

# Review of Important ECG Findings in Patients with Syncope

Joseph Toscano, MD

## Abstract

Guidelines recommend 12-lead ECG as an important test to perform in patients with syncope. Though the incidence of ECG abnormalities is quite low, urgent care clinicians must be knowledgeable about the findings which portend the highest risk for these patients. Having a structured approach to interpreting these patients' ECGs, and acting appropriately when abnormalities are found, should minimize morbidity and mortality in this patient group.

## Introduction

Syncope is an uncommon problem seen in urgent care practice, and most cases of syncope are self-limited and benign. Transient loss of consciousness is, however, occasionally a harbinger of a more severe underlying problem. Coronary ischemia, cardiac dysrhythmia and the potential for dysrhythmia, and cardiac outflow obstruction are among the cardiac causes of syncope that need to be considered when a patient presents after syncope, regardless of the healthcare setting. The following review summarizes the major conditions that need to be excluded by ECG for patients who present after having passed out (see Table 1).

## Epidemiology/Pathophysiology

There is little information about how frequently patients present to urgent care settings after syncope; however, it has been reported that up to 1.5% of emergency department visits<sup>1</sup> and 6% of hospital admissions are for this problem.<sup>1,2</sup> Data from the Framingham study showed an occurrence rate of first syn-

cope of 6.2 cases per 1000 patient-years.<sup>2</sup> Patients with cardiac syncope have a worse prognosis than those with noncardiac syncope,<sup>2</sup> and 12-lead ECG is an important test to be used

**Table 1:** Important ECG findings in patients with syncope

- Paroxysmal or sustained dysrhythmia on monitoring in clinic
- Non-sinus rhythm of any sort
- Nonspecific intraventricular conduction delay (QRS > 100 ms without left or right bundle branch pattern)
- Left bundle branch block or left anterior or posterior hemiblock
- ECG signs of coronary ischemia
- Long QT syndrome - QTc > 440-450 msec in men or > 460 msec in women
- Brugada sign - right bundle branch block and anterior ST elevation
- Left ventricular hypertrophy in someone with no reason to have it and/or Q waves in II, III, aVF, V5, and V6
- Pre-excitation syndromes (PR interval < 120 msec) with or without delta wave

for cardiac diagnoses. Because the large trials combine ECG abnormalities with other potentially important determinants of outcome (e.g., lab results, vital signs, age, comorbidities), it is hard to determine the independent effect of "picking-up" an

abnormal finding, and, to the author's knowledge, this has not been quantified. Although the number is surely small overall, for those affected it is obviously very important. Such "low-incidence, high-stakes" situations are common in acute care practice, and the basic principle of minimizing morbidity and mortality by appropriately interpreting ECGs is inferred from the overall data and case studies.

Syncope has many potential causes, with a common pathophysiology of transient, globally decreased cerebral blood flow. There are many disorders that need to be considered, including syncope mimics. The complete evaluation of these patients requires more complex decision-making than will be explained in this review. On the other hand, several authoritative guidelines<sup>2,3,4</sup> recommend ECG as the primary, and in most cases only, diagnostic tool for these patients, and along with a history and physical examination, a complete initial evaluation can be provided at most urgent care clinics. Clinicians need to be cognizant of the ECG findings that place patients at higher risk for ongoing or recurrent problems after syncope.

The cardiac causes of syncope, which may be apparent on ECG, include conditions which lead to coronary ischemia, cardiac outflow obstruction, dysrhythmias, and conduction system problems severe enough to decrease cardiac output. Paroxysmal dysrhythmias may rarely be observed in clinic. When these are not present, it is vital to look for ECG changes, which are known to predispose to recurrent dysrhythmia or other problems. Patients with non-sinus rhythm of any sort, conduction disorders of the left bundle (left bundle branch block, left anterior or posterior hemiblock), or nonspecific intraventricular conduction delay (prolonged QRS without left or right bundle pattern) are more likely to suffer significant adverse cardiac outcomes after syncope.<sup>5</sup>

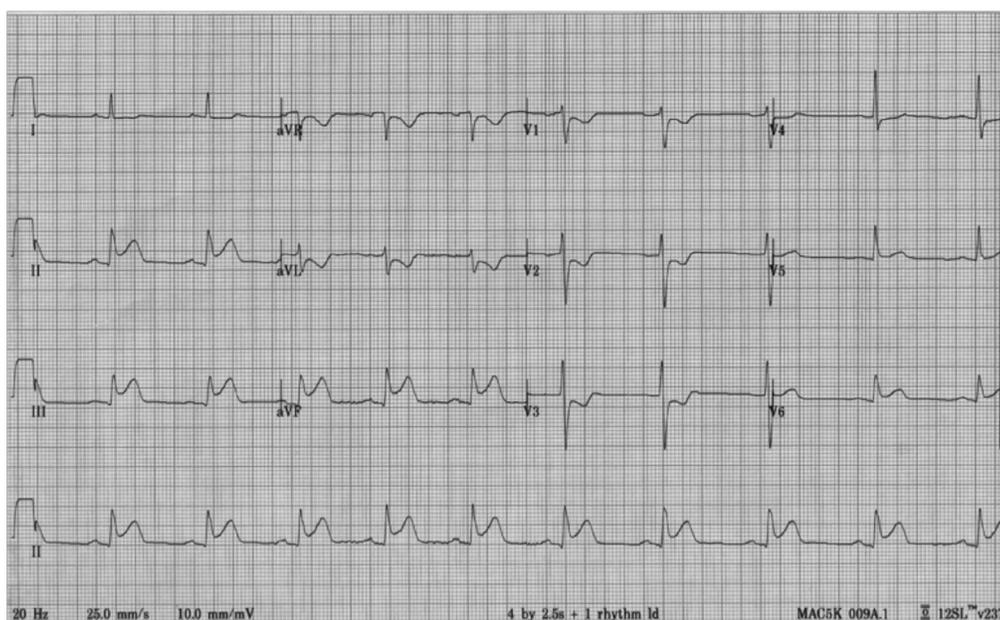
## Coronary Ischemia

Syncope associated with coronary ischemia can have many causes, including paroxysmal dysrhythmia, left ventricular pump failure, and acute mitral regurgitation. Careful cardiac and pulmonary physical exam may be revealing in these cases. As well, the clinician should review the 12-lead ECG for signs of ischemia. These include new and evolving ST segment changes, T wave inversions, and/or Q waves (see Figures 1 and 2). Comparison with any prior ECGs can increase the specificity of any abnormal findings for an acute problem, as can obtaining a second ECG in clinic 10 to 15 minutes after the first for stable patients. The sensitivity of ECG is less than perfect, however, so patients with a presentation that is clinically suspicious for ischemia should be promptly and safely transferred to a higher level of care, even with a normal or unchanged ECG.

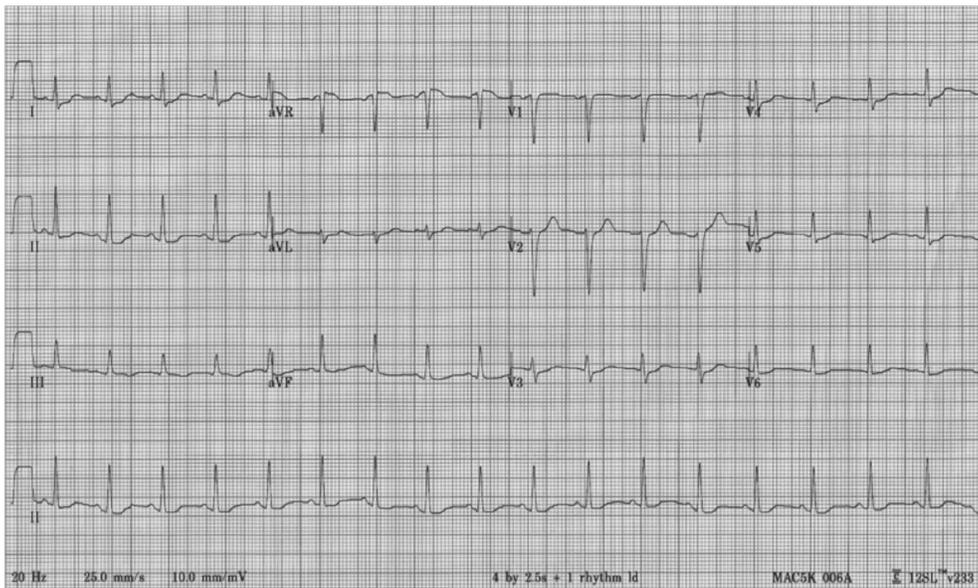
## Brady- and Tachydysrhythmias

Bradycardia results in syncope due to directly decreased cardiac output (cardiac output = stroke volume x heart rate). A slow heart rate should be obvious on ECG and may be due to sinus bradycardia or first-, second-, or third-degree heart block. Sinus bradycardia and lower grade heart block (first- or type I second-degree heart block) may be transient and generally carry a lower risk of adverse outcomes than higher grade heart block (type II second- or third-degree heart block). Sinus bradycardia or even lower grade heart block may be seen as part of a "vagal" response, and any slow heart rate may be due to intrinsic conduction system disease, medication (calcium channel blockers and beta blockers), or hyperkalemia.

Patients with persistent, symptomatic bradycardia of any sort or high-grade heart block even without symptoms generally require transfer to a higher level of care and may be candidates

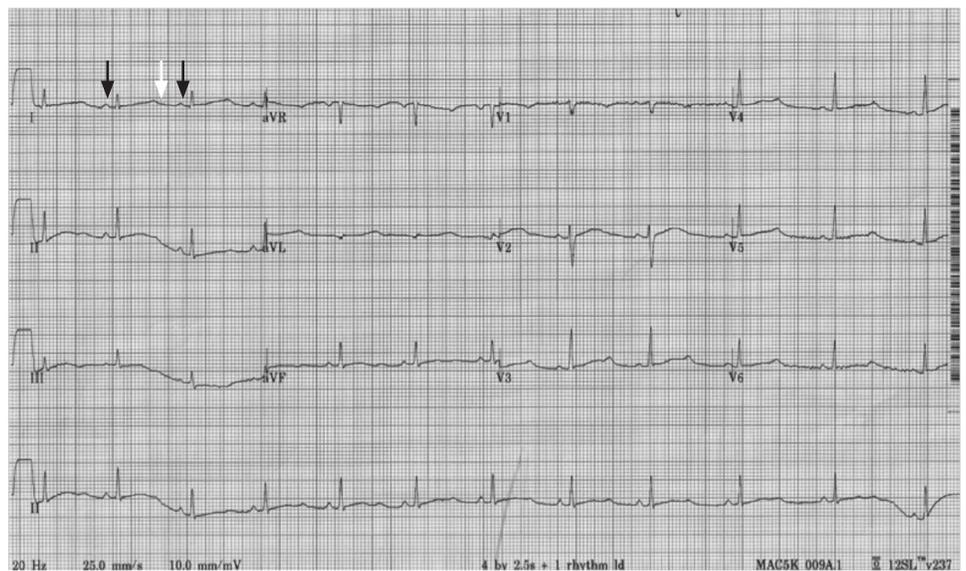


**Figure 1:** ST Segment Elevation Myocardial Infarction – STEMI. The pattern of ST segment elevations in contiguous inferior leads (II, III, aVF) with ST segment depressions in the reciprocal anteroseptal leads has a high specificity for acute myocardial infarction. Syncope in this patient, who otherwise complained only of left arm pain, may have been related to paroxysmal bradycardia or other dysrhythmia, but did not recur. Emergency transfer to the emergency department was followed by emergency cardiac catheterization showing a 100% right coronary artery lesion, which was successfully stented.



**Figure 2:** Cardiac Ischemia. This 41-year-old male presented after passing out while playing basketball. ST segment depressions in the lateral leads (I, aVL, V4, V5, V6) were subtle but persistent on serial ECG. He was transferred to the ED and admitted. His cardiac enzymes became positive, and cardiac catheterization revealed a 99% circumflex lesion.

**Figure 3:** Long QT syndrome. It is important to measure the QT interval and the QTc (or at least verify the computerized reading), but a general impression of whether the QTc is prolonged can be obtained by looking for the end of the T wave (white arrow). If it occurs over halfway between the R-R interval (from one black arrow to the other, above), then the QTc will be prolonged. An alternative method using the peaks of the R and T waves is easier to “train your eye to see” but may be less accurate. This 16-year-old patient presented after syncope. She had inducible ventricular tachycardia during electrophysiology study and had an automated internal cardioverter/defibrillator placed at the same time.



for pacemaker placement; associated symptoms are a more important criterion for pacemaker placement than heart rate, though documented periods of asystole for three or more seconds or any escape rate below 40 beats per minute are significant in asymptomatic patients. Those with asymptomatic sinus bradycardia or low grade heart block should be managed in consultation with a cardiologist, and most can have outpatient follow-up.

Tachycardia results in syncope when the heart rate reaches a threshold above which inadequate time for ventricular filling lowers stroke volume below a critical, brain-perfusing level. This threshold rate will be lower for patients with many types of underlying cardiac disease. Ventricular tachycardia or very rapid supraventricular tachycardia cause syncope more often than atrial fibrillation, atrial flutter, or multifocal atrial tachycardia do. Of course, any culprit dysrhythmia may have resolved by the time the patient presents for care to an urgent care clinic.

It may recur paroxysmally and be captured with monitoring in clinic, but more often, the clinician must examine the ECG for conditions that predispose to tachydysrhythmia (see below).

As with ischemia, ECG sensitivity is too low for it to be relied upon to completely “rule-out” a potential rhythm problem, particularly in an asymptomatic patient after syncope has resolved. Therefore, for patients with worrisome presentations, such as syncope during exercise or prominent palpitations preceding their syncopal episodes, the urgent care clinician should coordinate disposition and follow-up with a cardiologist, even if the 12-lead ECG in clinic is normal.

Though the heart rate in sinus tachycardia will usually not rise to the level where a patient will have syncope as a result of the elevated rate alone, it may more often indicate an underlying non-cardiac problem that needs evaluation, for example, blood loss, dehydration, infection, etc.

## Conditions Which Predispose to Dysrhythmia

Several conditions, in healthy patients without structural heart disease, are associated with a predisposition to tachydysrhythmias and syncope. These conditions are also associated with sudden cardiac death, so clinicians should look for the ECG findings of these disorders in all patients who present after syncope. Clinicians should immediately consult a cardiologist for any patient with any of these ECG findings after syncope. Hospital admission for telemetry and electrophysiology study is often advised. Any of these may be found on routine ECG as well, and without a history of recent or prior syncope, an outpatient workup can be pursued. The occurrence of syncope, however, portends higher risk, and these patients need immediate, or at least extremely close, specialty follow-up. These conditions are reviewed in greater depth elsewhere,<sup>6</sup> but the following section summarizes the most important details.

### Long QT syndrome (LQTS)

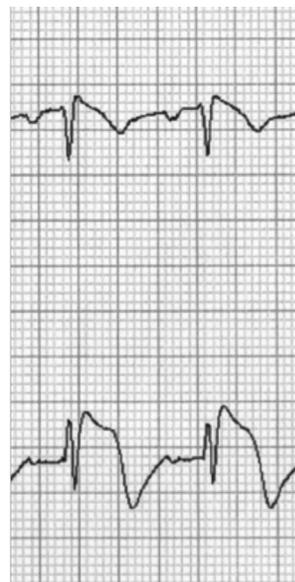
Several abnormalities of delayed ventricular repolarization are characterized by prolongation of the QT interval on ECG. These abnormalities include congenital membrane-ion-channel defects and some acquired causes related to medications, toxins, and neuropathies. Any of them may result in spontaneous or provoked (e.g., at the sound of a loud noise or physical or emotional stress) ventricular tachycardia, which can degenerate to fibrillation. The median age of those who die from LQTS is 32 years old.

Delayed ventricular repolarization is defined relative to heart rate (QTcorrected, or QTc) and LQTS is characterized by a QTc interval > 440-450 msec in men and > 460 msec in women. Though the correct formula to calculate QTc is part of most ECG units, computerized assessment of the QT interval may be unreliable and will result in a miscalculation. Any reading in a

syncope patient should be checked with a set of calipers. As a general “gestalt,” look for the end of the T wave to occur after the halfway point between the R-R interval (see Figure 3) if the patient has LQTS.

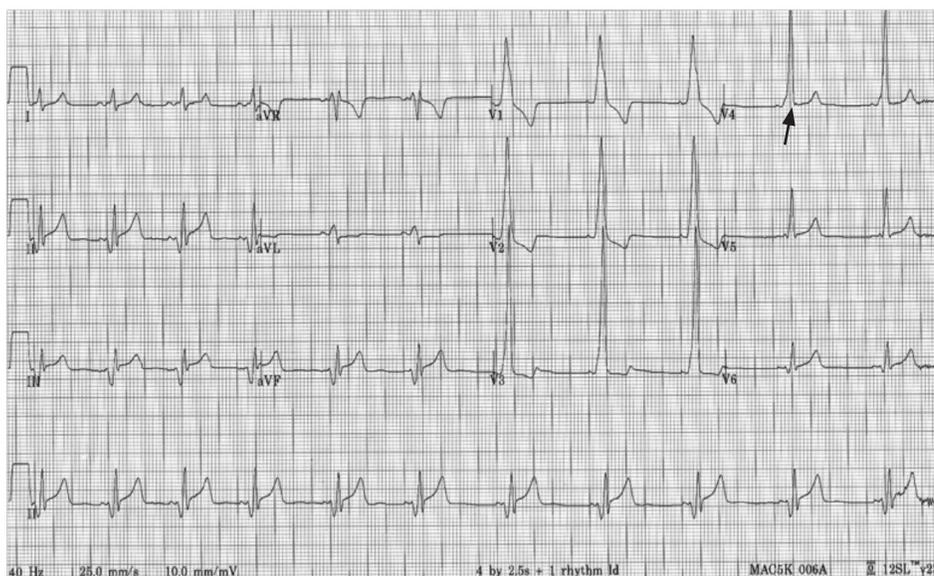
### Brugada syndrome

Patients with Brugada syndrome have a defective sodium membrane channel due to an inherited chromosomal mutation. The defective channel predisposes to ventricular tachycardia and fibrillation. The ECG hallmark of this disorder is a right bundle branch pattern with anterior ST segment elevation in either a coved-downsloping or saddle-shaped pattern (see Figure 4).



**Figure 4:** Brugada Sign. The pattern of right bundle branch block (RBBB) and “coved-downsloping” ST segment elevation in leads V1 (top) and V2 (bottom) comprise one form of Brugada sign. In a patient with syncope, this is suspicious for Brugada syndrome due to self-terminating ventricular tachycardia. A second form of Brugada sign includes more of a saddle-shape (coved but not downsloping) to the elevated ST segment. The basic abnormal pattern to remember for Brugada is “RBBB and ST elevation” which should not normally occur together.

The mortality of undiagnosed, untreated Brugada syndrome is reported to be 30% at two years; the average age at diagnosis is between 35 and 45. The ECG findings may be transient and are



**Figure 5:** Wolf-Parkinson-White Syndrome (WPW). The pattern of shortening of the PR interval to less than 120 milliseconds, widening of the QRS complex, and a slurred upstroke of the initial part of the R wave, called the delta wave (black arrow), comprise WPW. Lown-Ganong-Levine syndrome appears similar but lacks the delta wave.

reported to be unmasked by fever or some medications. A pseudo-Brugada sign can be produced by electrolyte disturbances, cocaine, cyclic antidepressants, class 1A or 1C antidysrhythmics, and right ventricular infarction or injury; however, all of these patients would require referral to a higher level of care in the setting of syncope.

## Ventricular pre-excitation syndromes

Several mechanisms of supraventricular tachycardia (SVT) exist, but those which incorporate conduction along an accessory pathway can permit SVT rates and other situations (e.g., very rapid conduction of atrial fibrillation) that are more likely to result in syncope. If the resting ECG captures conduction of the atrioventricular impulses along the accessory pathway, the classic pattern of Wolf-Parkinson-White (WPW) syndrome will be apparent. This includes shortening of the PR interval to less than 120 milliseconds, widening of the QRS complex, and a "delta wave," which describes a slurred upstroke of the initial part of the R wave (see Figure 5). Patients with WPW will not have these findings if atrioventricular impulses are traveling along the normal conduction system at the time the ECG is performed. Lown-Ganong-Levine syndrome includes shortening of the PR interval to less than 120 milliseconds due to an accessory pathway, but there is no delta wave.

**Table 2:** Several, of many, systems used for diagnosing left ventricular hypertrophy on ECG

### CORNELL CRITERIA

- Height of the S wave in V3 plus height of the R wave in aVL > 24 mm for men
- Height of the S wave in V3 plus height of the R wave in aVL > 20 mm for women

### COMMONLY USED

- Height of the R wave in V5 or V6 plus height of the S wave in V1 or V2 > 35mm

### OR

- Sum of tallest R wave and deepest S wave in precordial leads > 40 mm

### OR

- Height of R wave in aVL > 11 mm

## Cardiac Outflow Obstruction

Another genetic disorder, hypertrophic cardiomyopathy (HCM), comes in several forms, all involving abnormal left ventricular hypertrophy (LVH), either concentrically or including just a segment of the left ventricular wall. HCM has also been called idiopathic hypertrophic subaortic stenosis (IHSS) and hypertrophic obstructive cardiomyopathy and occurs in someone without the stressors of hypertension or valvular heart disease to produce the LVH. Many patients will never have symptoms, but there is potential for the hypertrophied segment to obstruct the outflow of blood from the left ventricle, particu-

larly during exercise. This can lead to syncope or worse: HCM is the most common cause of sudden death in young athletes. Physical exam findings include a bisferiens pulse and systolic murmur heard along the left sternal border, which increases with Valsalva maneuver. ECG findings include LVH and/or prominent Q waves in leads II, III, aVF, V5 and V6. Though there are many schemes for predicting LVH (see Table 2) and these tend to be highly specific (computerized LVH algorithms are reliable if positive), sensitivity of these calculations for LVH in general ranges between 25 and 50%. Still, the ECG is reported to be abnormal in about 90% of patients with HCM, so examining for abnormal Q waves and/or "LVH in someone who has no reason to have it" is important when evaluating a patient post-syncope.

## Conclusion

Though of limited sensitivity, 12-lead ECG is inexpensive and is uniformly recommended in the evaluation of patients after syncope. Patients with ECG findings of obvious dysrhythmia or the potential for dysrhythmia, conduction disturbances, coronary ischemia, or cardiac outflow obstruction require cardiology consultation, and many will require transfer, admission, and electrophysiology or other evaluation. These are uncommon occurrences but truly represent a chance to potentially save a life, if appropriate diagnosis and referral is made.

*Joseph Toscano, MD, has been practicing urgent care and emergency medicine in the San Francisco Bay area for more than 15 years. He develops and reviews continuing medical education material in urgent care and emergency medicine for a variety of publications and presents regularly at urgent care conferences. Dr. Toscano is a founding board member of both the Urgent Care College of Physicians and the Board of Certification in Urgent Care Medicine.*

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