

Evaluation and Management of Syncope

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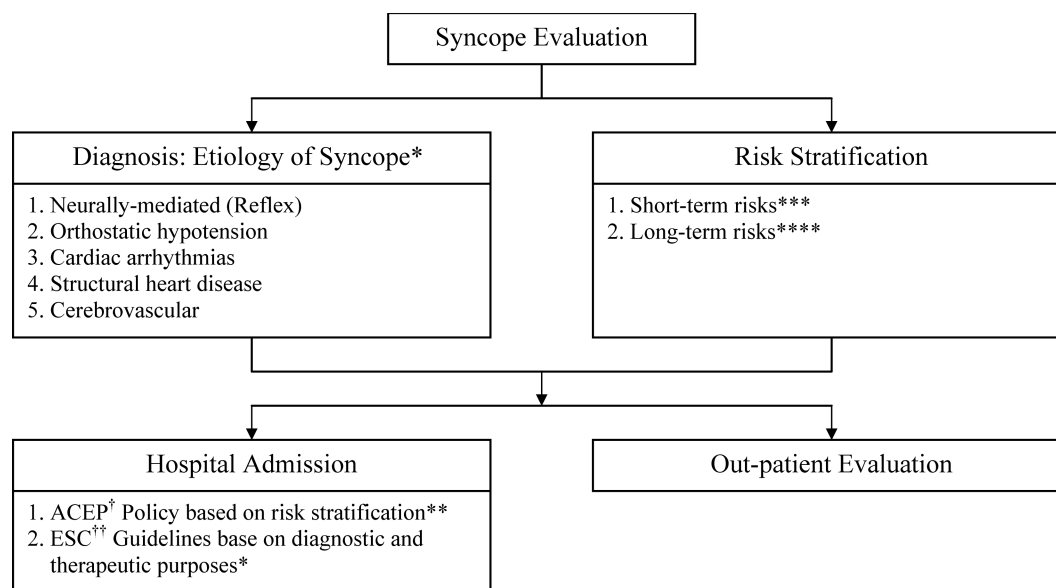
Syncope is a transient loss of consciousness precipitated by cerebral hypoperfusion, which is associated with a brief absence of postural tone and usually followed by a complete recovery. This clinical condition is a common medical problem and may be attributed to a multitude of disease processes. Risk stratification identifies the safest setting for the initial evaluation as well as which patients are most likely to have a life-threatening event. Establishing the diagnosis of syncope is important so that specific treatment can be instituted to prevent future recurrences and eliminate the underlying predisposing disease. This article reviews the etiology, risk stratification, diagnosis, and therapeutic management of syncope.

Keywords: syncope; cerebral hypoperfusion; cardiac syncope; risk stratification; syncope evaluation; driving guidelines

Syncope is a transient loss of consciousness (TLOC) precipitated by cerebral hypoperfusion, which is associated with the absence of postural tone and usually followed by a complete recovery within a few minutes (Brignole et al., 2004; Soteriades et al., 2002). This clinical condition is a common medical problem with an estimated incidence of 6.2 per 1,000 person-years and accounts for 1% of emergency department (ED) visits and 6% of all hospital admissions (Grossman et al., 2007; Soteriades et al., 2002). Syncope may be due to a multitude of disease processes, and the etiology of syncope may remain unknown in a large percentage of patients (Brignole et al., 2004; Disertori et al., 2003). Establishing the diagnosis of syncope is important so that specific treatment can be instituted to prevent future recurrences and eliminate the underlying predisposing disease (Ammirati, Colivicchi, & Santini, 2000; Brignole & Shen, 2008; Soteriades et al., 2002). Because of the sporadic and infrequent nature of syncopal events in a given patient, establishing a correct diagnosis remains the major challenge in managing these patients (Brignole et al., 2004; Kapoor, 2002; Strickberger et al., 2006).

Syncope Evaluation

The initial consideration in evaluating syncope is to differentiate true syncope from nonsyncopal events such as psychogenic pseudosyncope, seizures, metabolic disorders (hypoxia or hypoglycemia), and intoxications (Brignole et al., 2004; Kapoor, 2002). In psychogenic pseudosyncope, the patient has underlying psychogenic factors and manifests symptoms of conversion reaction with transient abnormal response without loss of consciousness. The diagnosis should be considered if the patient presents with a prolonged abnormal response (10–30 minutes), frequent episodes (up to several times a day), and lack of physical injury (Wieling, Ganzeboom, & Saul, 2004). Seizures, metabolic disorders, and intoxications may cause TLOC but not on the basis of cerebral hypoperfusion; seizure disorders are the most common nonsyncopal TLOC (Strickberger et al., 2006). There are two main reasons to evaluate patients with syncope: to determine the etiology of syncope and to stratify the risk of future adverse outcomes (Figure 1) (Brignole et al., 2004; Colivicchi et al., 2003; Costantino et al., 2008; Disertori et al., 2003; Quinn, McDermott, Stiell, Kohn, & Wells, 2004; Sun



* Brignole, Alboni et al., 2004
 ** Huff et al., 2007
 *** Grossman et al., 2007; Costantino et al., 2008; Quinn et al., 2004
 **** Colivicchi et al., 2003; Costantino et al., 2008; Martin et al., 1997
 † American College of Emergency Physicians
 ** European Society of Cardiology

Figure 1. Syncope evaluation.

et al., 2007). The result of this initial evaluation will determine further diagnostic or therapeutic strategies and the need for hospital admission or outpatient testing (Brignole et al., 2004; Huff et al., 2007).

Etiology of Syncope

According to the European Society of Cardiology (ESC), syncope may be classified into five major categories: neurally mediated, orthostatic, cardiac arrhythmia related, structural heart disease related, and cerebrovascular syncope (Table 1) (Brignole et al., 2004).

Neurally Mediated (Reflex) Syncope

Neurally mediated syncope (NMS) is caused by a reflex response with vasodilatation and bradycardia contributing to systemic hypotension and/or cerebral hypoperfusion. The classical vasovagal syncope, carotid sinus syncope, and situational syncope are included in this category. Vasovagal syncope is precipitated by emotions, unpleasant sights or sounds, pain, or orthostatic stress (prolonged standing in crowded or hot places) and is typically associated with postepisode fatigue, weakness, nausea, or vomiting (Alboni et al., 2001; Brignole et al., 2004; Strickberger et al., 2006). Carotid sinus syncope is related to accidental mechanical manipulation of the carotid sinuses, such as neck turning,

shaving, or tight collars (Kapoor, 2002; Strickberger et al., 2006). Situational syncope refers to those forms of NMS associated with specific scenarios, such as micturition, coughing, or defecating.

Orthostatic Syncope

Orthostatic syncope refers to syncope in which the upright position causes hypotension and cerebral hypoperfusion without bradycardia (Brady & Davis, 2003; Brignole et al., 2004). This type of syncope usually occurs after standing up, with exertion or associated with prolonged standing in crowded or hot places. Orthostatic hypotension occurs when the autonomic nervous system response to changes in position is faulty or if the patient is hypovolemic. Several causes of orthostatic hypotension include medications, neurogenic causes such as multisystem atrophy (MSA), Parkinsonism and diabetic neuropathy, and nonneurogenic causes such as impaired venous return, hypovolemia, and cardiac insufficiency (Brady & Davis, 2003; Brignole et al., 2004). MSA is a sporadic neurodegenerative disorder characterized by a combination of Parkinsonian, autonomic, cerebellar, or pyramidal signs and symptoms and may present with orthostatic hypotension as a result of autonomic failure (Colosimo, Tiple, & Wenning, 2005).

TABLE 1. Classification and Etiology of Syncope

1. Neurally mediated (reflex) syncope
• Vasovagal syncope
• Carotid sinus syncope
• Situational syncope
2. Orthostatic syncope
• Autonomic failure
• Drug-induced orthostatic hypotension
• Volume depletion
3. Cardiac arrhythmia-related syncope
• Sinus node dysfunction (bradycardia/tachycardia syndrome)
• Atrioventricular conduction system disease
• Paroxysmal supraventricular and ventricular tachycardias
• Wolff-Parkinson-White syndrome
• Inherited syndromes (Long QT syndrome, Brugada syndrome)
• Drug-induced proarrhythmias
4. Structural heart disease-related syncope
• Obstructive cardiac valvular disease
• Cardiomyopathy
• Atrial myxoma
• Coronary artery disease
5. Cerebrovascular syncope
• Vascular steal syndromes
• Vertebrobasilar artery disease
• Carotid artery disease

Cardiac Arrhythmia-Related Syncope

Cardiac arrhythmias may precipitate syncope because bradycardia or tachycardia causes a decrease in cardiac output regardless of circulatory demands (Brignole et al., 2004). The potential causes of syncope in this category are sinus node dysfunction, atrioventricular (AV) conduction abnormalities, paroxysmal supraventricular (SVT) and ventricular tachycardias (VT), Wolff-Parkinson-White (WPW) syndrome, and inherited syndromes, such as long QT syndrome (LQTS) or Brugada syndrome (Brignole et al., 2004; Strickberger et al., 2006).

Structural Heart Disease-Related Syncope

Structural heart diseases (SHD) such as aortic stenosis, obstructive cardiomyopathy, pulmonary hypertension, or atrial myxomas can precipitate syncope because circulatory demands outweigh the impaired ability of the heart to increase cardiac output (Brignole et al., 2004; Strickberger et al., 2006). Pulmonary embolism is also included in this category. Coronary artery disease (CAD)

and cardiomyopathy are other structural heart diseases that may cause syncope by predisposing patients to paroxysmal VT.

Cerebrovascular Syncope

Cerebrovascular syncope is also referred to as neurological syncope and occurs as a result of decreased cerebral perfusion associated with cerebrovascular disease. The steal syndrome occurs in subclavian obstruction when preferential blood flow is diverted from the brain to the arm during arm activity (Brignole et al., 2004). Patients with severe vertebrobasilar or bilateral carotid artery disease may experience syncope associated with focal neurological symptoms (Strickberger et al., 2006). The physical examination in these patients may reveal carotid bruits or weak or absent brachial or radial pulses. While uncommon (1%), this type of syncope should be entertained as a possible cause if suggested by history or physical findings (Alboni et al., 2001; Strickberger et al., 2006).

Risk Stratification of Syncope

Short-Term Risk (Table 2)

There are only a few studies that directly evaluate the short- and long-term risk of syncope (Colivicchi et al., 2003; Disertori et al., 2003; Quinn et al., 2004; Sun et al., 2007). The San Francisco syncope rule (SFSR) may offer some guidance in predicting which patients are likely to have short-term (7-day) serious outcomes and to guide hospital admission decisions (Quinn et al., 2004). The initial derivation study enrolled 684 patients with syncope and near syncope (Quinn et al., 2004). Patients with syncope are at 25% risk for serious outcomes if they present with one of these five clinical conditions: congestive heart failure (CHF), hematocrit <30%, electrocardiogram (ECG) abnormalities (nonsinus rhythm or new changes), shortness of breath, or systolic blood pressure <90 mm Hg. The CHESS acronym may assist in remembering these conditions. Serious outcomes include death, myocardial infarction, cardiac arrhythmias, pulmonary embolism, stroke, subarachnoid hemorrhage, significant hemorrhage, ED revisit, or hospital admission. Quinn et al. (2004) and Quinn, McDermott, Stiell, Kohn, and Wells (2006) reported a high sensitivity (96% and 98%) and moderate specificity (62% and 56%) of SFSR in both initial derivation and validation studies. However, an independent validation of the SFSR demonstrated a lower sensitivity (89%) and specificity (42%), suggesting that the rule has limited generalizability (Sun et al., 2007).

TABLE 2. Short-Term Risk Stratification of Syncope

San Francisco Syncope Rule (SFSR): ^a Predictors of short-term outcomes (7-day): CHES	Boston Syncope Criteria (BSC): ^b Predictors of short-term outcomes (30-day)	Short-Term Prognosis of Syncope (STePS): ^c Predictors of short-term outcomes (10-day) by multivariate analysis
Congestive heart failure Hematocrit <30% ECG abnormalities Shortness of breath Systolic blood pressure <90 mm Hg	History of ACS Suspicious cardiac history Abnormal cardiac examination Evidence of conduction disease) Family history of SCD Persistent abnormal vital signs Volume depletion (persistent dehydration, gastrointestinal bleeding or hematocrit <30)	Abnormal ECG Concomitant trauma Absence of prodromal symptoms Male gender
Outcomes (25% overall incidence with at least one risk factor) Death Myocardial infarction Arrhythmia Pulmonary embolism Stroke Subarachnoid hemorrhage Significant hemorrhage Readmission to emergency department or hospital admission	Primary outcomes (23% overall incidence) Intervention: Antiarrhythmic treatment Pacemaker/ICD placement Myocardial revascularization Cardiopulmonary resuscitation (CPR) Blood transfusion Endoscopy with intervention Correction of carotid stenosis Outcomes: Death Myocardial infarction Arrhythmia Pulmonary embolism Stroke Subarachnoid hemorrhage Significant hemorrhage Cardiac arrest Infection/sepsis Life-threatening sequelae (rhabdomyolysis, long bone or cervical spine fractures)	Outcomes (6.1% overall incidence) Death (0.7%) Major therapeutic procedures (5.4%) (CPR, pacemaker or ICD insertion, ICU admission) Early hospital readmission

^aQuinn et al. (2004). ^bGrossman et al. (2007). ^cCostantino et al. (2008).

The Boston Syncope Criteria (BSC) is a clinical strategy, which was developed to determine the prognosis of syncope based on the SFSR and the American College of Emergency Physicians' (ACEP) clinical policy (Disertori et al., 2003; Huff et al., 2007; Quinn et al., 2004). The BSC consists of eight categories that could affect primary outcomes over 30 days, consisting of a critical intervention or an adverse outcome. The incidence of primary outcomes in the original study of 384 patients was 23%, and the BSC demonstrated 97% sensitivity and 62% specificity in identifying these patients (Disertori et al., 2003). Clinical application of the BSC led to a 48% reduction in hospital admissions.

The Short-Term Prognosis of Syncope (STePS) study assessed severe outcomes of syncope in 676 patients, including death, major therapeutic procedures, and early hospital readmission (Costantino et al., 2008). The major therapeutic procedures were defined as cardiopulmonary arrest, pacemaker or implantable cardioverter defibrillator

(ICD) insertion, and intensive care unit admission. The overall incidence of severe short-term (10-day) outcomes was 6.1%, including 0.7% for mortality and 5.4% for major therapeutic procedures. The multivariate analysis identified abnormal ECGs, concomitant trauma, absence of symptoms of impending syncope, and male gender as predictors of short-term risk for serious outcomes (Costantino et al., 2008).

Long-Term Risk (Table 3)

Martin, Hanusa, and Kapoor (1997) developed a long-term risk stratification system for cardiac arrhythmia and 1-year mortality, including abnormal ECG, a history of ventricular arrhythmia or CHF, and age >5 years. Events ranged from 0% for those without risk factors to 27% for those with three or four risk factors. The OESIL (Osservatorio Epidemiologico della Sincope nel Lazio) reported age >5 years, lack of prodromes, history of cardiovascular

TABLE 3. Long-Term Risk Stratification of Syncope

Risk stratification of patients with syncope: ^a Risk factors Abnormal ECG History of ventricular arrhythmia History of congestive heart failure Age >45 years	Osservatorio Epidemiologico della Sincope nel Lazio (OESIL): ^b Risk factors Age >65 years History of cardiovascular disease Lack of prodromes Abnormal ECG	STePS: ^c Predictors of long-term outcomes (1-year) by multivariate analysis Ventricular arrhythmias Age >65 years Neoplasms Cerebrovascular disease Structural heart disease
Outcomes: Arrhythmia or 1-year mortality 0% incidence for no risk factors 27% incidence for three to four risk factors	Outcomes: 1-year mortality 0% incidence for no risk factor 0.8% incidence for one risk factor 19.6% incidence for two risk factors 34.7% incidence for three risk factors 57.1% incidence for four risk factors	Outcomes (9.3% overall incidence) Death (6%) Major therapeutic procedures (3.3%): CPR, pacemaker or ICD insertion, and ICU admission

^aMartin et al. (1997). ^bColivicchi et al. (2003). ^cCostantino et al. (2008).

disease, and abnormal ECGs as predictors of 1-year mortality (0%, 0.8%, 19.6%, 34.7%, and 57.1% incidence for zero, one, two, three, and four risk factors, respectively) (Colivicchi et al., 2008). STePS also assessed long-term (1-year) serious outcomes (overall incidence of 9.3%) of syncope and found age >5 years, ventricular arrhythmias, SHD, cerebrovascular disease, and neoplasm to be predictors of 1-year mortality (6.0%) and major therapeutic procedures (3.3%) (Costantino et al., 2008).

There is no optimal risk stratification to date, and the discrepancies in prior studies make it difficult to compare their clinical applicability. The BSC, which included more clinical predictors and adverse outcomes, reported a similar sensitivity and specificity to the initial study of SFSR, but its results have not been validated (Disertori et al., 2003). A lower specificity and sensitivity for the SFSR in one independent validation and a low positive predictive value of the STePS indicate the need for additional studies before they can be recommended as a standard of care for assessing the patient with syncope (Brignole & Shen, 2008; Costantino et al., 2008; Sun et al., 2007).

The risk of death and other adverse outcomes of syncope appears to be related to the underlying disease or patient's general risks rather than the syncope itself (Brignole & Shen, 2008; Colivicchi et al., 2003; Costantino et al., 2008; Martin et al., 1997). Patients with cardiac syncope have higher rates of sudden cardiac death (SCD) and all-cause mortality than those without a cardiac cause (Soteriades et al., 2002; Strickberger et al., 2006). The 5-year mortality rate in patients with cardiac syncope has been reported to approach 50%, with a 30% incidence of death in the first year (Soteriades et al., 2002; Strickberger et al., 2006). Based on mortality risk, the causes of syncope may be divided into cardiac, noncardiac, or unknown (Soteriades et al., 2002). SHD is a well-established major

risk for cardiac syncope and should be the primary focus of the initial syncope evaluation (Alboni et al., 2001; Chen et al., 2000).

Hospital Admission

Patient disposition after the initial evaluation is an important aspect of the management of syncope. Currently, there are two guidelines that can help with decisions in regard to hospital admission (Brignole et al., 2004; Huff et al., 2007). The ACEP developed a policy for hospital admission based on risk stratification (Huff et al., 2007). Patients with older age and comorbidities, an abnormal ECG, Hct <0%, and history or presence of CHF, ischemia, or other SHD are at high risk of having adverse outcomes and should be admitted to the hospital. The ESC provides another guideline that is based on the need for diagnostic or therapeutic interventions (Brignole et al., 2004). Hospital admission for diagnostic evaluation is recommended for patients with suspected or known significant SHD, an abnormal ECG suggestive of arrhythmic syncope, syncope occurring during exercise or supine position, syncope causing severe injury (skull or bone fractures, intracranial hemorrhage or internal organ injuries), family history of SCD, or suspected device malfunction (Brignole et al., 2004). Therapeutic indications for admission according to the ESC include syncope due to cardiac arrhythmias, ischemia, SHD, or cardiopulmonary disease or neurally mediated bradycardia requiring pacemaker implantation (Brignole et al., 2004).

Syncope management units (SMUs) were recently developed to improve patient care (Brignole et al., 2004; Brignole, Ungar, et al., 2006; Shen et al., 2004). This multidisciplinary unit is equipped with cardiac monitoring and patients have immediate access to echocardiogram,

tilt-table testing, and cardiology or neurology consultations. Patients are admitted directly to this SMU from the ED and receive their initial care for 6 hours before they are discharged for outpatient testing or admitted to the hospital for further evaluation (Shen et al., 2004). The Syncope Evaluation in the Emergency Department Study identified 103 intermediate-risk patients with syncope according to the AECOP clinical policy and randomized them to receive standard care or evaluation in an SMU (Huff et al., 2007; Shen et al., 2004). The SMU evaluation in this study increased presumptive diagnosis and actuarial survival and reduced hospital admission and length of hospital stay (Shen et al., 2004). A standardized-care pathway that incorporated Web-based interactive ESC guideline software was used in a different study to evaluate 745 patients with syncope in the ED (EGSYS-2) (Brignole et al., 2004; Brignole, Ungar, et al., 2006). Use of the standardized care pathway decreased hospital admission and length of hospital stay and improved diagnosis at a lower overall cost (Brignole, Ungar, et al., 2006).

Diagnosis of Syncope

Both American Heart Association/American College of Cardiology Foundation (AHA/ACCF) and ESC have established guidelines for the evaluation of syncope (Brignole et al., 2004; Strickberger et al., 2006). Other studies have also presented various diagnostic approaches (Ammirati et al., 2000; Kapoor, 2002; Shen et al., 2004). Based on these guidelines and prior studies, an algorithm for the evaluation of syncope is suggested (Figure 2) (Ammirati et al., 2000; Brignole et al., 2004; Kapoor, 2002; Shen et al., 2004; Strickberger et al., 2006). The initial clinical evaluation of a patient with syncope includes a careful history and a physical examination including supine and upright blood pressure measurements and ECG (Alboni et al., 2001; Ammirati et al., 2000; Brignole et al., 2004; Kapoor, 2002; Strickberger et al., 2006). Assessing the medication list is necessary to exclude agents that may precipitate orthostatic or arrhythmia related syncope (Strickberger et al., 2006). The patient's age is important information because the causes of syncope are highly

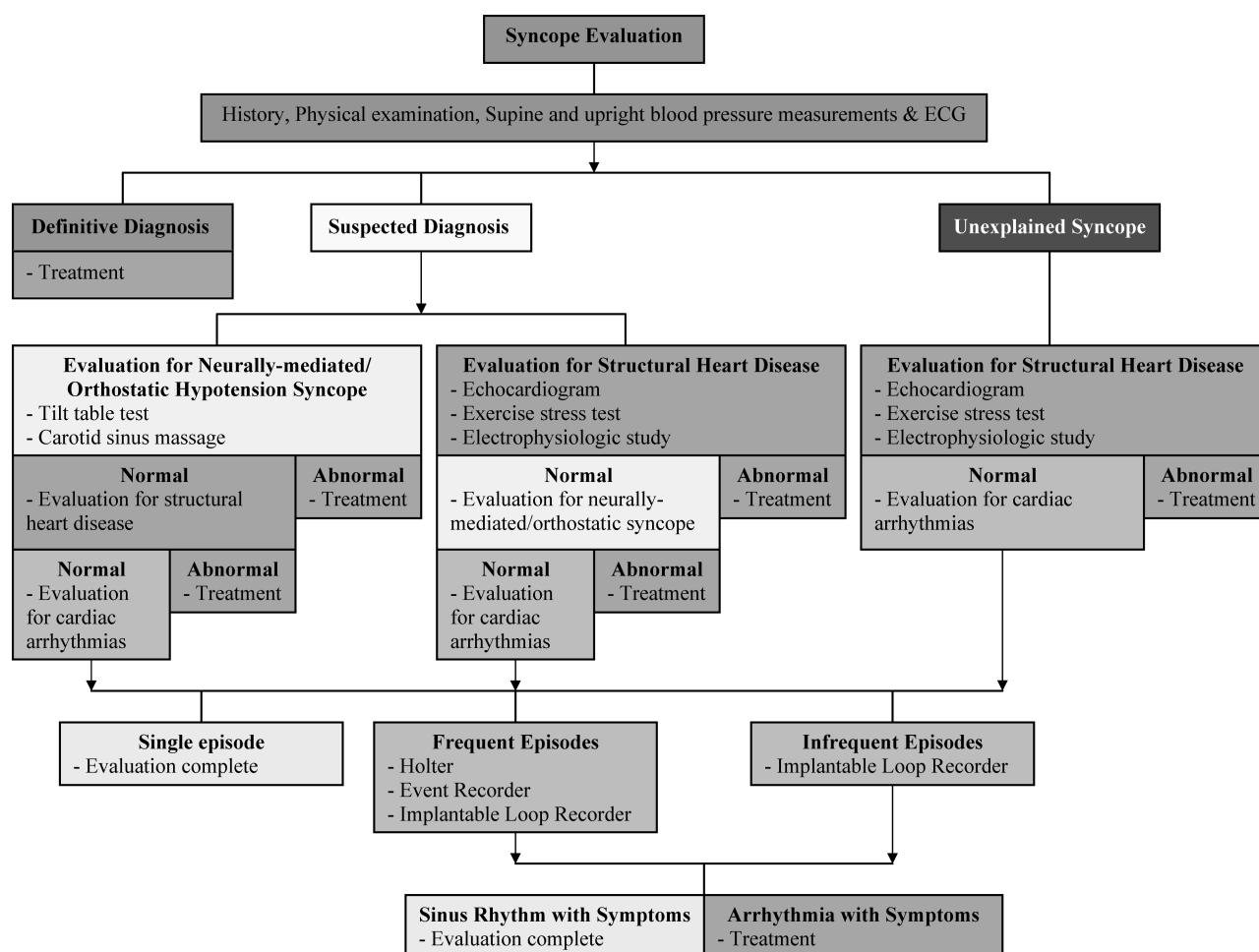


Figure 2. Diagnostic strategies for syncope.

age dependent (Strickberger et al., 2006). Pediatric and young patients are more likely to have NMS and cardiac arrhythmia-related syncope such as the LQTS or WPW syndrome. NMS is the most frequent cause of syncope in middle-aged patients. Elderly patients commonly have a higher incidence of cardiac syncope and may also experience situational NMS and orthostatic syncope.

A pertinent clinical history includes prodromal symptoms, precipitating events, prior cardiac history, family history of SCD, and the description of the event by any witness (Brignole et al., 2004; Strickberger et al., 2006). The diagnosis of NMS or orthostatic syncope is determined largely by the description of the precipitating event, clinical presentation, and characteristics of syncope (Brignole et al., 2004; Strickberger et al., 2006). Chest pain, shortness of breath, palpitations, and syncope in a supine position or during exercise is indicative of possible cardiac-related syncope (Alboni et al., 2001; Kapoor, 2002; Strickberger et al., 2006).

Physical examination may detect underlying cardiovascular disease, such as irregular pulses, heart murmurs, carotid bruits, or pulse differential (Strickberger et al., 2006). Additionally, the severity of syncope may be assessed by the evidence of physical injury and classified as benign with minor injury and malignant with severe injury (Strickberger et al., 2006). Blood pressure measurement in both arms and in supine and upright positions will help establish the diagnosis of subclavian obstruction or orthostatic hypotension. A blood pressure difference of 20 mm Hg between both arms is suggestive of subclavian obstruction (Lobato, Kern, Bauder-Heit, Hughes, & Sulek, 2001). Orthostatic hypotension is defined as a blood pressure drop of at least 20/10 mm Hg or more with a change in position from supine to standing (Brady & Davis, 2003). Blood pressure should be measured 5 minutes after the patient resumes a supine position and 3 minutes after the patient stands up (Kapoor, 2002). An abnormal ECG is helpful in establishing the presence of cardiac arrhythmia or SHD as the cause of the syncope. The ECG may demonstrate abnormal AV conduction, bundle branch block or abnormal Q waves, or ST-T changes consistent with ischemic cardiac disease, accessory pathways, LQTS, or Brugada syndrome (Brignole et al., 2004; Strickberger et al., 2006).

Diagnostic Strategies

After the initial evaluation patients may be categorized into three groups: patients with a definitive diagnosis, suspected diagnosis, and unexplained syncope (Figure 2)

(Brignole et al., 2004). The diagnosis of syncope is established after the initial evaluation in 50% of all patients and treatment strategies may be initiated (Brignole et al., 2004). In one study, the NMS accounts for up to two-thirds of the initial and subsequent diagnosis of syncope followed by cardiac related and orthostatic syncope (Brignole et al., 2004).

Patients with a suspected diagnosis will need specific testing to confirm or rule out the diagnosis (Brignole et al., 2004; Kapoor, 2002). Patients with possible NMS or orthostatic syncope are recommended to have a tilt-table test or carotid sinus massage (CSM) (Brignole et al., 2004; Strickberger et al., 2006). The long interval between episodes of NMS may contribute to a normal conventional testing (tilt-table test and CSM), and a negative test result does not necessary exclude the diagnosis (Alboni et al., 2001; Brignole, Sutton, et al., 2006; Maisel, 2004). Early application of an internal loop recorder (ILR) was recently found to establish more diagnoses with suspected NMS than conventional testing (Brignole, Sutton, et al., 2006). Patients with suspected cardiac syncope will require an echocardiogram and exercise stress testing (Brignole et al., 2004; Strickberger et al., 2006). Cardiac arrhythmias are the major causes of syncope in patients with SHD and prolonged cardiac monitoring or electrophysiologic (EP) studies may be necessary to uncover the plausible cardiac arrhythmia (Kapoor, 2002; Strickberger et al., 2006). If the initial work-up for suspected syncope is normal, reevaluation of the patients and testing results may warrant additional assessment for NMS or cardiac syncope or specialty consultation (Brignole et al., 2004; Kapoor, 2002).

Because of a high incidence of mortality associated with cardiac syncope, the first step in evaluating recurrent unexplained syncope is to exclude any underlying SHD and to make sure that patients do not have unrecognized cardiac syncope (Kapoor, 2002). An echocardiogram and stress test are recommended for elderly patients without clinical evidence of SHD and for patients with a history of exercise induced syncope (Strickberger et al., 2006). The testing strategies in patients with unexplained syncope and normal ECGs and cardiac examination depend on the severity and frequency of syncope (Brignole et al., 2004; Strickberger et al., 2006). Patients with a first benign episode of syncope and without evidence of SHD have a high probability of having NMS, a low risk for cardiac syncope, and a good prognosis (Soteriades et al., 2002). These patients do not require additional work-up for cardiac arrhythmias (Brignole et al., 2004; Kapoor, 2002; Strickberger et al., 2006). Because of the risk of

physical injury and diminished quality of life associated with frequent syncope, patients with multiple unexplained episodes (≥ 2) warrant further work-up for arrhythmic syncope (Brignole & Shen, 2008; Strickberger et al., 2006). Holter, external loop recorder (ELR) or ILR are appropriate tests for patients with frequent syncope, whereas ILR is a better choice for patients with infrequent episodes (Strickberger et al., 2006). Documented normal sinus rhythm during the episode of syncope excludes arrhythmia-related syncope as well as NMS; these patients are not at risk of having serious outcomes, and they do not require additional cardiac testing (Strickberger et al., 2006). Those with recorded arrhythmias during symptoms will need appropriate treatment for either bradycardia or tachycardia (Boersma, Mont, Sionis, García, & Brugada, 2004; Moya et al., 2001; Zipes et al., 2006).

Diagnostic Testing

Carotid Sinus Massage

Carotid sinus massage is performed to evaluate patients with suspected carotid sinus hypersensitivity. This test may be performed at the bedside with patients in the supine or upright positions under continuous ECG and blood pressure monitoring (Miller & Kruse, 2005; Strickberger et al., 2006). CSM is performed one side at a time by applying firm massage for 5 to 10 seconds at the site of most appreciable carotid pulsation. Carotid sinus hypersensitivity is diagnosed when CSM causes a ≥ 3 -second pause, a ≥ 50 mm Hg fall in systolic blood pressure, or both, associated with presyncope or syncope (Miller & Kruse, 2005). In the absence of a response, CSM is repeated on the other side 1 minute later. CSM should not be performed in patients with a history of recent transient ischemic attack or stroke or on a carotid artery that has a significant bruit or known stenosis (Strickberger et al., 2006).

Tilt-Table Test

Tilt-table testing is also referred to as head-up tilt, which is performed in patients with suspected NMS or orthostatic syncope. Tilt-table testing promotes venous pooling in the lower extremities and provokes vasovagal response through the Bezold-Jarisch mechanism leading to bradycardia and hypotension in NMS (Hainsworth, 2003). Patients are secured to a tilt table, and blood pressure and heart rate are monitored every 2 minutes for 10 minutes. The patients are then tilted upward at angles between 60 and 80 degrees for 30 to 60 minutes with regular monitoring of clinical response, blood pressure, and heart rates (Miller & Kruse,

2005). Pharmacologic provocation with sublingual nitroglycerine, Isuprel, or adenosine triphosphate infusion is occasionally administered during this test (Brignole et al., 2004; Barón-Esquivias & Martínez-Rubio, 2003). Tilt-table testing is considered positive if the patient develops hypotension and marked bradycardia or asystole associated with symptoms similar to those of spontaneous NMS (Hainsworth, 2003). Symptomatic hypotension without bradycardia is indicative of orthostatic syncope.

Echocardiogram

The echocardiogram provides diagnostic and prognostic information on SHD that predisposes patients to syncope, including the assessment of cardiac size, left-ventricular function, wall motion, and valvular heart disease (Cheitlin et al., 2003; Strickberger et al., 2006). It has also become an established tool for diagnosing CAD and is the primary method for the diagnosis of aortic stenosis, congestive or hypertrophic cardiomyopathy, and atrial myxoma (Cheitlin et al., 2003).

Exercise Stress Test

Exercise stress testing in patients with syncope is performed to identify CAD and exercise-induced cardiac arrhythmias such as sinus node dysfunction, AV block, or tachycardias (Gibbons et al., 2002). Failure to increase systolic blood pressure 10 to 30 mm Hg during exercise stress testing may indicate significant SHD, such as left main stenosis or obstructive cardiomyopathy (Gibbons et al., 2002; McKenna, & Behr, 2002).

Cardiac Monitoring

In-hospital cardiac monitoring is warranted when the patient has evidence of SHD and is at high risk of a life-threatening arrhythmia (Brignole et al., 2004). Prolonged ECG monitoring is indicated if the initial evaluation shows a high probability of cardiac arrhythmia-related syncope or unexplained syncope with normal testing for NMS and without SHD (Brignole et al., 2004; Strickberger et al., 2006). The choice of outpatient cardiac monitoring is based on the frequency of syncope.

Holter

The Holter monitor is an external device that is used to monitor the ECG tracing continuously for a period of 24 hours or longer. The device is small and may be fastened to the patient's belt. It requires two or more external leads that attach to electrodes on the chest. The device is

returned to a diagnostic center for data analysis at the end of the recording session. Holter monitoring is indicated to capture an arrhythmia that is suspected to occur on a daily basis, and the overall diagnostic yield of syncope is low (8.6%) (Kühne, Schaer, Moulay, Sticherling, & Osswald, 2007; Strickberger et al., 2006). Holter monitoring may also reveal QT-interval changes, T-wave alternans, or ST changes (Strickberger et al., 2006; Zipes et al., 2006).

External Loop Recorder

The external loop recorder is also referred to as event recorder. It is an external device that allows prolonged continuous ambulatory ECG monitoring for up to 60 days (Gula et al., 2004). The ELR is a pagerlike device and may be fastened to the patient's belt. The device has both automatic and manual activation to freeze the memory. The stored cardiac rhythm during the event may be transmitted by a telephone to a diagnostic center for analysis. The ELR is used to evaluate sporadic syncopal episodes, which is suspected to occur once every 1 to 2 months, and the diagnostic yield may vary between 15% and 50% (Strickberger et al., 2006).

Implantable Loop Recorder

The implantable loop recorder is an internal monitoring device that is useful for the diagnosis of suspected arrhythmia-related syncope, particularly when the episodes are infrequent and conventional noninvasive cardiac testing is negative or inconclusive (Assar, Krahn, Klein, Yee, & Skanes, 2003; Brignole et al., 2004; Brignole, Sutton, et al., 2006; Krahn, Klein, Yee, Hoch, & Skanes, 2003; Moya et al., 2001; Zipes et al., 2006). This device is very small and is typically implanted subcutaneously in the left pectoral region as an outpatient procedure (Lombardi et al., 2005). The ILR has electrodes on the back of the device to detect patients' cardiac rhythm and does not require intracardiac leads. The unit is activated manually by the patient during a symptomatic event or by automatic preprogramming for a ventricular pause of >3 seconds, lower (<40 bpm) or high ventricular rate (>165 bpm) (Farwell, Freemantle, & Sulke, 2006; Moya et al., 2001; Strickberger et al., 2006). Data are retrieved with a programmer, and some devices also have the capability to allow patients to send data directly to their health care providers. More than 50% of patients experience symptoms during the 14-month duration of ILR monitoring, and the cause of syncope is established more often with ILR (47% vs. 20%) and at a lower cost per diagnosis than conventional testing, which includes ELR, tilt-table, and

EP testing (Boersma et al., 2004; Krahn et al., 2003; Lombardi et al., 2005).

Electrophysiologic Study

The electrophysiologic study is an invasive procedure that is recommended when cardiac arrhythmias are suspected to be the cause of syncope and noninvasive diagnostic studies are not conclusive (Strickberger et al., 2006). The EP testing is indicated in patients who have unexplained syncope in the presence of impaired left-ventricular function or SHD (Strickberger et al., 2006; Zipes et al., 2006). This study is also recommended in patients with high-risk occupations in whom every effort is necessary to exclude a cardiac cause of syncope (Brignole et al., 2004). The yield of EP studies depends on the presence of underlying SHD: both bradycardia (34% vs. 10%) and VT (21% vs. 1%) are more inducible in patients with than those without heart disease (Linzer et al., 1997). EP induction of polymorphic VT or ventricular fibrillation (VF) is predictive of syncopal events in patients with Brugada syndrome, survivors of SCD with CAD, and idiopathic VF, but it is less predictive in patients with dilated cardiomyopathy (Brignole et al., 2004; Brilakis et al., 2001; Brugada et al., 2003).

Treatment

Neurally Mediated Syncope

It is important to inform the patient that NMS is normally not life threatening but that injuries can occur if preventive measures are not appropriately taken (Benditt & Nguyen, 2009). Patient education is an important part of the treatment for NMS. The patient's understanding of the mechanism and warning symptoms may reduce injuries and increase treatment compliance (Benditt & Nguyen, 2009; Kapoor, 2002). The mechanism of NMS is heterogeneous; vasodilatation is the central part of this clinical entity, and severe bradycardia or asystole contributes to about one-half of the events (Brignole, Sutton, et al., 2006; Kapoor, 2003). Medical treatment for NMS includes avoiding dehydration, physical counterpressure maneuvers (PCM) at the onset of prodrome (lying down with their feet popped up, squatting, isometric hand gripping, arm tensing, and leg crossing), increasing in intravascular volume by oral or intravenous fluids and dietary salt, wearing support hose, and physical tilt training (Benditt & Nguyen, 2009; Brignole et al., 2004; Kapoor, 2002, 2003; Tan & Parry, 2008). Tilt training (standing training) promotes neurovascular tolerance to orthostatic stress; the recommended standing

duration is 3 to 5 minutes twice daily at first with a gradual increase in standing duration every 3 to 4 days, up to 30 to 40 minutes twice daily (Benditt & Nguyen, 2009; Kapoor, 2002). Some patients may require pharmacotherapy, such as volume expanders (fludocortisone), beta blockers, or vasoconstrictors and venoconstrictors (Medodrine) (Benditt & Nguyen, 2009; Kapoor, 2002). Randomized controlled trials have demonstrated no clear clinical benefit of these agents, and the patient should be warned of possible associated side effects of hypertension (dietary salt, fludocortisone, or Medodrine) or urinary retention or urgency (Medodrine) (Benditt & Nguyen, 2009). Permanent pacemaker insertion is effective only for asystole (Kapoor, 2002). The recommendation of a permanent pacemaker insertion for bradycardia-related NMS remains controversial, and additional studies are necessary before pacing can be considered a standard therapy (Brignole et al., 2004; Kapoor, 2002, 2003; Tan & Parry, 2008).

Orthostatic Syncope

The treatment of orthostatic syncope consists of education regarding aggravating factors for orthostatic syncope, nonpharmacologic and pharmacologic corrections of hypovolemia, and autonomic imbalance (Benditt & Nguyen, 2009; Brady & Davis, 2003). The nonpharmacologic approach focuses on making slow and careful changes in position, avoiding dehydration, increasing in intravascular volume, wearing support hose, and a routine exercise program (Benditt & Nguyen, 2009; Brady & Davis, 2003). The patient may also benefit from PCM, tilt training, and sleeping with the head of the bed elevated to 20 to 25 cm (Benditt & Nguyen, 2009). Pharmacotherapy with volume expanders or vasoconstrictors may be prescribed for severe symptoms of orthostasis (Benditt & Nguyen, 2009; Brady & Davis, 2003).

Cardiac Arrhythmia-Related Syncope

Transient bradycardia is frequently responsible for cardiac arrhythmia-related syncope and a cardiac pacemaker is required (Benditt & Nguyen, 2009; Boersma et al., 2004; Moya et al., 2001). Tachycardia accounts for the remainder of arrhythmia-related syncope, and treatment options include antiarrhythmic therapy, catheter ablation, pacemaker, or ICD insertion (Zipes et al., 2006).

SHD-Related Syncope

Patients with SHD may require either medical therapy, catheter-related procedures, or surgical correction for the treatment of underlying cardiac disease such as myocardial

revascularization or valvular surgery (Benditt & Nguyen, 2009; McKenna & Behr, 2002). If cardiac arrhythmias are the culprit for syncope in patients with SHD, patients will also need therapy for bradycardia or tachycardia in addition to the treatment for underlying SHD (Boersma et al., 2004; Moya et al., 2001; Zipes et al., 2006).

Cerebrovascular Syncope

The treatment of cerebrovascular syncope may be accomplished with surgical or percutaneous revascularization. Carotid endarterectomy remains the gold standard for hemodynamically significant carotid stenosis and angioplasty, and stenting is presently being evaluated as a treatment option for high-risk patients (Biggs & Moore, 2007). Both percutaneous and surgical options appear to be safe and reasonably durable for subclavian stenosis (Rogers & Calhoun, 2007). Primary stent placement may be the treatment of choice for vertebral arterial disease (Cloud & Marcus, 2003).

Driving

The AHA established driving guidelines related to arrhythmias that may affect consciousness that were later amended to include drivers with ICD insertion for primary prevention (Epstein et al., 1996, 2007). The ESC also suggested a guideline for driving for patients with syncope (Brignole et al., 2004). Two groups of drivers are defined: private and commercial (Brignole et al., 2004; Epstein et al., 2007). Drivers of taxicabs, small ambulances, and other vehicles form an intermediate category (Brignole et al., 2004). Data suggest that the risk for a motor vehicle accident related to syncope is low (Akiyama, Powell, Mitchell, Ehlert, & Baessler, 2001). The efficacy of drug therapy for NMS remains inconclusive, and repeat tilt-table testing to assess therapy has no predictive value (Brignole et al., 2004; Kapoor, 2002). There is no evidence that allowing 3 asymptomatic months to elapse provides assurance that syncope will not recur (Brignole et al., 2004). The ESC guidelines have shortened or eliminated the waiting duration of asymptomatic period for severe NMS, postpacemaker insertion, or VT treatment for both private and commercial drivers (Brignole et al., 2004; Epstein et al., 2007).

Driving recommendations should be prescribed in conjunction with the collaborating physician or cardiologist (Table 4). In general, there are no restrictions or minimal restrictions for private drivers who suffer from syncope with a low incidence of recurrence or a low probability of serious outcomes during a recurrent episode; these include benign

TABLE 4. Driving Guidelines

Types of Syncope		Driving Restriction After Treatment	
		Private Driver	Commercial Driver
Neurally mediated syncope ^a			
Vasovagal	Benign (single/mild)	No restriction	1 month
	Severe (frequent/high-risk activity)	3 months	6 months
Carotid sinus		1 month	1 month
Cardiac arrhythmia-related syncope ^a			
Supraventricular tachycardia		No restriction	No restriction
Ventricular tachycardia	Nonsustained	3 months	6 months
	Sustained	6 months	6 months
Pacemaker implant	Non-pacemaker dependent	1 week	1 week
	Pacemaker dependent	1 week	4 weeks
Internal cardioverter defibrillator	Prophylactic	1 week	Permanent
	Therapeutic	6 months	Permanent
Unexplained syncope ^b	After testing and treatment	No restriction	3 months

^aEpstein et al. (2007). ^bBrignole et al. (2004).

vasovagal syncope, carotid sinus syncope, SVT, postpace-maker or prophylactic ICD implantation, or unexplained syncope (Brignole et al., 2004; Epstein et al., 2007). A longer period of restriction is recommended for patients with a high likelihood of causing an accident during an event; these include severe vasovagal syncope, ventricular tachycardia, or posttherapeutic ICD insertion. The driving guidelines for commercial drivers are more restricted to ensure public safety (Brignole et al., 2004; Epstein et al., 2007).

Conclusion

A high percentage of syncope remains undiagnosed, and an important task in evaluating patients with syncope is to exclude underlying cardiac disease because those with cardiac syncope are at risk of having poor outcomes, including sudden cardiac death and all-cause mortality. Nurse practitioners need a good understanding of the complex mechanisms of syncope and should follow an organized approach in evaluating this common clinical condition. For nurse practitioners with limited experience in managing these patients, seeking collaborative advice or referring the patient to a specialist is recommended. It is essential to make a correct diagnosis so that appropriate treatment may be provided to eliminate the underlying disease and prevent recurrent syncopal episodes.

References

Akiyama, T., Powell, J., Mitchell, B., Ehlert, F., & Baessler, C., (2001). Resumption of driving after life-threatening

ventricular tachycardia. *New England Journal of Medicine*, 345, 391–397.

Alboni, P., Brignole, M., Menozzi, C., Raviele, A., Del Rosso, A., Dinelli, M., et al. (2001). Diagnostic value of history in patients with syncope with or without heart disease. *Journal of the American College of Cardiology*, 37, 1921–1928.

Ammirati, F., Colivicchi, F., & Santini, M. (2000). Diagnosing syncope in clinical practice: Implementation of a simplified diagnostic algorithm in a multicentre prospective trial—The OESIL 2 Study (Osservatorio Epidemiologico della Sincope nel Lazio). *European Heart Journal*, 21, 935–940.

Assar, M., Krahn, A., Klein, G., Yee, R., & Skanes, A. (2003). Optimal duration of monitoring in patients with unexplained syncope. *American Journal of Cardiology*, 92, 1231–1233.

Barón-Esquivias, G., & Martínez-Rubio, A. (2003). Tilt table test: State of the art. *Indian Pacing Electrophysiology Journal*, 3, 239–252.

Benditt, D., & Nguyen, J. (2009). Syncope: Therapeutic approaches. *Journal of the American College of Cardiology*, 19, 1743–1751

Biggs, K., & Moore, W. (2007). Current trends in managing carotid artery disease. *Surgical Clinics of North America*, 87, 995–1016.

Boersma, L., Mont, L., Sionis, A., García, E., & Brugada, J. (2004). Value of the implantable loop recorder for the management of patients with unexplained syncope. *Europace*, 6, 70–76.

Brady, J., & Davis, K. (2003). Orthostatic hypotension. *Am Family Physician*, 68, 2394–2398.

Brignole, M., Alboni, P., Benditt, D., Bergfeldt, L., Blanc, J., Bloch Thomsen, P. et al. (2004). Guidelines on management

- (diagnosis and treatment) of syncope—Update 2004. *Europace*, 6, 467–537.
- Brignole, M., & Shen, W. (2008). Syncope management from emergency department to hospital. *Journal of the American College of Cardiology*, 51, 284–287.
- Brignole, M., Sutton, R., Menozzi, C., Garcia-Civera, R., Moya, A., Wieling, W., et al. (2006). Early application of an implantable loop recorder allows effective specific therapy in patients with recurrent suspected neurally mediated syncope. *European Heart Journal*, 27, 1085–1092.
- Brignole, M., Ungar, A., Bartoletti, A., Ponassi, I., Lagi, A., Mussi, C., et al. (2006). Standardized-care pathway vs. usual management of syncope patients presenting as emergencies at general hospitals *Europace*, 8, 644–650.
- Brilakis, E., Shen, W., Hammill, S., Hodge, D., Lexvold, N., & Friedman, P. (2001). Role of programmed ventricular stimulation and implantable cardioverter defibrillators in patients with idiopathic dilated cardiomyopathy and syncope. *Pacing and Clinical Electrophysiology*, 24, 1623–1630.
- Brugada, P., Brugada, R., Mont, L., Rivero, M., Geelen, P., & Brugada, J. (2003). Natural history of Brugada syndrome: The prognostic value of programmed electrical stimulation of the heart. *Journal of Cardiovascular Electrophysiology*, 14, 458–460.
- Cheitlin, M., Armstrong, W., Aurigemma, G., Beller, G., Bierman, F., Davis, J., et al. (2003). ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography. *Journal of the American College of Cardiology*, 42, 954–970.
- Chen L., Chen M., Larson M., Evans J., Benjamin E., & Levy, D. (2000). Risk factors for syncope in a community-based sample (the Framingham Heart Study). *American Journal of Cardiology*, 85, 1189–1193.
- Cloud, J., & Marcus, H., (2003). Diagnosis and management of vertebral artery stenosis. *Quarterly Journal of Medicine*, 96, 27–54.
- Colivicchi, F., Ammirati, F., Melina, D., Guido, V., Imperoli, G., & Santini, M. (2003). Development and prospective validation of a risk stratification system for patients with syncope in the emergency department: The OESIL risk score. *European Heart Journal*, 24, 811–819.
- Colosimo, C., Tiple, D., & Wenning, K. (2005). Management of multiple system atrophy: State of the art. *Journal of Neural Transmission*, 112, 1695–1704.
- Costantino, G., Perego, F., Dipaola, F., Borella, M., Galli, A., Cantoni, G., et al. (2008). On behalf of the STePS investigators: Short- and long-term prognosis of syncope, risk factors, and role of hospital admission results from the STePS (Short-Term Prognosis of Syncope) study. *Journal of the American College of Cardiology*, 51, 276–283.
- Disertori, M., Brignole, M., Menozzi, C., Raviele, A., Rizzon, P., Santini, M., et al. (2003). Management of patients with syncope referred urgently to general hospitals. *Europace*, 5, 283–291.
- Epstein, A., Baessler, C., Curtis, A., Estes, M., III, Gersh, B., Grubb, B., et al. (2007). Addendum to “Personal and public safety issues related to arrhythmias that may affect consciousness: Implications for regulation and physician recommendations.” *Circulation*, 115, 1170–1176.
- Epstein, A., Miles, W., Benditt, D., Camm, A., Darling, E., Friedman, P., et al. (1996). Personal and public safety issues related to arrhythmias that may affect consciousness: Implications for regulation and physician recommendations. *Circulation*, 94, 1147–1166.
- Farwell, D., Freemantle, N., & Sulke, N. (2006). The clinical impact of implantable loop recorders in patient with syncope. *European Heart Journal*, 27, 351–356.
- Gibbons, R., Balady, G., Bricker, J., Chaitman, B., Fletcher, G., Froelicher, V., et al. (2002). 2ACC/AHA 2002 guideline update for exercise testing. *Circulation*, 106, 1883–1892.
- Grossman, S., Fischer, C., Lipsitz, L., Mottley, L., Sands, K., Thompson, S., et al. (2007). Predicting adverse outcomes in syncope. *Journal of Emergency Medicine*, 33, 233–239.
- Gula, L., Krahn, A., Massel, D., Skanes, A., Yee, R., & Klein, G. (2004). External loop recorders: Determinants of diagnostic yield in patients with syncope. *American Heart Journal*, 147, 644–648.
- Hainsworth, R. (2003). Syncope: What is the trigger? *Heart*, 83, 123–124.
- Huff, J., Decker, W., Quinn, J., Perron, A., Napoli, A., Peeters, S., et al. (2007). Clinical policy: Critical issues in the evaluation and management of adult patients presenting to the emergency department with syncope. *Annals of Emergency Medicine*, 49, 431–444.
- Kapoor, W. (2002). Current evaluation and management of syncope. *Circulation*, 106, 1606–1609.
- Kapoor, W. (2003). Is there an effective treatment for neurally mediated syncope? *Journal of the American Medical Association*, 289, 2272–2275.
- Krahn, A., Klein, G., Yee, R., Hoch, J., & Skanes, A. (2003). Cost implications of testing strategy in patients with syncope: Randomized assessment of syncope trial. *Journal of the American College of Cardiology*, 42, 495–501.
- Kühne, M., Schaer, B., Moulay, N., Sticherling, C., & Osswald, S. (2007). Holter monitoring for syncope: Diagnostic yield in different patient groups and impact on device implantation. *QJM*, 100, 771–777.
- Linzer, M., Yang, E., Estes, N., III, Wang, P., Vorperian, V., & Kapoor, W. (1997). Diagnosing syncope, part 1: Value of history, physical examination, and electrocardiography: Clinical Efficacy Assessment Project for the American College of Physicians. *Annals of Internal Medicine*, 126, 989–996.
- Lobato, E., Kern, K., Bauder-Heit, J., Hughes, L., & Sulek, C. (2001). Incidence of coronary-subclavian steal syndrome in patients undergoing noncardiac surgery. *Journal of Cardiothoracic and Vascular Anesthesia*, 25, 689–692.
- Lombardi, F., Calosso, E., Mascioli, G., Marangoni, E., Donato, A., Rossi, S., et al. (2005). Utility of implantable loop

- recorder (Reveal Plus®) in the diagnosis of unexplained syncope. *Europace*, 7, 19–24.
- Maisel, H., (2004). Specialized syncope evaluation. *Circulation*, 110, 3621–3623.
- Martin, T., Hanusa B., & Kapoor W. (1997). Risk stratification of patients with syncope. *Annals of Emergency Medicine*, 29, 459–466.
- McKenna, W., & Behr, E. (2002). Hypertrophic cardiomyopathy: Management, risk stratification, and prevention of sudden death. *Heart*, 87, 169–176.
- Miller, T., & Kruse, J. (2005). Evaluation of syncope. *American Family Physician*, 72, 1492–1500.
- Moya, A., Brignole, M., Menozzi, C., Garcia-Civera, R., Tognarini, S., Mont, L., et al. (2001). Mechanism of syncope in patients with isolated syncope and in patients with tilt-positive syncope. *Circulation*, 104, 1261–1267.
- Quinn, J., McDermott, D., Stiell, I., Kohn, M., & Wells, G. (2004). Derivation of the San Francisco Syncope Rule to predict patients with short-term serious outcomes. *Annals of Emergency Medicine*, 43, 224–232.
- Quinn, J., McDermott, D., Stiell, I., Kohn, M., & Wells, G. (2006). Prospective validation of the San Francisco Syncope Rule to predict patients with serious outcomes. *Annals of Emergency Medicine*, 47, 448–454.
- Rogers, J., & Calhoun, R. (2007). Diagnosis and management of subclavian artery stenosis prior to coronary artery bypass grafting in the current era. *Journal of Cardiac Surgery*, 22, 20–25.
- Shen, W., Decker, W., Smars, P., Goyal, D., Walker, A., Hodge, D., et al. (2004). Syncope Evaluation in the Emergency Department Study (SEEDS): A multidisciplinary approach to syncope management. *Circulation*, 101, 3636–3645.
- Soteriades, E., Evans, J., Larson, M., Chen, M., Chen, L., Benjamin, E., et al. (2002). Incidence and prognosis of syncope. *New England Journal of Medicine*, 347, 878–885.
- Strickberger, S., Benson, D., Biaggioni, I., Callans, D., Cohen, M., Ellenbogen, K., et al. (2006). AHA/ACCF scientific statement on the evaluation of syncope. *Journal of the American College of Cardiology*, 47, 474–484.
- Sun, B., Mangione, C., Merchant, G., Weiss, T., Shlamovitz, G., Zargaraff, G., et al. (2007). External validation of the San Francisco Syncope Rule. *Annals of Emergency Medicine*, 49, 420–427.
- Tan, M., & Parry, S. (2008). Vasovagal syncope in older patients. *Journal of the American College of Cardiology*, 51, 599–607.
- Wieling, W., Ganzeboom, K., & Saul, J. (2004). Reflex syncope in children and adolescents. *Heart*, 90, 1094–1100.
- Zipes, D., Camm, A., Borggrefe, M., Buxton, A., Chaitman, B., & Fromer, M. (2006). ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Journal of the American College of Cardiology*, 48, 1064–1108.

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