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Selected Topics: Cardiology Commentary

ELECTROCARDIOGRAPHIC MANIFESTATIONS: VENTRICULAR TACHYCARDIA

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□ **Abstract—Ventricular tachycardia is a serious rhythm disturbance that originates from any part of the myocardium or conduction system below the atrioventricular node. Ventricular tachycardia (VT) presents with a wide QRS complex and a rate greater than 120 beats/min. Ventricular tachycardia is frequently encountered as a complication of coronary artery disease or cardiomyopathy; furthermore, VT is also seen in patients with medication adverse effect or electrolyte disturbance. Ventricular tachycardia presents electrocardiographically in several forms, including monomorphic and polymorphic VT. As is true in most Emergency Department presentations, the Emergency Physician must approach the dysrhythmic patient from an undifferentiated perspective, in this instance, the wide complex tachycardia (WCT) scenario. The electrocardiographic differential diagnosis of WCT classically includes VT and supraventricular tachycardia with aberrant intra-ventricular conduction. This article will review the electrocardiographic presentations encountered in patients with ventricular tachycardia.** © 2003 Elsevier Inc.

□ **Keywords—electrocardiogram (EKG); ventricular tachycardia; wide complex tachycardia; torsades de pointe**

INTRODUCTION

Ventricular tachycardia is a serious rhythm disturbance that originates from any part of the myocardium or conduction system below the atrioventricular node. In most instances, ventricular tachycardia (VT) presents with a wide QRS complex and a rate greater than 120 beats/min. In the rare case, VT may present with a "normal appearing" QRS complex; such cases involve either children with an age-related "narrow" QRS complex width or adult patients with cardiac glycoside toxicity. Ventricular tachycardia is frequently encountered as a complication of coronary artery disease (CAD) due to active ischemia or presence of scar tissue, which can create substrate for ventricular dysrhythmias. Patients with cardiomyopathy are the second most frequently encountered patient group experiencing VT. Medications, particularly type IA antidysrhythmic agents, can produce ventricular dysrhythmias; other drugs that may cause VT include digoxin, phenothiazines, tricyclic antidepressants, and the various sympathomimetic agents. Electrolyte disorders, particularly abnormalities of potassium, may produce VT.

The term *wide complex tachycardia* (WCT) describes the rhythm scenario characterized by a broad QRS complex (greater than 0.12 s) and a ventricular rate over 100 beats/min. The electrocardiographic differential diagnosis of WCT classically includes VT and supraventricular tachycardia (SVT) with aberrant intra-ventricular conduction. Numerous strategies aimed at assisting the cli-

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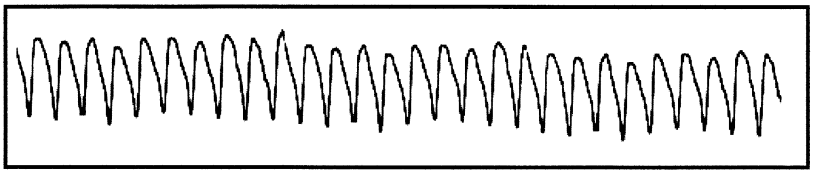

Figure 1. Case One: Wide complex tachycardia with a ventricular rate of approximately 180 beats/min. The QRS complex is quite wide with a single morphology, consistent with monomorphic VT.

nician with the proper diagnosis of WCT have been proposed, emphasizing various data including patient age, cardiovascular history, and the electrocardiogram (EKG) (1–9). This article will review the electrocardiographic presentations encountered in patients with ventricular tachycardia.

CASE PRESENTATIONS

Case 1

A 61-year-old man with past myocardial infarction noted the onset of chest pain followed by palpitations. The Emergency Medical Services (EMS) personnel were summoned and found the man alert and in no obvious distress. Vital signs included a blood pressure (BP) of 100/70 mm Hg, a pulse (P) of 175 beats/min (bpm), and a respiratory rate (R) of 30 breaths/min. The single-lead electrocardiographic monitor demonstrated a regular WCT at a rate of 180/min (Figure 1). Based upon the patient's age and medical history, as well as the nature of the QRS complex, the dysrhythmia was felt to be ventricular tachycardia.

A bolus of intravenous lidocaine was administered. While en route, the patient experienced a syncopal episode. A repeat determination of BP was palpable at approximately 70 mm Hg. Immediate direct-current cardioversion was performed and was successful in converting the VT to sinus tachycardia. The patient was admitted to the hospital after an uneventful Emergency Department (ED) course; subsequent 12-lead EKG analysis revealed normal sinus rhythm (NSR) with inferior ST segment depression. The patient demonstrated positive serial troponin T values consistent with acute myocardial infarction (AMI). Catheterization revealed mid-right coronary artery subtotal occlusion with thrombus that was successfully opened via angioplasty and stent placement. An automatic internal cardiodefibrillator was placed before discharge. The patient was discharged with non-ST segment elevation AMI complicated by VT.

Case 2

A 67-year-old man with a history of myocardial infarction presented to EMS personnel with the sudden onset of weakness and palpitation. He noted the onset of chest pain accompanied by palpitations. Paramedics found the patient to be alert with vital signs of a BP 130/100 mm Hg, a P of 170 bpm (regular), and a R of 24 breaths/min. The remainder of the examination was unremarkable. The single-lead EKG monitor revealed a regular WCT with atrioventricular dissociation (Figure 2). Transport time to the ED was less than 5 min; an i.v. was placed through which lidocaine was given and the patient was transported to the ED uneventfully.

On arrival, he remained alert with unchanged vital signs. Cardiopulmonary findings were significant for clear lung fields and a regular, rapid rhythm. Multi-lead rhythm strips and 12-lead EKG monitoring was initiated, revealing a regular WCT (Figure 3, A and B) at an

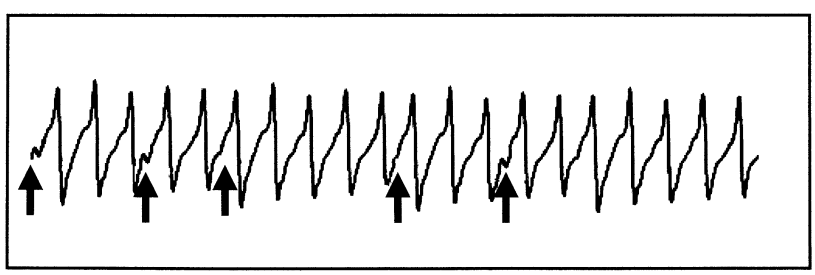

Figure 2. Case Two: Wide complex tachycardia with a ventricular rate of approximately 170 beats/min. The QRS complex is wide with a single morphology; additionally, atrioventricular dissociation is also seen (arrows). The above findings, particularly the presence of atrioventricular dissociation, are highly suggestive of VT.

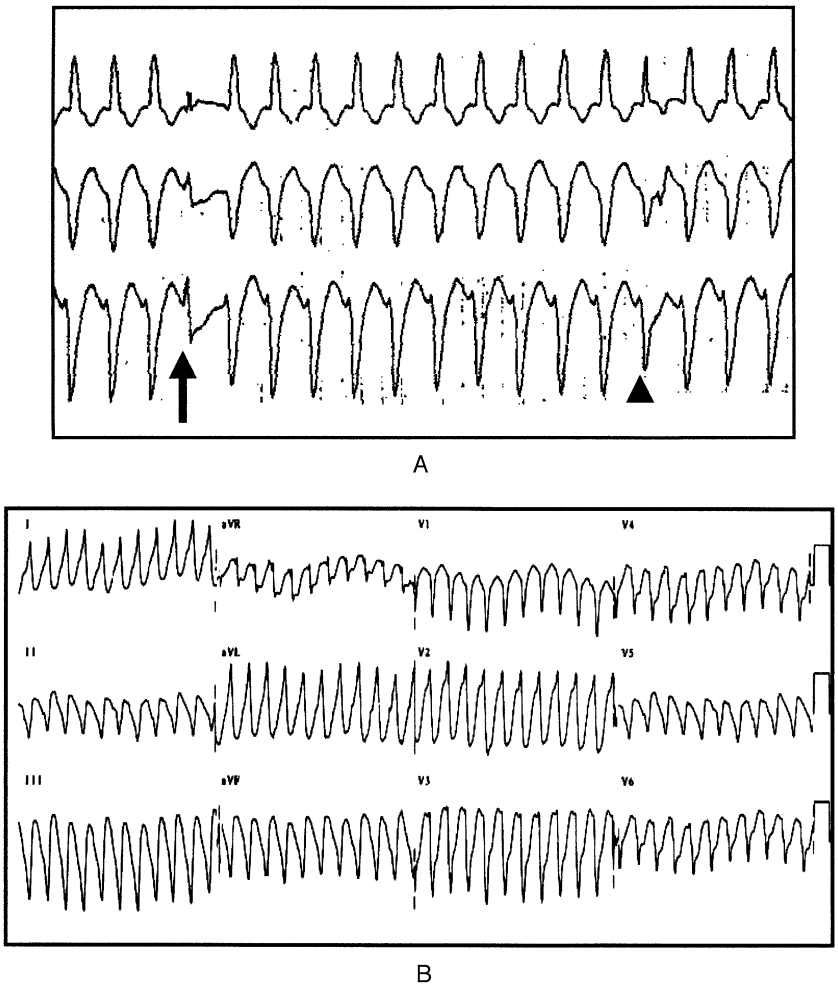

Figure 3. Case Two: (A) Multi-channel rhythm strip revealing a wide complex tachycardia. In addition, fusion (arrow head) and capture (arrow) beats are seen. The latter finding is highly suggestive of VT. (B) 12-lead EKG demonstrating a wide complex tachycardia. Negative QRS complex concordancy (all QRS complexes are predominantly negative across the precordium) is seen from leads V1 to V6, a finding that supports the diagnosis of VT.

approximate rate of 180/min. The dysrhythmia was felt to be VT due to the presence of fusion (Figure 3A, arrow-head) and capture (Figure 3A, large arrow) beats on the rhythm strip, negative concordancy on the 12-lead EKG, and the atrioventricular dissociation on the prehospital rhythm strip. The patient received i.v. procainamide, which ultimately converted the rhythm to NSR. He was admitted to the hospital with an unremarkable course including a negative evaluation for recurrent myocardial infarction.

Case 3

A 68-year-old woman with a history of angina, coronary artery disease, and atrial fibrillation experienced a witnessed syncopal event. Bystander cardiopulmonary resuscitation (CPR) was initiated. The EMS personnel were summoned and found an unconscious, pulseless, apneic elderly woman on the ground; the downtime was estimated to be 3 min. Cardiopulmonary resuscitation was initiated. The EKG monitor revealed polymorphic VT (Figure 4). The patient was defibrillated with 200 joules, which converted the patient to sinus tachycardia.

Subsequent monitor strips revealed sinus tachycardia with markedly prolonged QT interval. The patient was diaphoretic with vital signs of BP 175/87 mm Hg, pulse 115 bpm, and R 36 breaths/min. She was given i.v. magnesium for presumed torsade de pointes (TdP) variety of VT. The patient reportedly used oral quinidine sulfate and digoxin for the management of her atrial fibrillation. While awaiting intensive care unit admission, the patient once again lost consciousness with the development of pulseless polymorphic VT (Figure 5).

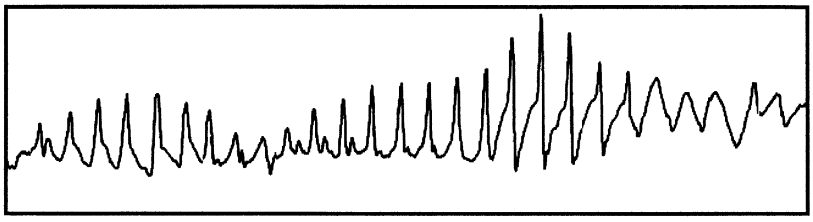

Figure 4. Case Three: A wide QRS complex tachycardia is seen; the QRS complex varies significantly from beat to beat. In addition to this marked variability to the QRS complex morphology, a "twisting" pattern of the QRS complexes is observed here, suggestive of torsades de pointes; ultimate confirmation of this rhythm diagnosis can be made only when abnormal repolarization (i.e., prolonged QT interval) is seen on the EKG when sinus rhythm is present.

Repeat defibrillation restored sinus rhythm; additional i.v. magnesium was administered. Hospital admission was significant for no dysrhythmia recurrence and a negative evaluation for acute coronary ischemia. The patient was felt to have experienced TdP version of VT, likely resulting from quinidine.

Case 4

A 59-year-old man was bowling when he suddenly developed chest pain and dyspnea. He called 911; upon paramedic arrival, the patient appeared diaphoretic but alert and in no distress. Monitoring revealed sinus bradycardia; prehospital 12-lead EKG demonstrated ST segment elevation in the inferior leads consistent with AMI. Appropriate therapy was initiated and the patient was transported to the ED. Upon arrival, he was awake and alert complaining only of chest discomfort. Soon after ED arrival, he suddenly became unresponsive while undergoing a 12-lead EKG (Figure 6, A and B); the monitor revealed polymorphic VT–he was pulseless. A single electrical defibrillation produced sinus tachycardia with the prior ST segment elevation in the inferior leads and a normal QT interval.

The patient was taken to the catheterization laboratory where multivessel coronary artery disease was found. Angioplasty was performed successfully. The patient was admitted to the intensive care unit with an uneventful hospital course. His ultimate discharge diagnoses included ST segment elevation AMI and polymorphic VT.

DISCUSSION

Ventricular tachycardia is defined as five or more ventricular beats in succession at a rate of more than 120 beats/min. The term *accelerated idioventricular rhythm* refers to ventricular rhythms with rates less than 120 beats/min. Ventricular tachycardia can be discussed educationally and approached therapeutically from several different viewpoints, including clinical, temporal, and morphologic perspectives. Ventricular tachycardia may be *stable* if it presents with intact circulation; conversely, it is *unstable* if the rhythm disturbance causes hemodynamic compromise, with pulseless VT the most extreme form. From the temporal perspective, it is *nonsustained* if self-terminating in less than 30 s; if the rhythm disturbance persists beyond this time period, it is considered *sustained*. Morphologically, VT may present electrocardiographically with a single QRS complex form and is therefore termed *monomorphic* VT; if the rhythm is characterized by numerous QRS complex morphologies, it is called *polymorphic* VT. Refer to Figure 7 for examples of monomorphic and polymorphic VT.

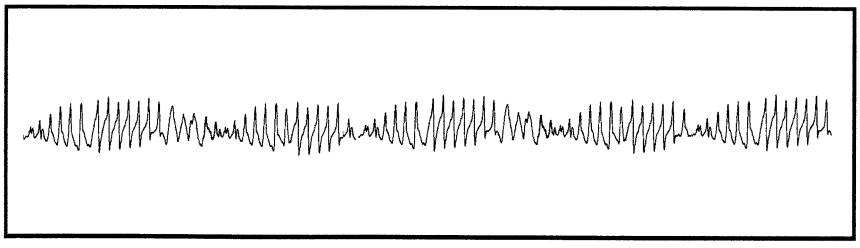

Figure 5. Case Three: Similar rhythm to that seen in Figure 4. This longer strip, however, more completely illustrates the "twisting or undulating" pattern typical of torsades de pointes; additionally, during a period of sinus rhythm, a markedly prolonged QT interval was seen, confirming the diagnosis of torsades de pointes polymorphic VT.

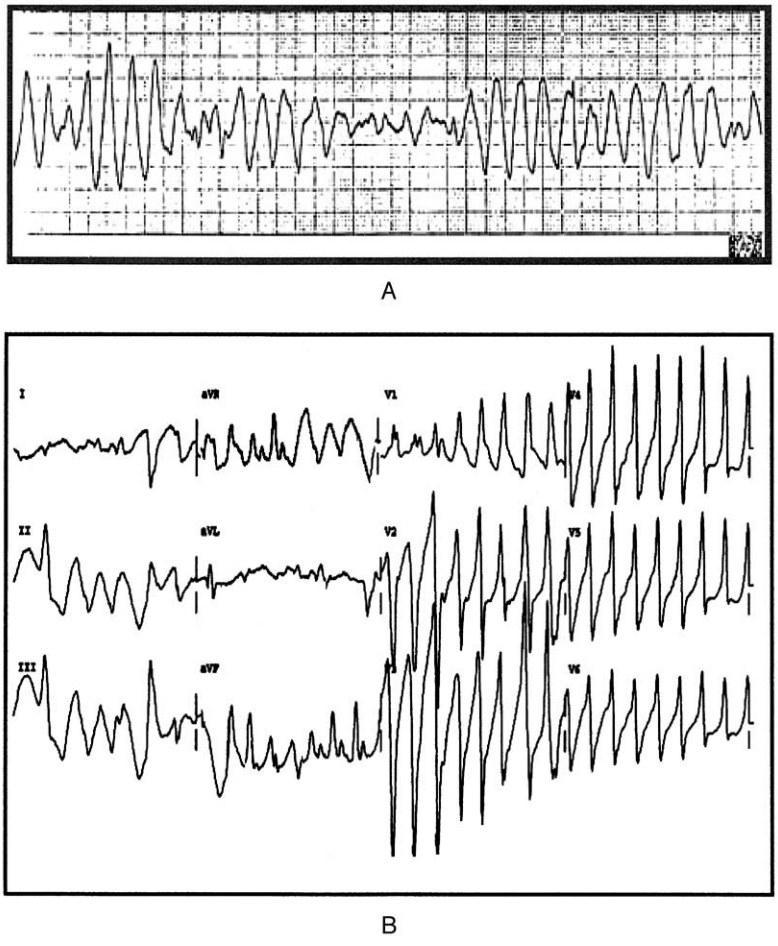

Figure 6. Case Four: (A) Polymorphic VT is seen in this example. The 12-lead EKG of this patient, obtained in sinus rhythm, demonstrated a normal QT interval. Therefore, this rhythm strip demonstrates polymorphic VT. (B) 12-lead EKG demonstrating polymorphic VT.

Regarding pathophysiology, VT usually results from either re-entry phenomenon or triggered automaticity. The properties of a re-entry circuit involve two pathways of conduction with differing electrical characteristics. These re-entry circuits that provide the substrate for VT usually occur in a zone of acute ischemia or chronic scar. The VT is usually initiated by an ectopic beat, although a number of other factors may initiate the dysrhythmia, including acute coronary ischemia, electrolyte disorders, and dysautonomia. Conversely, triggered automaticity of a group of cells can result from congenital heart disease, acquired heart ailments, or medication toxicity. These tachycardias tend to accelerate after initiation and slow markedly before termination.

Monomorphic Ventricular Tachycardia

Monomorphic ventricular tachycardia (MVT, Figure 8) is a form of ventricular tachycardia with a stable QRS complex configuration (a uniform beat-to-beat QRS complex morphology). It is the most commonly encountered form of VT in acute cardiac care and usually occurs after myocardial infarction. In the out-of-hospital setting, MVT is found in approximately 60–75% of prehospital patients with VT (10,11).

The EKG diagnosis of monomorphic ventricular tachycardia is based on the following features that may not be present in all instances. Monomorphic ventricular tachycardia is usually regular, with rates between 120 and 300 beats per minute (bpm), most often in the 140–180 bpm range. Certain medications, such as amiodarone, may slow the rate of VT; such patients may present with ventricular rates of 110–130 bpm. Supraventricular tachycardias (SVT) frequently present with more rapid rates when compared with VT; regarding ventricular rate, significant overlap unfortunately exists between VT and SVT such that rate is not a helpful criterion to differentiate origins. If the rhythm of a wide

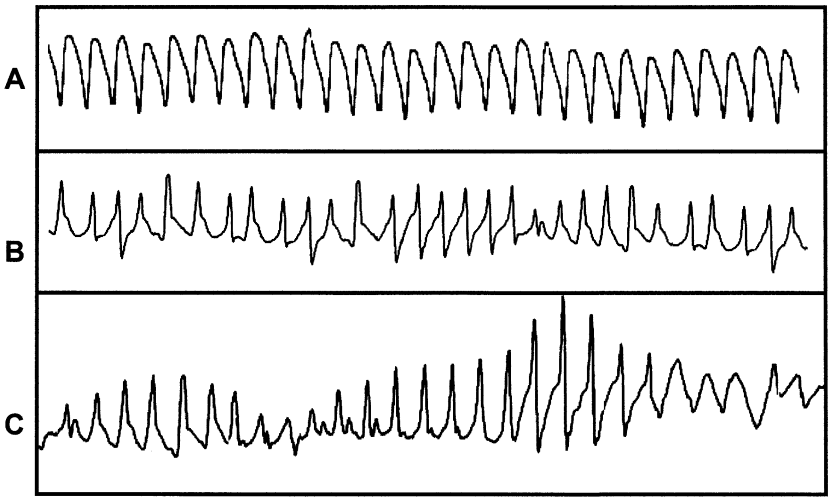

Figure 7. (A) Monomorphic VT. (B) Polymorphic VT. (C) Polymorphic VT with undulating pattern, suggestive of torsades de pointes. When interpreted with a sinus rhythm EKG demonstrating prolonged QT interval, this example of polymorphic VT can be termed torsades de pointes.

QRS complex tachycardia is obviously irregular (Figure 9, lower panel), the most likely diagnosis is atrial fibrillation with either aberrant conduction or pre-excitation; polymorphic ventricular tachycardia may also appear irregular. On rare occasions, MVT may present with an irregular rhythm; if irregular, monomorphic VT is minimally irregular as compared to an aberrantly conducted supraventricular tachycardia that ordinarily manifests marked irregularity.

In cases of ventricular tachycardia without retrograde conduction to the atria, the sinus node continues to initiate atrial depolarization. Because atrial depolarization is completely independent of ventricular activity, the resulting P waves will be dissociated from the QRS complexes—i.e., no association of the P wave will be noted with the QRS complex. The atrial rate is always slower than the ventricular rate. The EKG must be closely and carefully reviewed for evidence of atrial activity—the P wave. Unfortunately, P waves may be evident only in selected portions of the same lead (i.e., the P wave may be "lost" within a simultaneously occurring QRS complex); additionally, certain electrocardiographic leads may not demonstrate atrial activity. Indirect evidence of atrial activity may suggest the pres-

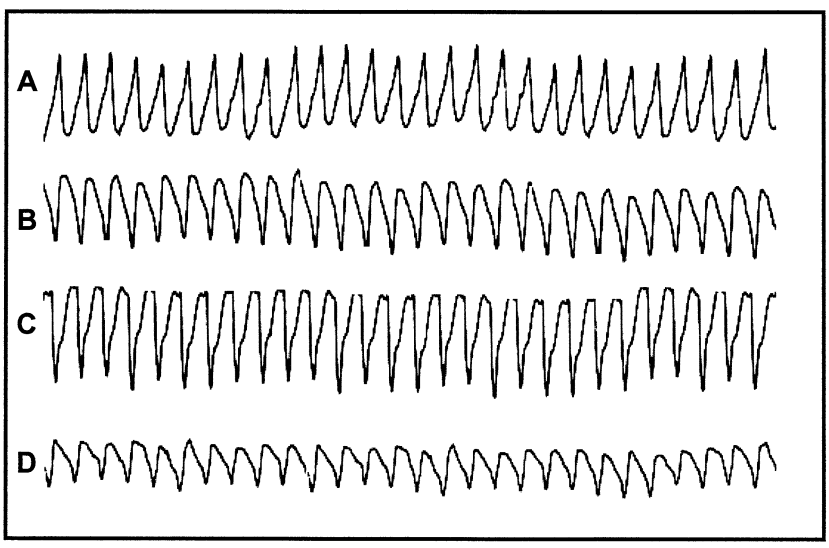

Figure 8. Different examples of monomorphic VT.

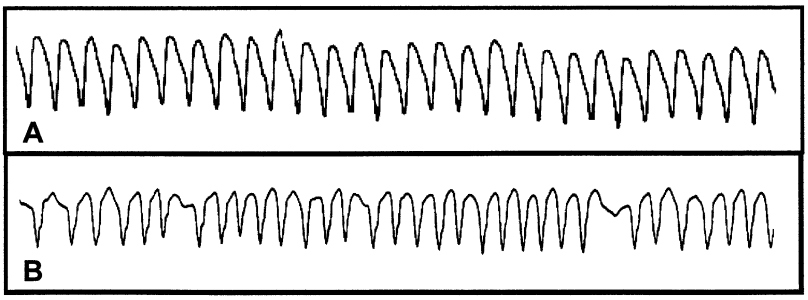

Figure 9. Upper panel—regular, wide QRS complex tachycardia consistent with monomorphic VT. Lower panel—irregular, wide QRS complex tachycardia consistent with atrial fibrillation with aberrant ventricular conduction.

ence of a P wave that is obscured by larger electrocardiographic structures. Beat-to-beat differences in the morphology of the electrocardiographic complexes, especially of the initial and terminal portions of the QRS complex and the ST segment, suggest the possibility of independent P wave activity, even though it may be impossible to pinpoint obvious P waves. If observed, such atrioventricular dissociation (Figure 2) is diagnostic for ventricular tachycardia; conversely, the lack of direct evidence of independent P wave activity does not exclude the diagnosis of VT. Atrioventricular dissociation is quite rare; it is noted in only 5–10% of patients with wide QRS complex tachycardia.

An independent atrial impulse may occasionally cause ventricular depolarization via the normal conducting system; such a supraventricular impulse, if conducted and able to trigger a depolarization within the ventricle, will result in unusual QRS complex morphology. If the resulting QRS complex occurs earlier than expected and is narrow, the complex is called a capture beat (Figures 3A and 10); the supraventricular impulse electrically captures the ventricle, producing a narrow complex. The presence of capture beats strongly supports a diagnosis of ventricular tachycardia. Fusion beats (Figures 3A and 10) occur when a sinus beat conducts to the ventricles via the atrioventricular node and joins—or fuses—with a ventricular beat originating from the abnormal ectopic focus. These two beats combine, resulting in a QRS complex of intermediate width and differing morphology when compared to the other beats of MVT. As with capture beats, the presence of fusion beats is strongly suggestive of ventricular tachycardia. Fusion and capture beats occur infrequently and are seen in fewer than 10% of patients with VT.

In the patient with VT, the QRS complex axis changes from that seen in sinus rhythm. In sinus rhythm, the normal QRS complex axis ranges from −30 degrees to +90 degrees; the majority of patients demonstrate an axis close to 60 degrees. In these instances, the QRS complex in lead aVR is usually negative (−210 degrees). When the QRS in lead aVR is entirely positive, the clinician must consider a ventricular origin of the tachycardia, i.e., ventricular tachycardia. Similarly, a change in the axis of more than 40 degrees to the left or right compared with an EKG recorded in sinus rhythm is suggestive of ventricular tachycardia.

The QRS complex duration, morphology, and the combination of these two features are valuable clues to the diagnosis of WCT in selected instances. Previous teaching held that a minimally widened complex occurred with SVT (less than 0.14 s), whereas VT was characterized by a significantly prolonged duration (6). Such a differentiation is not possible, in that VT may present with a relatively narrow QRS complex width, whereas SVT with aberrant conduction may manifest markedly wide complexes. The clinician should be cautioned in cases of WCT with a QRS complex width less than 0.14 s; in approximately 20% of VT, the QRS

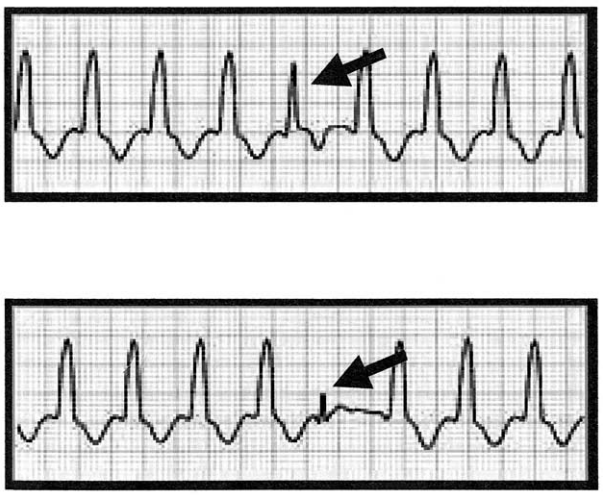

Figure 10. Rhythm strips demonstrating wide complex tachycardia with fusion (upper panel) and capture (lower panel) beats. The presence of either of these beats strongly suggests VT.

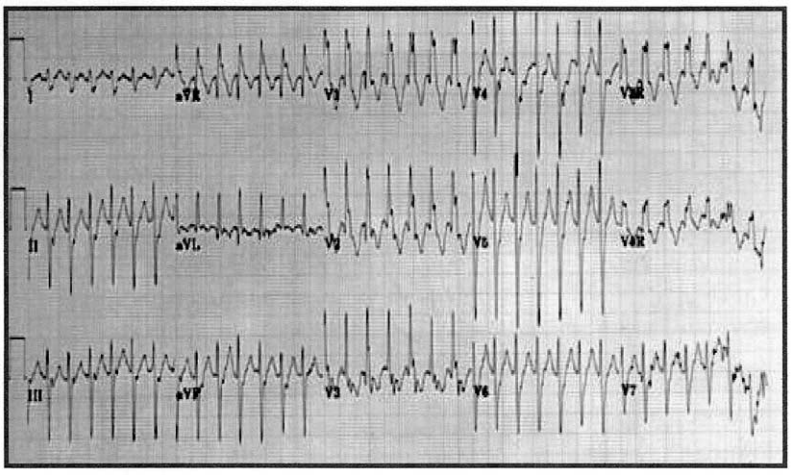

Figure 11. Ventricular tachycardia in a pediatric patient. Note the "relatively" narrow QRS complex compared to the adult-type broad QRS complex in VT. Children experiencing VT with relatively smaller amounts of cardiac tissue will depolarize over shorter periods of time and, consequently, demonstrate a "more narrow" QRS complex when compared to adults.

complex is less than 0.14 s. Further, narrow QRS complex VT has been described, usually in particular settings such as digitalis toxicity or in young children (3,8,12,13). Recall that QRS complex duration is a function of both total depolarization time and total tissue amount suitable for depolarization. As such, children experiencing VT with relatively smaller amounts of cardiac tissue will depolarize over shorter periods of time and, consequently, will demonstrate a "more narrow" QRS complex (Figure 11) when compared to adults. Conversely, QRS complex durations greater than 0.16 s are encountered in SVT with aberration in settings of hyperkalemia, tricyclic antidepressant overdose, therapy with antidysrhythmic agents, and Wolff-Parkinson-White syndrome. In general terms, the combination of QRS duration and bundle branch block (BBB) morphologies suggests VT: 1) a QRS complex width greater than 0.14 s with right BBB morphology (Figure 12); and 2) a QRS complex width greater than 0.16 s with left BBB morphology (Figure 13) (6,8).

Various QRS complex morphologies relative to BBB patterns are suggestive of the diagnosis; leads V1 and V6 are best used. With right BBB, a large R wave of varying morphologies in V1 is found with VT: monophasic R, QR, and RS patterns (3,6,8). With R-R' configurations in lead V1, an R wave with amplitude greater than the R' wave suggests VT, whereas the converse is true for the diagnosis of SVT (3,6). In lead V6, an R/S ratio of less than 1.0, QS, and QR patterns suggest VT (8). The various QRS complex morphologic findings with right BBB are noted in less than half of WCT patients with VT as the final rhythm diagnosis (8). With left BBB, fewer

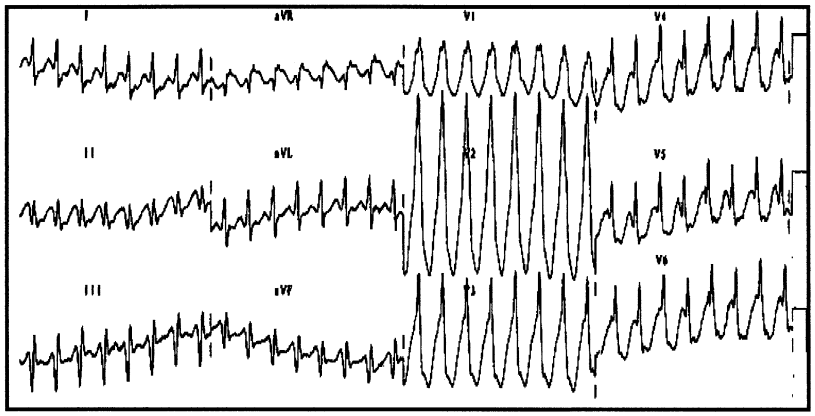

Figure 12. Wide complex tachycardia ultimately diagnosed as VT. Note the right BBB morphology with QRS complex duration greater than 0.14 s, positive QRS complex concordancy in the precordial leads, and presence of atrioventricular dissociation best seen in lead III.

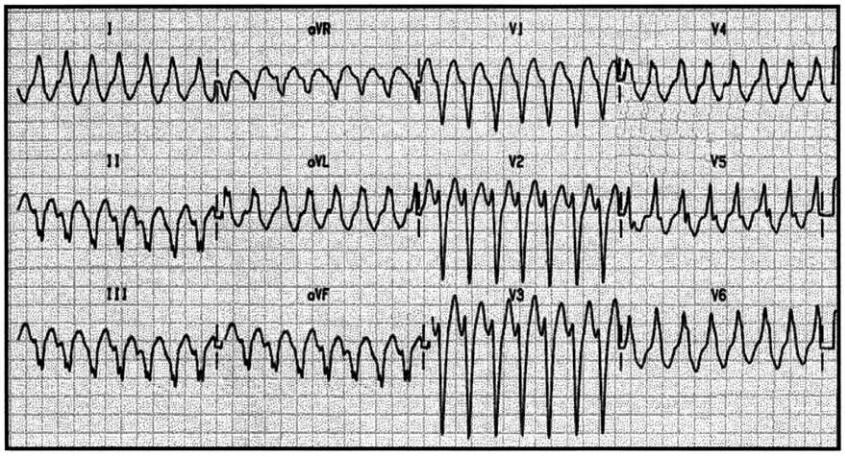

Figure 13. Wide complex tachycardia ultimately diagnosed as VT. Note the left BBB morphology with QRS complex duration greater than 0.16 s. Also note the indirect evidence of atrioventricular dissociation, seen best in leads I, II, III, V4, and V5. In each of these leads, distinct P waves are not noted; actually, beat-to-beat alterations in the QRS complex structure are seen and likely represent the P wave as it is "absorbed" into the larger QRS complex structure.

EKG clues are helpful in distinguishing the two rhythm disturbances. In lead V1, the following findings are highly suggestive of VT: R wave wider than 0.03 s, an RS interval greater than 0.07 s, slurred S wave, and notched S wave. Analysis of lead V6 is not helpful in the left BBB presentation (3,8,14).

Concordance addresses the relationship of the polarity of the QRS complexes across the precordium. Concordance of the QRS complexes in the chest leads that are either predominantly positive (Figures 12 and 14A) or negative (Figures 3A and 14B) suggests a ventricular origin for the tachycardia. It is suggested that positive concordance correlates with a ventricular focus for the tachycardia on the posterior ventricular wall, hence the wave of depolarization moves towards all the chest leads producing positive complexes. Similarly, negative concordance is thought to correlate with an anterior wall origin for the VT.

Polymorphic Ventricular Tachycardia

Polymorphic ventricular tachycardia (PVT) is defined as a ventricular tachycardia with an unstable (frequently varying) QRS complex morphology in any single EKG lead (Figures 4, 5, 6, 7B and C, and 15). Variation in both the R-R interval and electrical axis are also noted features of this ventricular dysrhythmia. Torsades de pointes (TdP) is identified when PVT occurs in the setting of delayed myocardial repolarization manifested on the EKG by a prolongation of the QT interval (Figures 4, 5, 7C, and 15A). TdP is therefore one subtype of PVT and not a synonym for the category of PVT. Multiple terms for PVT are found throughout the medical literature, causing confusion regarding rhythm recognition, etiology, and proper therapy. Torsades de pointes, cardiac ballet, atypical ventricular tachycardia, transient recurrent ventricular fibrillation, swinging ventricular tachycardia, and pleomorphic VT are such examples and are frequently used interchangeably to describe the category of PVT. It is most appropriate to use the term polymorphic ventricular tachycardia as the phrase to describe this subtype of VT; the term torsades de pointes should be employed only when delayed repolarization (prolongation of the QT interval) is noted on the EKG.

Polymorphic ventricular tachycardia is found in 20–25% of prehospital cardiac arrest patients; the subset of TdP is seen in 20% of patients in this setting (10,11). Polymorphic ventricular tachycardia has been found in victims of prehospital cardiac arrest, in survivors of sudden death subsequently studied in the electrophysiology laboratory, and in patients admitted to the critical care unit for the treatment of acute coronary ischemia. PVT, when noted in the above situations, frequently is the initial rhythm that then degenerates to either MVT or VF.

Polymorphic VT will present electrocardiographically with the following EKG characteristics: 1) a wide QRS complex (greater than 0.12 s); 2) changing R-R intervals (ranging from 0.2 to 0.4 s) with a frequency of 150 to 300 bpm; and 3) a QRS complex configuration that changes frequently, if not from beat to beat (15). This beat-to-beat variation in QRS complex configuration may not be apparent when viewed on relatively short rhythm strips or in a single lead as seen in Figure 6B. In fact, QRS configuration in PVT is in a constant state of change such that multiple leads may be required to note the true pattern. The distinction of PVT from MVT is often quite

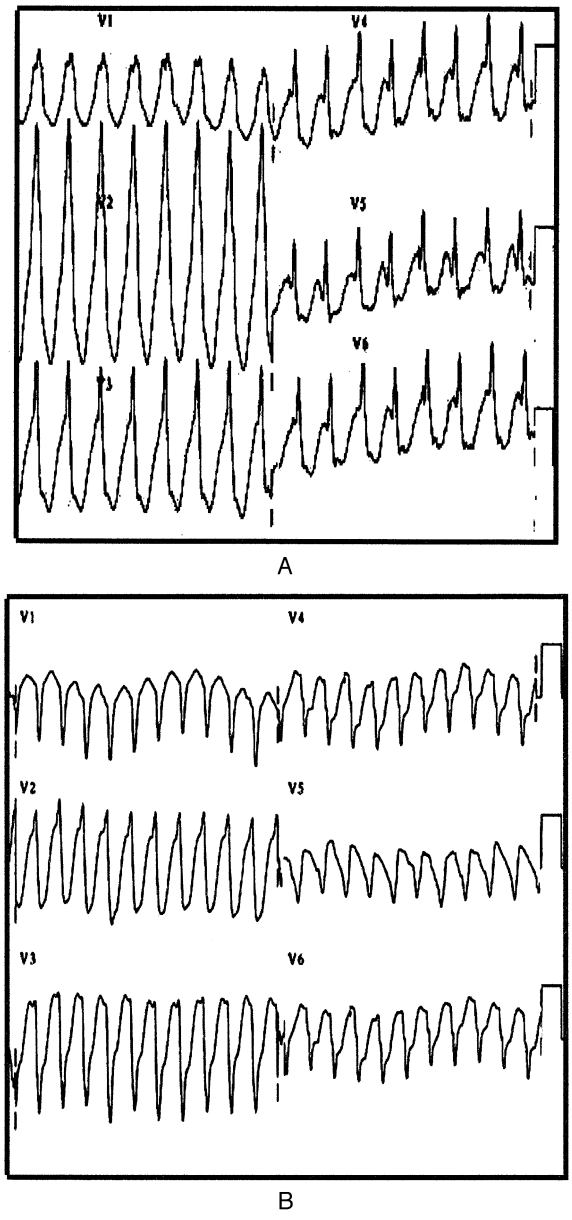

Figure 14. QRS complex concordancy in patients with VT. (A) Positive QRS complex concordancy. (B) Negative QRS complex concordancy.

straightforward, assuming the clinician has the capability of viewing several EKG leads. The separation of coarse ventricular fibrillation (VF) from PVT, however, is less easily performed. In fact, Clayton and Murray observed the electrocardiographic characteristics of PVT using frequency analysis, a process that distills the electrocardiographic signal into periodic components (15). They note that PVT differs from MVT and VF in the frequency, magnitude of the peak, and variability. They conclude that MVT, PVT, and VF are a continuum of electrical disorganization with MVT demonstrating the most organized electrical activity with VF the least; PVT represents an intermediate form of ventricular dysrhythmia. Clinically, the distinction of PVT from VF is not of major significance in that emergent electrical therapy is likely indicated in either case.

The PVT subtype, torsades de pointes, demonstrates polymorphous QRS complexes that vary from beat to beat. The variation is often quite pronounced with marked variation easily observed from any single beat to the subsequent beat. TdP also demonstrates a highly characteristic electrocardiographic pattern; the literal translation of the French term torsades de pointes—"twisting of the points"—elegantly describes the appearance of the QRS complex as it varies in amplitude, appearing to rotate about the isoelectric baseline in a semi-sinusoidal fashion (Figures 5 and 15A). As with PVT, such a pattern may not be apparent when viewed in any single lead; multiple lead analysis is usually required to discern the true pattern. Also required for the electrocardiographic (and clinical) diagnosis(es) of TdP is the demonstration of abnormal repolarization manifested by QT interval prolongation on the EKG.

Electrocardiographic Differential Diagnosis

Any discussion of ventricular tachycardia must also include a review of undifferentiated wide complex tachycardia with particular attention focused on the distinction of VT from SVT with aberrant intra-ventricular conduction. The electrocardiographic differential diagnosis of WCT includes VT versus SVT with aberrant ventricular conduction. Aberrant ventricular conduction may be due to a pre-existing BBB, a functional (rate-related) bundle malfunction resulting in a widened QRS complex when the heart rate exceeds a characteristic maximum for that patient, or accessory atrioventricular conduction as encountered in pre-excitation syndromes, such as that described by Wolff, Parkinson, and White. Other clinical syndromes less frequently encountered in this WCT differential include scenarios related to marked sinus tachycardia with pre-existing bundle branch block configuration, tricyclic-antidepressant overdose, hyperkalemia, recent electrical cardioversion, and ischemic electrocardiographic forms (the giant R wave) misinterpreted as a widened QRS complex with tachycardia. If one considers all patients encountered with WCT, approximately 80% ultimately will be diagnosed with VT; this preponderance of VT, however, probably reflects referral bias of difficult cases to electrophysiology centers (3,16–18). The true incidence of VT among ED patients with WCT is likely considerably less.

The limited usefulness of AV dissociation, fusion/capture beats, and other electrocardiographic character-

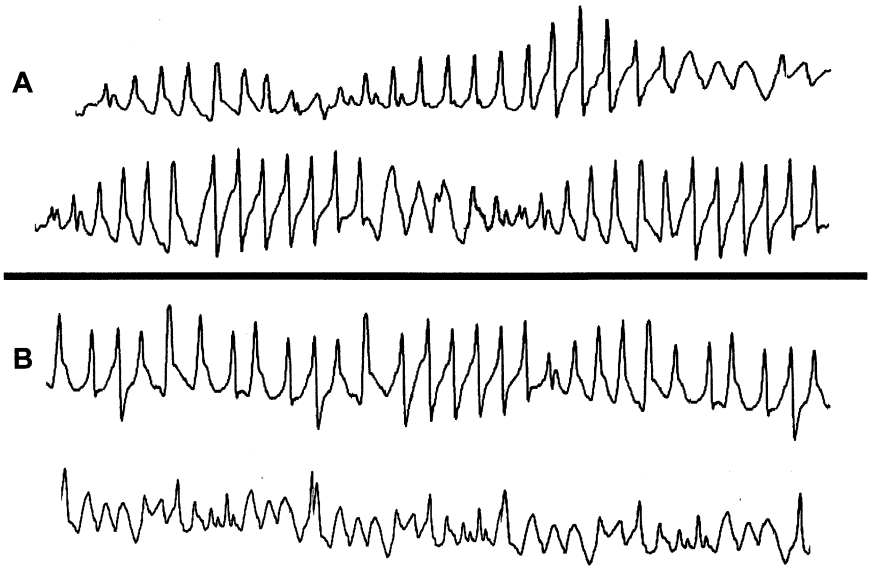

Figure 15. Polymorphic ventricular tachycardia. (A) Polymorphic VT, torsades de pointes type—note the twisting or undulating pattern. This specific subtype of polymorphic VT is diagnosed when abnormal repolarization (prolonged QT interval) is noted on the EKG. (B) Polymorphic VT.

istics has led to the development of several stepwise approaches to the diagnosis of WCT—the distinction of VT from SVT with aberrant conduction. For example, the approach suggested by Brugada and colleagues involves a four-step algorithm (5,7). Initially, the clinician examines the EKG for RS complexes in the precordial leads, the absence of which suggests VT. If RS complexes are present, the R to S interval (from onset of R wave to nadir of S wave) is examined. An RS interval greater than 0.10 s in any precordial lead suggests VT; an RS interval less than 0.10 s does not exclude VT and continues the clinician down the algorithm. The clinician then looks for AV dissociation, which, if present, is diagnostic of VT. If none of the above criteria is met, leads V1 and V6 are examined for morphologic criteria of VT as described above (5,19). This decision analysis enables the clinician to make the diagnosis of VT with a reported sensitivity and specificity of 0.99 and 0.97, respectively, when performed retrospectively by cardiologists (8). This tool, as well as numerous similar proposals, however, are somewhat cumbersome for the clinician and may not be as useful as suggested for the noncardiologist, particularly when applied infrequently. For example, the Brugada et al. approach was tested retrospectively by emergency physicians in the evaluation of regular WCT (8). Significant inter-observer disagreement with an incorrect rate of diagnosis of 22% was found in this study (20).

The principles discussed above in the diagnosis of WCT have focused largely on criteria suggestive of VT. The inherent flaw in this approach lies in the utilization of SVT with aberrancy as the default diagnosis in the event that none of the criteria for VT is met. Thus, misidentification of VT as SVT with aberrant conduction can, and does, occur with sometimes serious implications. In fact, this scenario has led some clinicians to suggest an approach that establishes criteria for the diagnosis of SVT, with a default diagnosis of VT if the parameters for SVT are not satisfied (21). The criteria for establishing a wide complex tachycardia as SVT with aberrancy, as set forth by Griffith and colleagues, centers on electrocardiographic evidence of BBB as follows: 1) left bundle morphology with an RS or QS wave in leads V1 and V2, delay to S wave nadir less than 70 ms, and R wave without Q wave in V6; and 2) right bundle morphology with an RSR' wave in V1 and RS wave in V6 with R wave height greater than S wave depth (21). If these rules are not satisfied, VT is diagnosed by default. Utilizing this approach, sensitivities and specificities for VT of approximately 90% and 85%, respectively, have been reported (21).

CONCLUSION

Ventricular tachycardia is a malignant dysrhythmia. Prompt, specific therapy is required to optimize outcome in any such presentation. Appropriate therapy assumes a correct diagnosis. The WCT presentation in the ED is a challenge. The differential diagnosis for such a presentation includes VT, SVT with aberrant conduction, and other dysrhythmias resulting from toxic or metabolic

etiologies. Clinical clues to aid in this diagnostic situation include patient age and history of cardiac disease with older age and past history of organic heart disease being predictors of VT. Electrocardiographic features suggestive of VT include atrioventricular dissociation and fusion/capture beats. Rate and regularity are not of great benefit with the exception of marked irregularity suggesting atrial fibrillation with aberrant conduction. Other features may be of assistance, including QRS morphology, axis, and the inter-relationship of the QRS complexes in the precordial leads. In many situations, the Emergency Physician (EP) may not be able to make a specific rhythm diagnosis. In these instances, the EP must treat the patient with respect to the clinical presentation; additionally, the default diagnosis should not be SVT with aberrant conduction. The EP must "assume the worst" and tailor a rapid evaluation and therapeutic approach with such thoughts in mind.

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