome.
Cardiac examination findings	
Systolic murmur at the left sternal border that increases with performance of the Valsalva maneuver and decreases with standing	This finding suggests the presence of hypertrophic obstructive cardiomyopathy.
Ejection systolic murmur at the aortic area, with a slow carotid upstroke	This finding suggests the presence of aortic stenosis.
Apical midsystolic nonejection click followed by a late systolic murmur	These findings indicate mitral valve prolapse.
Systolic ejection murmur in the pulmonic area and a single component S ₂	This finding suggests the presence of pulmonary stenosis.
Neurologic examination findings	
Focal weakness	This finding is most suggestive of either a transient ischemic attack or a stroke (as opposed to postictal focal weakness).
Vertigo, nystagmus, diplopia, ataxic gait	This combination of findings suggests vertebrobasilar insufficiency.
Signs of alcohol or drug abuse	Such findings can aid in diagnosis.

is inconclusive, the patient should undergo cardiac imaging, either with 2-dimensional echocardiography or with a perfusion nuclear scan. If the patient's symptoms are exertional or postexertional, the imaging study should be performed with stress in the form of exercise. Confirmation of an anatomic defect often requires cardiac catheterization. As previously suggested, the ability of any anatomic defect to induce arrhythmias should be assessed. Electrophysiologic testing is useful in this regard for risk stratification.

EVALUATION WHEN A NEUROLOGIC CAUSE OF SYNCOPE IS SUSPECTED

When their basic evaluation and clinical scenario suggest neurologic disease as the cause of syncope, patients

should be admitted to the hospital for further investigations and regular neurologic checks (**Figure 3**). If the index of suspicion is high for a seizure disorder, patients should undergo electroencephalography (or videoelectroencephalography, if syncope is episodic). After a seizure disorder is confirmed, further testing (including brain scans) should be performed to rule out secondary causes of seizure; for example, when a vascular event is suspected, a computed tomography (CT) scan should be obtained to exclude an acute intracranial hemorrhage. Syncope caused by transient ischemic attacks or stroke is more likely to involve the vertebrobasilar circulation and be accompanied by symptoms of vertigo and nystagmus; consequently, MRI should be used in these cases because it is superior for imaging the posterior circulation and

Table 3. Laboratory and Other Studies in Patients with Syncope

Study	Significance
Complete blood count	Results can reveal the presence of anemia, leukocytosis (indicating infection), and dehydration.
Basic metabolic panel	Results can reveal the presence of dehydration, electrolyte imbalance, uremia, hyperglycemia, and anion-gap acidosis typical of diabetic ketoacidosis.
Urine and serum toxicology screening	Such screening, which is optional, can reveal the presence of alcohol and cocaine in the system.
Serum pregnancy test	This study is mandatory for all female patients.
Arterial blood gases, chest radiograph	These tests, although optional, can aid diagnosis in patients with trouble breathing, hypoxia, shortness of breath, and suspicion of pulmonary embolism.
Thyroid function tests	These tests, although optional, can determine whether hypothyroidism is present.

Table 4. Results of Electrocardiography in Patients with Syncope

Findings	Significance
Increased/decreased heart rate	Tachycardia/bradycardia
Corrected QT interval of more than 0.44 s	Long QT syndrome
Right bundle branch block and ST-segment elevation in leads V ₁ through V ₃	Potential Brugada syndrome
Right bundle branch block with a QRS duration in lead V ₁ greater than 110 ms, an epsilon wave in V ₁ through V ₂ , and T-wave inversion in the right precordial leads	Potential right ventricular dysplasia
Short PR interval and a delta wave	Possible Wolff-Parkinson-White syndrome or supraventricular tachycardia
Prolonged PR interval and a bundle branch block	Possible atrioventricular block

fossa. Conventional angiography (and sometime magnetic resonance angiography) is required on occasion to distinguish between a blood clot and vasculitis. If a clot is diagnosed in a young person, the patient should undergo complete evaluation for thrombophilia and clotting disorders. On the other hand, the evaluation of a patient with vasculitis should focus on discovering rheumatologic diseases. Empiric CT scans in patients for whom the index of suspicion for neurologic disease is low have a very low diagnostic yield.

EVALUATION WHEN NEUROCARDIOGENIC SYNCOPE IS SUSPECTED

When syncope is recurrent but evaluations for cardiac and neurologic causes yield no definitive findings, a head-up tilt-table test may be appropriate to diagnose neurocardiogenic syncope, especially if there is a high index of suspicion. As previously mentioned, isoproterenol infusion has improved the sensitivity but lowered the specificity of the test in the diagnosis of neurocardiogenic syncope.⁶

EVALUATION WHEN NO SPECIFIC CAUSE OF SYNCOPE HAS BEEN IDENTIFIED

When extensive evaluation fails to suggest a cause of the syncopal events, especially in the presence of recurrent syncope, patients may require further monitoring with an implanted loop monitor for prolonged periods. Of course, in 10% to 20% of cases, no obvious cause of syncope is ever identified. In such cases, protective measures for the patient should be implemented to decrease morbidity.⁴

SYNCOPE IN ATHLETES

Young persons who regularly participate in sports and experience syncope pose a special diagnostic challenge. Clinicians should remember that normal ECG patterns in athletes can include sinus bradycardia, sinus tachycardia, sinus arrhythmia, a wandering atrial pacemaker, asymptomatic sinus pauses, and premature ventricular beats—assuming that structural heart disease has been excluded. The most common pathologic conditions associated with exertion-induced syncope in these

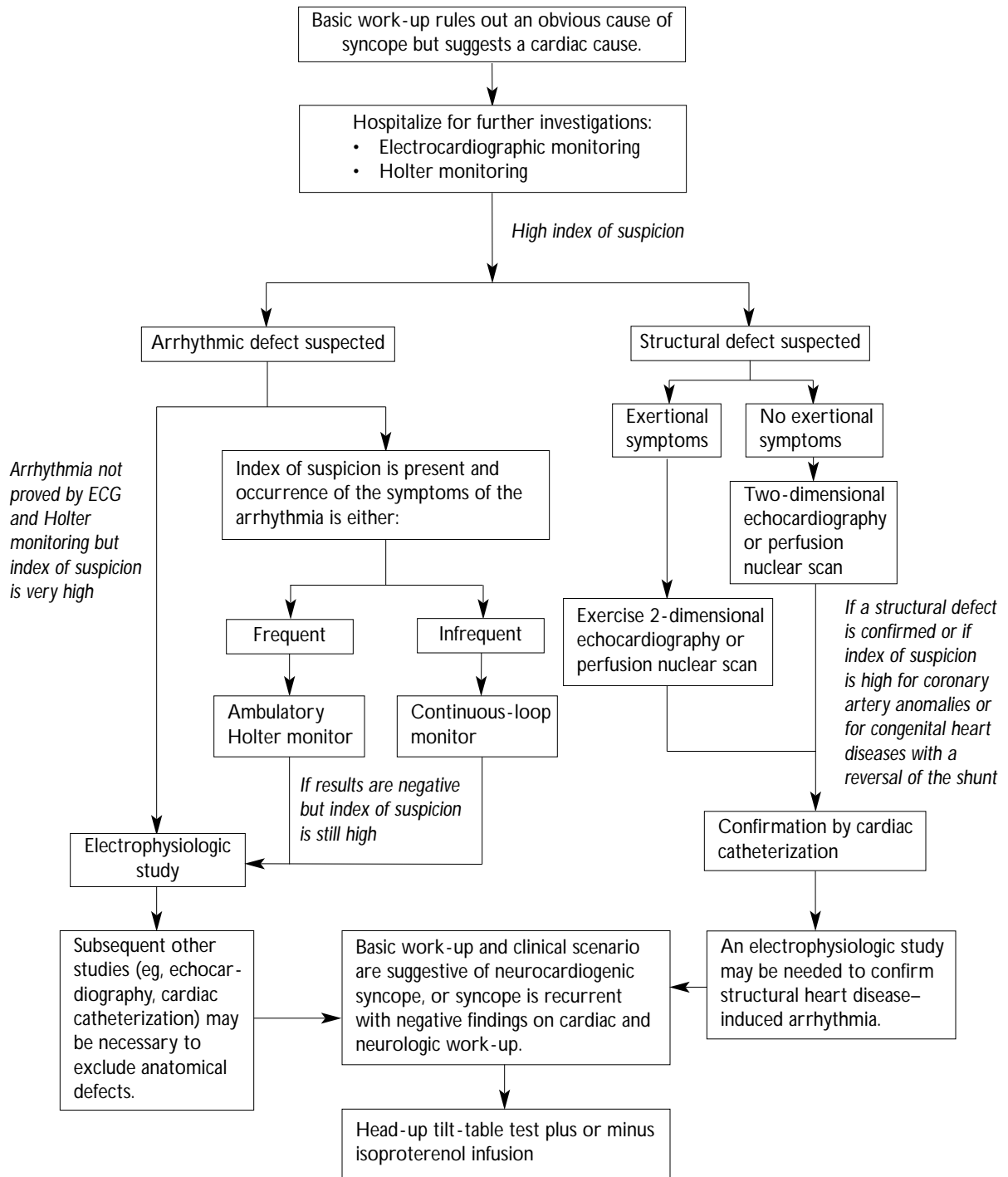


Figure 2. Algorithm illustrating the appropriate steps when a basic work-up has ruled out the obvious causes of syncope but suggests a cardiac cause. ECG = electrocardiography.

athletes are hypertrophic obstructive cardiomyopathy (HOCM), coronary artery anomalies, aortic stenosis, Wolff-Parkinson-White (WPW) syndrome, right ventricular dysplasia, and congenital cyanotic heart conditions.

Athletes who experience syncope because of HOCM and right ventricular dysplasia should not return to full participation in physical activity, even after they have been treated. In contrast, athletes who experience

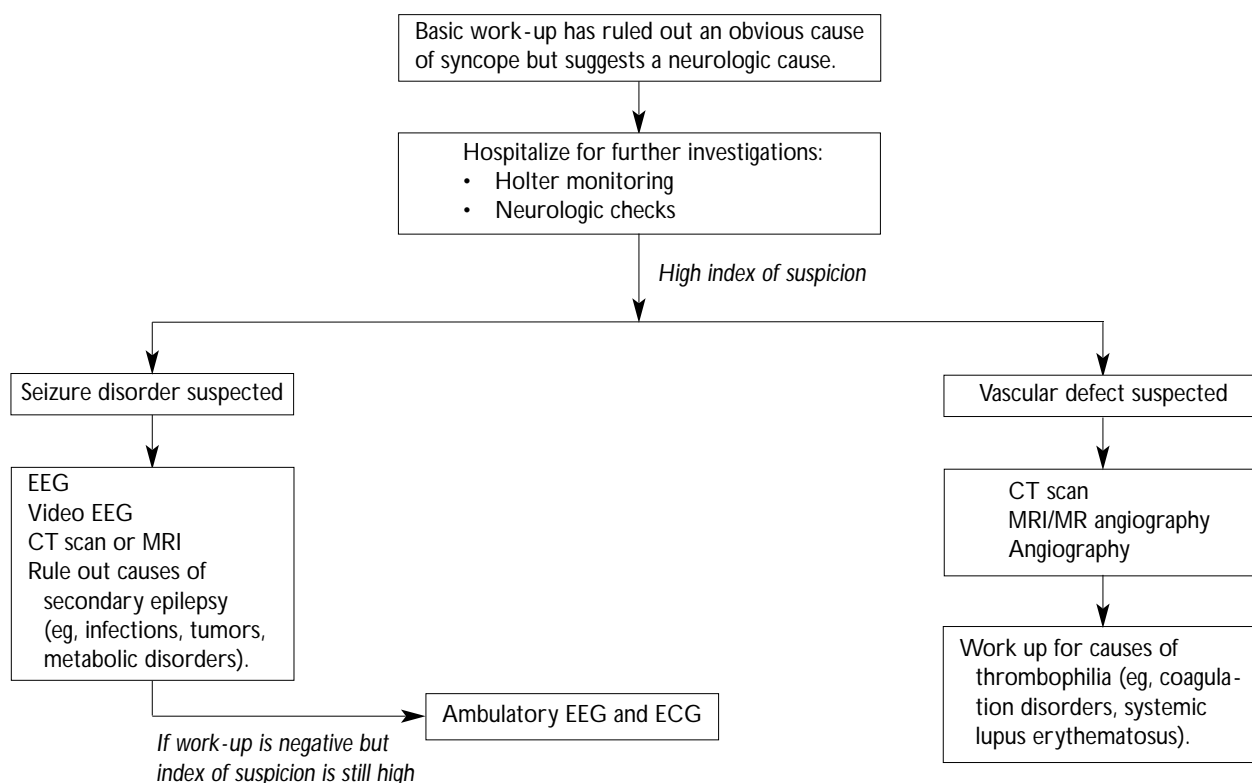


Figure 3. Algorithm illustrating the appropriate steps when a basic work-up has ruled out the obvious causes of syncope but suggests a neurologic cause. CT = computed tomography; ECG = electrocardiography; EEG = electroencephalography; MRI = magnetic resonance imaging.

syncope because of coronary artery anomalies, aortic stenosis, WPW syndrome, and other conditions can generally return to full participation gradually (and under strict supervision) after successful treatment.⁷

Sudden Cardiac Death

Despite a very low incidence in young adults (1 per 100,000 to 1 per 300,000),⁸ SCD in young athletes has received much attention recently and can cause significant distress to affected persons and their families. The primary goal of screening young athletes to determine risk for SCD is to identify those with either an underlying structural heart disease or a documented arrhythmia. Such screening is particularly useful in high-dynamic sports (eg, marathon running), in which the risk is greater. Characteristics of patients with increased risk for SCD include a family history of SCD, a personal history of survival after previous SCD, syncope (especially exertion-related syncope), symptomatic arrhythmias, and inducible ventricular arrhythmias on electrophysiologic study.

The most common cause of SCD in athletes varies between studies, according to geographic distribution. In a combined analysis of data for athletes in the United States, HOCM was the most common cause (23%), followed by coronary artery anomalies (16%), coronary artery disease (12%), myocarditis (9%), and right ventricular dysplasia (8%).^{8,9} In contrast, an Italian study reported right ventricular dysplasia as the most common cause (22%), followed by coronary atherosclerosis (18%) and coronary artery anomalies (12%); mitral valve prolapse and HOCM caused SCD much less commonly.¹⁰

Because of the risk for SCD, patients should not be allowed to participate in sports activities if they have symptomatic ventricular dysfunction, symptomatic arrhythmias, cyanosis (with an arterial saturation < 80%), or significant pulmonary hypertension.⁷

CONCLUSION

Syncope in young persons presents a considerable diagnostic challenge. Findings from the history, physical

examination, laboratory testing, and other investigations should be used to differentiate cardiac causes from non-cardiac causes. Treatment should be directed toward correcting the specific disease(s) responsible for the syncope and preventing any subsequent morbidity or mortality. Part 2 of this series will treat in greater detail the cardiac and noncardiac types of syncope, providing specific information on their etiology, clinical manifestations, diagnosis, and treatment. **HP**

REFERENCES

1. Kapoor WN. Evaluation and outcome of patients with syncope. *Medicine (Baltimore)* 1990;69:160–75.
2. Oh JH, Hanusa BH, Kapoor WN. Do symptoms predict cardiac arrhythmias and mortality in patients with syncope? *Arch Intern Med* 1999;159:375–80.
3. Mangione S. *Physical diagnosis secrets*. Philadelphia: Hanley & Belfus; 2000:4.
4. Kapoor WN. Syncope. *N Engl J Med* 2000;343:1856–61.
5. Zimetbaum PJ, Josephson ME. The evolving role of ambulatory arrhythmia monitoring in general clinical practice. *Ann Intern Med* 1999;130:848–56.
6. Morillo CA, Klein GJ, Zandri S, Yee R. Diagnostic accuracy of a low-dose isoproterenol head-up tilt protocol. *Am Heart J* 1995;129:901–6.
7. Graham TP Jr, Bricker JT, James FW, Strong WB. 26th Bethesda conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. Task Force 1: congenital heart disease. *J Am Coll Cardiol* 1994;24:867–73.
8. Libberthson RR. Sudden death from cardiac causes in children and young adults. *N Engl J Med* 1996;334:1039–44.
9. Maron BJ, Shirani J, Poliac LC, et al. Sudden death in young competitive athletes. Clinical, demographic and pathological profiles. *JAMA* 1996;276:199–204.
10. Corrado D, Basso C, Schiavon M, Thiene G. Screening for hypertrophic cardiomyopathy in young athletes. *N Engl J Med* 1998;339:364–9.

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