# CLINICAL GUIDELINE: Diagnosing Syncope: Part 2: Unexplained Syncope

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**Purpose:** To review the literature on diagnostic testing in syncope that remains unexplained after initial clinical assessment.

Data Sources: MEDLINE search.

**Study Selection:** Published papers were selected if they addressed diagnostic testing in syncope, near syncope, or dizziness.

Data Extraction: Studies were identified as population studies, referral studies, or case series.

**Data Synthesis:** After a thorough history, physical examination, and electrocardiography, the cause of syncope remains undiagnosed in 50% of patients. In such patients, information may be derived from the results of carefully selected diagnostic tests, especially 1) electrophysiologic studies in patients with organic heart disease, 2) Holter monitoring or telemetry in patients known to have or suspected of having heart disease, 3) loop monitoring in patients with frequent events and normal hearts, 4) psychiatric evaluation in patients with frequent events and no injury, and 5) tilt-table testing in patients who have infrequent events or in whom vasovagal syncope is suspected. Hospitalization is indicated for high-risk patients, especially those with known heart disease and elderly patients.

**Conclusions:** A flexible, focused approach is required to diagnose syncope. Features of the initial history and physical examination help guide diagnostic testing.

In the first part of this two-part study [1], the differential diagnosis of syncope was examined with respect to the information provided by results of the history, physical examination, and electrocardiography; an algorithmic approach to the diagnosis of syncope was also introduced. A careful history and physical examination are mandatory in all patients with syncope because they are the keys to determining whether additional diagnostic testing is required. Electrocardiography is recommended for almost all patients with syncope, whereas specialized neurologic testing is suggested only in certain circumstances: for example, computed tomography for patients with focal neurologic signs, electroencephalography for patients with seizure activity, or carotid or transcranial Doppler ultrasonography for patients with carotid bruits or a history of neurovascular symptoms. This paper addresses the workup of patients with syncope that is unexplained by the results of history, physical examination, or surface 12-lead electrocardiography.

## **Unexplained Syncope**

Syncope that remains unexplained after initial clinical assessment is of considerable concern to the practicing clinician. The algorithm that we developed provides three branches for unexplained syncope: one for patients known to have or suspected of having heart disease, one for elderly patients, and one for patients not known to have or suspected of having heart disease (<u>Figure 1</u>).

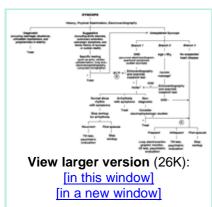


Figure 1. Algorithm for diagnosing syncope. \*
Carotid massage can be performed in an office setting only in the absence of bruits, a history of ventricular tachycardia, recent stroke, or recent myocardial infarction. Carotid hypersensitivity should be diagnosed only if clinical history is suggestive and massage is diagnostically positive (asystole ≥3 seconds, hypertension, or both). †May be replaced by inpatient telemetry if there is concern about serious arrhythmia. OHD = organic heart disease.

## Branch 1: Unexplained Syncope with Clinical Organic Heart Disease or Abnormal Electrocardiogram

Organic heart disease is often known, discovered, or suspected in patients who have sudden or exertional syncope. Evaluation of patients known to have or suspected of having heart disease often begins with echocardiography or an exercise stress test to determine and quantify the degree of heart disease. If the results of these tests are negative, further cardiac testing can often be avoided. If the results are positive, however, subsequent testing may include Holter monitoring or telemetry, signal-averaged electrocardiography, and intracardiac electrophysiologic studies.

#### **Echocardiography**

No studies have been specifically designed to assess the usefulness of echocardiography in syncope. In patients known to have or suspected of having heart disease, patients suspected of having arrhythmias, or patients with abnormal electrocardiograms, echocardiography is an important initial step in diagnostic testing. Unsuspected findings on echocardiography are reported in only 5% to 10% of unselected patients [2]. This yield is similar to that of 12-lead electrocardiography, but echocardiography is 7 times more expensive. The cost-effectiveness of echocardiography in diagnosing the cause of syncope has yet to be determined.

#### **Exercise Testing**

Exercise stress testing can be used for the evaluation of exertional syncope to diagnose ischemia or exercise-induced tachyarrhythmias or to reproduce exercise-associated or postexertional syncope. In one population study of patients with syncope, the yield of the exercise stress test was less than 1% [3]. No data are available to determine the yield for ischemia or exercise-induced tachyarrhythmias or to define the test's usefulness in diagnosing exercise-associated syncope. Tilt-table testing has been used to diagnose neurally mediated syncope, which may manifest as postexertional syncope [4, 5].

Exercise stress testing is recommended if patients have exercise-associated syncope and if the results of clinical evaluation suggest ischemic heart disease. In patients with exertional syncope, echocardiography should be done first to exclude hypertrophic cardiomyopathy.

#### 24-Hour Holter Monitoring

We summarize the results of ambulatory monitoring in syncope by determining the presence or absence of arrhythmias in patients who develop symptoms during monitoring [6]. In studies that evaluated syncope or presyncope with 12 or more hours of monitoring and reported on symptoms, 4% of patients had correlation of symptoms with arrhythmias (Table 1) [7-14]. In about 15% of patients, symptoms were not associated with arrhythmias; this finding excluded rhythm disturbance as a cause for syncope in these patients (overall diagnostic yield in 8 studies, 4% + 15% = 19%). No symptoms occurred in approximately 79% of patients, but arrhythmias were found in 14% [7-14]. The causal relation between most of these arrhythmias and syncope is uncertain, although certain uncommon asymptomatic arrhythmias (prolonged sinus pauses, Mobitz type II block, and sustained ventricular tachycardia during sleep) usually prompt appropriate treatment. If no

arrhythmias are found and no symptoms occur during monitoring, arrhythmic syncope is not necessarily excluded; this is because of the episodic nature of arrhythmias. In patients with a high pretest likelihood of arrhythmias (for example, patients who have brief loss of consciousness with short or absent prodrome, an abnormal electrocardiogram, or organic heart disease), further evaluation for arrhythmias should be pursued by event monitoring or electrophysiologic studies.

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## Table 1. Yield of Prolonged Electrocardiographic (Holter) Monitoring in Syncope

Only one study evaluated the effect of duration of monitoring on diagnostic yield [7]. Extending monitoring to 72 hours increased the number of arrhythmias detected (14.7% on the first day, an additional 11.1% the second day, and an additional 4.2% the third day) but not the yield for arrhythmias associated with symptoms.

A 24-hour Holter monitor or inpatient telemetry is recommended when symptoms suggest arrhythmic syncope (brief loss of consciousness, no prodrome, palpitations with syncope) and in patients who have syncope of unexplained cause, heart disease, or an abnormal electrocardiogram. Loop monitoring may be a reasonable alternative in patients with recurrent syncope and a normal heart.

#### **Intracardiac Electrophysiologic Studies**

Although they are relatively safe in patients with syncope [15], electrophysiologic studies are expensive and invasive. Such studies are associated with low risks for pulmonary embolism, cardiac perforation, arteriovenous fistulae, and myocardial infarction (cumulative risk < 3%) [16]. Electrophysiologic studies use electric stimulation and monitoring to discover conduction abnormalities that predispose patients to bradyarrhythmias and to determine a patient's propensity for developing tachyarrhythmias (both ventricular and supraventricular). Most protocols for programmed stimulation include three extrastimuli at one or two ventricular sites. More aggressive protocols, including the use of isoproterenol, may increase the sensitivity but decrease the specificity of tests for detecting tachyarrhythmias.

The most important outcome of electrophysiologic testing is the diagnosis of ventricular tachycardia. Other potentially important diagnostic outcomes include supraventricular tachycardias and bradyarrhythmias. Because only a few studies have used 24-hour Holter monitoring to confirm results of electrophysiologic studies [17, 18], the true diagnostic yield of this testing is generally unknown. Nevertheless, it is agreed that the results of an electrophysiologic test are considered positive if the test uncovers any of the following: 1) sustained monomorphic ventricular tachycardia [not including polymorphic ventricular tachycardia or ventricular fibrillation, which may be nonspecific responses], 2) a prolonged corrected sinus node recovery time longer than 1000 milliseconds, 3) markedly prolonged HV intervals longer than 90 milliseconds, 4) spontaneous or induced infra-Hisian block, and 5) supraventricular tachycardia with hypotension.

For the accompanying analysis, we used the above definitions wherever possible, excluding supraventricular tachycardias (which are relatively uncommon outcomes of electrophysiologic testing in syncope and can be diagnosed by other means). Our primary purpose was to classify study results to determine predictors of positive results on electrophysiologic studies. Key predictors that we assessed were presence of organic heart disease and brady-arrhythmic abnormalities (such as conduction-system disease) found on 12-lead electrocardiography.

Fourteen studies evaluating 1423 patients provided information on electrophysiologic outcomes but had insufficient detail to assess the importance of organic heart disease and baseline electrocardiography [19-32]. Heart disease was present in slightly more than half of the patients. Ventricular tachycardia was diagnosed in 14%, whereas a bradycardic outcome was observed in 21%. Because some patients (about

10%) had both tachycardic and bradycardic outcomes, the overall diagnostic yield in these studies (in which a high prevalence of patients had organic heart disease) was approximately 32% [14% ventricular tachycardia + 21% bradycardias –10% x (14 + 21)].

Table 2 summarizes eight additional studies in which the contribution of organic heart disease to a positive test result could be assessed [15, 17, 18, 33-37]. In these studies, 625 patients underwent electrophysiologic testing for syncope. Of the 406 patients with organic heart disease or an abnormal electrocardiogram, 21% had ventricular tachycardia and 34% had a bradycardia during the electrophysiologic study. Of the 219 patients with normal hearts, only 1% had ventricular tachycardia and 10% had a documented bradycardia (*P* < 0.001 for both comparisons). In these studies, approximately 14% of patients who could be given a diagnosis had both ventricular tachycardia and bradycardia. Thus, the diagnostic yield of electrophysiologic studies was almost 50% in patients with organic heart disease and about 10% in patients with a normal heart.

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Table 2. Diagnostic Yield of Intracardiac Electrophysiologic Studies in Syncope: Importance of Organic Heart Disease\*

These data are further elucidated by <u>Table 3</u>, which describes six referral studies of electrophysiologic testing in syncope [38-43]. In this table, the independent contributions of organic heart disease and electrocardiographic abnormalities are evaluated in predicting outcome. Most of the electrocardiographic abnormalities were evidence of serious conduction system disease (bundle-branch block, first-degree heart block, or sinus bradycardia). A few patients had nonspecific ST-segment and T-wave findings, left ventricular hypertrophy, or atrial and ventricular ectopy. Of 213 patients, 126 had organic heart disease; of the patients with organic heart disease, 19% had ventricular tachycardia and 17% had a bradycardia discovered during the electrophysiologic study. In the subgroup with conduction system abnormalities (n = 36), only 3% of patients had ventricular tachycardia but 19% received a diagnosis of bradycardia. Finally, of the 51 patients with normal hearts and normal electrocardiograms, 4% (n = 2) had ventricular tachycardia and 10% (n = 5) had bradycardia. None of the patients in these studies had multiple diagnoses. Although these differences are significant for ventricular tachycardia (P < 0.005), the differences in the prevalence of bradycardias among the three groups did not reach statistical significance (P > 0.2), perhaps because of small sample sizes.

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Table 3. Electrophysiologic Studies in Syncope: Importance of Electrocardiographic Abnormalities

These data show that positive results on electrophysiologic examinations occur predominantly in patients who have organic heart disease. Evidence of bradycardia is more likely to be found on electrophysiologic testing in patients who have conduction disease on their electrocardiograms; however, the yield of electrophysiologic studies in detecting bradyarrhythmias is limited [23]. Most important is that meaningful abnormal outcomes are rarely discovered during electrophysiologic testing in patients with clinically normal hearts and normal electrocardiograms.

Patients who have clinically normal hearts and normal electrocardiograms should rarely undergo electrophysiologic testing. Patients with syncope and serious organic heart disease, especially those who have had myocardial infarctions or congestive heart failure [26, 29, 31], and patients with preexcitation should be hospitalized and should probably undergo electrophysiologic studies. Patients with clinically normal hearts in whom conduction system disease (for example, first-degree heart block, sinus pauses, or bundle-branch block) is found on electrocardiography or Holter monitoring can be studied electrophysiologically or by loop monitoring. Elderly patients with conduction disease who are at high risk for morbid events (such as hip fracture) during syncope may be better evaluated initially with electrophysiologic studies than with noninvasive testing. These recommendations are concordant with the recent guidelines by the American College of Cardiology/American Heart Association Task Force [44]. Patients suspected of having arrhythmic syncope should not drive during their evaluation.

#### **Signal-Averaged Electrocardiography**

Low-amplitude signals (late potentials) are detected by signal-averaged electrocardiography. Three referral studies [45-47] show that this test has a sensitivity of 73% to 89% and a specificity of 89% to 100% for the prediction of inducible ventricular tachycardia in patients with syncope. However, no studies of the usefulness of signal-averaged electrocardiography have been done in unselected patients with unexplained syncope. It is also unclear whether it is safe to avoid electrophysiologic studies in patients with negative signal-averaged electrocardiography results and significant organic heart disease (such as depressed left ventricular function or previous myocardial infarction) [48].

Signal-averaged electrocardiography may be useful in selecting patients for electrophysiologic studies when coronary disease is present and ventricular tachycardia is suspected. This role should be ascertained in prospective studies of unselected patients with syncope before the use of this test is routinely recommended.

#### **Branch 2: Unexplained Syncope in Elderly Patients**

#### Causes

Syncope in elderly persons is associated with many daily situations (situational syncope). Syncope in association with micturition, defecation, postural changes, and meals was found in 20% of institutionalized elderly patients (mean age, 87 years) [49-52]. Other provocative situations include coughing, laughing, and swallowing. Postprandial hypotension can result in syncope during or after a meal [53]. Orthostatic hypotension is also common in elderly persons, particularly when it is caused by medications that may result in symptoms even if standard therapeutic doses are administered. In a study that compared community-dwelling elderly persons (mean age, 71 years) with young persons (mean age, 39 years), arrhythmias were diagnosed in 28% of the elderly persons and only 13% of the young persons [54]. Several entities, including aortic stenosis, myocardial infarction, transient ischemic attack, and carotid sinus syncope, were primarily found in the elderly persons [54].

#### **Diagnostic Testing**

In one population study of syncope in elderly persons (mean age, 71 years), a history and physical examination led to 40% of the diagnoses that could be assigned [54]. Furthermore, a diagnosis was suggested in an additional 15% of patients according to the results of a history and physical examination and was confirmed by specific tests, such as echocardiography or cardiac catheterization. Electrocardiography led to a diagnosis in 9% of elderly patients and 4% of younger patients.

Five recent referral studies of carotid sinus massage in syncope [55-59] show that carotid massage has its greatest clinical utility in elderly patients (mean age, 60 to 81 years). The test appears to be safe if it is done in the office in patients who do not have carotid bruits, recent myocardial infarction, recent stroke, or a history of ventricular tachycardia (incidence of neurologic complications < 0.2%) [60]. Intravenous access during the maneuver is not necessary. Patients who have cardioinhibitory hypersensitivity of the carotid sinus (that is, asystolic arrest lasting 3 seconds) were effectively treated by the implantation of an artificial pacemaker. The yield in these studies of referral-based populations was remarkably high (average yield for positive carotid massage, 46%). However, because the positive predictive value of carotid massage remains undefined (and may decline with age), a clinician who finds a sensitive carotid sinus should still consider

other prognostically important causes of syncope, depending on the nature of the episodes of syncope and the presence of comorbid conditions.

Arrhythmias were diagnosed by monitoring more frequently in elderly patients than in younger patients. In elderly patients, diagnosing heart disease may require such noninvasive diagnostic tests as stress tests or echocardiography. Referral studies of upright tilt-table testing in elderly patients with syncope (mean age ≥60 years) show positive responses to tilt in 54% of these patients (range, 26% to 90%) [61]. The rate of positive responses to tilt (number of positive test results divided by total number of patients tested) in elderly controls without syncope was 11% (range, 0% to 100%) [61].

In elderly patients, an inability to compensate for common situational stresses in the setting of several medical problems, medications, and physiologic impairments may combine to cause syncope [62]. If a single cause is not identified but many potential processes are found, management should be directed toward correcting these factors.

## Branch 3: Unexplained Syncope in Patients Not Known To Have or Suspected of Having Heart Disease

In patients not known to have or suspected of having heart disease and syncope, primary diagnostic tests include long-term ambulatory loop electrocardiography, tilt-table testing, and psychiatric evaluation. Considerations in making the decision to perform Holter monitoring, loop monitoring, or tilt-table testing are presented in Table 4.

View this table: [in this window] [in a new window] Table 4. Indications for 24-Hour Holter Monitoring, Long-Term Ambulatory Loop Electrocardiography, and Tilt-Table Testing

#### Long-term Ambulatory Loop Electrocardiography

Loop electrocardiographic monitoring can be done for 30 days or more. A loop recorder is a type of event monitor. Whereas some event monitors may be carried in a pocket and applied to the chest at the moment symptoms occur, loop monitors use two chest electrocardiographic leads that are continuously worn and connected to a small (beeper-sized) monitor. The monitor constantly records and erases the cardiac rhythm. Loop monitors can be activated after syncope by pressing a button that freezes in memory the previous 2 to 5 minutes and the subsequent 60 seconds of heart rhythm; the tracing can then be transmitted by telephone. Loop recorders are thus preferable to other types of event monitors because they can capture "retrospective" rhythm.

Three referral studies have evaluated loop monitoring in syncope, near syncope, dizziness, and palpitations [63-65]. The duration of monitoring ranged from 1 day to 4.5 months. True-positive test results (arrhythmia detected during syncope) were relatively frequent, occurring in 8% to 20% of patients. True negative results (normal cardiac rhythm during syncope) occurred in 12% to 27% of patients. The diagnostic yield (true-positive plus true-negative results) varied from 24% to 47%; the highest yield was seen in patients with palpitations.

Loop monitoring was most effective in patients with recurrent events (median number of events in two studies, 15 and 30 events/patient) [63, 64]. Because patients must comply with using the device (putting it on each morning, pushing a button after the episode of syncope, and transmitting the rhythm over the telephone), difficulties caused by human error limited diagnostic efficacy in as many as 32% of patients.

Loop recording is a noninvasive method of cardiac monitoring that requires a compliant patient. It is most beneficial in patients with frequent episodes of syncope. Loop monitoring is often deferred in favor of electrophysiologic studies in patients with serious organic heart disease who are at a high risk for fatal ventricular arrhythmias.

#### **Head-up Tilt-Table Testing**

In 25 studies of tilt-table testing in syncope, we assessed the most widely used procedures: passive tilt without pharmacologic stimulation [59, 66-73] and isoproterenol infusion after passive tilt [33, 58, 61, 74-85]. Studies that used other provocative or protective agents [86-88] were not reviewed because of limited data.

Methods. Most protocols for tilt-table testing use footboard support. During passive protocols and after baseline measurements of blood pressure and continuous monitoring of heart rate while patients are supine, patients are suddenly brought semiupright. Most studies used a tilt angle of 60 degrees. Fitzpatrick and colleagues [67] currently recommend that patients be kept in this position for 45 minutes; this period is two SDs from the mean time that is required to produce a positive response (approximately 24 minutes) [67].

All testing protocols that incorporate isoproterenol also include a passive phase of testing that usually lasts 10 to 15 minutes; in this phase, the patient is tilted upright without receiving intravenous medication. If an end point (syncope or hypotension) is not reached during tilting, the patient is brought to the supine position and isoproterenol infusion is started at 1 micro g/min. The patient is then retilted and isoproterenol infusion is continued. If an end point is still not reached, the patient is again brought to the supine position, the infusion rate is increased, and the patient is retilted. This procedure is continued with increasing doses of isoproterenol until a positive response or another end point (such as maximum dose, adverse effects, or development of severe tachycardia) is reached. The maximum dose of isoproterenol is 3 to 5 micro g/min.

Sensitivity and specificity. The sensitivity of tilt-table testing in two small referral-based studies of patients who had clinical vasovagal syncope was 67% to 83% [89, 90]. Specificity has been evaluated by performing upright tilt-table testing in patients without previous syncope. With passive tilt-table testing, specificity has ranged between 0% and 100%, although an overall rate is approximately 90% [61]. As protocols with longer duration are used and the dose of isoproterenol is increased, specificity declines. The overall specificity of upright tilt-table testing with isoproterenol is approximately 75% (range, 35% to 100%). The specificity was lowest when isoproterenol was used in young patients.

Positive responses in patients with unexplained syncope. In studies that used passive tilting, 49% of 425 patients (range, 26% to 90%) had a positive response to tilt-table testing. In 806 patients studied with isoproterenol, however, positive responses were seen in 62% (range, 39% to 87%). Approximately two thirds of the positive responses occurred during the isoproterenol phase. When either type of testing was used, approximately two thirds of the responses seemed to be cardioinhibitory; the rest were pure vasodepressor hypotensive reactions. Greater tilt angles and longer durations of testing were associated with a greater rate of positive responses.

On the basis of a recently published analysis of pooled data [61] and the data discussed above, passive upright tilt-table testing at 60 degrees for 45 minutes is recommended in patients with unexplained recurrent syncope in whom cardiac causes of syncope, including arrhythmias, have been excluded. In patients with negative results on a passive tilt-table test who have a high pretest probability of neurally mediated syncope (for example, young patients with a prodrome of nausea or warmth), tilt-table testing with isoproterenol is recommended. The test results should be considered positive only if a patient's typical symptoms are reproduced. Many laboratories suggest that women of childbearing age should have a pregnancy test and that men older than 45 years of age and women older than 55 years of age have stress testing before tilt-table testing. Positive test results would preclude tilt-table testing.

#### **Psychiatric Evaluation**

Aside from psychological issues that would predispose a patient to vasovagal syncope [91], psychiatric disorders as causes of syncope were previously considered to be uncommon [92-95]. In 1989, a high prevalence of psychiatric disorders (24%), especially anxiety and depressive disorders, was shown in a referral study of patients with syncope [96]. Syncope had been unexplained in many of the patients, and a large proportion of the patients who received treatment for their psychiatric disorder showed a marked diminution in syncope. More recently, a population-based study [97] showed a high prevalence of psychiatric

disorders (35%) in unselected patients with syncope. The most common disorders were generalized anxiety disorder (8.6%), panic disorder (4.3%), and major depression (12.2%). Alcohol dependence was found in 9.2% of patients. Patients with psychiatric disorders were younger, generally did not have underlying heart disease, and had more frequent syncope. At a 1-year follow-up examination, patients with psychiatric illnesses were found to have a higher rate of recurrence of syncope than were those with other causes of syncope [97].

As Hackel and associates [69] and Grubb and colleagues [98] have shown, such psychiatric disorders as conversion disorders can be reproduced with a psychosomatic response to tilt-table testing (apparent syncope with normal vital signs). Thus, the manifestations of at least three types of psychiatric disorders, beyond the vasovagal response, can include syncope: anxiety, depression, and conversion disorders.

Two referral studies examined the usefulness of a hyperventilation maneuver in syncope [99, 100]. These studies showed a significant correlation between a positive maneuver (open-mouthed hyperventilation for 2 to 3 minutes, resulting in near syncope or true syncope) and psychiatric causes of syncope. This was particularly useful in young patients (positive predictive value, 59%) [100].

Psychiatric disorders as potential causes of syncope should be sought in young patients who faint frequently, patients in whom syncope does not cause injury, and patients who present with many symptoms (for example, nausea, lightheadedness, numbness, and fear or dread) [96, 97]. The hyperventilation maneuver and screening instruments for common mental disorders are recommended [101].

#### **Miscellaneous Tests**

Lung ventilation-perfusion scanning should be reserved for patients in whom pulmonary embolism is likely (for example, patients who have recently had surgery or patients who have syncope with dyspnea or chest pain, abnormal arterial blood gases, or signs of pulmonary hypertension on physical examination). However, the overall prevalence of pulmonary embolism as a cause of syncope is low. Glucose tolerance testing is rarely indicated [102] because hypoglycemia is an uncommon cause of syncope (prevalence < 1%).

## **Special Issues in Evaluating Patients with Syncope**

#### **Risk Stratification**

Patients with syncope are admitted to the hospital primarily for observation and prevention of the consequences of a more serious subsequent episode or to rule out myocardial infarction or a new stroke. In the United States, the degree of variation in rates of hospitalization for syncope is high [103]. Eagle and colleagues' early research on prognostic classification of patients who present to the emergency department with syncope [95] began to address the issue, but the conclusion was that patients with cardiac syncope (which could not always be diagnosed on presentation) were at the highest risk for dying within 1 to 6 months.

The situation is clearest for patients who are at risk for myocardial infarction or malignant arrhythmias. On the basis of data from six population-based series of patients with syncope who presented to emergency departments (Table 5), we suggest that patients with syncope be hospitalized if they have evidence of organic heart disease, chest pain, or a history of arrhythmias or if they take medications that are associated with malignant arrhythmias [54, 94, 95, 104-106]. In patients who have myocardial infarction presenting as syncope, the electrocardiogram is usually abnormal [105]. This was seen in a study of 251 patients with syncope who presented to emergency departments, 18 of whom had acute cardiac ischemia and an abnormal electrocardiogram (sensitivity of electrocardiography, 100%; lower limit of 95% CI, 78%). Trauma caused by syncope has not been uniformly shown to correlate with prognostic seriousness [95]. Patients with syncope and neurologic symptoms that suggest a transient ischemic attack or stroke should be hospitalized [104].

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Table 5. Indications for Hospital Admission in Patients with Syncope\*

Elderly patients are often hospitalized for syncope, especially when it is serious or of recent onset. Elderly patients with situational syncope (such as syncope with micturition) may not require hospitalization. Research in this area would be valuable. Patients should also be hospitalized if rare causes of syncope (such as pericardial tamponade or pulmonary embolism) that would be imminently dangerous are suspected.

### **Driving**

The occurrence of syncope during driving could have serious consequences for the patient and other persons who might be harmed by the patient's vehicle. The physician and patient should carefully consider the risks when deciding whether the patient should continue to drive while syncope is being evaluated. State laws vary with respect to the physician's and the patient's responsibilities for reporting medical conditions that affect the ability to drive. Physicians should be aware of the pertinent laws in their own state.

### **Charges for Diagnostic Studies**

Table 6 shows current charges for tests that are used to diagnose syncope. The most efficient test is the combination of history and physical examination. In elderly patients, carotid sinus massage is inexpensive but may be nonspecific. Twelve-lead electrocardiography is recommended for most patients with syncope. Loop electrocardiography and psychiatric evaluations are appropriate for patients who have normal hearts. Echocardiography may be useful in detecting occult cardiac abnormalities, but it is moderately expensive. Most of the other tests are comparatively expensive (>\$400) and should be used only when specifically indicated.

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View this table: Table 6. Summary of Charges for Diagnostic Tests in Syncope\*

#### **Future Research**

To improve our knowledge of the efficacy of diagnostic tests in syncope, future studies should have strong study designs and should focus on such topics as implantable loop recorders [107], testing protocols that provide a rapid diagnosis, risk stratification, and the cost-effectiveness of echocardiography [108].

## **Conclusions**

The prognosis in syncope ranges from benign to life endangering. Risk stratification should be based on the results of history, physical examination, electrocardiography, and selected noninvasive tests.

Electrophysiologic studies should be reserved for high-risk patients who have organic heart disease. Patients at low risk who nonetheless have frequent episodes of syncope may have serious impairments in quality of life [109, 110]; an aggressive (although noninvasive) approach may be warranted in such patients. An individualized approach is required to diagnose syncope, but the core of the syncope workup remains a detailed history and physical examination.

## **Appendix**

The following are members of the Clinical Efficacy Assessment Subcommittee of the Health and Public Policy Committee of the American College of Physicians: George E. Thibault, MD, Chair, John R. Feussner, MD, Co-Chair, Anne-Marie J. Audet, MD; Gottlieb C. Friesinger Jr., MD; Daniel L. Kent, MD; Keith I. Marton, MD; Valerie Anne Palda, MD; John J. Whyte, MD; and Preston L. Winters, MD.

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Note: The Clinical Efficacy Assessment Project (CEAP) of the American College of Physicians is designed to evaluate and inform College members and others about the safety and efficacy of diagnostic and therapeutic methods.

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