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Diagnostics

The electrocardiogram in the patient with syncope

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Abstract Syncope is a common and challenging presentation for the emergency physician. Various investigators have developed clinical risk score and clinical decision rules which are designed to identify the population at highest risk for adverse events. In each of these clinical decision tools, the electrocardiogram (ECG) is one of the key clinical variables used to evaluate the patient. Certain electrocardiographic presentations in the patient with syncope will not only provide a reason for the loss of consciousness but also guide early therapy and disposition in this individual. Bradycardia, atrioventricular block, intraventricular conduction abnormality, and tachydysrhythmia in the appropriate clinical setting provide an answer to the clinician for the sync opal event. Morphologic findings suggesting the range of cardiovascular malady are also encountered; these entities are far ranging, including the various ST-segment and T-wave abnormalities of acute coronary syndrome, ventricular preexcitation as seen in the Wolff-Parkinson-White syndrome, Brugada syndrome with the associated tendency for sudden death, prolonged QT interval common in the diverse long QT interval presentations, and right ventricular hypertrophy suggestive of hypertrophic cardiomyopathy. This review discusses the ECG in the patient with syncope. The general use of the 12-lead ECG in this patient population is discussed. Furthermore, specific electrocardiographic presentations seen in the patient with syncope are also reviewed.

1. Introduction

Syncope is a common and challenging presentation for the emergency physician. It is estimated that 1% to 3% of visits to the ED and 6% of hospital admissions are related to syncope; furthermore, 20% to 50% of adults will experience a syncopal episode at least once in their lifetime [1]. Although most causes of syncope are benign and require no further evaluation, there is a small subset of patients for whom a syncopal episode may herald a potentially life-threatening condition [2-4]. Investigators have developed a clinical risk score [4] and clinical decision rules [2,3] which are designed to identify the population at highest risk for adverse events. In each of these clinical decision tools, an abnormal electrocardiogram (ECG) is one of the key clinical variables used to evaluate the patient.

Four basic diagnostic categories of syncope are encountered, including reflex-mediated, orthostatic, cerebrovascular, and cardiac. Of these 4 generally accepted categories of syncope, cardiac causes represent 10% to 30% of cases [1]. The ECG may be helpful in all 4 categories, yet the cardiac subtype is likely to yield the highest rate of electrocardio-

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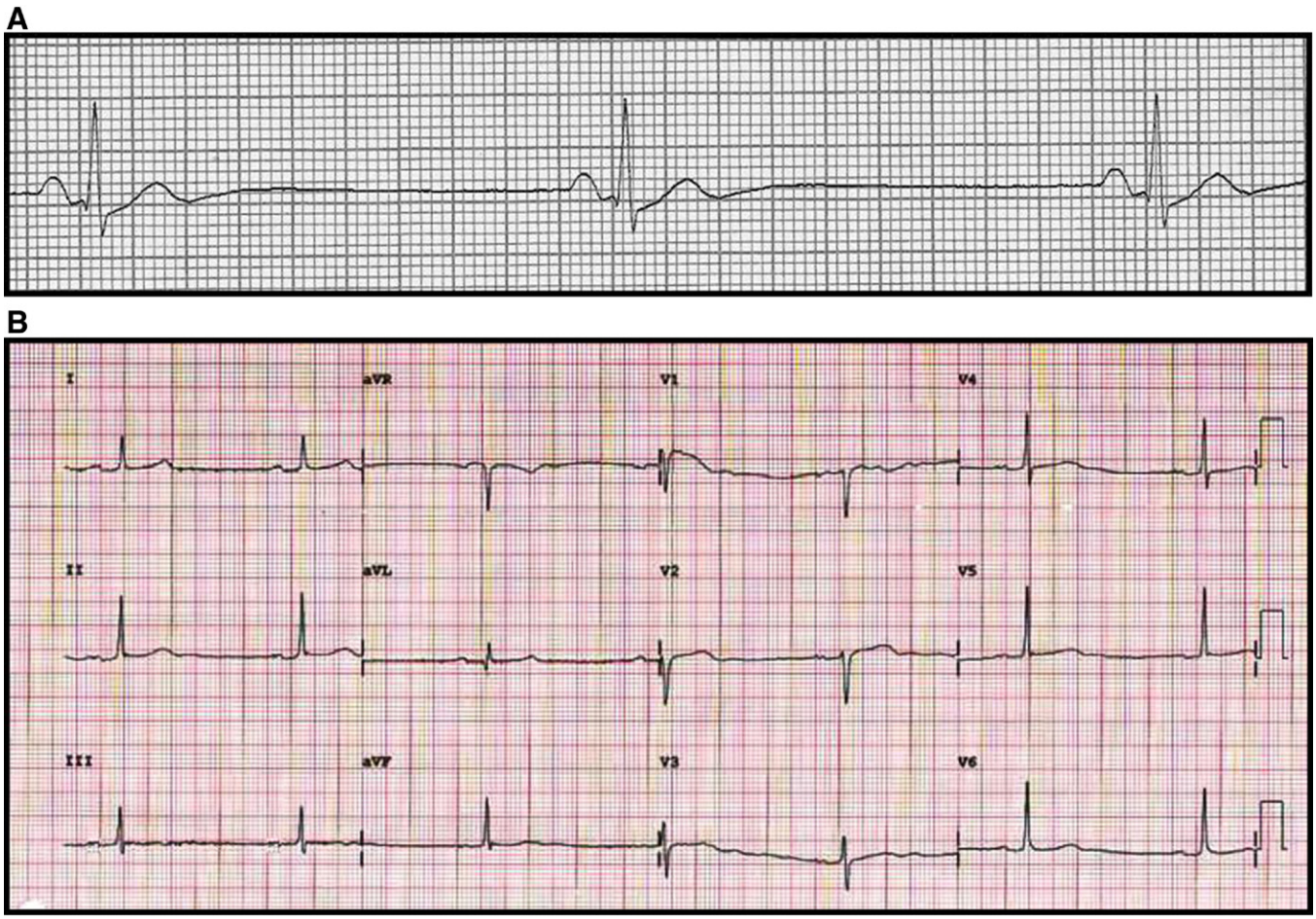

Fig. 1 A, Sinus bradycardia at approximately 30 bpm. B, Sinus bradycardia at approximately 30 bpm with first-degree AV block. Two defects in impulse generation/conduction are noted here, including the AV block and the bradycardia.

graphic abnormality. Regardless of the syncope presentation, the ECG is a noninvasive, inexpensive tool for identifying many of these important causes, as well as for predicting prognosis and risk stratification of these patients.

Current guidelines from the American College of Cardiology state that the assessment of a patient with syncope should begin with a careful history, physical examination, and ECG; furthermore, these guidelines state that the history and physical examination alone can lead to a diagnosis in more than 60% of patients [1,5]. The history, not surprisingly, can guide the clinician in many important areas of the syncope evaluation; for instance, historical points of interest that differentiate a syncopal episode from other potentially similar phenomena (ie, seizure) include a transient loss of consciousness, falling, and a rapid, spontaneous recovery [1,6,7]. Physical examination should focus on the vital signs as well as the cardiovascular and neurologic systems. In conjunction with the history and examination, the 12-lead ECG is the "procedure of first choice" according to the American College of Cardiology and American Heart Association [6,7]. Despite its obvious benefits, a 2004 study revealed that electrocardiographic testing was documented in only 59% of ED visits for syncope [8].

This review discusses the ECG in the patient with syncope. The general use of the 12-lead ECG in this patient population is discussed. Furthermore, specific electrocardiographic presentations seen in the patient with syncope are also reviewed.

2. Case presentations

2.1. Case 1

A 68-year-old woman presented to the ED with 2 days of progressive dizziness followed by syncope on the day of presentation; she denied chest pain or dyspnea. She had a history of subarrachnoid hemorrhage and hypertension managed with diltiazem. Examination was unremarkable with the exception of bradycardia at a rate of approximately 35 beats per minute (bpm); cardiac monitoring demonstrated a profound sinus bradycardia (Fig. 1A). The 12-lead ECG (Fig. 1B) revealed sinus bradycardia with first-degree atrioventricular (AV) block. The patient was admitted to

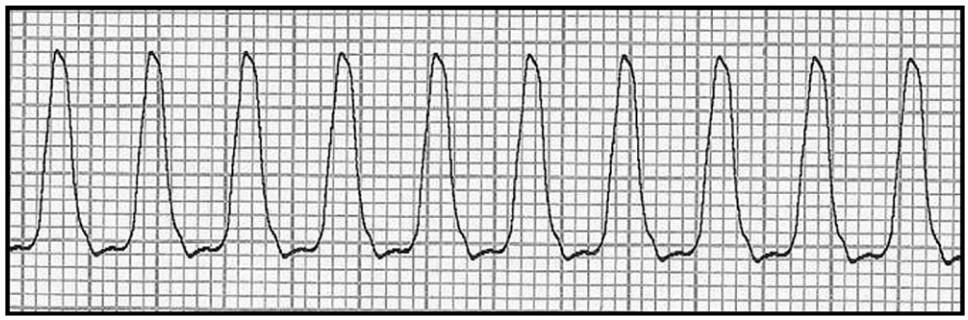

Fig. 2 Wide complex tachycardia in a young patient. The ventricular rate is very rapid, in excess of 240 bpm.

the hospital to a critical care unit with placement of transcutaneous pacer pads. Further monitoring demonstrated continued bradycardia. Serum cardiac markers were negative for myocardial infarction. The patient underwent placement of a permanent right ventricular pacemaker without complication. She was discharged from the hospital with bradycardia likely related to chronic conduction system disease exacerbated by the calcium channel blocking agent. She was well at follow-up.

2.2. Case 2

A 24-year-old man with no known medical history was evaluated by emergency medical service for acute weakness. Vital signs were significant for a blood pressure of 70 mm Hg, a pulse of 200 bpm, and a respiratory rate of 32 per minute. The cardiac monitor revealed a wide complex tachycardia at a rate of 200 bpm (Fig. 2). Immediate direct current cardioversion was administered with an initial energy of 100 J which successfully converted the rhythm to sinus tachycardia. He was transported uneventfully to the ED. Upon ED arrival, the patient lost consciousness; the cardiac monitor revealed a recurrent wide complex tachycardia. Immediate electrical cardioversion was performed with the return of sinus tachycardia. Twelve-lead ECG (Fig. 3) demonstrated normal sinus rhythm with shortened PR interval, widened QRS complex, and delta wave—all findings consistent with Wolff-Parkinson-White (WPW) syndrome. The patient received intravenous procainamide and was admitted to the hospital. Electrophysiologic study revealed an accessory pathway (AP) with anterograde conduction properties. Because of the patient's initial presentation and electrophysiologic study result, he underwent ablative therapy (transcatheter radiofrequency technique) with good results; no further rhythm disturbances were noted during 2 years of follow-up.

2.3. Case 3

A 38-year-old man presented to the ED complaining of 3 brief episodes of severe lightheadedness; approximately 30 minutes before presentation, he noted a full syncopal

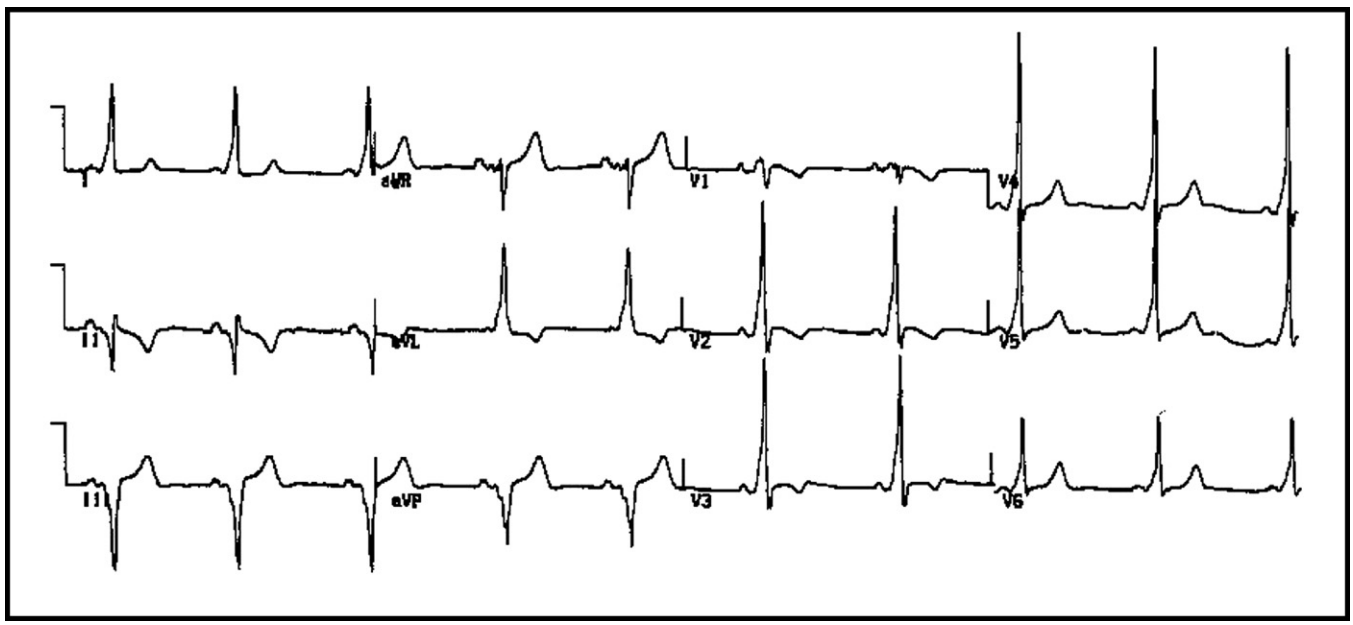

Fig. 3 Wolff-Parkinson-White syndrome. Note the classic triad, including the shortened PR interval, delta wave, and widened QRS complex. Pseudo-ischemic findings, such as Q waves and T-wave inversion, are also seen and do not represent sequelae of coronary artery disease.

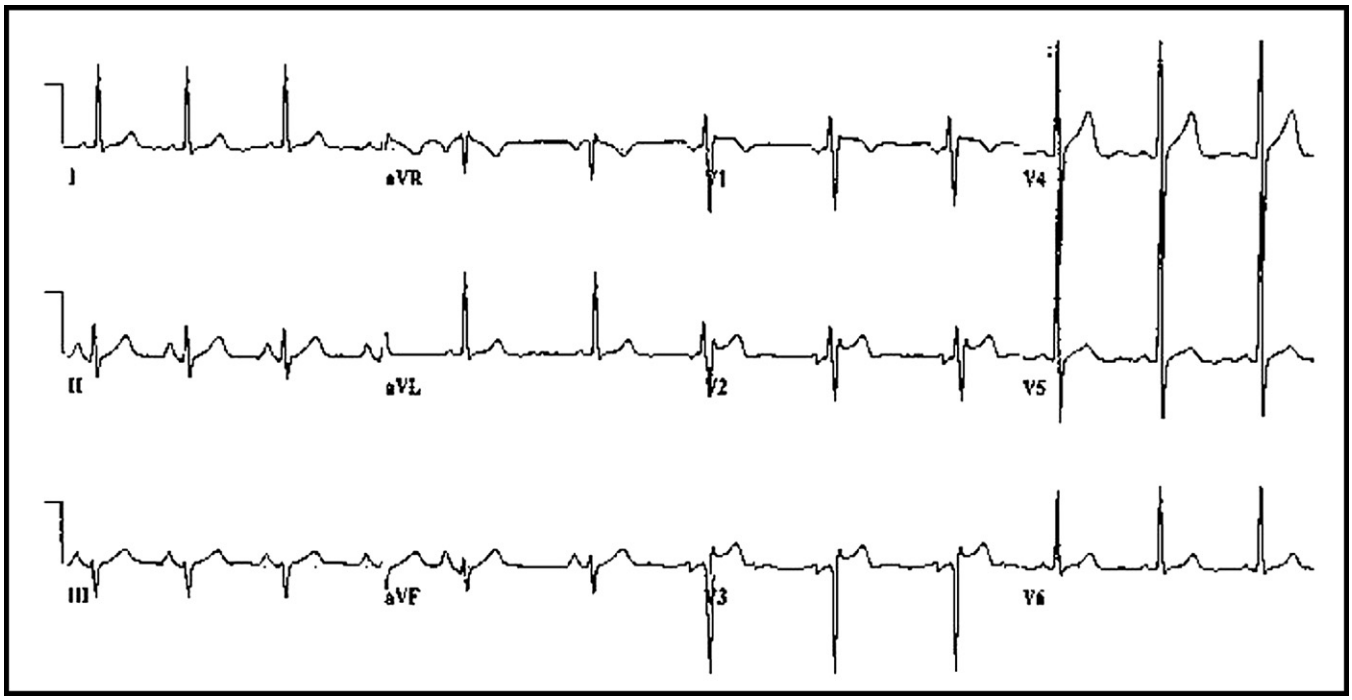

Fig. 4 Brugada syndrome. Note the incomplete RBBB with ST-segment elevation in leads V1 to V3.

episode. He denied any prior medical history or family history of cardiac disease. On arrival to the ED, his vital signs and physical examination were normal. An ECG was obtained (Fig. 4) and interpreted as normal sinus rhythm with incomplete right bundle branch block (RBBB) and ST-segment elevation in the right precordial leads. While in the ED awaiting serum chemistry results, he experienced recurrent syncope with ventricular fibrillation (Fig. 5). After approximately 45 seconds, the dysrhythmia converted to sinus rhythm after a single electrical cardioversion of 100 J; the patient regained consciousness. He was admitted to the hospital with pulseless ventricular tachycardia. At electrophysiology study, inducible polymorphic ventricular tachycardia (PVT) was noted. He was ultimately diagnosed with Brugada syndrome and received an internal cardiac defibrillator. He was well at 12-month follow-up.

2.4. Case 4

A 12-year-old boy without past medical history experienced a syncopal event while playing basketball. Upon regaining consciousness, the patient felt well and attempted to return to the game with recurrence of the syncope. He was transported to the ED by his parents for evaluation. Family history was significant for the sudden, unexplained death of a paternal uncle in the teenage years. Examination in the ED revealed a young male patient, alert and in no obvious distress. A 12-lead ECG demonstrated normal sinus rhythm with a prolonged QT interval; the QTc interval was approximately 560 milliseconds (Fig. 6). Serum chemistries were within normal limits. The patient was admitted to the hospital under the care of a cardiologist. Because of the patient's presentation (syncope, family history, and abnor-

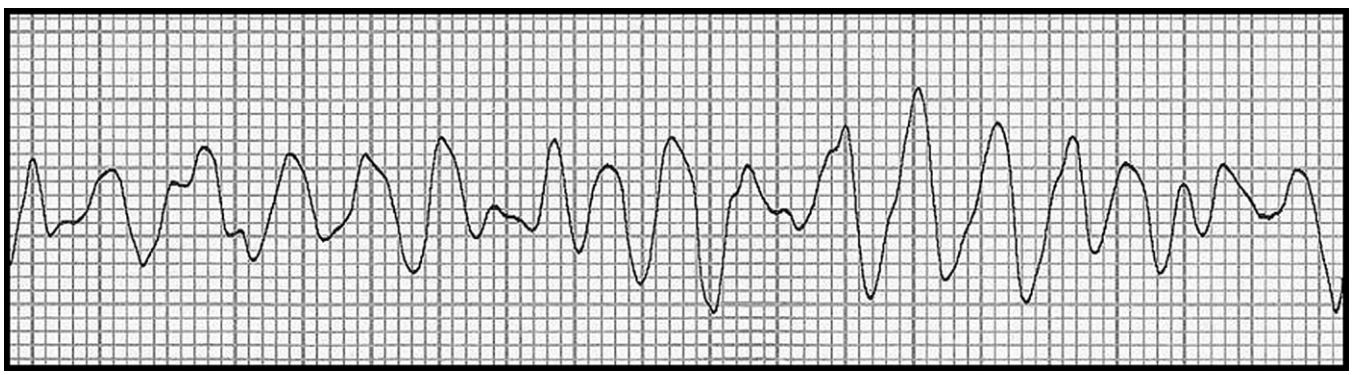

Fig. 5 Coarse ventricular fibrillation. This malignant dysrhythmia is seen in many cardiac arrest scenarios. Regarding the "syncope" patient, it is also encountered in the Brugada syndrome as well as the LQTS and hypertrophic cardiomyopathy.

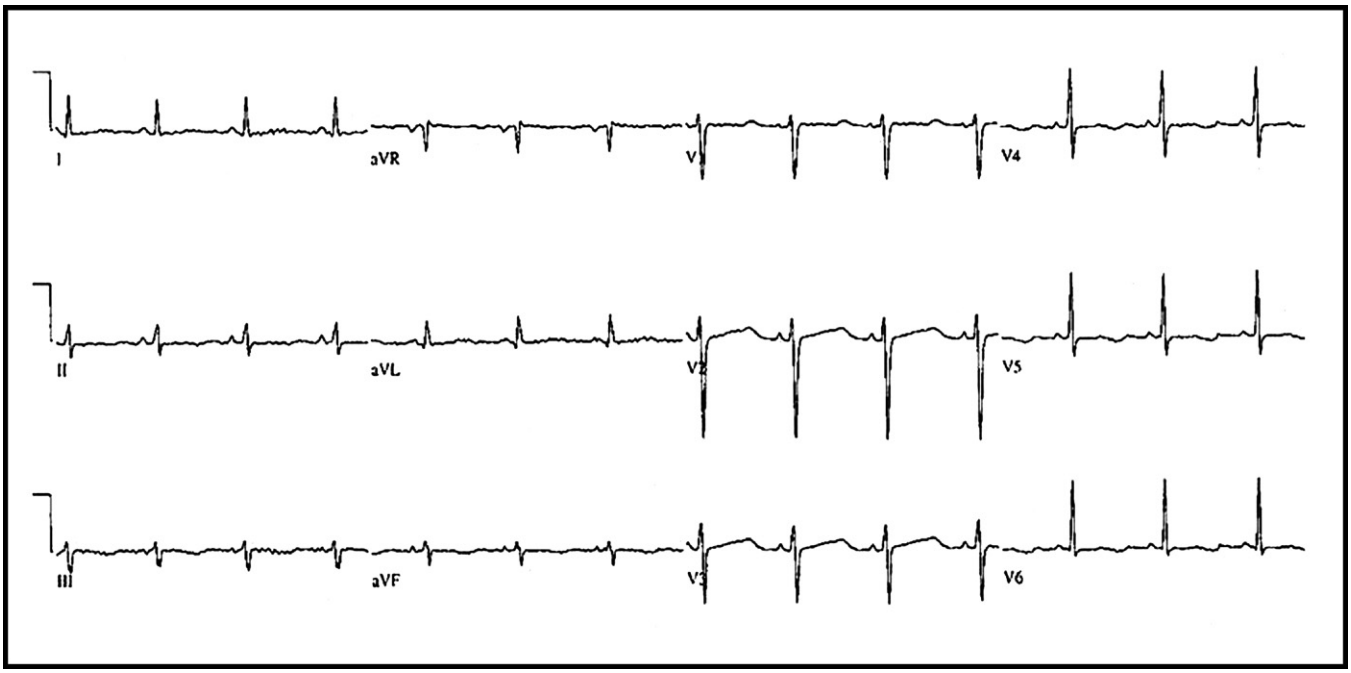

Fig. 6 Long QT syndrome.

mal ECG), a familial long QT syndrome (LQTS) was suspected; as such, metoprolol therapy was initiated. The patient was discharged home and was well at follow-up without recurrent complaint.

2.5. Case 5

A 31-year-old woman presented to the ED complaining of syncope; the syncope was preceded by palpitations and occurred during physical exertion. The patient reported that she had presented to another ED on separate occasions in the past 6 months for similar complaints. These episodes were preceded by palpitations. The patient denied any past medical history, family history of early cardiac disease, or drug use. The patient remained asymptomatic at the time of ED arrival. Her vital signs and physical examination, including cardiac examination, were normal. An ECG was obtained (Fig. 7). The ECG was suggestive of hypertrophic cardiomyopathy (HCM). An echocardiogram confirmed the diagnosis. The patient was then admitted to the hospital and treated with metoprolol. At 6-month follow-up, she was well without recurrent symptoms.

3. Discussion

Certain electrocardiographic presentations in the patient with syncope will not only provide a reason for the loss of consciousness but also guide early therapy and disposition. Bradycardia, AV block, intraventricular conduction abnormality, and tachydysrhythmia in the appropriate clinical setting provide an answer to the clinician for the syncopal event. Morphologic findings suggesting the range of cardiovascular malady are also encountered; these entities are far ranging, including the various ST-segment and T-wave abnormalities of acute coronary syndrome (ACS), ventricular preexcitation as seen in the WPW syndrome, Brugada syndrome with the associated tendency for sudden death, prolonged QT interval common in the diverse long QT interval presentations, and right ventricular hypertrophy suggestive of HCM. It must be noted, however, that ACS presentations are rarely manifested solely by syncope with the exception of the very elderly patient who might present with a multitude of symptoms or signs.

The most appropriate electrocardiographic approach to these patients is an initial review aimed at the detection of malignant dysrhythmia; this first electrocardiographic evaluation most often involves the electrocardiographic rhythm strip (Fig. 1A). If the electrocardiographic rhythm strip does not yield an answer, then the 12-lead ECG can be performed. The 12-lead ECG can provide not only a more detailed review of a challenging rhythm presentation but also the various morphologic findings as noted above. Of course, a "negative" ECG itself does not "rule out" either cardiac pathology or serious etiology. Refer to Fig. 8 for electrocardiographic rhythm examples of patients presenting with syncope.

Investigators have studied the ECG in the patient with syncope with the aim of identifying the individual at risk for adverse outcome. For instance, Martin et al [9] endeavored to develop and validate a risk classification system for patients presenting to the ED with syncope. In a 2-step analysis, the investigators reviewed the presentation in

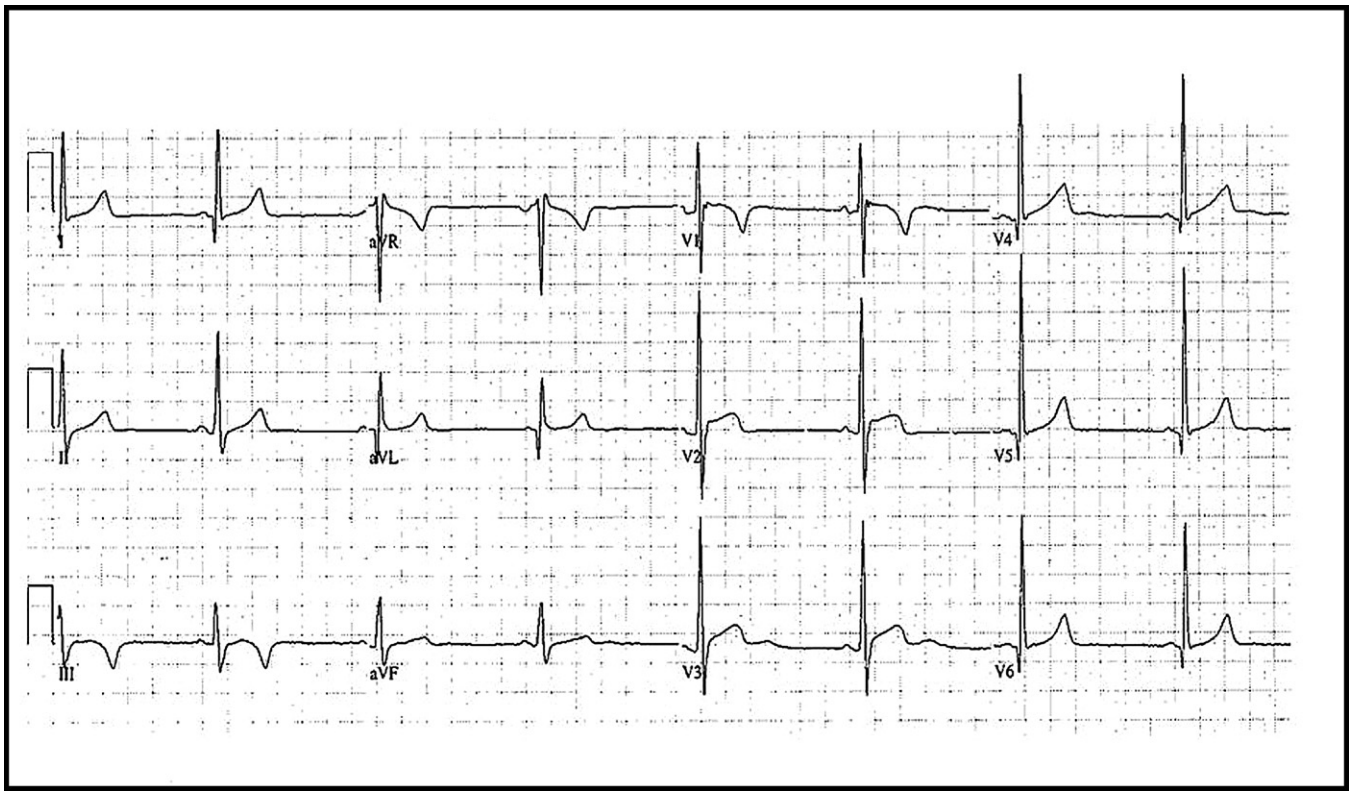

Fig. 7 Hypertrophic cardiomyopathy. Characteristic findings of left ventricular hypertrophy are common and include high-voltage R waves in the anterolateral leads (V4, V5, V6, I, and aVL). Prominent R waves may be present in leads V1 and V2 as well. The most frequent abnormalities found are large amplitude QRS complexes consistent with LVH and associated ST-segment and T-wave changes. Deep, narrow Q waves are common in the inferior (II, III, aVF) and lateral (I, aVL, V5, and V6) leads. The morphology of the Q waves—deep and very narrow—is perhaps the most characteristic and specific finding of HCM. Although large amplitude QRS complexes are the most common finding in HCM, deep narrow Q waves in the lateral leads are the most *specific* finding.

612 patients; they found that an abnormal ECG was associated with arrhythmia or death with an odds ratio (OR) of 3.2; other factors suggestive of a poor outcome included histories of congestive heart failure (OR, 3.2) and ventricular arrhythmia (OR, 4.8) [9]. Additional work performed by Sarasin et al [4] considered the subset of patients with unexplained syncope after an initial ED evaluation. In 344 patients, the investigators found that an abnormal ECG was a predictor of arrhythmia with an OR of 8.1; other factors of significance associated with arrhythmic syncope included older age (OR, 5.4) and a history of congestive heart failure (OR, 5.3). In patients with 1 risk factor, arrhythmia was encountered rarely (0%-2%); in patients with identified risk factors, arrhythmia occurred at the following frequencies: 1 risk factor, 0% to 2%; 2, 35-41%; and 3, 27% to 60%. The San Francisco Syncope Rule [3] incorporates the ECG in the evaluation of the patient with syncope; in this study, the investigators considered 684 presentations of syncope and reviewed clinical variables with the intent of identifying the patient at risk of poor short-term outcome. They not only found that an abnormal ECG was associated with an increased risk of short-term adverse event but also noted that dyspnea, low hematocrit, and hypotension were also predictors of poor outcome.

3.1. Wolff-Parkinson-White syndrome

Approximately 80 years ago, Wolff et al [10] reported the combination of bundle branch block, abnormally short PR interval, and paroxysms of tachycardia occurring in young, healthy patients with normal hearts. This original statement nicely describes the syndrome that now bears their names. Wolff-Parkinson-White syndrome is an uncommon but potentially serious form of ventricular preexcitation caused by an accessory conduction pathway linking the atria to the ventricles. It is thought to occur in 0.1% to 3% of the population and to account for 2.4% of cases of supraventricular arrhythmia seen in the ED [11]. The importance of recognizing this syndrome is that patients with WPW are prone to develop a variety of supraventricular tachyarrhythmias which may lead to unpleasant, disabling symptoms, and, in the extreme, sudden cardiac death.

Wolff-Parkinson-White syndrome is a form of ventricular preexcitation involving an accessory conduction pathway [12]. The AP bypasses the AV node, creating a direct electrical connection between the atria and ventricles. Four electrocardiographic features characterize WPW (Figs. 3 and 9): (1) a PR interval less than 0.12 seconds during sinus rhythm; (2) a slurring of the initial phase of the QRS

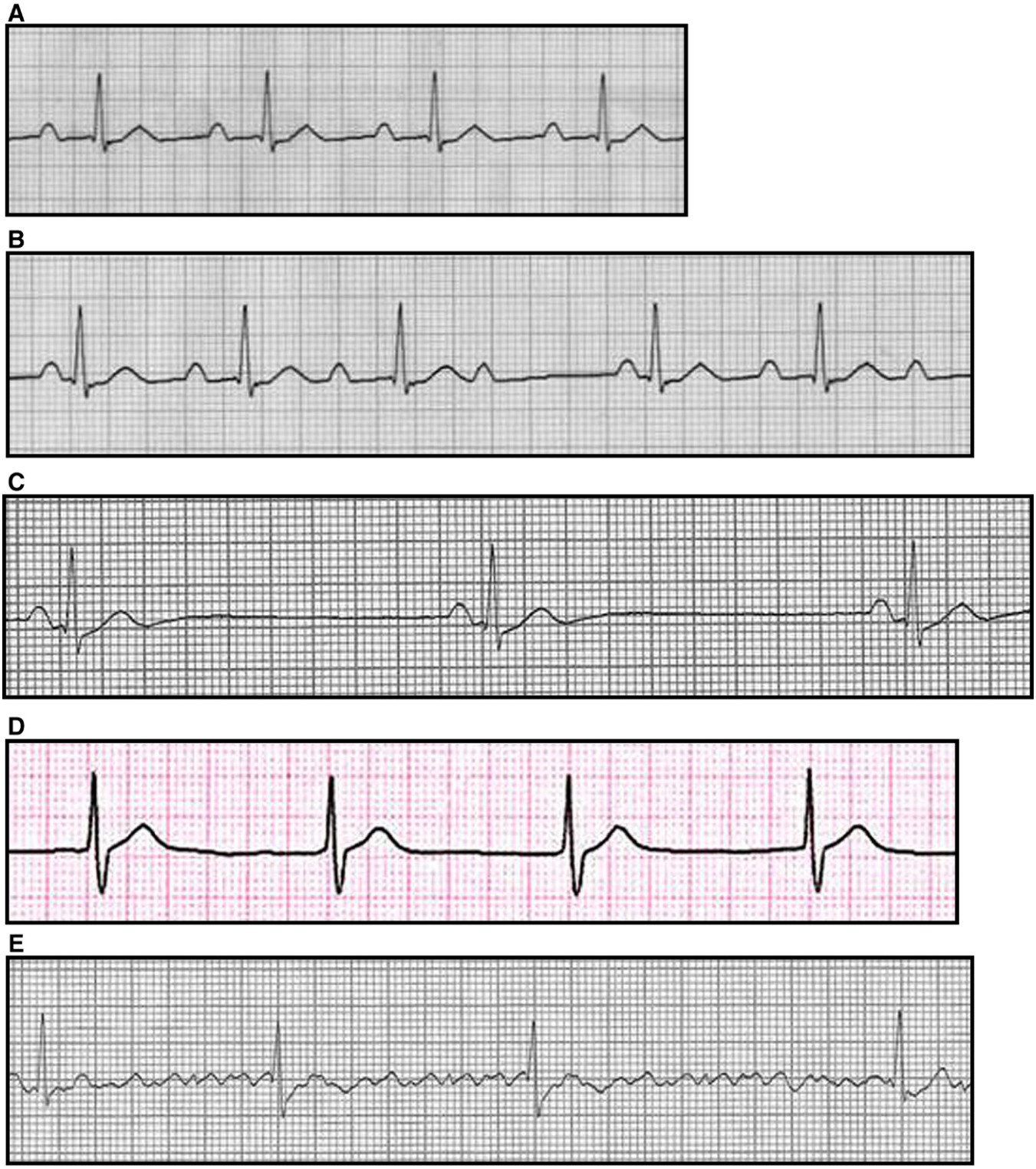

Fig. 8 Other electrocardiographic findings in the syncope patient. In addition to these findings, other more obvious findings can also be seen, such as ventricular tachycardia, third-degree AV block, and various ACS abnormalities. Specific findings indicative of impulse generation and conduction abnormality include the following: A, Sinus rhythm with first-degree AV block. B, Sinus rhythm with second-degree AV block. C, Sinus bradycardia. D, Junctional rhythm. E, Atrial fibrillation with slow ventricular response.

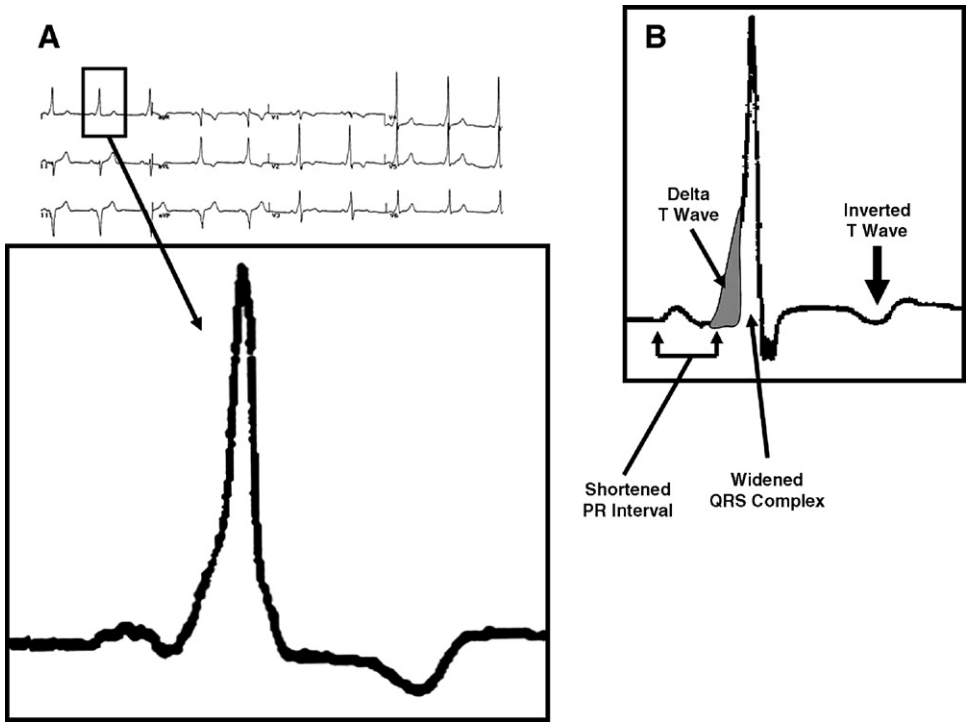

Fig. 9 Wolff-Parkinson-White syndrome. A, Note the classic triad, including the shortened PR interval, delta wave, and widened QRS complex. B, The delta wave is shaded in gray, shortened PR interval, widened QRS complex, and inverted T wave.

complex, called a delta wave; (3) QRS complex duration greater than 0.12 seconds; and (4) secondary ST-segment–T-wave changes directed opposite to the major delta wave and QRS complex changes [11,13].

The electrophysiology producing these electrocardiographic changes is easily explained by the mechanism of the AP. To begin with, the shortened PR interval represents the lack of conduction delay in the AP that would normally occur within the AV node. Once the aberrantly conducted impulse reaches the ventricular myocardium along with the normal impulse, the result is a widened QRS complex representing a fused activation via the 2 pathways. The delta wave illustrates the AP impulse, whereas the normal terminal portion of the QRS complex represents activation via the His-Purkinje system resulting from both the normal impulse and that of the AP [11].

Recognition of the ECG pattern is essential in the patient with syncope as these patients are susceptible to various supraventricular tachyarrhythmias, including paroxysmal supraventricular tachycardia (PSVT, 70%), atrial fibrillation (25%), atrial flutter (5%), and, rarely, ventricular fibrillation (Figs. 2 and 10) [11]. The most frequently encountered rhythm disturbance seen in the patient with WPW is PSVT—also known as AV reciprocating tachycardia. In the setting of such PSVT, activation of the ventricle occurs through either the normal conduction system and/or the AP with return of the impulse to the atrium by the other pathway. Such PSVT is referred to as either orthodromic or antidromic. The orthodromic, or anterograde, AV reciprocating tachycardia is a reentrant tachycardia in which the atrial stimulus is conducted to the ventricle through the AV node with a return of the impulse to the atria through the AP. Such tachycardia represents approximately 90% of PSVT cases seen in the patient with WPW. The conduction characteristics of this tachycardia are shown in the ECG in Fig. 10A—a narrow QRS complex, without a delta wave (as the ventricles are being activated through the normal conduction pathway), with ventricular rates ranging from 160 to 220 bpm in the adult and 160 to 260 bpm in the child.

In approximately 10% of patients with PSVT with WPW, an antidromic (retrograde) reciprocating tachycardia is observed. In this instance, the reentrant circuit conducts in the opposite direction, with anterograde conduction down the AP and return of the impulse retrograde to the atria via the bundle branches, His-Purkinje fibers, and AV node. With this pathway, the QRS complexes appear wide (essentially, an exaggeration of the delta wave) and the 12-lead ECG displays a very rapid, wide-complex tachycardia that is nearly indistinguishable from that of ventricular tachycardia (Figs. 2 and 10B).

Atrial fibrillation occurs more frequently in patients with WPW and is found in up to 20% of patients with symptomatic arrhythmia. This dysrhythmia is diagnosed with a very rapid, wide, bizarre, and irregular tachycardia. The AP in this instance lacks the feature of slow, decremental conduction—thus, the pathway can conduct atrial beats at a rate that can approach or exceed 300 bpm, subjecting the ventricle to very rapid rates (Fig. 10C). The important electrocardiographic clues for preexcited atrial fibrillation are the irregularity of the rhythm, the rapid

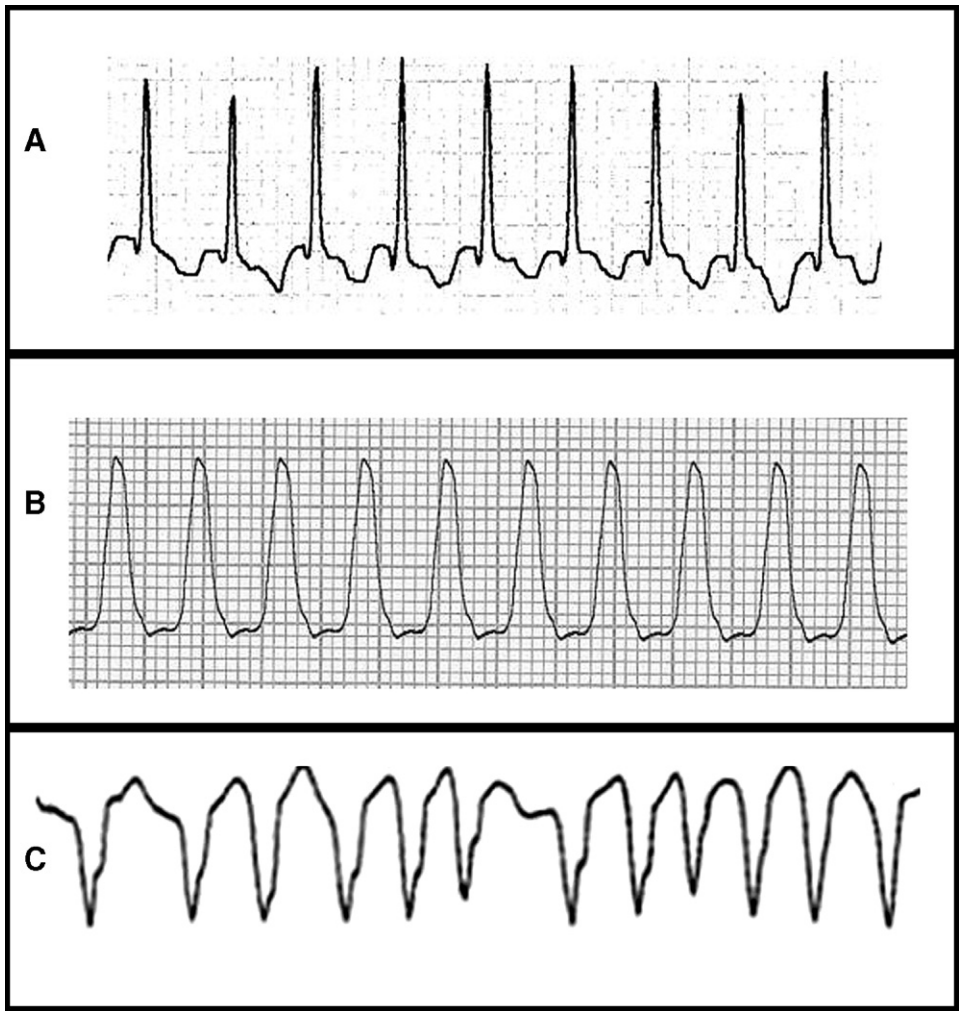

Fig. 10 Dysrhythmias seen in the WPW syndrome. A, Narrow complex tachycardia, an orthodromic tachycardia. B, Wide complex tachycardia, an antidromic tachycardia. C, Atrial fibrillation with wide QRS complex.

ventricular response (much too rapid for conduction down the AV node), and the wide, bizarre QRS complex, signifying conduction down the aberrant pathway.

3.2. Brugada syndrome

The Brugada syndrome was first described in 1992 as a unique set of electrocardiographic abnormalities associated with sudden death in otherwise healthy adults without structural heart disease [14]. Patients with this syndrome were noted to have a distinct set of electrocardiographic abnormalities, characterized by an RBBB pattern with ST-segment elevation in the right precordial leads (Fig. 4). This syndrome is known as the "Brugada syndrome." [14] Although originally thought to be primarily a disease in men of Southeast Asian descent, more recent reports have noted the presence of this deadly syndrome in women and children in most other areas of the world [15-18]. The Brugada syndrome is now suspected to be responsible for a large portion of patients with what was felt to be "idiopathic ventricular fibrillation" [19].

Patients with the Brugada syndrome have recurrent episodes of ventricular tachycardia (most commonly polymorphic) [15]. If this dysrhythmia is persistent, it eventually degenerates into ventricular fibrillation and results in sudden death (unless promptly treated); in other cases, ventricular fibrillation is the initial cardiac arrest rhythm. Conversely, if the dysrhythmia is self-terminating, it usually results in syncope or near syncope depending on its duration. Patients with self-terminating episodes of ventricular tachycardia will often present for evaluation of these symptoms.

The hallmark electrocardiographic findings, known as the "Brugada sign," include (1) RBBB and (2) ST-segment elevation in the right precordial leads (Fig. 11). It is important to note, however, that the RBBB may be incomplete and that the ST elevation in V1 to V3 may be minimal [20]. In addition, these electrocardiographic changes may be transient over time [20,21]. The pathologic mechanism leading to these electrocardiographic changes and the clinical syndrome is a mutation in the SCN5A cardiac voltage-gated sodium channel gene on chromosome 3 [21,22]. This mutation leads to a reduction in the fast

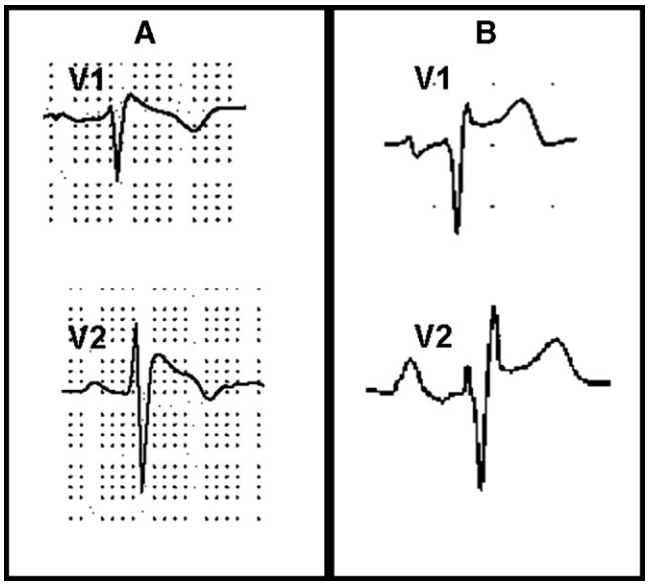

Fig. 11 Brugada syndrome. ST-segment elevation is found in the right precordial leads. This elevation can take one of two distinct formats, including the coved, or downsloping, and saddle-shaped types. A, Coved, downsloping type. B, Saddle-shaped type.

sodium channel current and a premature termination of the right ventricular epicardial action potential [21-23]. This loss of action potential does not affect the endocardium and the resulting epicardial-endocardial voltage gradient is thought to produce the observed electrocardiographic changes as well as the clinical syndrome of PVT [21,22].

Because various shapes of ST-segment elevation have been observed, there has been a recent attempt to standardize these electrocardiographic findings by type [22]. For example, type I refers to a coved-shaped ST-segment elevation of greater than or equal to 2 mm in more than one right precordial lead, followed by a negative T wave (Fig. 11A). Type II describes a saddle-shaped ST-segment elevation (Fig. 11B), whereas type III refers to elevations less than 2 mm [22]. At this time, only type I electrocardiographic changes are classified as being diagnostic of the Brugada syndrome, although a type II morphology ST-segment change in the appropriate patient should prompt consideration for the diagnosis [22].

The clinical consequences of Brugada syndrome are unpredictable bouts of PVT which, if continuous, may lead to ventricular fibrillation (Fig. 5) and sudden cardiac death [20]. Electrophysiologic testing is recommended to confirm the diagnosis in any patient with the appropriate clinical presentation, suggestive electrocardiographic findings, and risk factors, including aborted sudden cardiac arrest, unexplained syncope, family history of sudden cardiac death, and Southeast Asian ethnicity [21]. Prompt placement of an internal defibrillator in those patients with a confirmed diagnosis leads to an excellent prognosis with a 10-year mortality of 0% in one study [24], whereas failure to diagnose and treat has a mortality rate of approximately 30% at 2 years [15].

Other clinical syndromes may mimic the Brugada-type electrocardiographic changes. The Brugada sign has been shown to occur in cases of right ventricular pathology such as mediastinal tumor compressing the right ventricular free wall as well as hemopericardium compressing the right ventricle [21]. It is also seen in the hyperacute stage of acute right ventricular infarction and contusion of the right ventricle [21]. The Brugada sign is also known to be produced by drugs such as class 1A and 1C anti-arrhythmic agents, cocaine, and tricyclic antidepressants [21]. Hyperkalemia and hypercalcemia have also on occasion been responsible for the Brugada-like changes on the ECG [21]. Certainly, the EP must consider these potential clinical entities in the patient with the appropriate electrocardiographic findings. Yet, according to recent expert opinion, patients with an incidental Brugada sign and without appropriate symptoms or risk factors do not require further diagnostic testing [21].

3.3. Long QT syndrome

Long QT syndrome is an electrophysiologic cardiac disorder in which the repolarization phase of the ventricular action potential is lengthened. It is manifested as a prolongation of the QT interval on the ECG (Fig. 6) and is clinically significant in that it is associated with the development of PVT (Fig. 12) and sudden cardiac death. Along the same lines as the Brugada syndrome, the LQTS has been identified as another cause of sudden cardiac death

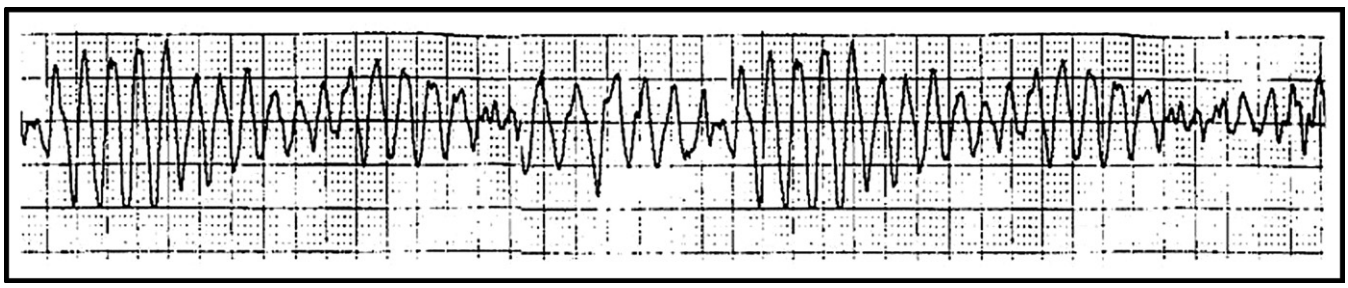

Fig. 12 Polymorphic ventricular tachycardia, not uncommonly seen in patients with the LQTS. This subtype of PVT is called torsade de pointes and is diagnosed most appropriately in the setting of abnormal repolarization states manifested by prolongation of the QT interval on the ECG. Torsade de pointes is rather elegant in appearance with a progressively varying polarity and amplitude of the QRS complex.

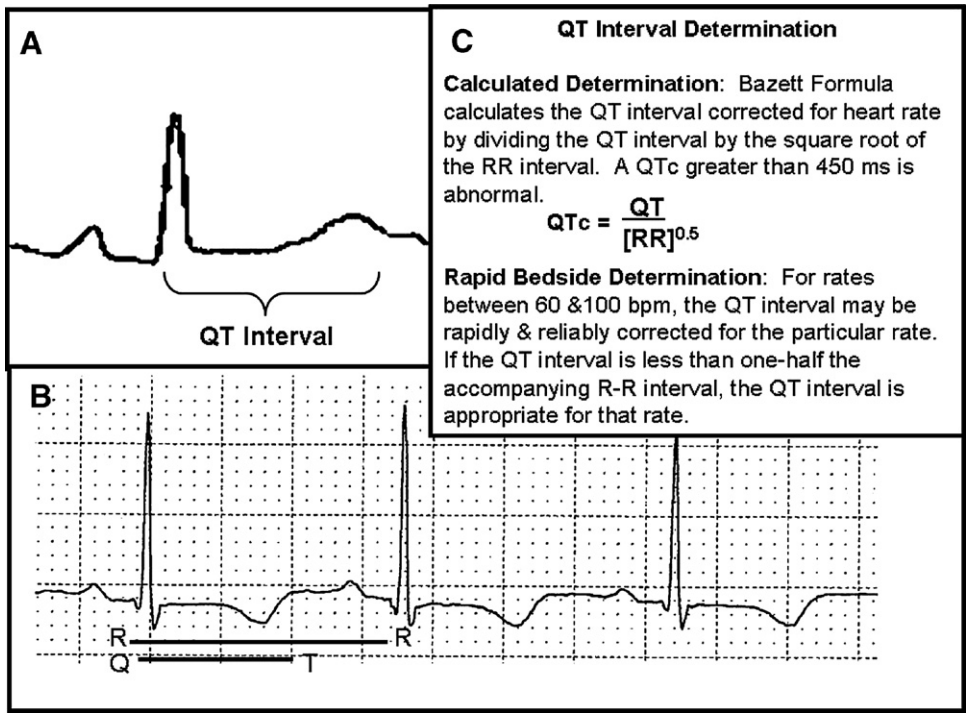

Fig. 13 Long QT syndrome. A, Prolonged QT interval. B, Prolonged QT interval. A simple bedside test can be used to determine whether the QT interval is prolonged for the individual patient. If the QT interval is longer than one half the accompanying RR interval, then the QT interval is excessively long. C, QT Interval determination. Calculated determination: Bazett formula calculates the QT interval corrected for heart rate by dividing the QT interval by the square root of the RR interval. A QTc greater than 450 milliseconds is abnormal. Rapid bedside determination: for rates between 60 and 100 bpm in sinus rhythm, the QT interval may be rapidly and reliably corrected for the particular rate. If the QT interval is less than one half the accompanying R-R interval, the QT interval is appropriate for that rate.

in otherwise healthy people with structurally normal hearts. Unlike the Brugada syndrome, patients with LQTS, however, usually present in the first or second decade of life, with 32 years being the median age of those who die of LQTS [25]. Furthermore, patients with LQTS tend to experience symptoms when precipitated by physical or emotional stress.

Congenital and acquired forms of LQTS are encountered, the former due to abnormalities in transmembrane ion channels and the latter due to multiple causes including medications and toxins, electrolyte imbalance, bradycardia, central nervous system event, ACS, autonomic neuropathy, and the human immunodeficiency virus [26].

As the name implies, the most obvious electrocardiographic finding in LQTS is a prolonged QTc interval (Fig. 13A and B). Typically, the QT interval lengthens with age and is longer in women than in men; it also varies inversely with heart rate. Regarding sex issues, the QT

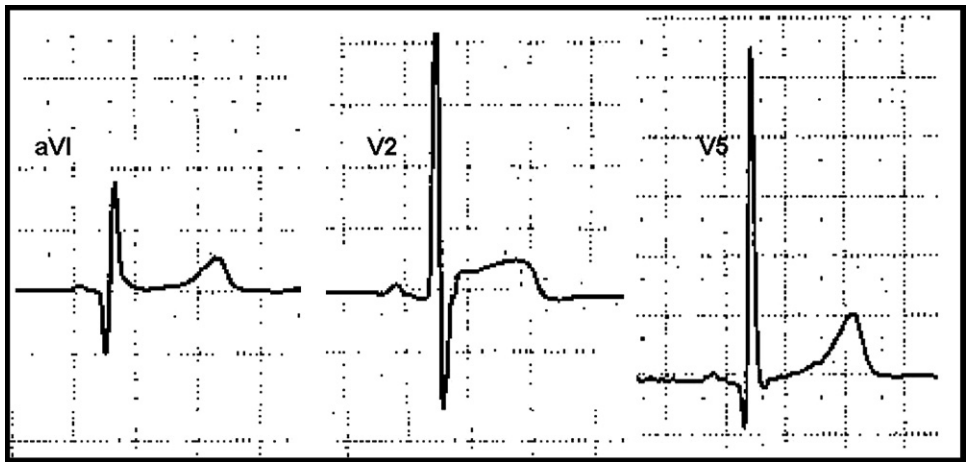

Fig. 14 Electrocardiographic findings of hypertrophic cardiomyopathy. In lead aVl, the prominent, narrow Q wave is seen. In lead V2, a very prominent R wave is also seen accompanied here by ST-segment elevation. Lead V5 reveals a significant Q wave with a very large R′ wave.

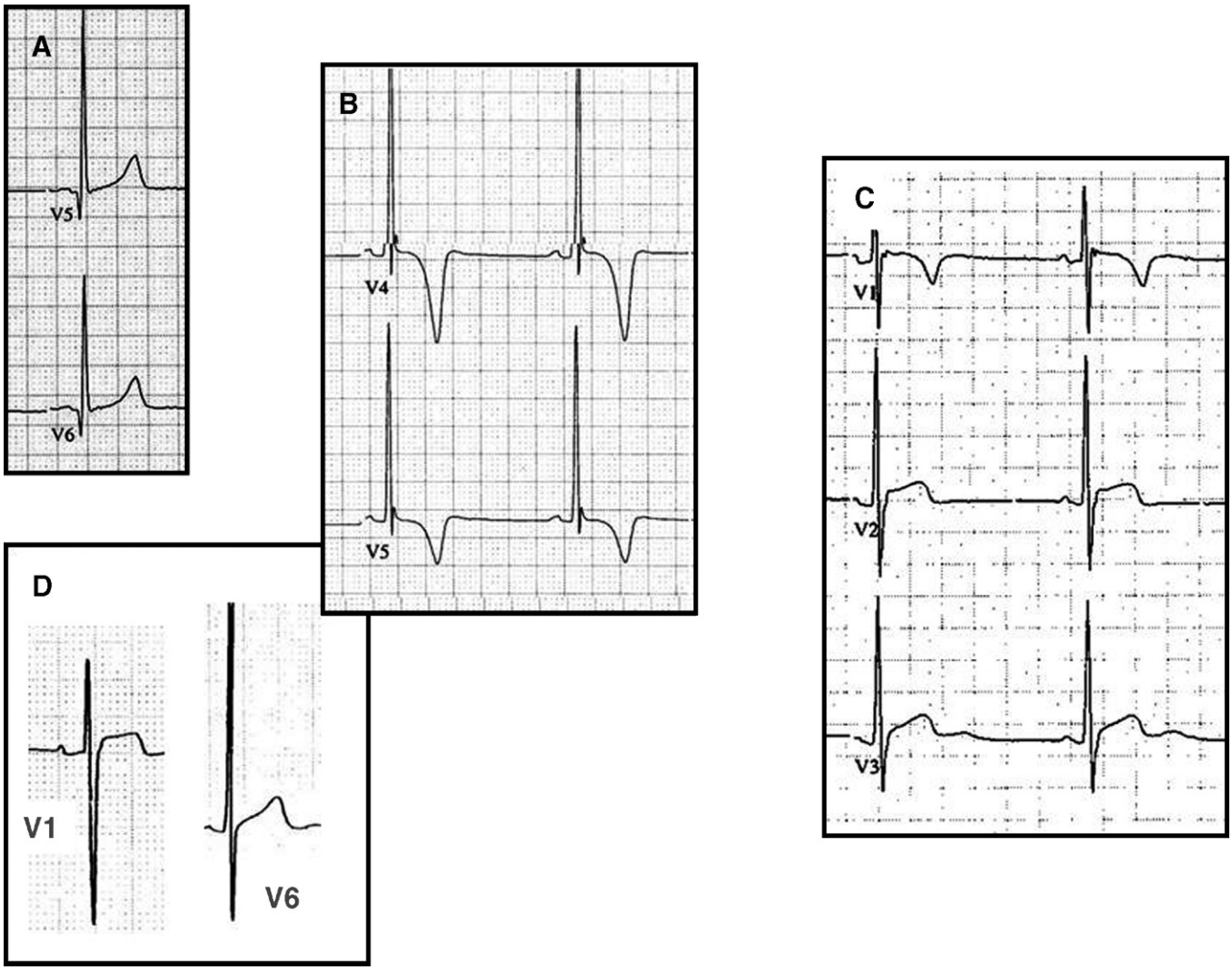

Fig. 15 Electrocardiographic features of hypertrophic cardiomyopathy. A, Narrow Q waves seen in leads V5 and V6. B, Deeply inverted T waves. C, ST-segment and T-wave changes resembling those findings encountered in ACS. D, Electrocardiographic LVH pattern with prominent S wave in lead V1 and R wave in lead V6.

interval is considered abnormal if greater than 440 milliseconds in men and greater than 460 milliseconds in women—the generally accepted maximum being approximately 450 milliseconds [25]. This finding, however, is not present in all patients with the syndrome. In fact, the majority of patients with LQTS will have a QTc greater than 440 milliseconds. Approximately 6% to 12% of patients with LQTS, however, will actually have a normal QTc interval. Prior studies have shown that in congenital LQTS, a 100% positive predictive value for the disease (ie, rule-in the syndrome) occurs with a QTc greater than 470 milliseconds in men and greater than 480 milliseconds in women [27]. Likewise, the 100% negative predictive value (ie, rule-out the syndrome) occurs with a QTc less than 390 milliseconds in men and less than 410 milliseconds in women [27]. These last statements assume, of course, that this electrocardiographic finding is noted in the appropriate clinical presentation. The simple observation of QT interval prolongation itself does not necessarily identify a patient at risk—it must be interpreted within the context of the clinical scenario.

Other electrocardiographic findings supporting the diagnosis can assist in the evaluation, particularly when the QTc interval is normal. These other electrocardiographic findings suggestive of the LQTS include T-wave and U-wave abnormalities. The QT interval may be more variable and T waves may be larger, prolonged, or bizarre looking, and may have a bifid, biphasic, or notched appearance. T-Wave alternans is a rare but diagnostic characteristic of LQTS where beat-to-beat variability is seen in the T-wave amplitude. This variability is due to the increased electrical instability during repolarization. The U-wave abnormalities seen include bizarre-looking and pronounced U waves. U-Wave alternans may also be seen.

The QTc interval may be calculated using the Bazett formula or via comparative measurements to the accompa-

nying R-R interval. These techniques are depicted in Fig. 13B and C.

3.4. Hypertrophic cardiomyopathy

Another cause of syncope with potentially lethal consequences is HCM. Hypertrophic cardiomyopathy is a heterogeneous genetic disorder that affects proteins of the cardiac sarcomere and is seen in 1 of every 500 adults in the general population [28]. Many patients remain asymptomatic throughout their lives; there is potential, however, for dyspnea, exercise intolerance, angina, syncope, and sudden death due to the mechanical effects of this disorder [28,29]. The classic pattern of hypertrophy occurs in an asymmetric manner along the ventricular septum, although apical, midventricular, and concentric hypertrophy have also been observed [29]. Symptoms occur when the hypertrophied basal septum partially blocks the outflow tract of the left ventricle creating a functional obstruction [28,29]. In addition, patients with severe obstruction may experience anterior motion of the mitral valve into the left ventricle during systole, further reducing outflow and leading to mitral regurgitation [28,29]. Tragically, sudden cardiac death may be the first presenting symptom for patients with this disease; in fact, HCM is known to be the most common cause of sudden death in young athletes [28].

Key elements of the history in patients with HCM at risk for sudden cardiac death may include a history of unexplained syncope and a family history of sudden cardiac death, although frequently patients will report neither [30]. Other factors which have been related through various studies to an increased risk of sudden cardiac death in patients with HCM include severe left ventricular hypertrophy (LVH; maximum wall thickness >30 mm), resting peak instantaneous outflow tract gradient greater than 30 mm Hg, abnormal blood pressure response during exercise, and the presence of nonsustained ventricular tachycardia during 48-hour ambulatory ECG monitoring [30].

The ECG is abnormal in approximately 90% of patients with HCM [29]. Yet, a majority of the electrocardiographic abnormalities that are encountered in the patient with HCM are not specific for the diagnosis. In all patients with HCM, the most common electrocardiographic changes (Figs. 7, 14, and 15) include increased QRS complex voltage, QRS complex widening, Q waves, and ST-segment/T-wave changes consistent with ventricular hypertrophy [29]. In younger patients, these electrocardiographic findings are more specific for the diagnosis and should certainly be cause for concern in the appropriate setting [29]. In the apical form of HCM, giant T-wave inversions are typical and may be seen in other forms as well [29]. In the older patient in whom HCM is being considered, these electrocardiographic findings must be distinguished from those occurring in conditions such as chronic hypertension, ACS, ischemic heart disease, and various conduction abnormalties [29].

Perhaps the most specific electrocardiographic finding (Figs. 14 and 15), if present, in young patients with HCM is the appearance of Q waves in leads II, III, aVF, V5, and V6 in the early teenage years. A recent study suggests that this finding is the earliest electrocardiographic manifestation of certain patients with HCM, preceding both wall hypertrophy and other echocardiographic abnormalities [31]. In a large population of patients with HCM studied, ST-segment/T-wave changes were found in greater frequency after age 20 years; furthermore, the electrocardiographic LVH pattern increased with age, whereas conduction disturbances were primarily seen after age 40 years [31]. The appearance of these abnormal Q waves in the teens studied had a reported sensitivity of 67%, specificity of 100%, positive predictive value of 100%, and negative predictive value of 78% for the diagnosis of HCM [31]. Of course, these electrocardiographic findings, if considered of significance in the patient, must be noted in the appropriate clinical presentation.

References

[1] Miller TH, Kruse JE. Evaluation of syncope. Am Fam Physician 2005;72:1492-500.

[2] Quinn J, McDermott D, Stiell I, et al. Prospective validation of the San Francisco syncope rule to predict patients with serious outcomes. Ann Emerg Med 2006;47:448-54.

[3] Quinn J, Stiell I, McDermott D, et al. Derivation of the San Francisco Syncope Rule to predict patients with short-term serious outcomes. Ann Emerg Med 2004;43:224-32.

[4] Sarasin FP, Hanusa BH, Perneger T, et al. A risk score to predict arrhythmias in patients with unexplained syncope. Acad Emerg Med 2003;10:1312-7.

[5] Strickberger SA, et al. AHA/ACCF Scientific statement on the evaluation of syncope. J Am Coll Cardiol 2006;47:473-84.

[6] AHA/ACC guidelines summary ACC/AHA Cardiology Guideline Summaries, vol 101, 2001. p. 126-7.

[7] Lee TH. Braunwald's Heart Disease: a textbook of cardiovascular medicine. 6th ed. Saunders.

[8] Sun BC, Edmond JA, Camargo Jr CA. Inconsistent electrocardiographic testing for syncope in United States emergency departments. Am J Cardiol 2004;93:1306-8.

[9] Martin TP, Hanusa BH, Kapoor WN. Risk stratification of patients with syncope. Ann Emerg Med 1997;29:459-66.

[10] Wolff L, Parkinson J, White PD. Bundle-branch block with short PR interval in healthy young people prone to paroxysmal tachycardia. Am Heart J 1930;5:685-704.

[11] Rosner MH, Brady Jr WJ, Kefer MP, Martin ML. Electrocardiography in the patient with the Wolff-Parkinson-White syndrome: diagnostic and initial therapeutic issues. Am J Emerg Med 1999;17:705-14.

[12] Kent AFS. Researches on the structure and function of the mammalian heart. J Physiol 1893;14:233.

[13] Zipes D. Braunwald's heart disease: a textbook of cardiovascular medicine. 7th ed Saunders.

[14] Brugada P, Brugada J. Right bundle branch block persistent ST segment elevation and sudden death: a distinct clinical and electrocardiographic syndrome. A multicenter report. J Am Coll Cardiol 1992;20:1391-6.

[15] Brugada P, Brugada R, Brugada J. The Brugada syndrome. Curr Cardiol Reports 2000;2:507-14.

[16] Monroe MH, Littmann L. Two-year case collection of the Brugada syndrome electrocardiogram pattern at a large teaching hospital. Clin Cardiol 2000;23:849-51.

[17] Hermida J, Lemoine J, Aoun FB, et al. Prevalence of the Brugada syndrome in an apparently healthy population. Am J Cardiol 2000; 86:91-4.
[18] Priori SG, Napolitano C, Giordana U, et al. Brugada syndrome and sudden cardiac death in children. Lancet 2000;355:808-9.
[19] Alings M, Wilde A. "Brugada" syndrome—clinical data and suggested pathophysiological mechanism. Circulation 1999;99: 666-73.
[20] Mattu A, Rogers RL, Kim H, Perron AD, Brady WJ. The Brugada syndrome. Am J Emerg Med 2003;21:146-51.
[21] Littman L, Monroe MH, Kerns II WP, Svenson RH, Gallagher JJ. Brugada syndrome and "Brugada sign": clinical spectrum with a guide for the clinician. Am Heart J 2003;145(5):768-78.
[22] Ott P, Marcus FI. Electrocardiographic markers of sudden death. Cardiol Clin 2006;24:453-69.
[23] Poelzing S, Forleo C, Samodell M, et al. SCN5A polymorphism restores trafficking of a Brugada syndrome mutation on a separate gene. Circulation 2006;114:360-2.
[24] Brugada J, Brugada P, Brugada R. The syndrome of right bundle branch block ST segment elevation in V1 to V3 and sudden death—the Brugada syndrome. Cardiovasc Drugs Ther 2002;16: 25-7.
[25] Meyer JS, Mehdirad A, Salem BI, et al. Sudden arrhythmia death syndrome: importance of the long QT syndrome. Am Fam Physician 2003;68:483-8.
[26] Khan IA. Clinical and therapeutic aspects of congenital and acquired long QT syndrome. Am J Med 2002;112:58-66.
[27] Vincent GM, Timothy KW, Leppert M, Keating M. The spectrum of symptoms and QT intervals in carriers of the gene for the long-QT syndrome. N Engl J Med 1992;327:846-52.
[28] Nishimura RA, Holmes Jr DR. Hypertrophic obstructive cardiomyopathy. N Engl J Med 2004;350:1320-7.
[29] Popjes ED, Sutton MSJ. Hypertrophic cardiomyopathy: pathophysiology, diagnosis, and treatment (The heart). Geriatrics 2003;58(3):41-50.
[30] Frenneaux MP. Assessing the risk of sudden cardiac death in a patient with hypertrophic cardiomyopathy. Heart 2004;90:570-5.
[31] Shimizu M, Ino H, Yamaguchi M, et al. Chronologic electrocardiographic changes in patients with hypertrophic cardiomyopathy associated with cardiac troponin I mutation. Am Heart J 2002; 143(2):289-93.