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Differentiating Types Of Wide-Complex Tachycardia To Determine Appropriate Treatment In The Emergency Department

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Abstract

Wide-complex tachycardia is a rare disease entity among patients presenting to the emergency department. However, due to its potential life-threatening nature, emergency clinicians must know how to assess and manage this condition. Wide-complex tachycardia encompasses a range of cardiac dysrhythmias, some of which can be difficult to distinguish and may require specific treatment approaches. This review summarizes the etiology and pathophysiology of wide-complex tachycardia, describes the differential diagnosis, and presents an evidence-based approach to identification of the different types of tachycardias through the use of a thorough history and physical examination, vagal maneuvers, electrocardiography, and adenosine. The treatment options and disposition for patients with various wide-complex tachycardias are also discussed, with attention to special circumstances and select controversial/contemporary topics.

Case Presentations

You're dictating a chart when EMS brings in an ashen-looking middle-aged man clutching his chest and gasping, "I can't breathe." You follow him into the room, and, as he's attached to the monitor, you see a wide complex tachycardia with a rate of 146 beats/min. His blood pressure is 86/50 mm Hg, and his pulse oximetry is 86%. The tech is already attaching ECG leads, and you note a sternotomy scar on the patient's chest. You ask for pads to be placed for direct cardioversion; as you're getting ready to shock, you think, "Should I wait for the ECG? Is this ventricular tachycardia or supraventricular tachycardia? Do I even need to know that before shocking him?"

Later during your shift, you are called into the room by the nurse of a patient you've just admitted for syncope. The nurse points at the monitor and yells, "He's in v-tach!" The patient is looking at you quizzically and asks, "What's going on?" The monitor shows a wide-complex tachycardia. His blood pressure is 135/96 mm Hg, and his pulse oximetry is 94%. You calmly ask for a stat ECG and pads to be placed on the patient. As you watch his ECG print out, you think, "I'm not sure that's ventricular tachycardia, but it's possibly supraventricular tachycardia with aberrancy. I need to check his potassium levels. Should I use amiodarone first or should I try adenosine?"

In the next room, there is a young female patient you've seen every shift for the past 3 days who was checked in with "anxiety." You think that she is possibly looking for benzodiazepines except, this time, her triage vital signs show a heart rate of 190 beats/min. There is no blood pressure recorded. You go to see her immediately, and she's hyperventilating and appears anxious. You hook her up to the monitor yourself and note an irregular wide-complex tachycardia. Her blood pressure is 102/69 mm Hg. As you're calling for a nurse and a tech, you think, "She's too young for ventricular tachycardia, isn't she? Maybe she overdosed, or maybe she has a conduction issue. I really need to see this ECG."

Introduction

Wide-complex tachycardia (WCT) describes a rhythm in which the heart rate is > 100 beats/min and the QRS complex is > 120 milliseconds. It is essential for emergency clinicians to have a comprehensive understanding of the recognition and management of the different types of WCTs, as many of these rhythms may progress to other sudden, life-threatening dysrhythmias or progressive cardiomyopathy without prompt treatment. Furthermore, if a WCT is misdiagnosed, subsequent delayed or inappropriate therapeutic decisions can portend further hemodynamic compromise.

Unfortunately, despite the numerous algorithms published in the literature, there are no electrocardiographic criteria that can definitively diagnose a specific WCT, and agreement with regard to rhythm identification is inconsistent.[1,2] This issue of *Emergency Medicine Practice* will provide you with the tools that are required to recognize, diagnose, and treat common etiologies of WCT.

Critical Appraisal Of The Literature

A PubMed literature search was performed using the search terms *wide complex tachycardia, broad complex tachycardia, wide complex dysrhythmia, wide QRS complex tachycardia, broad QRS complex tachycardia, ventricular tachycardia, supraventricular tachycardia,* and *ventricular arrhythmia.* These terms were also used in conjunction with the terms *emergency department, prehospital, clinical signs, clinical history, vagal maneuvers, hyperkalemia,* and *tricyclic antidepressant.* This search yielded a total of 949 references from 1964 to June 2014, which were reviewed for relevance. A literature search of the Cochrane Database using the same search terms yielded a total of 7 reviews, only 1 of which was relevant for the current topic. The position statements of the American Heart Association (AHA), the American College of Cardiology (ACC), the European Society of Cardiology (ESC), and the European Resuscitation Council (ERC) were also reviewed.

Current consensus guidelines from the cardiology literature, including those put forth by the AHA, ACC, ESC, and ERC, focus on the identification and treatment of ventricular tachycardia rather than wide-complex tachycardia alone.[3-5] The 2010 AHA[5] and 2010 ERC guidelines[4] provide protocols for cardiopulmonary resuscitation, but they do not address the nuances of ventricular dysrhythmia management.

A combined position statement from the ACC, AHA, and ESC on the management of ventricular arrhythmias was issued in 2006, and it provides a considerably more detailed discussion.[3] For this guideline, the collaborative performed a comprehensive literature review of specific topics and then issued a consensus statement for each. While broader in scope than the AHA and ERC resuscitation guidelines, it does not include current evidence.

Etiology And Pathophysiology

WCT can result from either an extrinsic or an intrinsic etiology. The primary extrinsic causes of WCT involve sodium-channel blockade. Drugs that inhibit cardiac sodium channels will slow phase 0 depolarization in the myocardium, which will significantly widen the QRS complex on an electrocardiogram (ECG).[6,7] Hyperkalemia also inhibits cardiac sodium channels, but it does so through the passive influx of potassium intracellularly, which alters the resting membrane potential.[8,9] In addition, acute, complex

medical illnesses (such as sepsis and congestive heart failure) can give rise to an associated WCT.[10] These illnesses may present as supraventricular tachycardias (SVTs), such as atrial fibrillation (AF) and atrial flutter, and will manifest as a WCT when combined with aberrant conduction.

Intrinsic causes include ventricular tachycardia (VT), the most common etiology of regular WCT, which can be monomorphic or polymorphic.[11] Monomorphic ventricular tachycardia is generated from a single focus of re-entry, and it is most commonly seen in patients with prior myocardial infarction (MI) and secondary scar formation.[11] Polymorphic VT originates from multiple foci or a primary re-entrant circuit that migrates within the ventricular myocardium. It is encountered most commonly as torsades de pointes or as a consequence of acute myocardial ischemia.

An SVT that originates above the atrioventricular (AV) node will typically present with a narrow QRS complex. However, all types of SVT can present as WCT when in the presence of aberrant conduction.

Differential Diagnosis

VT should be the first rhythm considered when a patient presents with WCT, but, in reality, the differential diagnosis is quite broad. Furthermore, misdiagnosis of the specific WCT rhythm can be catastrophic if the wrong treatment is administered as a result. **Table 1** provides a summary of the differential diagnosis for WCT.

The Toxic Myocardium

Cardiac Sodium-Channel Inhibitors

Among the many drugs that are associated with WCT, tricyclic antidepressants (TCAs) are prototypical. TCAs antagonize sodium channels to prolong cardiac depolarization, demonstrated by widening of the QRS complex. This phenomenon, when combined with tachycardia due to the anticholinergic effect of TCAs, will result in WCT. Other drugs that cause sodium-channel blockade are cocaine, diphenhydramine, and class Ia and Ic antidysrhythmic drugs, such as procainamide and flecainide. Cocaine (and other sympathomimetic) toxicity may also give rise to tachydysrhythmias other than WCT, and these can result in demand (type 2) myocardial ischemia.

Hyperkalemia

Hyperkalemia causes QRS widening in a manner similar to sodium-channel-blocking drugs. Increased serum potassium levels lead to passive influx of these ions into myocardial cells and alter the resting potential, making it less negative. This, in turn, leads to inactivation of sodium channels and results in abnormal, delayed depolarization and a wide QRS. As a general rule, potassium levels > 8 mEq/L will present with QRS widening.[9] **(See Figure 1, page 4.)** This abnormality in ventricular depolarization and the resultant QRS widening are progressive and can eventually result in ventricular fibrillation and cardiac arrest. In fact, hyperkalemia is a significant cause of death in patients with end-stage renal disease, estimated at 1.9% to 5%.[12]

Table 1. Types Of Wide-Complex Tachycardia With Historical Factors And Electrocardiographic Findings

Type of WCT	History	Physical Examination	Regularity	ECG Features
Sodium-channel blockade	Drug overdose (ie, TCA, cocaine use)	Altered mental status, mydriasis, hyperpyrexia	Regular	Axis right, terminal R wave in aVR (TCA toxicity)
Hyperkalemia	Renal insufficiency, acute kidney injury	Arteriovenous fistula, dialysis catheter	Regular	Peaked T waves
SVT with aberrancy	None	None	Regular or irregular	Baseline BBB or transient BBB (transient BBB is often right BBB with rSR' wave in V_1)
SVT with accessory pathway	Pre-excitation syndrome (ie, WPW)	None	Regular	QRS narrow or wide, more-rapid rate
Atrial fibrillation with accessory pathway	Pre-excitation syndrome (ie, WPW)	None	Irregular	QRS narrow or wide, more-rapid rate
Monomorphic VT	Myocardial infarction	Cannon A waves, variable S_1	Regular	AV dissociation, axis -180° to -90°, QRS > 160 ms, precordial concordance
Polymorphic VT	Myocardial infarction, long QT syndrome, methadone use	None	Irregular	QRS of variable morphology, baseline long QTc
Paced tachycardia	Pacemaker placement	Pacemaker	Regular	Pacing artifact, slower rate

Abbreviations: AV, atrioventricular; BBB, bundle branch block; ECG, electrocardiogram; SVT, supraventricular tachycardia; TCA, tricyclic antidepressant; VT, ventricular tachycardia; WCT, wide-complex tachycardia; WPW, Wolff-Parkinson-White.

Supraventricular Tachycardia

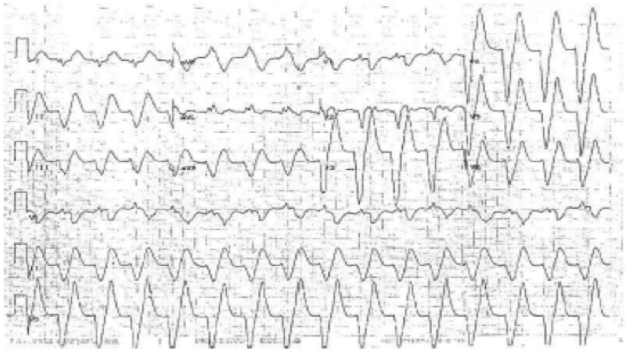
Supraventricular Tachycardia With Aberrancy

Aberrant conduction refers to delayed impulse propagation in some portion of the His-Purkinje system distal to the AV node. Any SVT with associated aberrant conduction can be mistaken for VT. In addition, aberrant conduction can be baseline or rate-related, further complicating the diagnosis.

In patients with a baseline bundle branch block (BBB), any SVT, including sinus tachycardia, will manifest as a WCT. However, the morphology of a patient's baseline BBB may change with more-rapid rates and be mistaken for VT. If treatment is given for VT, the patient may be at risk for further deterioration. Datino et al induced supraventricular tachycardia in 59 patients with baseline BBB; 76% of the patients demonstrated morphological changes in the QRS complex and BBB pattern, including the appearance, amplitude, and duration of the QRS complex.[13] Interestingly, none of the patients developed a contralateral BBB, which suggests that a WCT with a new contralateral BBB may be specific for VT. The opposite situation must also be considered, as VT may occur in patients with baseline BBB with QRS morphology that appears identical to the underlying BBB, thus leading to misdiagnosis of SVT with aberrancy.[14,15] In summary, although helpful, the comparison of QRS and BBB morphology from current and baseline ECGs will not definitively differentiate VT from SVT with aberrancy.

Even in the absence of baseline BBB, patients with SVT involving rapid atrial rates may develop rate-related aberrant conduction.[16] This phenomenon may be seen in any SVT that involves conduction through the AV nodal-His-Purkinje system (ie, atrial flutter, ectopic atrial tachycardia, AV nodal re-entrant tachycardia, and orthodromic [anterograde] AV re-entrant tachycardia). **(See Figure 2.)** Frequently, these rhythms present as narrow-complex tachycardia. However, conduction delay can occur in the setting of more-rapid heart rates, and rate-related refractory (or fatigue) conducting fibers of the AV nodal-His-Purkinje system will lead to QRS widening and, therefore, WCT on ECG.[8,17,18] Rate-related aberrancy is best confirmed by comparing current ECGs with baseline ECGs, as the QRS morphology should be altered. As previously mentioned, rate-related aberrancy may be present even in patients with baseline BBB; this is suggested by changes in BBB morphology.[13] However, in patients without BBB on prior ECG, it can be difficult to identify rate-related aberrancy as the cause of WCT.

Figure 1. Wide-Complex Tachycardia Due To Hyperkalemia



Widened QRS with peaked, steep T waves (a rapid/steep slope of the T wave, particularly the downslope, is most correlated with elevated serum K).

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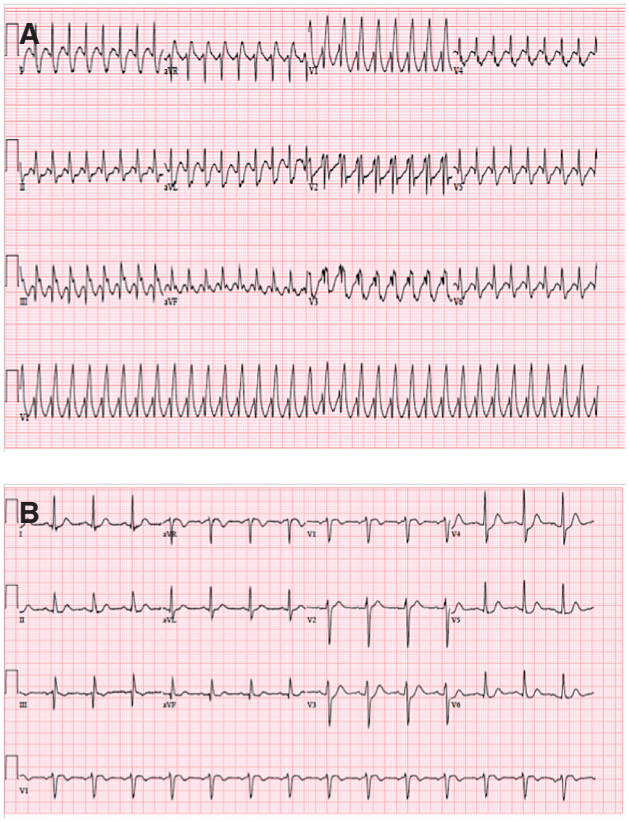
Figure 2. Wide-Complex Tachycardia With Right Bundle Branch Block Pattern Before And After Adenosine



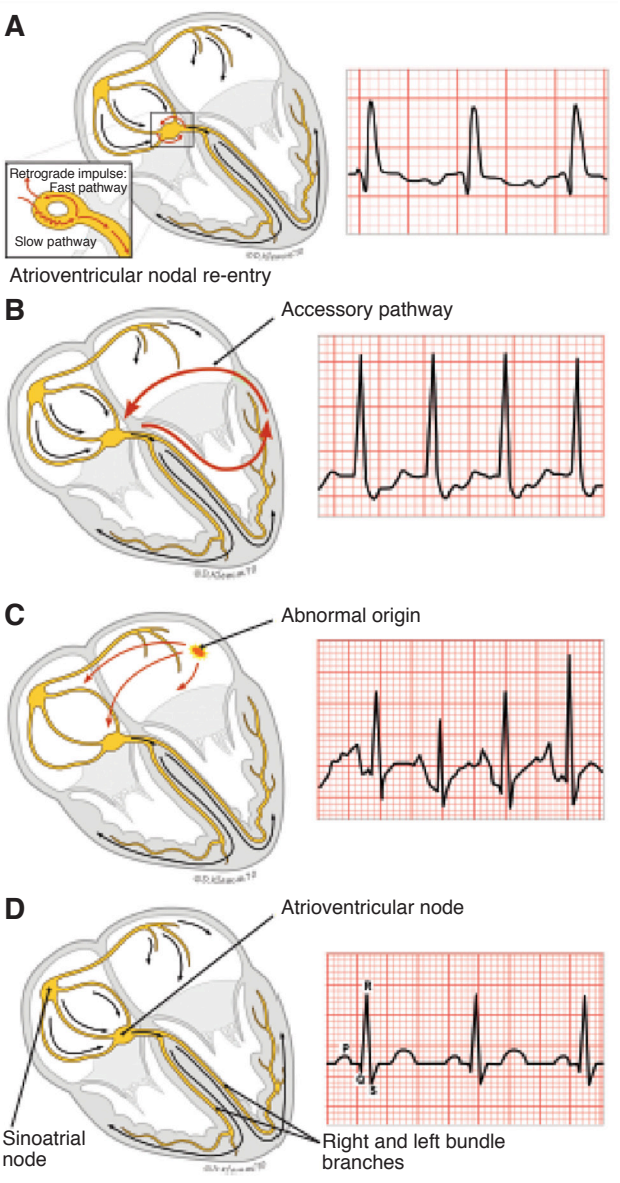
(A) Regular wide complex tachycardia with right bundle branch block pattern. (B) Adenosine administered with conversion to sinus rhythm and resolution of bundle branch block pattern; this suggests that the rhythm in Figure 2A is a re-entrant supraventricular tachycardia involving the atrioventricular node with rate-related aberrancy.

Courtesy of Ian deSouza, MD.

Supraventricular Tachycardia With An Accessory Pathway

SVTs that are conducted anterograde through a bypass tract will present as WCT. **(See Figure 3)** for types of SVT.) An example is Wolff-Parkinson-White (WPW) syndrome with antidromic AV re-entrant tachycardia. In this rare case, an atrial impulse is conducted from the atrium to the ventricle via an anatomic bypass tract, and depolarization of the ventricles then proceeds regularly, but inefficiently, as it propagates without the benefit of the rapidly conductive His-Purkinje system. The impulse then returns to the atria in a retrograde manner through the AV node to complete the re-entry circuit. As anterograde conduction does not occur via the rapidly conductive Purkinje fibers, ventricular depolarization occurs slowly, and this is manifested by a wide QRS complex on an ECG. Antidromic AV re-entrant tachycardia can, therefore, mimic monomorphic VT, and differentiation between the two may be difficult.[8,11,19] Furthermore, SVTs can proceed solely from the atria to the ventricles via a bypass tract, as this pathway often involves refractory periods that are shorter than those of the AV node. These situations usually involve rapidly generated atrial impulses (as in AF or atrial flutter). The patient with both AF and a fast-conducting accessory pathway is particularly worrisome, as this combination may result in rapid ventricular rates[5,20] and associated instability.[8]

Figure 3. Types Of Supraventricular Tachycardia



(A) Typical atrioventricular nodal tachycardia; retrograde P is often hidden in QRS but may be seen as pseudo r in V_1. (B) Orthodromic atrioventricular re-entrant tachycardia; a longer RP interval may distinguish this rhythm from Figure 3A and is dependent on the site and properties of the accessory pathway. (C) Ectopic atrial tachycardia; P axis and morphology is dependent on site and mechanism of focus. (D) Normal sinus rhythm.

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Ventricular Tachycardia

Despite the numerous forms of SVT that may masquerade as VT, approximately 80% of all WCTs will actually be VT.[3] Presumptive treatment of a regular WCT as VT is encouraged, as it can minimize the consequences of misdiagnosing VT as SVT, an error that can lead to catastrophic inappropriate therapy or delay in appropriate treatment.[21,22] VT is further divided into monomorphic and polymorphic forms.

Monomorphic Ventricular Tachycardia

Monomorphic VT originates from a single re-entry circuit or focus within the ventricle, resulting in wide QRS complexes with uniform appearance. The single focus is often secondary to myocardial scarring from previous MI, and this abnormal myocardium then provides the required slow-conducting limb of a re-entrant circuit. Another common cause of re-entrant monomorphic VT is from a circuit involving the bundle branches of the His-Purkinje system. These circuits can be secondary to structural heart disease or idiopathic in nature.[23]

Polymorphic Ventricular Tachycardia

Polymorphic VT originates from multiple ventricular foci or a single, migrating scroll wave within the ventricle, leading to beat-to-beat variability in the appearance of the QRS complex. One type of polymorphic VT is torsades de pointes. In contrast to the typical re-entrant mechanism involved in monomorphic VT, torsades de pointes is a triggered dysrhythmia that occurs in the setting of QT prolongation. This prolonged QT can be due to a number of factors, including medications or illicit drugs, metabolic disturbances

(such as decreased serum potassium, magnesium, or calcium), or congenital syndromes. When the baseline QT interval is normal in duration, polymorphic VT is most commonly seen in association with acute myocardial ischemia or cardiac arrest.

Paced Tachycardia

Sinus Tachycardia With Paced Ventricular Response

Implantable pacemakers depolarize the ventricular myocardium without the use of the His-Purkinje system conduction, and, therefore, they can cause iatrogenic QRS widening. Patients with pacemaker-conducted sinus tachycardia will appear to have WCT, though the rate should not surpass the programmed rate threshold.

Pacemaker-Mediated Tachycardia

Pacemaker-mediated tachycardia occurs when a dual-chamber pacemaker itself provides a re-entry circuit through which impulses travel. A native, premature complex is sensed by the atrial lead of the pacemaker. The pacemaker responds by generating a ventricular impulse that, in turn, is sensed by the atrial lead. This results in a re-entrant circuit where the pacemaker itself serves as a limb. Differentiating between sinus tachycardia with paced ventricular response and pacemaker-mediated tachycardia may require interrogating the patient's device.[24]

Prehospital Care

As with other cases involving cardiac illness, many of the patients presenting with WCT will arrive to the emergency department (ED) via emergency medical services (EMS). The prehospital care of the patient with WCT focuses on rapid transport. Personnel with advanced life-saving training may be able to provide electrical therapy for unstable tachycardia. In select cases, performing vagal maneuvers is a rapid intervention that is easy to perform and may be appropriate for patients presenting with tachycardia in the prehospital arena.[25] Vagal maneuvers transiently induce slowing of AV nodal conduction by increasing vagal tone. These maneuvers include the Valsalva maneuver, the diving response, and carotid sinus massage.[26] Studies examining the efficacy of Valsalva conversion of SVT have demonstrated variable success rates, ranging from 6% to 54%.[25,27,28]

Prehospital administration of adenosine, both with and without direct physician control, is also commonly performed.[29,30] Although adenosine may be useful in differentiating WCT in the ED, it is of uncertain utility in the prehospital arena.[31,32] Goebel et al examined the accuracy of paramedic diagnosis of SVT in the field.[31] This study included 224 patients, 20% of whom received adenosine inappropriately after misdiagnosis of SVT. Thirty-one patients (14%) suffered adverse effects, with 12 of those patients suffering rhythm disturbances, including VT and AF. Furthermore, adenosine administration in the field may preclude the physician's interpretation of the response and underlying rhythm, thus limiting adenosine's diagnostic value.

Emergency Department Evaluation

The history and physical examination is an important first step in evaluating all ED patients, including those with WCT. The 12-lead ECG also provides critical information in cases of WCT and can be performed simultaneously.

History

The history of present illness provides important clues as to the duration and stability of the WCT. It can also point toward a specific cause, as in the setting of sodium-channel-blocker overdose. Most commonly, however, it will not distinguish the underlying rhythm mechanism. Patients with VT can present with both hemodynamic stability and minimal symptomatology. Studies report that as few as 30% of patients with sustained VT are symptomatic upon ED arrival.[33-35]

A past medical history of renal insufficiency or acute kidney injury should suggest the possibility of WCT secondary to hyperkalemia, particularly in the setting of nonadherence to scheduled hemodialysis. Review the patient's complete medication list, including nonprescribed medications that may have been ingested in overdose. Pay particular attention to the possibility of poisoning from sodium-channel-blocking drugs, including TCAs, class Ia and Ic antidysrhythmics, and diphenhydramine. Inquire specifically about potassium-sparing diuretics and potassium supplements, as these preparations can cause hyperkalemia.[36,37]

The factors in the patient's history that can distinguish VT from SVT are also limited. However, a prior history of VT is highly predictive of subsequent VT.[35] The Leiden Out-of-Hospital Cardiac Arrest evaluation study by Kies et al included 300 consecutive patients who presented in cardiac arrest due to VT or ventricular fibrillation (VF).[38] Eighty-eight of the patients (29%) had a recurrence of VT/VF within the 5-year follow-up period. In addition to prior episodes of VT, a history of MI,[34,35,39] structural heart disease,[40] and reduced ejection fraction[35] were also more predictive of VT than SVT. Male sex[40] and age > 35 years[34] were found to be somewhat weaker predictors. **(See Table 2, page 7.)** However, male sex and age > 35 years were also found to be predictive of VT in 2 studies that examined patients with syncope.[41,42]

Physical Examination

Patients with WCT who are hemodynamically unstable require immediate direct-current cardioversion (DCCV). Therefore, obtaining the patient's vital

signs is the single most important component of the physical examination. However, pay close attention to the more subtle signs of end-organ hypoperfusion, such as transient alterations in mental status, rales/wheezes, and skin findings such as pallor, mottling, coldness, or diaphoresis. Despite normal vital signs, the presence of these signs should prompt the consideration of electrical therapy.

In patients with WCT secondary to toxins or metabolic disturbances, the physical examination may aid in diagnosis of the underlying etiology. Arteriovenous fistulas, grafts, and other points of access for hemodialysis are suggestive of hyperkalemia. In patients with toxic ingestions, a pure WCT in the absence of other symptoms would be very unusual, as the patient should exhibit signs and symptoms of the toxidrome associated with the ingested agent. TCA overdoses, for example, would present with WCT in the setting of an anticholinergic toxidrome. WCT secondary to complex medical illnesses (such as sepsis or congestive heart failure) may present with various stigmata specific to those conditions as well.

The utility of the physical examination in distinguishing between the etiologies of intrinsic WCT is to identify atrioventricular dissociation (the hallmark criterion for VT) and to determine the response to vagal maneuvers. Garratt et al examined the ability of clinicians to identify AV dissociation in order to distinguish VT from SVT.[43] Only jugular venous pulse amplitude (cannon A waves) and variability in the intensity of the first heart sound were associated with VT. Variability in the intensity of the arterial pulse was less frequently associated with accurate prediction of VT. The performance of vagal maneuvers during the physical examination may aid in diagnosis of an underlying WCT rhythm; if it is re-entrant in mechanism, this may terminate the rhythm.

Diagnostic Studies

The Diagnostic Utility Of Electrocardiography

A 12-lead ECG can allow the emergency clinician to differentiate between the types and mechanisms of WCT and, therefore, can guide treatment. However, most of the data obtained from ECG analysis have yielded test characteristics that have high specificity but poor sensitivity for the diagnosis of VT.

Rate

Heart rate does not accurately distinguish VT from SVT.[44] Nevertheless, there are some exceptions and important caveats. A wide or narrow tachycardia with the heart rate fixed at or near 150 beats/min should suggest the possibility of atrial flutter with 2:1 conduction. In the setting of WCT, this could be due to atrial flutter with aberrant conduction in the His-Purkinje system or anterograde conduction down an accessory pathway. Furthermore, the rare case of WCT at the rate of 300 beats/min would undoubtedly suggest atrial flutter with 1:1 anterograde conduction through an accessory pathway that, in contrast to the AV node, may have conduction properties conducive to such a rapid rate.

Axis

Both left-axis deviation and right-axis deviation can be associated with VT.[40] However, patients with

Table 2. Point Estimates Of Pooled Data: Historical Factors And Electrocardiogram Findings In Patients With Ventricular Tachycardia Or Supraventricular Tachycardia

Factors And Findings	VT/Total[a]	SVT/Total[a]	Sensitivity (%)	Specificity (%)	LR+	LR-
Male sex[34,35,39,40]	288/363	126/201	79	37	1.3	0.55
Prior VT[35]	21/81	2/116	26	98	15	0.75
Prior MI[34,35,39]	156/216	32/173	72	82	3.9	0.34
CABG[34,35]	27/143	20/138	19	86	1.3	0.94
Structural disease[40] or reduced EF[35]	148/196	33/132	76	75	3.0	0.32
Axis -180° to -90°[35,39,40,47]	66/286	10/175	23	94	1.0	0.90
QRS > 140 ms[39,40,47]	529/693	78/195	76	60	1.9	0.40
QRS > 160 ms[39,47]	366/571	29/167	64	83	3.7	0.40
Precordial concordance[40,47]	99/595	12/160	17	93	2.2	0.90
AV dissociation[35,39,40,47]	205/774	0/179	26	100	∞[b]	0.74

[a]The ratio indicates the number of subjects with the row characteristic divided by the number of subjects where this characteristic was assessed and reported.

[b]The LR+ approaches infinity because, based on pooled data from these particular studies, there were no cases of SVT in which ECG demonstrated AV dissociation. However, this point estimate is unlikely to be truly correct for the greater population. It may be more appropriate to provide the 95% confidence interval for this estimate, which is 15.7 to infinity.

Abbreviations: AV, atrioventricular; CABG, coronary artery bypass graft; CI, confidence interval; EF, ejection fraction; LR+, positive likelihood ratio; LR-, negative likelihood ratio; MI, myocardial infarction; SVT, supraventricular tachycardia; VT, ventricular tachycardia,

a baseline conduction delay, including BBB, will frequently demonstrate axis deviation as well, so axis alone is minimally useful in differentiating VT from SVT.[45,46] Although axis deviation is not sensitive, extreme right-axis deviation (between -180° and -90°) can be predictive of VT. **(See Figure 4.)** Extreme right-axis deviation has poor sensitivity, with reported ranges of 13% to 23%, but it has good specificity at 87% to 96%.[35,39,40,45,47] A more recent study by Marill et al found the presence of extreme right-axis deviation to increase the odds of VT by a likelihood ratio of 2.6.[48]

QRS Duration And Morphology

The QRS duration can be moderately helpful in differentiating the etiology of a WCT, but it is by no means definitive. A QRS duration > 140 milliseconds increases the probability that a WCT is ventricular in origin, with a reported sensitivity ranging from 43% to 79% and specificity from 58% to 73%.[39,40,45-47] However, patients with BBB or antidromic SVT can also present with a QRS duration > 140 milliseconds.[40,45,46] A QRS > 160 milliseconds has a better predictive value for VT, particularly with left bundle branch block (LBBB) morphology, with sensitivity ranging from 64% to 65% and specificity ranging from 79% to 97%.[40,46,49]

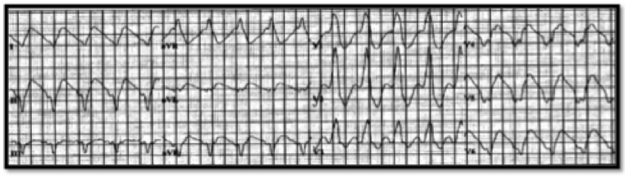
Brugada et al focused on the RS interval, which measures the onset of the R wave to the nadir of the S wave in the precordial leads.[50] They found that an RS interval > 100 milliseconds was 66% sensitive and 98% specific for VT. The logic of this approach is that the initial depolarization of the ventricle may occur more slowly with VT than with SVT with aberrant conduction.

A right BBB is typical in rate-related aberrant conduction, as the right bundle branch characteristically has a longer relative refractory period than its counterpart. More specifically, Wellens et al determined that the presence of a triphasic rSR' pattern in lead V_1 was 90% more likely to indicate aberrant conduction rather than a ventricular rhythm.[44]

Concordance

QRS concordance across the precordial leads (QRS complexes that are entirely positive or entirely negative in leads V_1-V_6) is also predictive of VT, with specificity reported between 90% and 100%.[40,45,47,49]

Figure 4. Extreme Right Axis Deviation (Between -180° And -90°)



Courtesy of Keith A. Marill, MD.

However, precordial concordance can be quite rare, with a reported sensitivity of 0% to 15% among ECGs demonstrating VT.[40,45,49] **(See Figure 5.)**

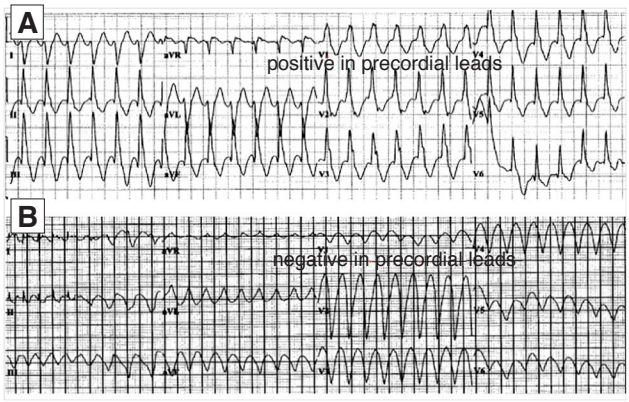
Atrioventricular Dissociation, Capture Beats, And Fusion Beats

When P waves are visible on the 12-lead ECG but are completely independent of regular grouping with QRS complexes, this is known as AV dissociation and is nearly 100% specific for VT. **(See Figure 6).** However, visible AV dissociation is a rare finding in VT, particularly when the ventricular rate is rapid, with reported sensitivity between 5% and 24%.[39,40,47,51]

Capture beats and fusion beats can signify AV dissociation and are highly specific for VT, but they are also rare, occurring in approximately 0.5% of VT cases.[39,47] Capture beats occur when an atrial impulse is conducted through the AV nodal-His-Purkinje system at a fortuitous moment, rapidly depolarizing the ventricles via the His-Purkinje system prior to slow, adjacent depolarization from the ventricular focus. This results in a narrow QRS complex with markedly different morphology than the predominant wide QRS of WCT. **(See Figure 7, page 9.)**

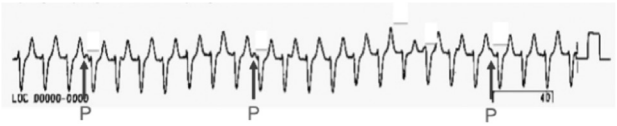
Similarly, fusion beats occur when an atrial impulse propagates through the AV node and reaches the ventricles but then simultaneously depolarizes the myocardium along with the impulse from the ventricular focus. This concurrent depolarization of

Figure 5. Precordial Concordance



(A) Positive concordance. (B) Negative concordance.
Courtesy of Ian deSouza, MD.

Figure 6. Atrioventricular Dissociation



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the ventricles from the combining of both impulses gives rise to an intermediate QRS morphology. **(See Figure 7.)** Although capture beats and fusion beats on the ECG are near-diagnostic of VT, SVT with aberrancy may demonstrate ECG findings similar to fusion complexes, occurring in 7 of 24 tracings in a study by Greenstein and Goldberger.[52]

Comparison To Prior Electrocardiograms

If prior ECGs are available for comparison, patients with baseline BBB can be identified. For patients with baseline BBB in which the WCT involves a QRS morphology that is identical to the baseline BBB, you can generally presume the rhythm to be SVT with aberrancy.[53] Nevertheless, exceptions where VT demonstrated a QRS morphology similar to an underlying BBB have been reported.[14] Evidence of a previous MI on a prior ECG suggests the presence of myocardial scar and may also support the diagnosis of VT.[40]

Diagnostic Algorithms

Algorithms have been developed to assist in differentiating VT from SVT in patients with WCT. Brugada et al developed a multistep approach that combined some of the aforementioned characteristics in order to identify VT: (1) absence of an RS complex in all precordial leads (concordance); (2) RS interval duration > 100 milliseconds; (3) AV dissociation; and (4) QRS morphology characteristics.[50] This algorithm was highly sensitive and specific in their initial sample; however, the Brugada criteria have not been found to be consistent across observers, as reflected by kappa values of 0.42 to 0.58.[1,2] Likewise, the criteria developed by Vereckei et al that assess the presence of an R wave, its duration, and its shape have a reported sensitivity and specificity of 92% and 65%, respectively;[54,55] however, validation is pending. In a recent study, the criteria by Brugada et al were compared to those developed by Vereckei et al in ECGs from 51 patients with induced WCT.[56] The overall sensitivity and specificity were similar, but the use of step 1 (the presence of an initial R in aVR) in the Vereckei algorithm alone yielded a positive likelihood ratio of 18 and required < 10 seconds to assess.

Perhaps the most simple, practical, and appealing algorithm has been developed by Griffith et al.[53] They suggest that, due to its associated risk of deterioration, VT should be the default diagnosis in patients with WCT, unless evidence suggests otherwise. They also suggest that clinicians assess QRS morphology for evidence of typical LBBB or right bundle branch block (RBBB) characteristics; if it is not consistent with either typical BBB pattern, then VT should be assumed. A sensitivity and specificity of approximately 90% and 75%, respectively, were calculated with this approach.

The Role Of Adenosine

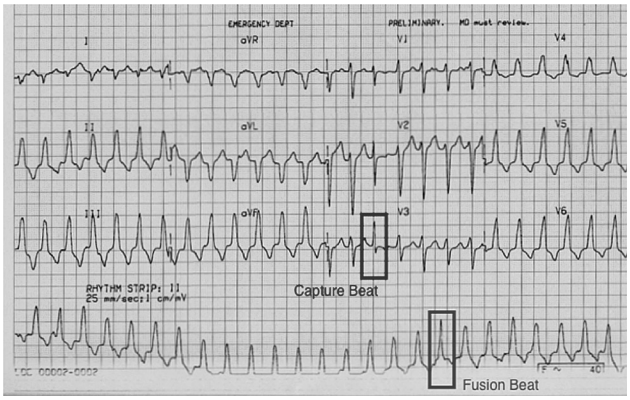
Diagnostic Utility

In WCT, the administration of adenosine can aid in both diagnosis and management by transiently inducing AV-nodal block, which should terminate most re-entrant dysrhythmias that involve the AV node as part of the circuit. **(See Figure 8, page 10.)** These rhythms include AV nodal re-entrant tachycardia (AVNRT)[44] in which there is a *functional* accessory pathway within the AV node, and atrioventricular re-entrant tachycardia (AVRT), both orthodromic and antidromic,[45] which involves an *anatomic* accessory pathway. Adenosine can also reveal the underlying atrial rhythm (such as AF or atrial flutter) through AV-nodal block.[57]

Furthermore, a lack of response to adenosine can also be diagnostic. In a retrospective study of 197 patients with WCT by Marill et al, nonresponse to adenosine increased the odds of a patient having VT by a factor of 9.4, while any response to adenosine increased the odds of SVT by a factor of 36.3.[35] However, in certain rare cases, a response to adenosine can confirm VT. In a case report from Schuller et al, adenosine transiently induced retrograde ventriculoatrial nodal block, supporting the diagnosis of VT.[58]

Although primary studies[35,59] and a review[60] have extolled the safety of adenosine administration in patients with WCT, there are situations in which adenosine is contraindicated.[35,59,60] The quintessential WCT rhythm for which adenosine is contraindicated is AF in the setting of WPW syndrome. Approximately 39% of WPW cases may be associated with AF.[20] Adenosine may induce AV block and accelerate conduction of atrial fibrillatory impulses through the bypass tract, which can lead to very rapid ventricular arrhythmias that degenerate to VF.[19,35,59,60] There are also case reports[61,62] in which adenosine administration in other WCTs may have precipitated VT and VF.

Figure 7. Capture And Fusion Beats



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Laboratory Analysis

Laboratory testing is of limited value in the early management of WCT, but focused testing can be useful in order to prevent deterioration in a previously stabilized patient.

Electrolytes

In the setting of WCT, obtain both a potassium level and a magnesium level. Correction of both hypokalemia and hypomagnesemia can aid in prevention of recurrent VT.[63-65]

Cardiac Biomarkers

The measurement of cardiac biomarkers in patients with WCT, specifically troponin levels, may be reasonable, as most of these patients will have VT due to prior MI, and, therefore, they will be at risk for recurrent MI. However, when VT is associated with minor elevations of troponin, it should not be assumed that this WCT is a result of an acute MI.[3] In fact, these troponin elevations are of limited diagnostic value and are likely due to type 2 MI (caused by ischemia from a mismatch in supply and demand); the patient should be treated for VT in the same manner as a patient without such a laboratory finding. A patient with WCT who is determined to have SVT with aberrancy after termination with adenosine may not require troponin testing unless the patient exhibits symptoms of an acute coronary syndrome or there is uncertainty in rhythm diagnosis.[66]

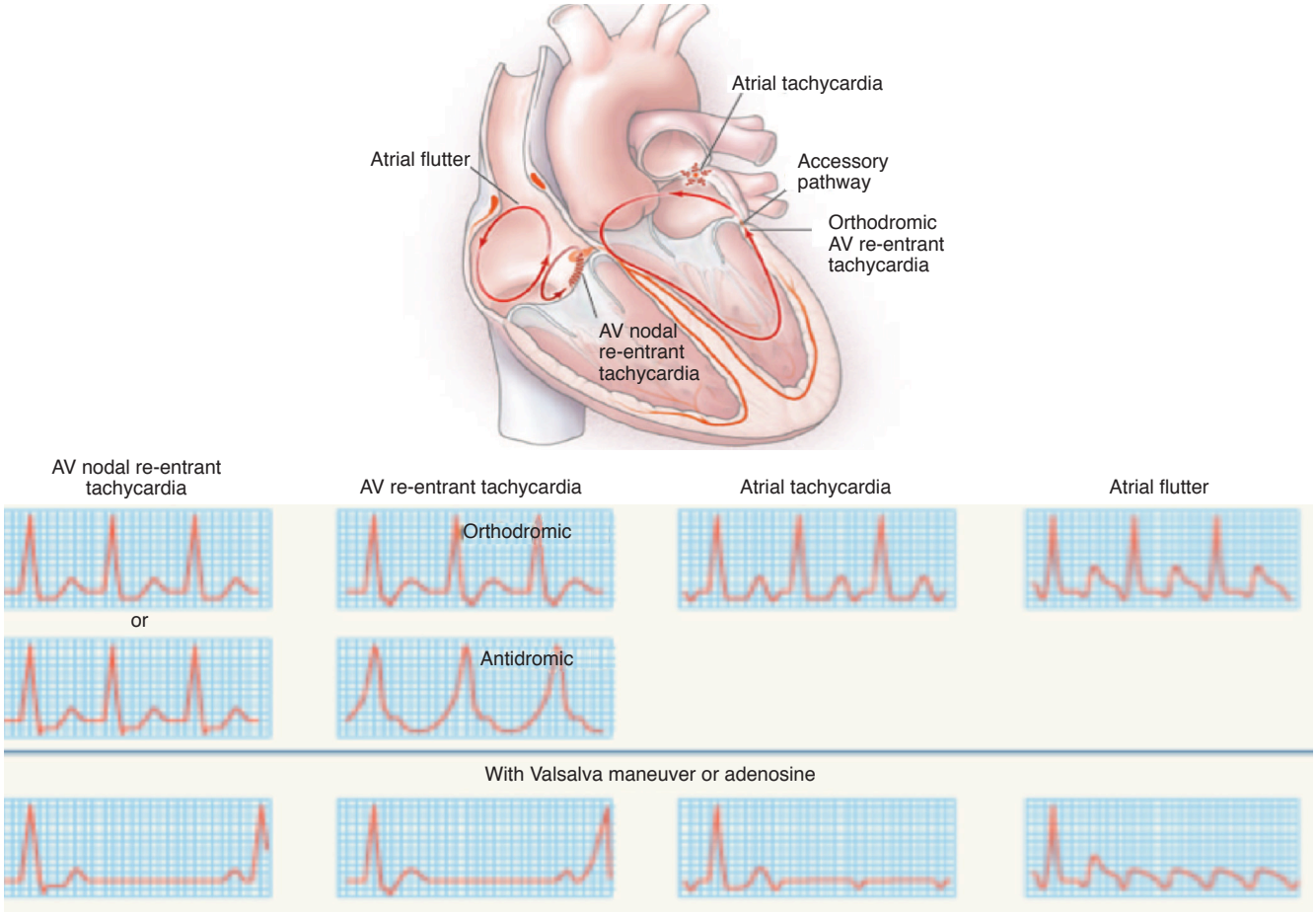
Toxicology Screening

The urine drug screen may be of some utility in patients presenting with WCT that is thought to be related to drug ingestions. Some drugs that result in sodium-channel blockade can also induce tachydysrhythmias. The urine drug screen can aid in detection of WCT secondary to cocaine, but will be of limited use to detect drugs for which urine is not routinely used for testing.

Treatment

In cases of pulseless VT, circulation must take priority, and prompt defibrillation should be performed with 200 J (biphasic) or 360 J (monophasic), followed

Figure 8. Supraventricular Tachycardia And Response To Adenosine



Abbreviation: AV, atrioventricular.

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by immediate chest compressions.[5,67] Minimizing time to defibrillation is the leading predictor of survival after a cardiac arrest due to pulseless VT or VF.[68] In this subgroup of malignant ventricular dysrhythmias, assessment of airway and ventilation should follow the first cycle of uninterrupted compressions. In all other cases, assess airway, breathing, and circulation, and intervene accordingly.

Unstable Wide-Complex Tachycardia

Whereas pulselessness should be self-evident, determining hemodynamic stability in a patient with a WCT may be more subjective and involves an assessment of end-organ (ie, heart, brain, skin) perfusion. Do not rely solely on the presence or absence of hypotension, but consider other symptoms and signs of hypoperfusion such as ischemic chest discomfort, altered mental status, and pallor/diaphoresis.

Unstable WCT, whether ventricular or supraventricular in origin, regular or irregular, should be treated initially with synchronized DCCV.[5,69] If the patient is conscious and the situation allows, sedation/analgesia should precede electrical therapy. Defibrillator pads may be placed on the chest in an anterior-lateral or anterior-posterior configuration.[67] The defibrillator should be set to synchronize shock delivery with the R wave of the cardiac cycle. This is recommended in order to avoid shock delivery on the upslope of the T wave, which may precipitate another triggered, malignant dysrhythmia.[3] DCCV should be administered at an initial dose of 100 J (biphasic) and may be escalated, if necessary, when there is lack of response.[3] This approach to treatment must be modified in cases of polymorphic VT, in which the defibrillator may not recognize the varying morphologies of QRS complexes, fail to deliver the shock, and ultimately delay appropriate therapy. Therefore, it is recommended that initial therapy be defibrillation at 200 J (biphasic) for an unstable polymorphic VT.[3]

Failure Of Initial Therapy

DCCV terminates VT in approximately 90% of cases.[70] Occasionally, VT may fail to terminate with DCCV. This may occur for a number of reasons, including correctable variables such as poor conduction at the interface of the skin and electrical pad or paddle; poor internal conduction due to the presence of pneumothorax, electrolyte abnormality, hypoxia, or severe acidosis; or an insufficient cardioverter energy setting. Some variables, such as large body mass index, are not rapidly correctable. Following assessment for correctable variables, attempt DCCV again. Medical therapy (such as procainamide) may also be considered if the patient is stable, as this may facilitate subsequent DCCV (as in cases of AF).[71] Vagal stimulation via Valsalva maneuver, having the patient cough, or performing laryngoscopy can occasionally terminate VT,[72,73] but they are not generally recommended. In the absence of obviously correctable factors, consultation with the electrophysiology service for emergent overdrive pacing in the electrophysiology laboratory should be considered. Ultimately, VT ablation may be the definitive therapeutic modality, although it is generally not performed emergently.

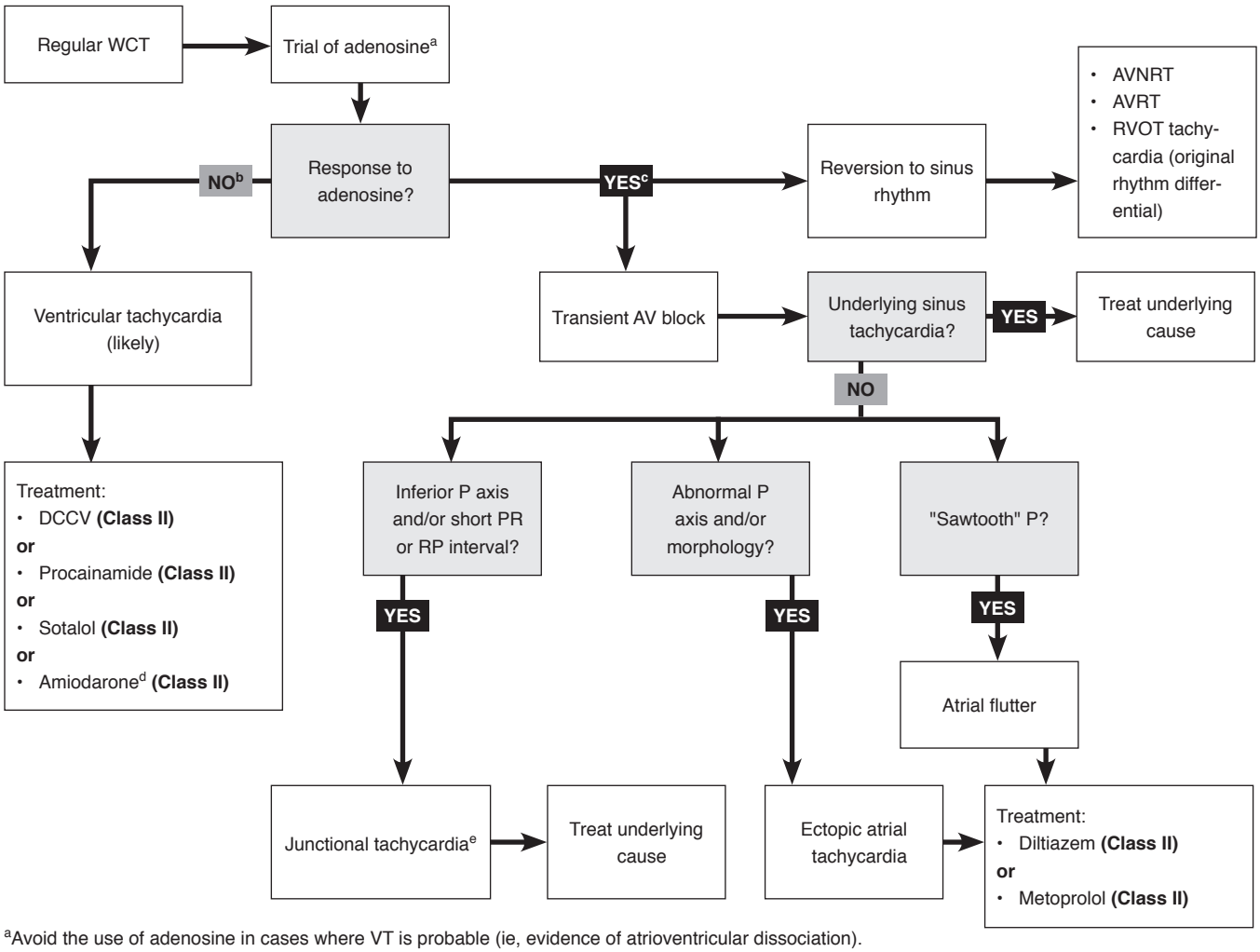
Medications For Stable Wide-Complex Tachycardia

Management of stable WCT begins with measuring electrolytes and liberally supplementing potassium and magnesium levels.[3] If the rhythm is regular, adenosine is safe and may be diagnostic (and even therapeutic) in SVT with aberrancy.[35] These various regular SVTs, if not terminated with adenosine, may then be treated with rate-controlling agents such as calcium-channel antagonists or beta-adrenergic antagonists. Ultimately, radiofrequency catheter ablation of the causative culprit will be curative.[74]

If response (diagnostic or therapeutic) to adenosine is absent or, in rare cases, results in retrograde ventriculoatrial block, the rhythm is more likely VT.[35] In these cases, the initial choice of therapy is less clear. Typical drug options include Vaughn-Williams class I antidysrhythmics (such as lidocaine and procainamide) and class III drugs (such as amiodarone and sotalol). Procainamide and sotalol will prolong the ventricular myocardial refractory period in order to terminate re-entry. Lidocaine predominantly affects automaticity, and amiodarone—when given intravenously—has no significant acute effect on ventricular refractoriness and repolarization. Its antidysrhythmic efficacy is largely time-dependent and due to accumulation of its active metabolite.[75,76] Therefore, lidocaine and amiodarone may be more effective for the treatment of VT when the mechanism is abnormal automaticity, such as in acute myocardial ischemia. In a systematic review of drug comparison trials for treatment of stable, monomorphic VT, it was found that procainamide and sotalol were superior to lidocaine.[77] In the same review, procainamide was not found to be more effective than amiodarone, but 2 noncomparison studies have shown that amiodarone is relatively ineffective for treatment of stable, monomorphic VT.[78,79] Therefore, current guidelines recommend procainamide as the first-line agent.[5] Despite this recommendation, however, even when stable, VT is likely most effectively treated by sedation and cardioversion.[79,80]

If the rhythm is irregular, it may be polymorphic VT. If the baseline QTc is prolonged, the rhythm is torsades de pointes. Management should start with discontinuing all offending medications and administering intravenous magnesium.[5] In cases of torsades de pointes preceded by bradycardia or pauses, electrical pacing (either transcutaneous or transvenous) may be employed to maintain

Clinical Pathway For Initial Management Of Stable, Regular Wide-Complex Tachycardia



[a]Avoid the use of adenosine in cases where VT is probable (ie, evidence of atrioventricular dissociation).

[b]Ventricular tachycardia may respond to adenosine with transient AV nodal block.

[c]There are 2 different responses to adenosine; the character of the response will help determine the underlying rhythm.

[d]Amiodarone has a delayed antidysrhythmic effect and may be relatively less successful (see "Medications for Stable Wide-Complex Tachycardia" on page 14).

[e]This is a rare type of SVT that may be seen in patients with enhanced AV nodal automaticity, digoxin toxicity, or myocarditis.

Abbreviations: AVNRT, atrioventricular nodal re-entrant tachycardia; AVRT, atrioventricular re-entrant tachycardia; DCCV, direct-current cardioversion; RVOT, right ventricular outflow tract; WCT, wide-complex tachycardia.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

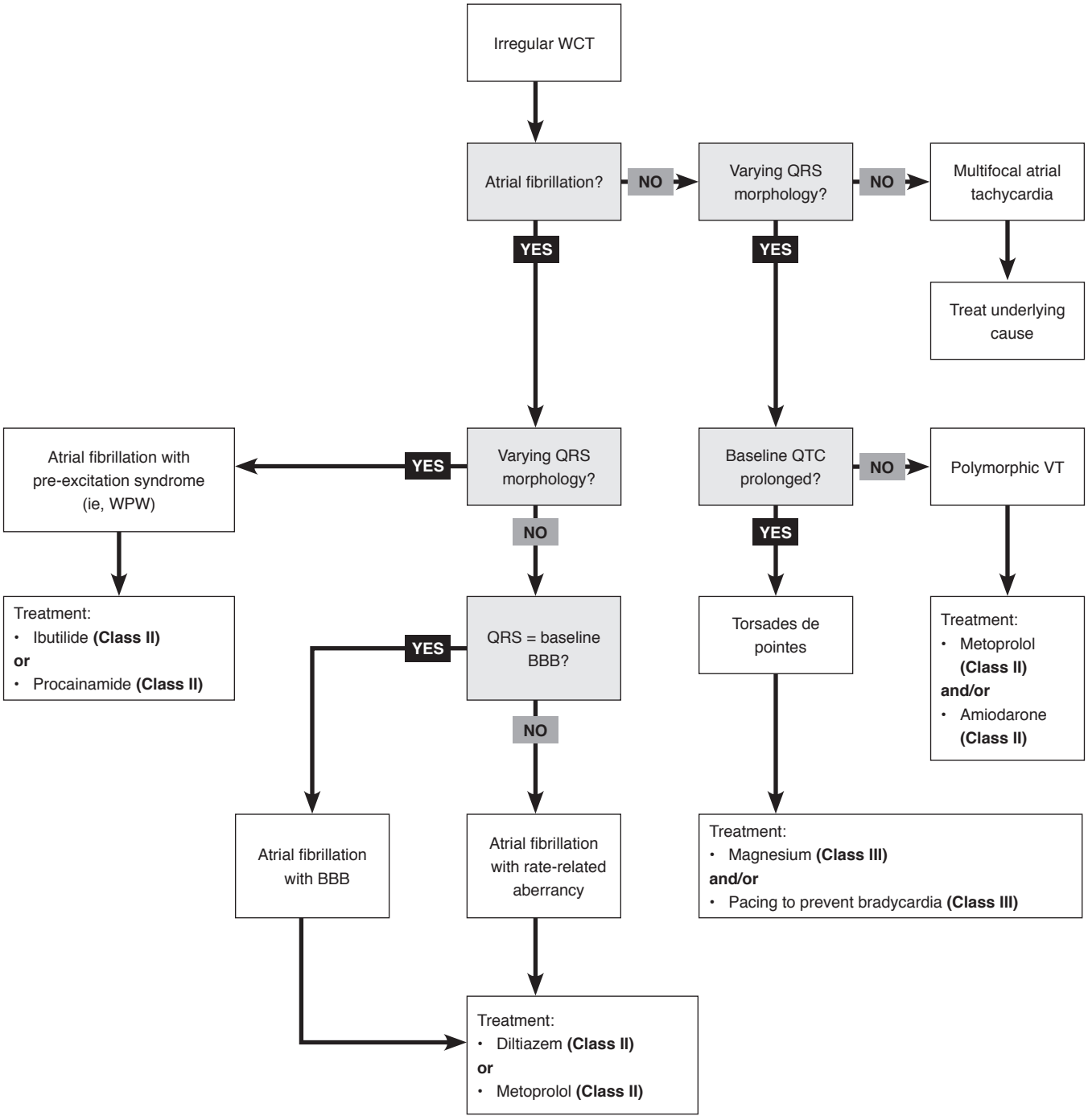
Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Clinical Pathway For Medical Management Of Stable, Irregular Wide-Complex Tachycardia



Abbreviations: BBB, bundle branch block; VT, ventricular tachycardia; WCT, wide-complex tachycardia; WPW, Wolff-Parkinson-White syndrome.
For Class of Evidence definitions, see page 12.

normal heart rate and, consequently, to shorten the QT interval.[81] Another irregular WCT is pre-excited AF. In contrast to treatment of regular SVTs, avoid using AV-nodal blocking agents, including short-acting adenosine.[5] In this situation, AV-nodal agents may lead to increased ventricular response through preferential conduction of atrial impulses by the accessory tract. DCCV is recommended for the patient with unstable wide-complex AF. However, if chemical conversion is preferred, rhythm control may then include procainamide or ibutilide.[82-85] Catheter ablation of the accessory tract will ultimately provide definitive treatment.[74] Lastly, if the patient has a baseline, pre-existing conduction abnormality with wide QRS morphology identical to that of the presenting rhythm, it is likely that this rhythm is simply AF with BBB. Rate control may be attempted with agents such as calcium-channel antagonists or beta-adrenergic antagonists.

Treatment Of Extrinsic Causes

Extrinsic causes of WCT may include hyperkalemia and drug overdose, which should be treated in a case-specific manner. WCT that is related to drug overdose is typically due to TCAs. Rarely, cases can be caused by digoxin and may involve a characteristic ECG rhythm known as bidirectional VT.[70] Each drug in overdose has its preferred antidote (eg, sodium bicarbonate for TCA and digitalis-Fab for digoxin). WCT (such as SVT with aberrancy) is often due to an underlying acute medical illness; the specified illness should be addressed earlier on in management, and the rhythm should be treated later if necessary. Treating the WCT rhythm first in patients with rate or rhythm control is less likely to be successful and may be associated with a greater risk of adverse events.[10]

Implantable Cardioverter-Defibrillators

Most patients who suffer from VT will undergo evaluation for implantable cardioverter-defibrillator (ICD) placement in order to prevent recurrent, sustained dysrhythmias. In patients who require ICD implantation, concomitant antidysrhythmic therapy is often used to reduce ICD shocks, but this is sometimes ineffective. In cases of electrical storm, catheter ablation has been found to be an effective strategy, both to reduce recurrence of electrical storm and the frequency of VT episodes.[73] Some experts have also begun to advocate primary ablation without ICD placement in select patients with hemodynamically stable VT with preserved ejection fraction.[86]

Special Circumstances

Irregular Wide-Complex Tachycardia With Wolff-Parkinson-White Syndrome

The diagnosis of AF in conjunction with WPW syndrome is critical, as it will dictate therapy, but the recognition of this dysrhythmia may be difficult. Rates are often rapid, and the rhythm may appear regular to the undiscerning eye. Evidence on ECG that it is, indeed, AF with WPW may be the presence of beat-to-beat variability in QRS duration.[20] The narrower complexes are due to anterograde conduction of atrial impulses through the AV node and His-Purkinje system. In contrast, the wider complexes are a result of the conduction of atrial impulses through the accessory tract to the ventricles without the use of the rapidly conducting His-Purkinje fibers. These 2 pathways of AV conduction continue to compete for capture of the ventricles, and this, along with the additional potential for rate-related aberrancy, will give rise to QRS complexes of varying duration. **(See Figure 9, page 15.)**

Current guidelines recommend avoiding agents that slow conduction through the AV node in cases of pre-excited AF, as conduction of rapid atrial impulses may then occur preferentially through the accessory tract to yield rapid, untenable ventricular rates.[5] Drugs that slow conduction through the accessory tract are preferred; procainamide and ibutilide have both demonstrated success.[83,85]

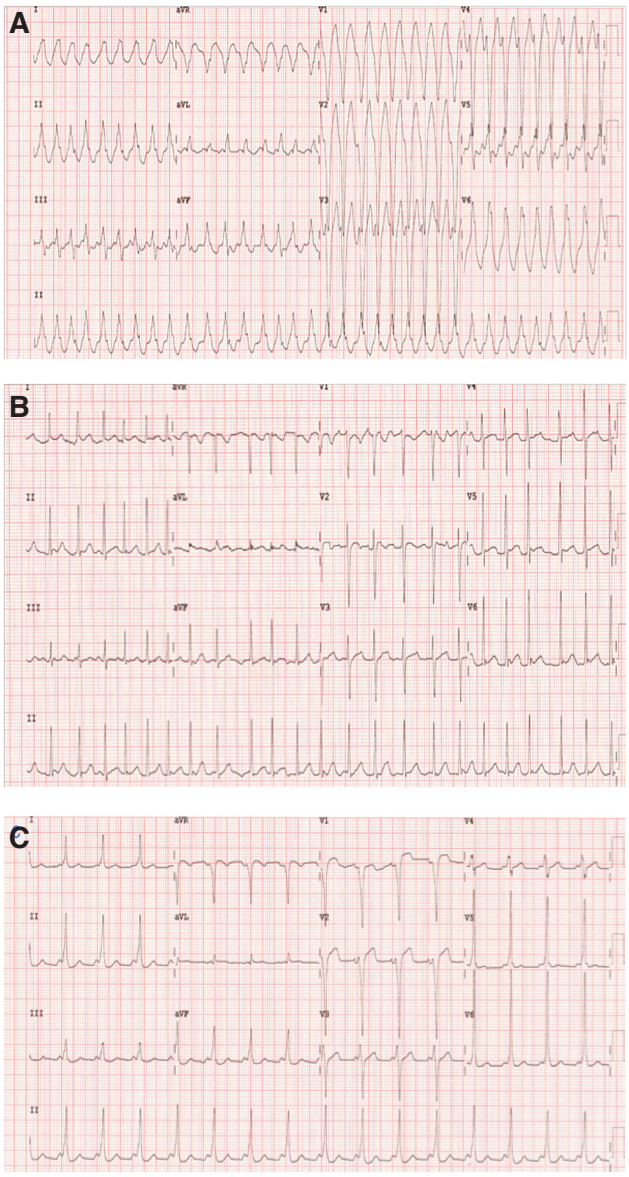
Ventricular Tachycardia As A Consequence Of Acute Myocardial Infarction

VT and ventricular fibrillation secondary to an acute MI has markedly decreased in incidence, coincident with advances in reperfusion therapy for ST-segment elevation MI.[87,88] This is most likely due to rapid restoration of patency in the culprit vessel that reduces ischemia and, therefore, prevents subsequent myocardial scar formation. In cases where infarction progresses without reperfusion therapy, the development of scarring with its associated slow conduction will provide the substrate for re-entry and increase the risk of subsequent VT. Interestingly, the mechanism of VT induction appears to vary, dependent on the time from the onset of MI. During the acute ischemic event, VT will typically occur as a result of abnormal automaticity; afterward, the mechanism is usually re-entrant.[89] Late onset (> 48 hours after MI occurs) sustained VT has a particularly poor prognosis, reflecting irreversible myocardial injury that may lead to VT recurrence and increased mortality.[90] Although the use of amiodarone for stable VT has been de-emphasized,[5] it may be an effective treatment for polymorphic VT in the setting of MI.[91]

Right Ventricular Outflow Tract Ventricular Tachycardia

Right ventricular outflow tract tachycardia is rare, and it is unique in that it may occur in the absence of structural heart disease. The rhythm typically has a LBBB pattern and an inferior QRS axis, suggesting an origin from the right ventricular outflow tract.[92] Through the use of programmed stimulation and measured response to drugs, VT has been postulated to be triggered by activity mediated by cyclic adenosine monophosphate.[92] The dysrhythmia is also known as adenosine-sensitive, catecholamine-dependent VT. Adenosine terminates this VT through its effect of antagonizing catecholamine-stimulated cyclic adenosine monophosphate elevation in ventricular myocardium.[93] Patients with this particular VT have a good prognosis, provided that right ventricular dysplasia (a potential structural etiology that can lead to sudden death) has been excluded.[94,95] When medical therapy is not well tolerated, catheter ablation may provide a long-term solution.[74,96]

Figure 9. Wide-Complex Tachycardia In Special Populations



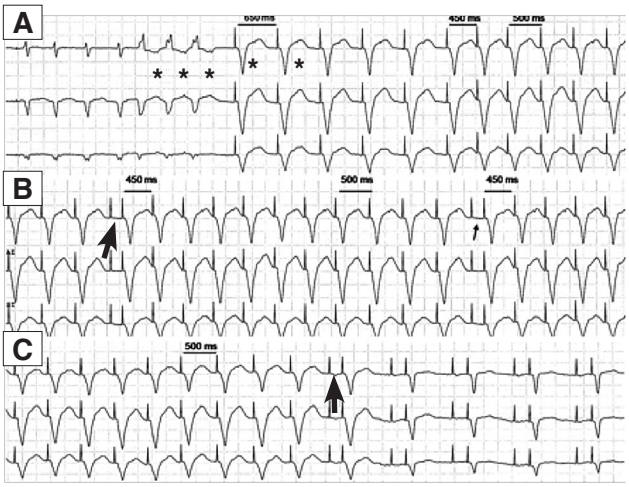
(A) Wide-complex tachycardia in a patient with Wolff-Parkinson-White Syndrome. Note the subtle irregularity in the rhythm, suggesting atrial fibrillation. (B) Procainamide infusion slows conduction in accessory pathway, and atrial impulses resume propagation primarily through the AV nodal-His-Purkinje system. (C) Continued procainamide infusion and rhythm conversion to sinus tachycardia with short PR and delta.

Courtesy of Ian deSouza, MD.

Wide-Complex Tachycardia In The Patient With An Implantable Pacemaker

Pacemaker-mediated tachycardia is a WCT that is observed exclusively in patients with dual-chamber pacemakers. **(See Figure 10.)** Pacemaker-mediated tachycardia is a re-entrant dysrhythmia that is similar to others except that the pacemaker itself forms part of the re-entry circuit. This re-entrant tachycardia will not exceed the pacemaker's programmed upper rate limit; however, tachycardia may be significant enough to cause symptoms necessitating emergent treatment. A trial of adenosine may or may not be effective.[94] On the other hand, placing a magnet over the pacemaker will definitively terminate this re-entrant dysrhythmia.[97] After a pacemaker-mediated tachycardia event that is successfully treated, the device will need to undergo adjustments to its atrial sensing thresholds. Most

Figure 10. Pacemaker-Mediated Tachycardia



(A) First 3 asterisks indicate PVCs with retrograde P wave that then triggered the onset of PMT. (B) Arrow shows lack of atrial capture, a failed attempt at termination by the device. (C) Arrow indicates successful atrial capture and termination of PMT.

Abbreviations: PMT, pacemaker-mediated tachycardia; PVC, premature ventricular contraction.

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modern devices include programming to prevent pacemaker-mediated tachycardia. (For more information on managing patients with pacemakers, see the September 2014 issue of *Emergency Medicine Practice*, "Managing Pacemaker-Related Complications And Malfunctions In The Emergency Department" at www.ebmedicine.net/pacemakers.)

Slow VT may be seen in up to 30% of patients with an ICD.[98] Slow VT is defined as VT with a rate of 101 to 148 beats/min. The dysrhythmia may persist without therapy by the ICD because the rate may lie below the device's tachycardia detection rate (the rate above which therapy is applied). Patients with slow VT, as in other tachycardias, may exhibit signs and symptoms of hypoperfusion, but usually without clinical consequences, as the dysrhythmia is typically short lasting. If necessary, drug therapy may be instituted in order to suppress further tachycardia episodes.

Controversies/Cutting Edge

Predicting Tachydysrhythmias

The ability to predict the occurrence of tachydysrhythmias remains limited. In addition to a prior dysrhythmic or MI event, compromised ejection fraction is the major factor used to identify increased risk of life-threatening VT; an ejection fraction < 30% is an indication for prophylactic ICD placement.[99] Congestive heart failure may precede the development of both ventricular and atrial tachydysrhythmias, including AF and atrial flutter. Valvular disease may also portend the development of atrial tachydysrhythmias.

A number of advanced electrocardiographic techniques (including signal averaged electrocardiography to detect late ventricular potentials, measurement of heart rate variability, and T-wave alternans detection using spectral and modified moving average techniques) have been developed. These have shown some benefit in predicting VT and have been used to identify patients at both relatively higher and lower risk, but they are not definitive. Regarding short-term tachydysrhythmia prediction, recurrent nonsustained VT may herald an imminent sustained VT in the monitored intensive care unit setting, but the use of more-advanced predictive technologies awaits further development.

Electrical Therapy

Electrical therapy is the treatment of choice for patients with pulseless and other unstable tachydysrhythmias. It is highly effective and considered safe. Nevertheless, the safety of recurrent shocks has long been questioned.[100] Recent intriguing data from the Multicenter Automatic Defibrillator Implantation Trial (MADIT) group suggests that ICD shock therapy is associated with increased mortality.[101] The mechanism of this association is uncertain. It is also uncertain whether current energies employed with a biphasic waveform are ideal for transthoracic DCCV or defibrillation to achieve maximal effectiveness with minimal harm. Overdrive, or antitachycardia pacing, is generally less painful and may be less detrimental than a single electrical shock for tachycardia termination.[101] Today, antitachycardia pacing is delivered by ICDs via an automated algorithm programmed at the time of implantation or by manual control at the bedside by an electrophysiologist. If antitachycardia pacing is proven to be safer in the long term, then its use could increase through either ICD or transthoracic delivery in the ED.

New Medications

Vernakalant is an agent developed to treat AF and atrial flutter by specifically prolonging the atrial action potential to block atrial re-entry. It accomplishes this by antagonizing the ultra-rapid delayed rectifier and other potassium currents in the atria at high heart rates with relatively less effect on ventricular repolarization and the QT interval.[102] This drug has been approved for use in Europe but not in the United States. Development and approval of an agent such as vernakalant that specifically targets atrial repolarization would represent an advance in treatment over the most effective agents currently available in the United States. The most effective medicines used today, such as ibutilide, cause global repolarization delay and QT prolongation, and they risk the development of potentially lethal torsades de pointes. A drug that specifically targets atrial repolarization could be useful to terminate a variety of wide-complex supraventricular tachycardias, including AF, atrial flutter, atrial tachycardia, and possibly AVNRT.

Disposition

Patients Appropriate For Discharge

Patients without symptoms of acute coronary syndromes or other comorbidity who are ultimately diagnosed with SVT can safely be discharged home. Luber et al retrospectively studied 111 patients who were diagnosed with SVT in the ED.[103] Of these patients, 79 (71%) were ultimately discharged, and only 3 returned to the ED with recurrent SVT within the 3-year study period. None of these recurrences were complicated by hemodynamic instability. Close electrophysiology or cardiology follow-up should be urged for patients with known pre-excitation syndromes or new-onset SVT. Discussion with a patient's electrophysiologist or cardiologist, if available, can also help determine appropriate disposition and/or arrange further testing (such as Holter monitoring).

Risk Management Pitfalls In Managing Wide-Complex Tachycardia

1. **"There's no way that's VT. The patient is completely stable."**
   A patient's hemodynamic status is a poor predictor of VT. The diagnosis should be made based on examination of the 12-lead ECG, and treatment should be tailored appropriately.

2. **"Maybe if I just try to shock him at 200 J again, it'll work."**
   Be cautious when repeating treatments, including electrical cardioversion, if the patient is not responding as expected. Hyperkalemia and drug toxicity can mimic VT, as can sinus tachycardia with a BBB, and all such cases will be refractory to DCCV.

3. **"She's got a pacemaker, so I'll bet that's just sinus tachycardia on the monitor."**
   Patients with pacemakers that trigger ventricular contraction will, by definition, demonstrate a WCT while in sinus tachycardia. However, these patients may also have underlying ischemic or structural heart disease that can put them at risk for slow VT. Always obtain a 12-lead ECG and do not rely solely on the single lead displayed on the monitor.

4. **"This kid's barely 18 years old, so this irregular WCT has to be WPW with AF, right?"**
   In young adults, be sure to consider the risk of VT before assuming that the dysrhythmia is secondary to SVT. Long-QT syndrome, congenital heart defects, and drugs can increase the risk of ventricular dysrhythmias.

5. **"This QRS complex doesn't look that different from the BBB seen on prior ECGs, so I'm sure it's just SVT with aberrancy."**
   VT in patients with underlying BBB can demonstrate major changes, minor changes, or no change at all in QRS morphology. Look for other ECG criteria associated with VT before assuming that it is SVT with aberrancy.

6. **"I know this has to be torsades de pointes! Let's just give 2 g of magnesium."**
   Remember that torsades de pointes secondary to prolonged QT is only 1 etiology of polymorphic VT, so consider acute cardiac ischemia when a patient presents in this rhythm.

7. **"Well, he's stable, so let's avoid electrical cardioversion and just use amiodarone."**
   Although amiodarone is the first-line agent for treatment of VT in the ERC guidelines, several studies have suggested that amiodarone may be less effective than other drugs, and procainamide is the first-line treatment recommended by the ACC/AHA.

8. **"He didn't respond to adenosine, so it must be VT."**
   Although the vast majority of SVTs will demonstrate some response to adenosine, there are some SVTs that will not respond. Furthermore, right ventricular outflow tract tachycardia may terminate with adenosine administration. Keep in mind that adenosine must be pushed rapidly with a flush and that some patients will respond to a 12-mg dose, even when they don't respond to 6 mg.

9. **"This patient's WCT could be related to hyperkalemia, but she's pretty stable, so I'll just start procainamide and wait for her potassium level to come back from the lab."**
   WCT secondary to hyperkalemia will not respond to antidysrhythmics, and it carries a high risk of hemodynamic decompensation and cardiac arrest. If hyperkalemia is suspected, administer calcium to stabilize the myocyte membrane. Calcium administration will have minimal adverse effects on other mechanisms of WCT.

10. **"This is new-onset SVT, so this patient has got to be admitted."**
    Young patients with a primary SVT who do not have symptoms or signs of myocardial ischemia, and who are stable throughout the ED course after conversion to sinus rhythm, can be safely discharged home.

Patients To Consider For Admission

Patients who present with new-onset WCT, regardless of the outcome of ED management, should be observed and considered for admission to a monitored bed for further evaluation and management. Since differentiating between VT and SVT is difficult, maintain a high suspicion of VT in the differential diagnosis. It should also be reiterated that some VTs may respond to adenosine with termination of the dysrhythmia, so be careful of considering the response to adenosine as reassurance of a safe discharge. Discharge can be considered after consultation with an electrophysiologist or cardiologist and after discussion of the risks and alternatives with the patient.

Emergent consultation should also be considered for older patients with new-onset SVT, patients with syncope or other severe symptoms, drug-resistant SVT, or patients with pre-excitation syndromes. Patients with SVT secondary to infection, drug ingestion, or decompensated heart disease (both ischemic and structural) may benefit from admission for treatment of the specific underlying etiology.

Patients with VT should be admitted to a medicine or cardiology service and to a monitored floor bed or step-down unit. Patients with recurrent tachydysrhythmias or severe hemodynamic compromise should be admitted to the intensive care unit. If available, patients with WCT secondary to extrinsic causes (eg, drugs, hyperkalemia) should be admitted to the appropriate service (toxicology, nephrology).

Summary

WCT can be a life-threatening emergency, and it can also be difficult to manage. The management of unstable tachydysrhythmias focuses on ensuring adequate oxygenation and perfusion as well as prompt synchronized cardioversion. However, if time and patient stability allow, appropriate medical therapy may be an effective strategy. The most important tool in assessing the patient with WCT is the ECG. Adjunctive diagnostic tools, such as the history and physical examination and response to Valsalva maneuvers or adenosine infusion, may also aid in diagnosis and therapeutic decisions.

If the underlying rhythm is identified to be VT in a stable patient, electrical cardioversion may still be the most effective first choice in management. However, if drug therapy is preferred, remember that current guidelines recommend procainamide as the first-line agent, despite a lack of high-quality evidence demonstrating superiority of one drug over another. For WCT that is found to be SVT, the diagnosis of the underlying rhythm is also important for choosing the correct treatment. Adenosine can be used first for suspected regular wide-complex SVT, and it may be therapeutic or diagnostic; however, adenosine and other AV-nodal blockers should be avoided if the WCT is irregular and pre-excited AF is suspected. Irregular WCT should be treated with agents such as procainamide or ibutilide that preferentially act upon the bypass tract. Finally, when pre-excitation has been excluded, calcium-channel antagonists or beta-adrenergic antagonists can also be used to slow AV-nodal conduction to the ventricles from an intra-atrial rhythm (such as AF or atrial flutter).

Once the tachydysrhythmia has been controlled and the patient has been stabilized and observed, patients with primary SVT and no significant comorbidities can be discharged with close follow-up with cardiology or electrophysiology. Patients with unstable WCT rhythms and patients with tachydysrhythmias of uncertain origin or secondary to underlying illness should be admitted to the hospital for further management. Patients with recurrent tachydysrhythmias should be admitted to the intensive care unit.

Selected Abbreviations

ACC	American College of Cardiology
AF	Atrial fibrillation
AHA	American Heart Association
AV	Atrioventricular
AVNRT	Atrioventricular nodal re-entrant tachycardia
AVRT	Atrioventricular re-entrant tachycardia
BBB	Bundle branch block
DCCV	Direct-current cardioversion
ECG	Electrocardiogram
ERC	European Resuscitation Council
ESC	European Society of Cardiology
ICD	Implantable cardioverter-defibrillator
MI	Myocardial infarction
PMT	Pacemaker-mediated tachycardia
PVC	Premature ventricular contraction
RVOT	Right ventricular outflow tract
SVT	Supraventricular tachycardia
TCA	Tricyclic antidepressant
VF	Ventricular fibrillation
VT	Ventricular tachycardia
WCT	Wide-complex tachycardia
WPW	Wolff-Parkinson-White

Case Conclusions

For the middle-aged man who came in unable to breathe, after the tech finished placing the ECG leads, you glanced down at the ECG as it was printing and noticed that there was a leftward axis and QRS duration of 136 milliseconds. There was no evidence of AV dissociation and no concordance across the precordial leads. The urgency of the situation precluded further analysis at that moment, and you proceeded with cardioversion, but you placed SVT with aberrancy on the differential. Direct cardioversion failed even at 200 J. You performed a bedside pulmo-

nary sonogram that demonstrated pulmonary edema. The patient's pulse oximetry continued to drop. You proceeded with intubation, with a resultant reduction in sympathetic tone, and the patient's heart rate decreased and revealed a sinus rhythm with left bundle branch block. The patient was treated for new-onset congestive heart failure and admitted to the ICU.

Your syncope patient remained hemodynamically stable, and you had plenty of time to review his ECG. You noted negative concordance across the precordial leads, a QRS of 166 milliseconds, and possibly a fusion beat in the rhythm strip. "So it is VT," you thought to yourself. You administered procainamide, which successfully converted the rhythm. Review of his chart demonstrated a previous MI and a previous recommendation by his cardiologist to have an ICD placed. The patient was admitted to the ICU for placement of an ICD.

You observed that the young woman appeared ill and wondered if you had time to get an ECG. You cycled her blood pressure, which was stable at 105/66 mm Hg. The tech handed you the ECG. Peering over your shoulder, the nurse said, "Looks like SVT. Do you want me to grab some adenosine?" However, you noted that there was irregular WCT in all leads, without torsades de pointes morphology. Given her age, you assumed that this was AF due to WPW syndrome, so you replied, "No, get me a crash cart and some procainamide." You placed the pads on the patient, recycled her blood pressure, which was still stable, and started a procainamide infusion. Ultimately, despite appropriate medical treatment, the patient developed significant hypotension and required synchronized cardioversion to return her back to normal sinus rhythm. She was admitted to a monitored step-down bed with a plan for an electrophysiology study the following morning.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

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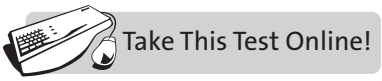
CME Questions



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1. All of the following ECG characteristics are suggestive of VT with WCT EXCEPT:
a. Upward right QRS axis
b. QRS duration > 160 milliseconds
c. Positive concordance of the ECG chest leads
d. P wave associated with each QRS complex

2. A 60-year-old woman with a history of hypertension presents with acute chest pain radiating to the left shoulder as well as nausea. Her pain is relieved with aspirin and nitroglycerin therapy. Vital signs include: heart rate, 78 beats/min; blood pressure, 160/100 mm Hg; respiratory rate, 20 breaths/min; and temperature, 36.5°C. The patient is mildly diaphoretic. Cardiac examination reveals a regular rhythm, and lungs are clear to auscultation. The abdomen is soft and nontender, and there is no peripheral edema. ECG demonstrates normal sinus rhythm with newly inverted lateral T waves, but no frank ST-segment elevation, and a normal corrected QT interval. In the ED on the cardiac monitor, the patient develops a polymorphic VT at a rate of 150 beats/min. She remains alert and awake with no recurrent pain. While preparing to take the patient to the cardiac catheterization lab, initial therapy might include:
a. Adenosine
b. Procainamide
c. Amiodarone
d. Emergent defibrillation
e. Calcium chloride

3. A 57-year-old man presents to the ED with a chief complaint of chest pain, palpitations, and mild confusion. Vital signs include: heart rate, 172 beats/min; blood pressure, 89/50 mm Hg; respiratory rate, 20 breaths/min; and temperature, 36.1°C. Respirations are normal, and heart sounds are rapid and regular. ECG demonstrates a wide QRS-complex tachycardia with QRS duration of 162 milliseconds and LBBB pattern. Initial therapy includes:
a. Adenosine
b. Procainamide
c. Amiodarone
d. Synchronized DCCV
e. Defibrillation

4. A 69-year-old woman with a history of MI, congestive heart failure, and VT presents with palpitations. She is ambulatory with mild shortness of breath on exertion. Vital signs include: heart rate, 165 beats/min; blood pressure, 102/75 mm Hg; respiratory rate, 18 breaths/min; and temperature, 37.2°C. Physical examination is remarkable for 3-cm jugular venous distention, rapid regular heart sounds, bibasilar crackles on lung examination, soft and nontender abdomen, and 1+ bilateral lower extremity edema. Mental status and neurologic examination are normal. ECG demonstrates regular WCT at a rate of 165 beats/min with QRS duration of 170 milliseconds and QRS with an upward right axis. Initial therapy includes:
a. Adenosine
b. Procainamide
c. Amiodarone
d. Synchronized DCCV
e. Defibrillation

5. **A 40-year-old woman presents with palpitations and mild light-headed sensation while driving today. She has experienced a few similar episodes, but she has never lost consciousness. There is no associated chest pain or shortness of breath. She has no known history of cardiac or metabolic illness. She takes amitriptyline at night for depression and insomnia. There is no family history of heart disease before the age of 50 or sudden death. Vital signs include: heart rate, 160 beats/min; blood pressure, 110/80 mm Hg; respiratory rate, 22 breaths/min; and temperature, 37.3°C. The patient is mildly anxious but alert, oriented, and cooperative. Heart sounds are regular and tachycardic, and lungs sounds are clear. Extremities are normal. ECG demonstrates regular WCT at a rate of 160 beats/min with RBBB morphology and QRS duration of 126 milliseconds. There are no P waves evident. Initial therapy includes:**
   a. Adenosine
   b. Procainamide
   c. Amiodarone
   d. Synchronized DCCV
   e. Calcium chloride

6. **A 35-year-old man with a history of hypertension, diabetes mellitus, and chronic renal failure on hemodialysis presents with a light-headed sensation and shortness of breath. There is no chest pain and no known heart disease. Vital signs include: heart rate, 130 beats/min; blood pressure, 98/70 mm Hg; respiratory rate, 24 breaths/min; and temperature, 37.5°C. Physical examination is remarkable for regular heart sounds, mild bibasilar crackles on lung examination, no peripheral edema, and AV fistula in the left upper extremity. The patient is alert and cooperative, with a normal neurologic examination. ECG demonstrates WCT with a rate of 130 beats/min and a QRS duration of 160 milliseconds with rounded morphology. Initial therapy includes:**
   a. Adenosine
   b. Procainamide
   c. Amiodarone
   d. Synchronized DCCV
   e. Calcium chloride

7. **A 33-year-old man with a history of WPW syndrome and prior AF presents with palpitations and nausea. There is no vomiting, diarrhea, chest pain, shortness of breath, or fever. Vital signs include: heart rate, 180 beats/min; blood pressure, 130/90 mm Hg; respiratory rate, 20 breaths/min; and temperature, 37.0°C. The patient is alert and oriented. Cardiac examination reveals regular tachycardia without murmur. Lungs are clear to auscultation, and the abdomen is soft and nontender. There is no peripheral edema. Initial therapy includes:**
   a. Adenosine
   b. Procainamide
   c. Amiodarone
   d. Synchronized DCCV
   e. Calcium chloride

8. **A 55-year-old man with a history of prior MI and moderate congestive heart failure presents with a chief complaint of syncope. The patient briefly felt nauseous with palpitations, and then he fell from a standing position. There was no seizure activity or incontinence. He awoke spontaneously within a few minutes. He is brought in by ambulance, alert and oriented but mildly diaphoretic. Upon sliding over into a gurney, the patient passes out. The cardiac monitor reveals a regular WCT at a rate of 195 beats/min. You do not feel a pulse. Initial therapy includes:**
   a. Procainamide
   b. Amiodarone
   c. Synchronized DCCV
   d. Endotracheal intubation
   e. Defibrillation

Physician CME Information

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Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals: Upon completion of this activity, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

Objectives: Upon completion of this article, you should be able to: (1) identify the ECG characteristics that define wide-complex tachycardia and differentiate between ventricular tachycardia and supraventricular tachycardia; (2) apply the indicated treatment based on the rhythm determined to be present; and (3) determine the appropriate disposition and care for patients after termination of wide-complex tachycardia.

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