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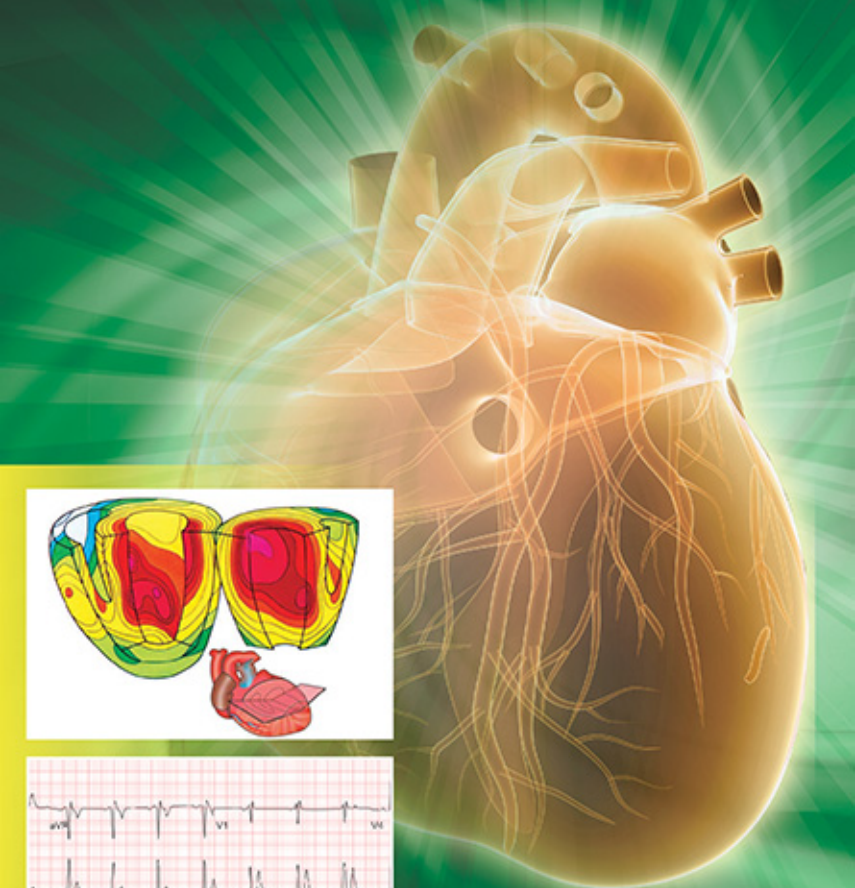
Manual of
ELECTROPHYSIOLOGY



Kanu Chatterjee
Mark Anderson
Donald Heistad
Richard E Kerber

JAYPEE

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CHAPTER

4

Syncope

Vijay Ramu, Fred Kusumoto, Nora Goldschlager

Chapter Outline

- Epidemiology
  - Incidence and Prevalence of Syncope
  - Economic Burden of Syncope
  - Causes and Classification of Syncope
- Diagnostic Tests
  - History and Physical Examination
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- Guidelines

INTRODUCTION

Syncope is a sudden and transient loss of consciousness associated with loss of postural tone, followed by complete and spontaneous recovery. The term "syncope" originates from the Greek word "Synkoptein" which means—cutting short (koptein- "to cut"). In the first six centuries, many Greek philosophers and physicians speculated on the causes of syncope. Claudius Galen, a famous Greek physician, suggested that syncope was a problem of both the stomach and the heart.[1] The mechanism for transient loss of consciousness associated with syncope is cerebral hypoperfusion with reduced blood flow to the reticular activating system. A common phrase used in clinical medicine is presyncope which is considered to represent a warning or prodrome for frank syncope. In the case of presyncope, symptoms, such as dizziness and graying out, are not followed by frank loss of consciousness. Many physicians evaluate and treat presyncope in a similar manner to syncope; although a reasonable approach, there is no strong clinical data to support similar etiologies and outcomes.

It is important to acknowledge that the definition of syncope (and thus etiologic classification) varies even among experts. Using a more general definition of transient loss of consciousness, some experts include neurologic (e.g. seizure and concussion), metabolic (e.g. hypoxia) and psychiatric conditions as forms of syncope, while others who emphasize cerebral hypoperfusion consider syncope as one of the several causes of transient loss of consciousness and classify some

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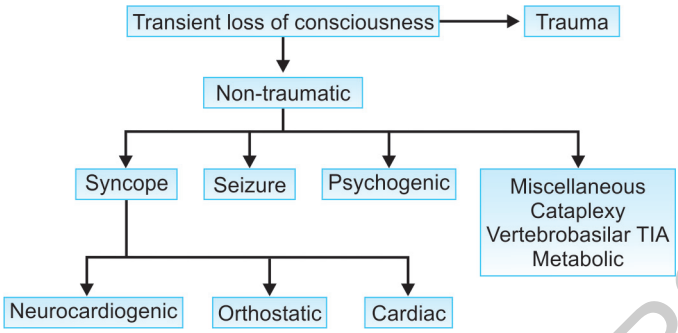
FLOWCHART 1: Classification of mechanistic causes for transient loss of consciousness and syncope



neurologic, metabolic and psychiatric mechanisms as separate entities **(Flowchart 1)**.[2,3] Using this more restrictive definition of syncope, the three most important causes of transient loss of consciousness are: (i) syncope; (ii) seizure, and (iii) psychogenic blackouts. Other rare causes include metabolic disorders, such as hypoglycemia or hypoxia, intoxication, and psychiatric problems such as cataplexy or pseudosyncope. Determining the correct cause of syncope is the key to approaching therapy, if the initial working clinical diagnosis is erroneous, subsequent investigations and even the final diagnosis and treatment may also be incorrect.[4] Regardless of definition, syncope may represent a harbinger for sudden death, and often the diagnostic evaluation focuses on identifying or ruling out potential life-threatening causes of syncope, such as ventricular arrhythmias or aortic stenosis. For this reason, the diagnostic workup for the patient with syncope revolves around two different but related issues: (i) identification of the specific mechanism for syncope and (ii) risk stratification to estimate short-term and long-term risk of adverse outcomes. It is important for the clinician to remember that the diagnostic workup of syncope requires a patient-specific approach and diagnostic tests must carefully be chosen. Several medical societies, including the European Society of Cardiology (ESC) and the American Heart Association/American College of Cardiology Foundation (AHA/ACCF) have provided comprehensive guidelines or scientific statements for the diagnosis and management of syncope, although some aspects are not without controversy.[2-5]

EPIDEMIOLOGY

Incidence and Prevalence of Syncope

Several studies have attempted to evaluate the incidence and prevalence of syncope. Obtaining accurate figures is difficult,

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since it has been estimated that only 25–50% of syncopal episodes are reported to medical professionals and only 2–5% of episodes are evaluated in emergency department settings.[2] In the most recent report from the Framingham study, the incidence of a first report of syncope was 6.2/1000 person-years follow-up and a ten-year cumulative incidence of 6%.[6] In addition, a sharp increase in the incidence of syncope after age 70 years, particularly in the presence of cardiovascular disease, was reported. Several studies suggest that syncope is quite common in younger populations.[7–9] For example, in a cohort of 62 medical students, 32% reported a prior episode of syncope, with a higher rate in women than in men (42% and 31% respectively).[9] Collectively, the data suggest a bimodal distribution of a first episode of syncope, with a first peak between the first and the second decade (with a female predominance) and another peak that begins after the age of 60 years and is gender-independent.[2] To summarize, up to approximately 30–40% of people will have an episode of syncope during their lifetime, and of these 30–40% will have a recurrent episode within three years.[1–9]

Although syncope may be the first symptom for a patient at high risk for sudden death and adverse clinical outcomes, examination of cohort studies suggests that the mortality rate ranges from 1–2% at 30 days to 7–8% at one year.[6,10] Examination of large hospital databases suggests that in-hospital mortality for syncope is also low (0.28%) with almost all deaths occurring in patients over 60 years of age.[8] However, in the Framingham report, patients who were thought to have a cardiac cause of syncope had a six month mortality rate of 10%.[6] The additional risk conferred simply by the presence of syncope is controversial and has varied from no additional risk to a 30% increase in risk in cohort studies using matched controls.[6,11] For example, Kapoor et al. found that mortality was the same in patients with or without syncope and was instead dependent on the presence and type of underlying cardiac disease and other comorbidities.[11] In contrast, in a population based study from the Framingham data, syncope was associated with a 30% increase in mortality compared to patients without syncope **(Fig. 1)**.[6]

Economic Burden of Syncope

It has been estimated that syncope accounts for 1–3% of emergency department evaluations and 1–6% of hospital admissions.[12,13] Several studies have estimated that the annual cost of management and treatment of patients with syncope ranges 1.7–2.4 billion dollars in the United States.[8,14] The cost for the management of patients with syncope varies widely,

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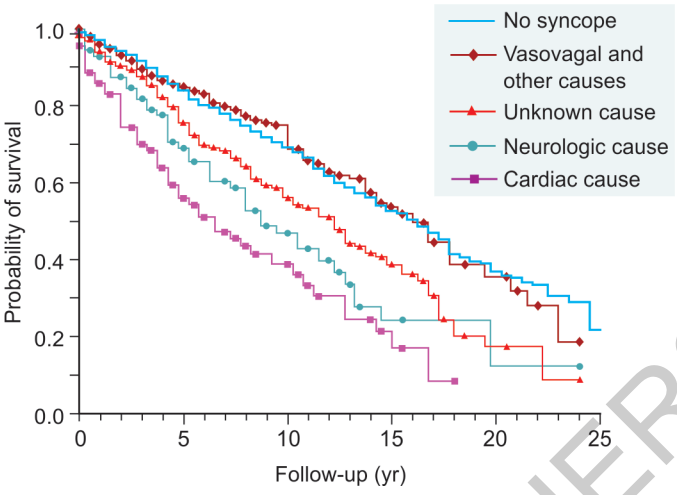

FIGURE 1: Survival curves from Framingham data for patients with different types of syncope. (*Source:* Soteriades ES, Evans JC, Larson MG, et al. Incidence and prognosis of syncope. N Engl J Med. 2002;347: 878-85)

dependent on the diagnostic tests ordered and whether or not an implantable cardiac rhythm device (pacemaker or ICD) is used.[8] In addition to the cost of diagnostic evaluation and specific therapies, syncope can be associated with injuries and significant psychological disability that can increase cost and have a significant impact on quality-of-life.[15–17] Major injuries, such as fractures and motor vehicle accidents were reported in about 6% of patients.[15] Minor injuries, including bruises and lacerations were reported in 27–29% of patients with syncope.[15] Elderly patients have a higher incidence of injuries when compared to younger patients, with a dramatic increase after age of 70 years, the incidence almost doubles between the sixth and the seventh decades of life.[15–17] Syncope is associated with a significant reduction in quality-of-life indices, particularly in the presence of recurrent episodes, associated comorbidities and in women.[15–17]

Causes and Classification of Syncope

As discussed earlier, syncope is but one cause of transient loss of consciousness. Syncope can further be classified into three general causes: (i) reflex or neurally mediated syncope; (ii) syncope due to orthostatic hypotension, and (iii) cardiac syncope **(Flowchart 1)**.

Reflex or neurally mediated syncope is the most common cause of syncope. One form, often called vasovagal syncope or the "common faint", is the single most common cause of syncope. The pathophysiological basis for vasovagal syncope

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was described by Sir Thomas Lewis in the early 1900s and importantly noted that although the bradycardia component could be reversed by atropine, hypotension and altered state of consciousness persisted. Although vasovagal syncope is the most common cause of syncope in younger patients, accounting for up to 50% of cases, it is important to recognize that even in patients more than 65 years old vasovagal syncope accounts for approximately 30% of cases. In all forms of reflex syncope, triggering of the afferent limb of a reflex arc leads to hypotension due to vasodilation (vasodepressor effect) and decreased heart rate (vagal effect). In vasovagal syncope the afferent limb can be triggered by a variety of conditions such as heat, hypovolemia, pain, or fear and anxiety while in conditions often called—situational syncope, triggering occurs from specific actions such as micturition, cough or swallow. Finally, particularly in older patients, the afferent limb can be triggered by carotid sinus stimulation.

A second cause of syncope is orthostatic hypotension. Normally with standing, accumulation of fluid in the legs results in the initiation of a complex neurologic reflex response that maintains systemic blood pressure. In orthostatic hypotension this response is insufficient, leading to a decrease in systemic blood pressure. An abnormal response is usually defined as a 20–30 mm Hg drop in systolic blood pressure on standing. Reduction in blood pressure and the increase in heart rate (which may be attenuated in older patients and in those with diabetes) are usually observed immediately after standing but can be delayed for a short period of time and thus blood pressure measurements should continue for several minutes after standing. Orthostatic hypotension can be due to a primary abnormality of the autonomic nervous system (either isolated or affecting multiple systems) or secondary (e.g. diabetes, Parkinson's disease and uremia). Diabetes is the most common cause of autonomic neuropathy in the United States and can be associated with relatively high mortality rates (25–50% mortality at 5–8 years).[18] Another form of autonomic dysfunction that can be associated with syncope is the postural orthostatic tachycardia syndrome (POTS); up to 20–30% of patients with POTS will report a prior history of syncope or presyncope.[19] Investigators believe that in POTS, hypotension on standing does not occur but maintenance of upright blood pressure requires an abnormal increase in heart rate, leading to sustained sinus tachycardia (usually >110 bpm). Although patients with POTS and orthostatic hypotension can present with symptoms of syncope they more commonly complain of symptoms such as fatigue, palpitations, rapid and pounding heart rates, and dizziness.

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The third cause of syncope is a cardiac abnormality. Etiologies of cardiac syncope can broadly be divided into abnormal heart rhythms and obstruction to flow. Both rapid heart rates and slow heart rates can cause cerebral hypoperfusion and syncope. Obstruction to blood flow can be due to aortic stenosis, pulmonary valve stenosis or dynamic left ventricular outflow tract obstruction in some patients with hypertrophic cardiomyopathy (HCM). Patients with anomalous coronary arteries can sometimes present with exertional syncope, particularly in those whose right coronary artery originates from the left system with the right coronary artery passing between the aorta and pulmonary artery.[20] Large population studies have shown that of the multiple causes of syncope, a cardiac etiology carries the worst prognosis **(Fig. 1)**.[6]

DIAGNOSTIC TESTS

The evaluation of syncope is often challenging and in up to 40% of cases no specific cause can be identified; this is specially true in older patients.[2,3,6] The history and physical examination play an essential role in evaluating patients with syncope. Several studies have attempted to evaluate the diagnostic yield of different tests in clinical practice.[2,3,21] It is important to acknowledge that the highest diagnostic yield of any test in unselected populations of patients with syncope is at best probably 25–30% with no test providing a "gold standard", underscoring the importance of careful initial evaluation by the history and the physical examination and choosing subsequent tests based on this initial assessment.[2–4]

History and Physical Examination

The history and physical examination play an important role in establishing cause of transient loss of consciousness and, in particular, for differentiating between syncope and seizure as this distinction can be difficult **(Flowchart 1)**. For example, up to 40% of patients with syncope will have generalized seizure-like activity and myoclonic jerking due to cerebral hypoperfusion.[22] Symptoms, such as prolonged confusion after the episode (postictal state) and tongue biting, are suggestive of seizure.[23,24] An altered sense of smell, taste or an aura, such as a sense of déjà vu prior to the event are suggestive of a temporal lobe seizure.[24] Focal neurologic signs and symptoms during or after the event (Todd's palsy), also make seizure more likely than syncope. Urinary incontinence, although more commonly observed with seizures, does not completely rule out syncope. In syncopal patients, recovery is complete and often, but not always, rapid, as contrasted with seizures in which recovery

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is slow due to the postictal state and associated confusion. Petit mal or absence seizures are occasionally misdiagnosed as syncope. A key feature that favors the diagnosis of absence seizures is preserved postural tone despite unresponsiveness. Temporal lobe seizures can have a long duration with varied levels of consciousness. Several investigators have evaluated specific characteristics of the history for differentiating among the various causes of syncope and between syncope and seizure.[24,25] In a cohort of 539 patients a simple point score system correctly differentiated seizure from syncope in 94% of patients with a sensitivity of 94% and a specificity of 94%.[24] Tongue biting, postictal confusion, head turning to one side and prodromal déjà vu or jamais vu were more suggestive of seizures while presyncope, diaphoresis prior to the episode, or loss of consciousness associated with standing made seizure less likely.

Once the clinician has decided that an episode of transient loss of consciousness is most likely due to syncope, the history and physical examination can provide further clues as to its specific cause.[26,27] Pertinent questions should include a history prior of cardiac disease and diagnosis, if known; family history of arrhythmias, syncope and sudden death; knowledge of an abnormal electrocardiogram, medications, positional changes that occurred prior to the syncopal spell (including head-turning), prodromal symptoms, and history of prior syncopal events. Features of the clinical history that are more commonly associated with significant arrhythmias such as ventricular tachycardia or bradycardia due to advanced or complete heart block include male sex, age older than 50 years, fewer than three episodes of syncope and duration of warning prior to syncope of less than 6 seconds.[26–28] Conversely, symptoms, such as blurred vision, nausea, vomiting, warmth, diaphoresis and prolonged fatigue after syncope, have been associated with neurally mediated syncope rather than ventricular tachycardia or complete heart block. In a cohort of 341 patients, the presence of suspected cardiac disease was the strongest predictor of cardiac cause of syncope and absence of cardiac disease had a negative predictive value for a cardiac cause of 97%.[26] However, a more recent study found that the value of clinical history for distinguishing between cardiac and neurally mediated syncope was significantly reduced in older patients.[21] Finally, syncope associated with exertion has traditionally been identified as "high-risk" due to its association with valvular aortic stenosis, HCM, congenital coronary anomalies and channelopathies such as Long QT syndrome, but no large studies have been performed in these groups.[29] Using a similar approach for distinguishing between syncope and seizure, Sheldon and his coworkers identified several historical features that can help

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FIGURE 2: Simultaneously recorded leads aVR and aVF rhythm strips obtained during carotid sinus massage (CSM). Initially sinus slowing is observed and ultimately a 3.5 second sinus pause is noted. A pause with ventricular asystole more than 3 seconds, due either to sinus pause/arrest or AV block, defines carotid sinus hypersensitivity

to differentiate between vasovagal syncope and cardiac causes of syncope **(Fig. 2)**.[30,31] Symptoms such as lightheadedness associated with pain or medical settings or with prolonged sitting or standing, and a sensation of warmth or sweatiness prior to the episode made vasovagal syncope more likely. Conversely, a history of diabetes, prior arrhythmia, no recollection of the episode and palpitations preceding syncope more likely made a cardiac cause. Similarly, a first event over 35 years of age or bystanders describing the patient "turning blue" were also associated with a cardiac cause of syncope.

The physical examination has a central role in evaluation of syncope. A complete cardiac examination should be performed to assess the presence of structural cardiac disease. Orthostatic vital signs are an essential part of the physical examination in a patient. A drop of systolic blood pressure exceeding 20 mm Hg or a decrease in diastolic blood pressure of more than 10 mm Hg is considered to be diagnostic for orthostatic hypotension. The POTS is defined as an increase in heart rate more than 25–30 beats per minute within 5 minutes of standing with symptoms. Cardiac auscultatory findings, such as murmurs or gallops, are important for identifying the presence and severity of structural cardiac disease such as aortic stenosis and HCM. On palpation, findings, such as a left ventricular heave or a sustained left ventricular impulse, can alert the clinician to the presence of structural heart disease, such as left ventricular hypertrophy or ventricular dilation, due to cardiomyopathy or past myocardial infarction that will make more likely a cardiac cause of syncope.

Finally, performing carotid sinus massage (CSM) is important in patients with syncope over 40 years of age.[2] Neurologic complications during CSM occur rarely (0.17–0.45% of patients) and it is important to confirm the absence of carotid bruits or neurologic symptoms suggestive of a stroke or transient ischemic attack and before performing the maneuver.[2] Continuous electrocardiographic monitoring is essential and continuous blood pressure monitoring is highly desirable. To perform CSM, firm continuous pressure for 5–10 seconds should be applied to the right carotid artery at the level of the cricoid cartilage and after several minutes the same maneuver should be repeated on the left side. Ideally, CSM should be

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performed in both the supine and upright positions since an abnormal vasodepressor response may be detected only when the patient is upright. An abnormal response (carotid sinus hypersensitivity) is usually defined as ventricular asystole more than 3 seconds (due either to sinus pause or AV block) or a fall in systemic blood pressure more than 50 mm Hg **(Fig. 2)**. Formal CSM is often included as a part of the tilt-table test in some institutions.

It is important to remember that the history and physical examination play a critical role for risk stratification of the patient with syncope.[2,3] As outlined previously, patients with a cardiac cause of syncope have a far worse short-term and long-term prognosis compared to syncope due to other causes. For this reason, the history and physical examination help the clinician decide whether structural cardiac disease is present and provide an initial estimate for the likelihood of a cardiac cause for syncope.

Blood Tests

Routine blood tests including electrolytes, tests for anemia (hematocrit or hemoglobin) and glucose, although commonly performed, generally have a low diagnostic yield in evaluation of syncope.[2,3] The exception may be the presence of anemia; anemia has been incorporated into several prognostic algorithms used for risk stratification of patients with syncope.[32,33] Frequently, patients with syncope are evaluated for myocardial infarction, even in the absence of an infarction pattern on the electrocardiogram. Despite this approach, the yield of diagnostic evaluation for myocardial infarction for patients with syncope is less than 1%.[34] In a small study investigators found that higher brain natriuretic peptide (BNP) levels could be used to identify patients with syncope due to cardiac causes or worse outcomes, and a recently published risk stratification schema for patients with syncope used a BNP more than 300 pg/mL as one criteria for identifying high-risk patients.[33,35] It is important to note that the most recent guidelines do not specifically recommend any blood tests for the evaluation of syncope.[2,3]

Electrocardiogram

A 12 lead ECG is a basic part of the workup in all patients with syncope.[2,3] Although the diagnostic yield of a baseline ECG is low (5–10%), it is an inexpensive and widely available test that can be used to quickly risk stratify patients, particularly if it is abnormal.[2,3,29] Baseline sinus bradycardia, atrioventricular block and intraventricular conduction block (left or right bundle branch block) suggest the possibility of bradycardia as a cause for syncope. Presence of Q waves that suggest the possibility

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of a prior myocardial infarction or other findings, such as left ventricular hypertrophy make structural heart disease, and thus a cardiac cause of syncope, more likely. The presence of premature ventricular depolarizations may have some prognostic information in patients with syncope. In an older study of 235 patients with syncope, the presence of frequent or paired premature ventricular contractions was associated with higher mortality and risk of sudden death.[36]

Finally, there are some ECG patterns that can be used to identify potential causes for syncope: Wolff-Parkinson-White syndrome, Long QT syndrome, Brugada syndrome, arrhythmogenic right ventricular cardiomyopathy and HCM **(Figs 3 to 7A and B)**.

Echocardiography

Both the AHA/ACCF and ESC guidelines state that the echocardiogram plays a central role in syncopal patients with suspected cardiac disease.[2,3] In a review of over 2,000 elderly patients admitted for syncope at a single center, echocardiograms were obtained in 40% of patients and abnormalities were identified in almost 70%.[21] However, results from the echocardiogram affected management in less than 5% of the cases.[21]

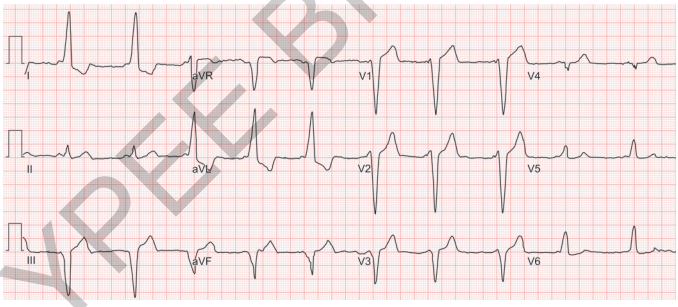

FIGURE 3: ECG from a patient with Wolff-Parkinson-White syndrome due to the presence of a right freewall accessory pathway. Preexcitation of the right ventricle leads to a short PR interval and a QRS complex with a left bundle branch block morphology and a negative delta wave in aVR [*Source:* Kusumoto FM. ECG Interpretation. Pathophysiology to Clinical Application. New York: Blackwell-Springer; 2009 (with permission)]

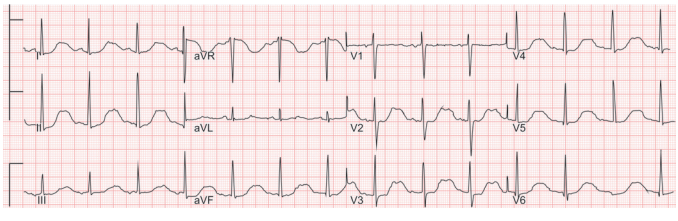

FIGURE 4: ECG from a patient with syncope and long QT syndrome (*Source:* Kusumoto FM. ECG Interpretation. Pathophysiology to Clinical Application. New York: Blackwell-Springer; 2009)

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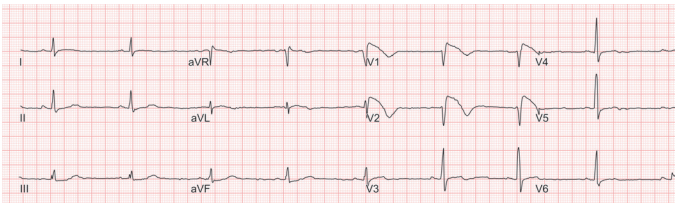


FIGURE 5: ECG from a patient with Brugada syndrome showing the characteristic type I right bundle branch block pattern and downsloping ST segment elevation in V_1 and V_2. Type I Brugada syndrome is more specific than the "saddle back" ST segment contour in the type II pattern. ECG patterns in Brugada syndrome can be quite variable, even over short periods of time; questionable diagnoses can be clarified during the intravenous infusion of a sodium-channel blocking drug (*Source:* Kusumoto FM. ECG Interpretation. Pathophysiology to Clinical Application. New York: Blackwell-Springer; 2009)

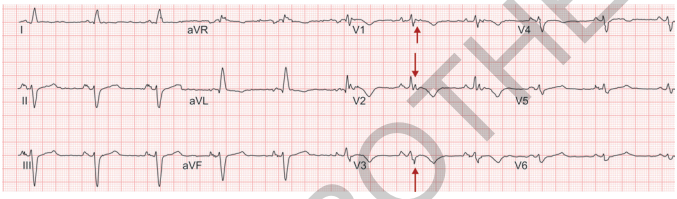
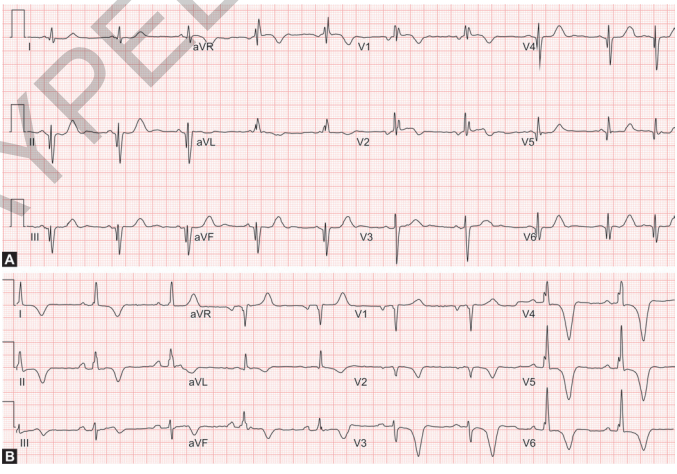


FIGURE 6: ECG from a patient with arrhythmogenic right ventricular cardiomyopathy. There are prominent anterior forces and precordial T wave inversion is present in addition to the highly specific epsilon waves (arrows) that represent delayed conduction in the right ventricle. (*Source:* Kusumoto FM. ECG Interpretation. Pathophysiology to Clinical Application. New York: Blackwell-Springer; 2009)



FIGURES 7A AND B: (A) ECGs from two patients with hypertrophic cardiomyopathy. Hypertrophy predominantly affecting the septum, leading to a larger than expected R wave in V1 and "pseudo Q waves in the inferolateral leads. (B) Hypertrophy affecting the cardiac apex, leading to deep lateral T wave inversions (*Source:* Kusumoto FM. ECG Interpretation. Pathophysiology to Clinical Application. New york: Blackwell-Springer; 2009)

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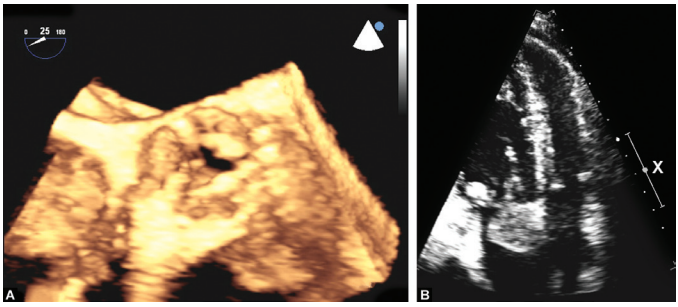

FIGURES 8A AND B: (A) Diagnostic echocardiographic images in patients that presented with syncope. Three-dimensional transesophageal echocardiographic image of a patient with severe aortic stenosis. (B) Four chamber transthoracic echocardiographic image of a sessile left atrial myxoma attached to the interatrial septum. (*Source:* Emery Kapples, Jeannine Hiers and Carolyn Landolfo)

Echocardiography has a low diagnostic yield in patients with a normal physical examination and normal ECG and need not necessarily be obtained in all patients with syncope. A structural abnormality noted during echocardiography does not per se establish a diagnostic cause for syncope. However, the presence of severe aortic stenosis or rarer conditions, such as atrial myxoma, is usually diagnostic of etiology **(Figs 8A and B)**.

Exercise Testing

Exercise testing has a low diagnostic yield in the evaluation of syncope (<5%).[29] However, it may particularly be useful in those patients with exertional syncope. Published guidelines are not uniform in their recommendations; however, the AHA/ACCF scientific statement (but not the ESC guidelines) suggest that exercise testing should be more widely applied to any patient with unexplained syncope, particularly those with coronary artery disease or those at risk for coronary artery disease.

Perhaps even more useful than the identification of ischemia in the patient with syncope is the evaluation of hemodynamic and heart rhythm responses to exercise testing. Development of atrioventricular block with exercise is always abnormal and suggests bradycardia as the mechanism for symptoms. An abnormal decrease in blood pressure during or after exercise may be an important clue for the mechanism of exertional syncope in a patient with HCM.

Continuous ECG Monitoring

External Devices (24 Hours Ambulatory ECG Recorders, Event Recorders)

Since intermittent bradycardia or tachycardia are the most common cardiac etiologies for syncope, an ECG obtained

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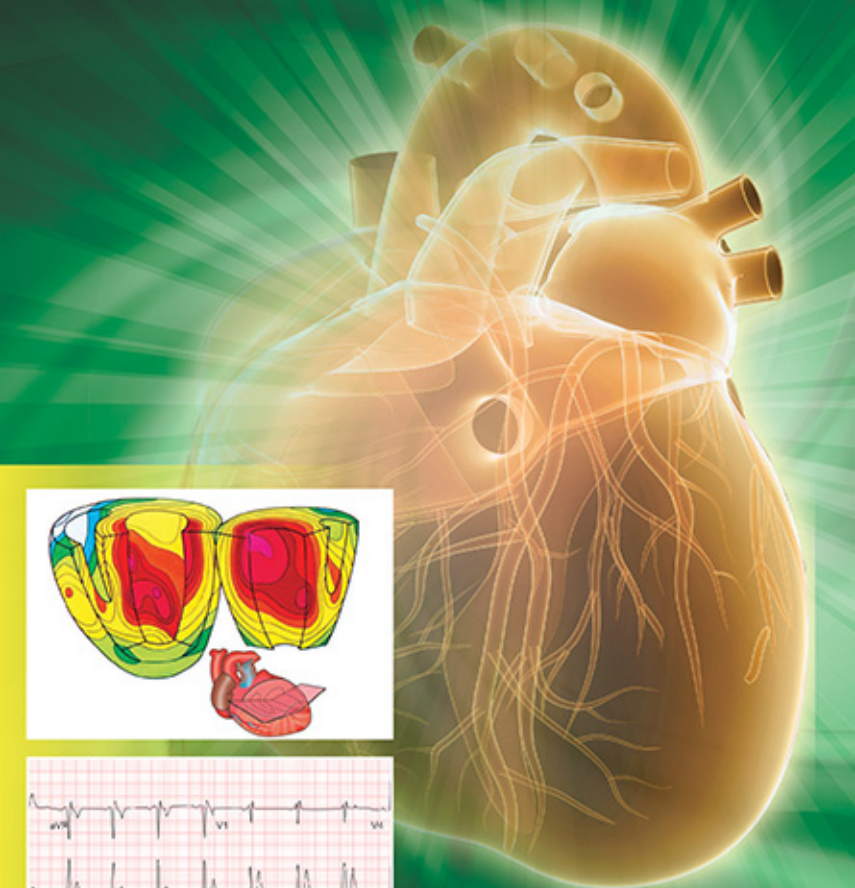

Kanu Chatterjee
Mark Anderson
Donald Heistad
Richard E Kerber

JAYPEE