

Emergency Medicine Practice

Evidence-Based Education • Practical Application

CLINICAL CHALLENGES:

- Which electrocardiogram findings are most specific for ventricular tachycardia in patients with wide complex tachycardia?
- What factors should guide the initial management of patients presenting with stable versus unstable wide complex tachycardia?
- Which patients presenting with wide complex tachycardia are at highest risk for hemodynamic deterioration?

Authors

Robert Allen, MD

Clinical Assistant Professor of Emergency Medicine, Los Angeles General Medical Center, Keck School of Medicine of USC, Los Angeles, CA

Caleb J. Bailie, MD

Department of Emergency Medicine, SUNY Downstate Health Sciences University, Kings County Hospital Center, Brooklyn, NY

Ian S. deSouza, MD

Professor of Clinical Emergency Medicine, SUNY Downstate Health Sciences University, Kings County Hospital Center, Brooklyn, NY

Prior to beginning this activity, see "CME Information" on page 2.



Wide Complex Tachycardia in the Emergency Department: An Updated Approach to Diagnosis and Management

■ Abstract

Wide complex tachycardia encompasses a range of cardiac tachydysrhythmias, several of which can be life-threatening, difficult to distinguish, and require distinct treatment approaches. This review summarizes the etiologies and pathophysiology of wide complex tachycardia, outlines the differential diagnosis, and provides an evidence-based approach to distinguishing the various types using the history, physical examination, electrocardiography, and response to intravenous adenosine. Management strategies and disposition decisions are also reviewed, with attention to special clinical circumstances and selected areas of ongoing controversy, including emerging artificial intelligence tools.



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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) identify areas in practice that require modification to be consistent with current evidence in order to improve competence and performance; (2) develop strategies to accurately diagnose and treat both common and critical ED presentations; and (3) demonstrate informed medical decision-making based on the strongest clinical evidence.

CME Objectives: Upon completion of this activity, you should be able to: (1) differentiate ventricular tachycardia from supraventricular causes of wide complex tachycardia (WCT) using key historical features, physical examination findings, and electrocardiographic criteria; (2) apply evidence-based algorithms and guideline recommendations to determine appropriate initial management of stable versus unstable WCT in the emergency department; and (3) formulate a management strategy for special clinical scenarios involving WCT.

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Planners

- Daniel J. Egan, MD, MBA (Course Director and Editor-in-Chief)
- Kaushal Shah, MD (Associate Editor-in-Chief)

Faculty

- Robert Allen, MD (Author)
- Caleb J. Bailie, MD (Author)
- Ian S. deSouza, MD (Author)
- Jennifer Kahoud, MD, MBA (Peer Reviewer)
- James Morris, MD, MPH (Peer Reviewer)
- Aimee Mishler, PharmD (Pharmacology Editor)
- Joseph D. Toscano, MD (Research Editor)
- Angie Wallace (Content Editor)
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Direct all inquiries to:

Phone: 678-366-7933

Fax: 770-500-1316

5600 Spalding Drive, Unit 921697

Norcross, GA 30010-1697

E-mail: ebm@ebmedicine.net

Website: www.ebmedicine.net

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Case Presentations

CASE 1

A 72-year-old man presents to the emergency department with shortness of breath...

- He is attached to the monitor and you see a wide complex tachycardia at a rate of 158 beats/min.
- His blood pressure is 128/73 mm Hg, and his pulse oximetry is 96% on ambient air.
- The patient sees you looking at the defibrillator and says, "I don't want to be shocked. Are there any meds you can give me?"
- You consider which medications you should use for pharmacologic cardioversion of his ventricular tachycardia...

CASE 2

A 28-year-old woman presents to the emergency department with vomiting...

- She is treated with multiple rounds of antiemetics. As she is being prepared for transfer to the observation unit due to persistent oral intolerance, a nurse suddenly calls out for assistance.
- You arrive and check the monitor, where you see that the patient has a wide complex tachycardia with variable QRS morphology. You diagnose her with polymorphic ventricular tachycardia.
- She has a pulse. Her blood pressure is 60/30 mm Hg.
- As the nurses reach for pads to apply to the patient, you consider whether you need to synchronize the shock or defibrillate...

■ Introduction

Wide complex tachycardia (WCT) describes a rhythm in which the heart rate is faster than 100 beats/minute and the QRS complex duration is >120 milliseconds. Emergency clinicians must have a comprehensive understanding of how to identify and manage the different types of WCTs. Without prompt therapy, many of these rhythms may progress to other life-threatening tachydysrhythmias, even if they were initially benign. Misdiagnosis of WCT can lead to delayed or inappropriate therapeutic decisions that can result in further hemodynamic compromise. Unfortunately, there are no electrocardiographic criteria that can definitively diagnose a specific WCT, and agreement regarding rhythm identification is inconsistent.¹ This issue of *Emergency Medicine Practice* reviews the diagnostic and therapeutic approaches for evaluating and managing the various WCTs. It updates the 2015 review,² with a focused emphasis on the management of patients with WCT who present with a pulse.

■ Critical Appraisal of the Literature

A search of MEDLINE through PubMed was performed with the terms *wide complex tachycardia*, *broad complex tachycardia*, *wide complex dysrhythmia*, *wide QRS complex tachycardia*, *broad QRS complex tachycardia*, *ventricular tachycardia*, *supraventricular tachycardia*, *ventricular arrhythmia*, *ventricular dysrhythmia*, *ventricular tachydysrhythmia*, *aberrant conduction*, and *aberrancy*. The search was limited to references published from 2014 to 2025. These search terms were also used in conjunc-

tion with the terms *emergency department*, *ED*, *prehospital*, *clinical signs*, *clinical history*, *vagal maneuvers*, *hyperkalemia*, *tricyclic antidepressant*, *artificial intelligence*, *AI*, *machine learning*, *deep learning*, *neural networks*, and *interpretable machine learning*. The search yielded a total of 948 studies that were reviewed for relevance. An updated literature search of the Cochrane Database using the same search terms yielded a total of 36 reviews, none of which were relevant to the current topic. Guidelines and 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.

■ Etiology and Pathophysiology

WCT can result from an extrinsic or an intrinsic etiology. The primary extrinsic causes of WCT involve sodium channel blockade. Agents that can inhibit cardiac sodium channels will slow phase 0 depolarization of the cardiac action potential and reduce His-Purkinje and myocardial conduction velocity, which will significantly widen the QRS complex on an electrocardiogram (ECG).³ Hyperkalemia also inhibits cardiac sodium channels, but it does so by altering the resting membrane potential.⁴ Acute medical illnesses (eg, sepsis and congestive heart failure) can give rise to WCT.⁵ These illnesses may present as supraventricular tachycardias (SVTs) such as atrial fibrillation and atrial flutter, and will manifest as a WCT in the presence of aberrant conduction. Aberrant conduction (or aberrancy) occurs when there is delayed conduction within

the His-Purkinje system due to absolute or relative refractoriness of one of the bundle branches.⁶

Intrinsic causes of WCT include ventricular tachycardia (VT), which is the most common etiology of regular WCT and can be monomorphic or polymorphic.⁷ Monomorphic VT is usually 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.⁸ Polymorphic VT is a triggered tachydysrhythmia that originates from multiple foci or a primary re-entrant circuit that then migrates within the ventricular myocardium. It is encountered most commonly as torsades de pointes or as a consequence of acute MI.⁹

■ Differential Diagnosis

VT should be the first rhythm considered when a patient presents with WCT, since VT can deteriorate from a stable to unstable tachydysrhythmia, but the differential diagnosis of WCT is quite broad. 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.

Toxic Myocardium

Cardiac Sodium-Channel Inhibitors

Among the many medications that are associated with WCT, tricyclic antidepressants (TCAs) are prototypical.¹⁰ TCAs antagonize sodium channels to prolong cardiac depolarization and widen the QRS complex. This phenomenon, when combined with tachycardia due to the anticholinergic effect of TCAs, will result in WCT. Other agents that cause sodium channel blockade include cocaine, diphenhydramine, and class Ia and Ic antidysrhythmic medications such as procainamide and flecainide.¹¹

Hyperkalemia

Hyperkalemia causes QRS widening in a manner similar to sodium channel-blocking agents. 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 complex. (See **Figure 1, page 5**.) As a general rule, potassium levels >8 mEq/L will present with QRS widening.¹² This abnormality in ventricular depolarization and the resultant QRS widening may be progressive and can eventually result in VT and cardiac arrest.¹³

Table 1. Types of Wide Complex Tachycardia, With Historical Factors, Physical Examination Findings, and Electrocardiographic Characteristics

WCT Type	Historical Factors	Physical Examination Findings	Regularity	ECG Characteristics
Sodium-channel blockade	Toxic ingestions (eg, tricyclic antidepressants, illicit substance use [eg, cocaine], diphenhydramine)	Altered mental status, mydriasis, hyperpyrexia	Regular	Right-axis deviation, terminal R in aVR
Hyperkalemia	Chronic renal insufficiency, acute kidney injury	Arteriovenous fistula/graft, dialysis catheter	Regular	Peaked T waves, prolonged PR interval
SVT with aberrancy	None	None	Regular or irregular	Baseline or rate-related bundle branch block
Non-atrial fibrillation SVT with accessory pathway	Pre-excitation syndrome (eg, WPW syndrome)	None	Regular	QRS wide (with anterograde AV conduction through accessory pathway)
Atrial fibrillation with accessory pathway	Pre-excitation syndrome (eg, WPW syndrome)	None	Irregular	Varying QRS width, very rapid ventricular rate
Monomorphic VT	Previous MI, ischemic cardiomyopathy	Cannon A waves, variable S1	Regular	AV dissociation
Polymorphic VT	Occlusion MI, long QT syndrome, QT-prolonging medication use	None	Irregular	Varying QRS morphology/axis, baseline long QTc
Bidirectional VT	Digoxin toxicity, symptoms with exertion (CPVT)	None	Regular	Beat to beat alteration in QRS axis
Paced tachycardia	Pacemaker placement	Pacemaker	Regular	Pacing artifact, relatively slower ventricular rate

Abbreviations: AV, atrioventricular; CPVT, catecholaminergic polymorphic ventricular tachycardia; ECG, electrocardiogram; MI, myocardial infarction; SVT, supraventricular tachycardia; VT, ventricular tachycardia; WCT, wide complex tachycardia; WPW, Wolff-Parkinson-White.

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Supraventricular Tachycardia

Supraventricular Tachycardia With Aberrancy

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

In patients with a baseline bundle branch block, any SVT (including sinus tachycardia) will manifest as a WCT; however, the morphology of a patient's baseline bundle branch block may change with more rapid rates and be mistaken for VT. Misdiagnosis of VT may subject the patient to the risks of antidysrhythmic agents (eg, malignant tachydysrhythmia, hypotension) without benefit. In a 2008 prospective study, SVT was induced in 59 patients with baseline bundle branch block; 76% of the patients demonstrated morphological changes in the QRS complex and bundle branch block pattern, including the appearance, amplitude, and duration of the QRS complex.¹⁴ None of the patients developed a contralateral bundle branch block, which suggests that a WCT with a new contralateral bundle branch block may be specific for VT. However, the opposite scenario must also be considered, as VT may occur in patients with baseline bundle branch block with QRS morphology that appears identical to the underlying bundle branch block, potentially leading to the misdiagnosis of SVT with aberrancy.^{15,16} Although helpful, comparison of QRS and bundle branch block morphology in current and baseline ECGs cannot definitively differentiate VT from SVT with aberrancy.

Figure 1. Wide Complex Tachycardia Due to Hyperkalemia

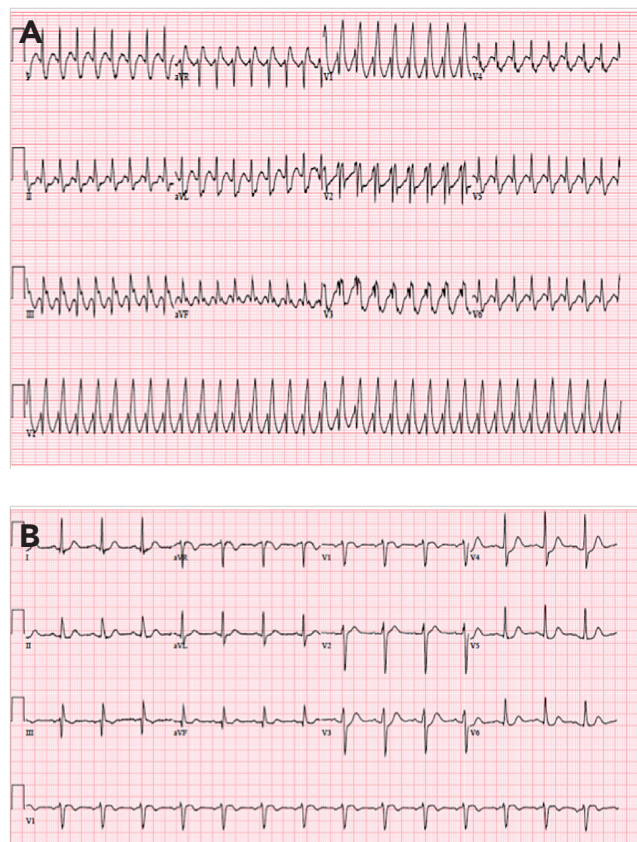


Electrocardiogram showing widened QRS with peaked T waves (rapid/steep slope of the T wave, particularly the downslope) is most correlated with elevated serum potassium.

Reprinted from *American Journal of Emergency Medicine*, Volume 18, Number 6, Amal Mattu, William J. Brady, David A. Robinson, *Electrocardiographic Manifestations of Hyperkalemia*, pages 721- 729, Copyright © 2000, with permission from Elsevier.

Even in the absence of baseline bundle branch block, patients with an SVT may develop rate-related aberrant conduction.¹⁷ This phenomenon may be seen in any SVT that involves anterograde conduction through the AV node and His-Purkinje system (eg, atrial flutter, ectopic atrial tachycardia, AV nodal re-entrant tachycardia, and orthodromic AV re-entrant tachycardia). (See Figure 2.) Typically, these rhythms present as a narrow QRS complex tachycardia. However, in the setting of more rapid atrial rates, refractory (or fatigued) conducting fibers of the His-Purkinje system will delay impulse propagation and ventricular depolarization, leading to QRS widening and, therefore, WCT observed on ECG.^{18,19} Right bundle branch block pattern is more common in rate-related bundle branch block, as the right bundle branch's refractory period is longer than that of the left bundle

Figure 2. Wide Complex Tachycardia With Right Bundle Branch Block Pattern, Before and After Adenosine Administration



A: Electrocardiogram showing regular, wide complex tachycardia with right bundle branch block pattern (rSR').

B: The same patient's electrocardiogram following adenosine administration, with subsequent conversion to sinus rhythm and resolution of the bundle branch block pattern; this suggests that the original tachydysrhythmia shown in Figure 2A is an atrioventricular nodal re-entrant tachycardia with rate-related aberrancy.

Images courtesy of Ian deSouza, MD.

branch.²⁰ Rate-related aberrancy may occur even in patients with baseline bundle branch block and may be suggested by changes in bundle branch block morphology.¹⁴ However, even in patients without bundle branch block on baseline ECG, it can be difficult to definitively diagnose rate-related aberrancy as the cause of the wide QRS complex.

Supraventricular Tachycardia With an Accessory Pathway

SVTs that are conducted anterograde through an accessory pathway, also known as *pre-excitation syndromes*, will present as WCT. An example is antidromic AV re-entrant tachycardia (AVRT) with Wolff-Parkinson-White (WPW) syndrome. This is a rare WCT occurring in only 8% of patients with pre-excitation referred for electrophysiology study.²¹ In AVRT, an atrial impulse is conducted from the atria to the ventricles via an anatomical accessory pathway, and depolarization of the ventricles subsequently proceeds inefficiently, propagating without the use of the rapidly conductive His-Purkinje system. On the ECG, this appears as a wide QRS complex. The wave of depolarization then returns to the atria in a retrograde manner through the AV node to complete the re-entry circuit. (See Figure 3.) Antidromic AVRT can, therefore, mimic monomorphic VT, and differentiation of these conditions may be difficult.²² In contrast, orthodromic AVRT involves anterograde conduction through the AV node and His-Purkinje system and retrograde conduction through an accessory pathway, demonstrating a narrow QRS complex on an ECG unless there is simultaneous aberrant conduction. SVTs can proceed exclusively from the atria to the ventri-

cles via an accessory pathway, which typically involves a shorter refractory period than that of the AV node. These situations usually involve rapidly generated atrial impulses, as in atrial fibrillation or atrial flutter. The patient with both atrial fibrillation and a fast-conducting accessory pathway is particularly worrisome, as this combination may result in very rapid ventricular rates²³ and associated hemodynamic instability.

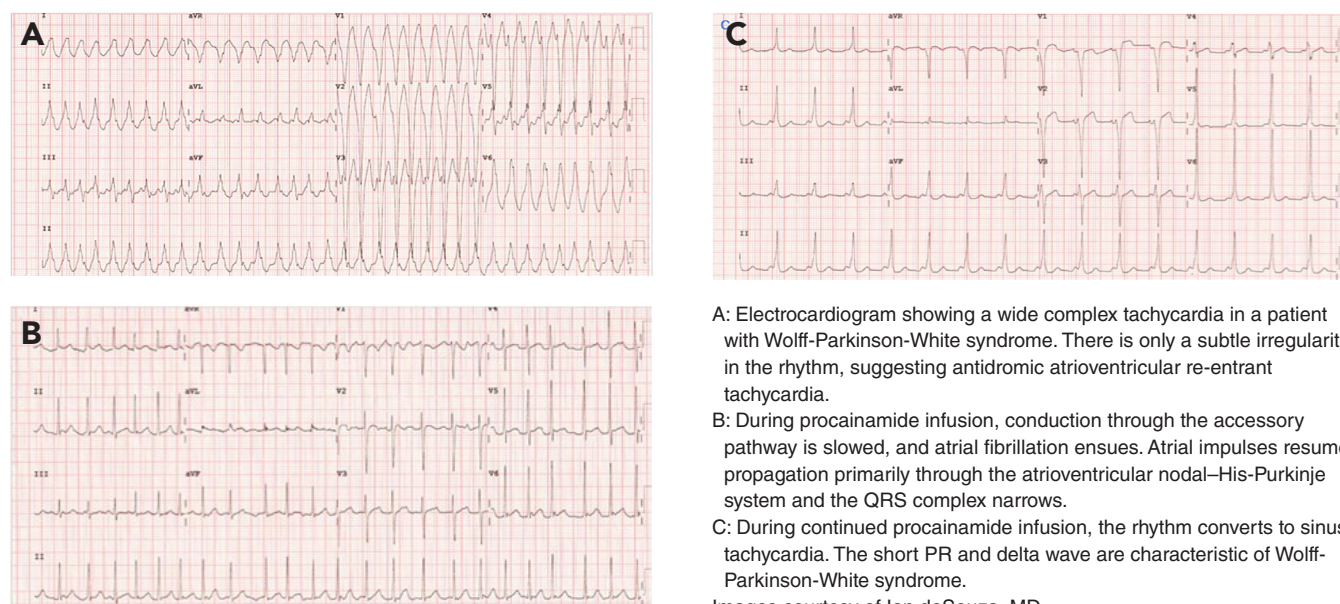
Ventricular Tachycardia

Despite the numerous forms of SVT that may masquerade as VT, it is reported that approximately 80% of all WCTs will actually be VT.²⁴ However, this estimate comes from the cardiology setting, in which clinicians will be more likely to see VT than in the emergency department (ED).²⁴ The true estimate of VT prevalence in the ED is likely to be lower. Presumptive treatment of a regular WCT as VT is advised, as this can minimize the consequences of misdiagnosing SVT with aberrancy, an error that can delay appropriate therapy or lead to catastrophic, inappropriate therapy.²⁵⁻²⁷ 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 or fibrosis from previous MI or cardiomyopathy,²⁸ and this abnormal myocardium provides the required slow-conducting limb of a re-entrant circuit. These circuits can be secondary to structural heart disease or idiopathic in nature.²⁹ Unlike classic VT, which

Figure 3. Antidromic Atrioventricular Re-entrant Tachycardia With Wolff-Parkinson-White Syndrome



typically occurs in older patients with structural heart disease, idiopathic VT encompasses different subtypes of VT, including outflow tract VT, fascicular VT, and bundle branch re-entry VT.

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 tachydysrhythmia that occurs in the setting of prolonged repolarization. This can be due to a number of factors, including medications or illicit substance use, metabolic disturbances (eg, hypokalemia, hypomagnesemia, or hypocalcemia), or cardiac channelopathies. Prolonged repolarization generates a prolonged QT interval on an ECG; however, when the baseline QT interval is normal in duration, polymorphic VT is most commonly associated with acute MI.³⁰⁻³³ With prompt reperfusion and improved medical therapy to reduce infarct size, the incidence of VT in the setting of acute MI has decreased to 1%, from a previous range of 3% to 5%.⁹ In the setting of acute ischemia, hypoxia and the products of anaerobic glycolysis result in intracellular acidosis. Alterations in the activity of electrolyte exchange channels result in increased intracellular calcium and increased extracellular potassium.³⁴ VT in the setting of acute MI is thought to arise from abnormal automaticity due to calcium- and potassium-related changes in transmembrane potential and, consequently, cardiac myocyte excitability.^{33,34}

Bidirectional Ventricular Tachycardia

Bidirectional VT is a rare cause of WCT in which there is beat-to-beat alteration in the direction of the QRS complex.²⁰ (See Figure 4.) Causes include digoxin toxicity and catecholaminergic polymorphic VT. A history of digoxin use in the setting of bidirectional VT would strongly suggest digoxin toxicity. Catecholaminergic polymorphic VT is more often found in younger patients and may be associated with physical exertion or strong emotional triggers.

Paced Tachycardia

Sinus Tachycardia With Paced Ventricular Response

Implantable pacemakers depolarize the ventricular myocardium without the use of the His-Purkinje system, and, therefore, can cause iatrogenic QRS widening; for example, single-lead pacing in the right ventricle depolarizes the right ventricle first and the left ventricle afterwards, generating a left bundle branch pattern on an ECG.²⁰ Patients with pacemaker-conducted sinus tachycardia will appear

to have WCT, though the rate should not surpass the device's programmed maximum rate.

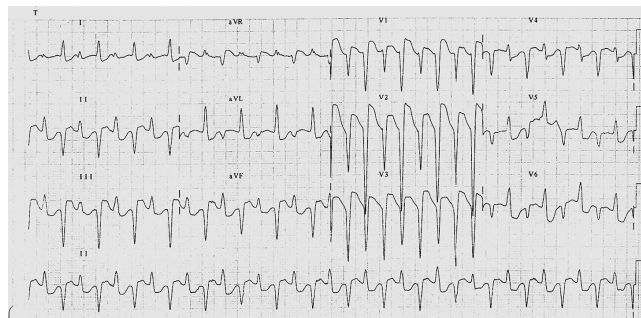
Pacemaker-Mediated Tachycardia

Pacemaker-mediated tachycardia occurs when a dual-chamber pacemaker provides a re-entry circuit through which the impulse travels. A native, premature complex, typically originating from the ventricle, is conducted retrograde through the AV node and sensed by the atrial lead of the pacemaker. The pacemaker responds by generating a ventricular impulse that is again conducted retrograde through the AV node and sensed by the atrial lead, prompting the device to generate another ventricular impulse and perpetuate the cycle. This results in a re-entrant circuit in which the pacemaker serves as one of the pathways. Differentiating between sinus tachycardia with paced ventricular response and pacemaker-mediated tachycardia may require device interrogation.³⁵

■ Prehospital Care

As with other cases involving acute cardiac illnesses, many of the patients presenting with WCT will arrive at the ED via emergency medical services (EMS). The prehospital care of the patient with WCT focuses on oxygenation, ventilation, cardiac monitoring, and rapid transport. Personnel with advanced life-saving training may be able to provide electrical therapy for unstable WCT. Other therapies, such as medications, will vary based on regional protocols; for example, the 2024 New York City EMS protocol allows paramedics to administer either intravenous (IV) amiodarone (150 mg over 10 min) or IV lidocaine (1 mg/kg over 2 min, max 100 mg) for patients with stable WCT.³⁶

Figure 4. Bidirectional Ventricular Tachycardia



Electrocardiogram demonstrating bidirectional ventricular tachycardia. Used with permission of Life in the Fast Lane (<https://litfl.com>). Available at <https://litfl.com/bidirectional-ventricular-tachycardia-bvt-ecg-library/>

■ Emergency Department Evaluation

History

The history of present illness provides important clues about the duration and stability of the WCT. It can also point toward a specific cause, such as a toxic ingestion that involves sodium channel blockade. Most commonly, however, the history will not distinguish the underlying rhythm mechanism. Patients with VT can present with both hemodynamic stability and minimal symptomatology.³⁷ As few as 30% of patients with sustained VT are symptomatic upon ED arrival.³⁸⁻⁴⁰

A past medical history of renal insufficiency should suggest the possibility of WCT secondary to hyperkalemia, particularly in the setting of nonadherence to scheduled hemodialysis. The current medication list may reveal prescribed or nonprescribed medications that may have been ingested in overdose, such as sodium channel-blocking agents (including TCAs), antidysrhythmics, and diphenhydramine. Potassium-sparing diuretics and potassium supplements can cause hyperkalemia.^{41,42} Loop and thiazide diuretics such as furosemide and hydrochlorothiazide can cause hypokalemia and prolong the QT interval, which will increase the risk for VT.

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.⁴⁰ The Leiden out-of-hospital cardiac arrest evaluation study included 300 consecutive patients who presented in cardiac arrest due to VT or ventricular fibrillation (VF). Eighty-eight patients (29%) had a recurrence of VT/VF within the 5-year follow-up period.⁴³ In addition to prior episodes of VT, a history of MI,^{39,40,44,45} structural heart disease,⁴⁶ and reduced ejection fraction⁴⁰ were also more predictive of VT than SVT.

Physical Examination

Assessment of end-organ perfusion is the single most important component of the physical examination. Subtle signs of end-organ hypoperfusion include transient alterations in mental status, respiratory insufficiency and rales, and pallorous or cool extremities with delayed capillary refill. Even with a normal blood pressure, the presence of these signs suggests hemodynamic instability and should prompt consideration of immediate electrical therapy.

For patients with WCT secondary to toxic ingestions or metabolic disturbances, the physical examination may aid in diagnosis of the underlying etiology. Arteriovenous fistulas, grafts, and other points of access for hemodialysis will be suggestive of hyperkalemia. Patients with toxic ingestions will exhibit signs and symptoms of the toxidrome associated with the ingested agent; for example, a TCA overdose would present with WCT in the setting of an anticholinergic toxidrome. WCT secondary to complex medical illnesses (eg, sepsis or congestive

heart failure) may present with symptoms and signs specific to those conditions.

The physical examination can aid in distinguishing between intrinsic WCTs by identifying signs of AV dissociation, which is the hallmark criterion for VT. A prospective randomized controlled trial of 26 patients examined the ability of clinicians to identify AV dissociation in order to distinguish VT from SVT.⁴⁷ 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.^{48,49}

Identification of an implantable cardiac device such as a pacemaker, cardioverter-defibrillator (ICD), or loop recorder can enable expedited interrogation for rhythm diagnosis and ICD-directed therapy.

■ Diagnostic Studies

The Diagnostic Utility of Electrocardiography

A 12-lead ECG can help the emergency clinician differentiate between the types and mechanisms of WCT and, thus, 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.

Heart Rate

Heart rate does not accurately distinguish VT from SVT.⁵⁰ While VT typically has a rate >130 beats/min, slower rates (100-120 beats/min) can occur in the setting of end-stage dilated cardiomyopathy or heart failure, and in patients on long-term amiodarone treatment.²⁸ There are some exceptions and important caveats, however. A WCT with the heart rate fixed near 150 beats/min would suggest the possibility of atrial flutter with 2:1 AV conduction, and the conduction may be aberrant. WCT at the rate of 300 beats/min would suggest atrial flutter, with 1:1 anterograde conduction through an accessory pathway, which, in contrast to the AV node, may have conduction properties that permit such a rapid ventricular rate.

Axis

Both left-axis deviation and right-axis deviation can be associated with VT.⁴⁶ However, patients with a baseline conduction delay, including bundle branch block, will frequently demonstrate axis deviation as well, so axis alone is minimally useful in differentiating VT from SVT.^{51,52} Although axis deviation is not sensitive, extreme right-axis ("northwest axis") deviation (between -180° and -90°) can be predictive of VT. (See Figure 5, page 9.) Extreme right-axis deviation has poor sensitivity, with a reported range of 13% to 23%, but it has good specificity at 87% to 96%.^{40,45,46,51,53} A methodological analysis of diagnostic accuracy found the presence of extreme right-

axis deviation to increase the odds of VT by a factor of 2.6.⁵⁴ Left-axis deviation in the setting of WCT with right bundle branch block pattern and right-axis deviation in the setting of WCT with left bundle branch block pattern can suggest VT.⁵⁰

QRS Duration and Morphology

The QRS duration can help differentiate the etiology of a WCT, but it is not 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%.^{45,46,51-53} However, patients with bundle branch block or antidromic AVRT can also present with a QRS duration >140 milliseconds.^{46,51,52} A QRS duration >160 milliseconds has a better predictive value for VT, particularly with left bundle branch block morphology, with sensitivity ranging from 64% to 65% and specificity from 79% to 97%.^{6,46,52}

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.⁵⁵ They found that an RS interval >100 milliseconds was 66% sensitive and 98% specific for VT. The reasoning behind this approach is that the initial depolarization of the ventricle may occur more slowly with VT than with SVT with aberrancy. A retrospective study found that R wave peak time ≥ 50 milliseconds at lead II is a highly sensitive criterion that discriminates VT from SVT in patients with WCT (area under the curve, 0.97 [95% confidence interval, 0.95 to 0.99]; positive likelihood ratio, 34.8; and kappa coefficient, 0.86).⁵⁶

A right bundle branch block is typical in rate-related aberrant conduction, as the right bundle branch characteristically has a longer relative refractory period than its left bundle branch counterpart. More specifically, a 1978 retrospective study by Wellens et al determined that a triphasic rSR' pattern in lead V1 is 10 times more likely to indicate SVT with aberrancy than VT.⁵⁰ A 2021 prospective study of 111 patients found that an RS/QRS interval ratio >0.41 can help differentiate fascicular VT (an idiopathic VT that presents with a right bundle branch block morphology) from SVT with right bundle branch block.⁵⁷

Concordance

QRS concordance across the precordial leads (QRS complexes that are entirely positive or entirely

negative in leads V1-V6) is also predictive of VT, with specificity reported between 90% and 100%.^{6,46,51,53} However, precordial concordance can be quite rare, with a reported sensitivity of 0% to 15%.^{6,46,51} (See **Figure 6, page 10.**)

Atrioventricular Dissociation, Capture Complex, and Fusion Complex

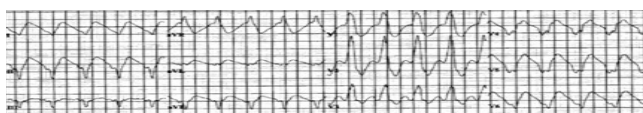
When P waves are visible on 12-lead ECG but are completely independent of regular grouping with QRS complexes, this is known as AV dissociation. (See **Figure 7, page 11.**) While the presence of AV dissociation is highly specific for VT,⁵⁰ it can also rarely be seen in AV nodal re-entrant tachycardia (AVNRT).²⁸ However, visible AV dissociation is a rare finding in VT, particularly when the ventricular rate is rapid, with reported sensitivity between 5% and 24%.^{45,46,53} AV dissociation at the onset of a WCT may be easier to identify if captured on ECG or telemetry.⁵⁸

Capture and fusion complexes (or “beats”) can signify AV dissociation and are highly specific for VT, but they are also very rare, occurring in approximately 0.5% of VT cases.^{45,53} Although capture and fusion complexes on 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 retrospective study.⁵⁹ A capture complex occurs when an atrial impulse is conducted by the AV nodal-His-Purkinje system at a fortuitous moment, rapidly depolarizing (“capturing”) the ventricles through the His-Purkinje system before the occurrence of slow, adjacent depolarization from the ventricular focus. This results in a narrow QRS complex with markedly different morphology than the predominantly wide QRS of the WCT. Similarly, a fusion complex occurs when an atrial impulse propagates through the AV node and reaches the His-Purkinje system but then simultaneously depolarizes the myocardium, along with the impulse from the ventricular focus. This concurrent depolarization of the ventricles from the combination of both impulses gives rise to an intermediate (“fused”) QRS morphology. (See **Figure 8, page 11.**)

Comparison to Prior Electrocardiography

In cases in which the WCT involves a QRS morphology that is identical to the baseline bundle branch block, the rhythm can generally be presumed to be SVT with aberrancy.^{60,61} However, exceptions have been reported in which VT demonstrated a QRS morphology similar to an underlying bundle branch block.¹⁵ QRS complexes during the episode of WCT that are narrower than the QRS complex during sinus rhythm are suggestive of VT.⁶² Evidence of a previous MI on a prior ECG suggests the presence of myocardial scar and may also support the diagnosis of VT.⁴⁶ Prior ECGs may also show evidence of congenital causes that can predispose a patient to VTs (eg, Brugada syndrome, hypertrophic

Figure 5. Extreme Right-Axis Deviation



Electrocardiogram showing extreme right-axis deviation (between -180° and -90°).

Image courtesy of Keith Marill, MD.

cardiomyopathy, arrhythmogenic right ventricular dysplasia, or long QT syndrome).⁶³

Diagnostic Algorithms

Multiple algorithms have been developed to assist in differentiating VT from SVT in patients with WCT.^{1,55,56,64-68} A simple, practical, and appealing algorithm was developed by Griffith et al⁶⁴ and is supported by some guidelines.⁶³ Griffith et al suggest that VT should be the default diagnosis in patients with WCT because of the associated risk of clinical deterioration.⁶⁴ They also suggest that clinicians should assess QRS morphology for evidence of typical left bundle branch block or right bundle branch block characteristics; if the QRS morphology is not consistent with either typical bundle branch block pattern, then VT should be assumed. A sensitivity of 90% and specificity of 75% were calculated with this approach.⁶⁴ Unfortunately, among the large number of currently available algorithms, none have been developed or validated in an ED setting, limiting their applicability.^{69,70} As such, citing the complexity and limited accuracy, several authors have suggested that algorithms should not be recommended for clinical use at this time.^{20,24,28}

Adenosine Response

Diagnostic Utility

The administration of IV adenosine can aid in both diagnosis and treatment of regular WCT by transiently inducing AV nodal block, an effect which should terminate re-entrant tachydysrhythmias that involve the AV node as part of the circuit. These rhythms include AVNRT,⁵⁰ in which there is a functional accessory pathway within the AV node, and AVRT, both orthodromic and antidromic,⁵¹ which involves an anatomic accessory pathway. Adenosine can also uncover other underlying supraventricular rhythms (eg, atrial fibrillation, atrial flutter, atrial tachycardia) through transient AV nodal block.⁷¹

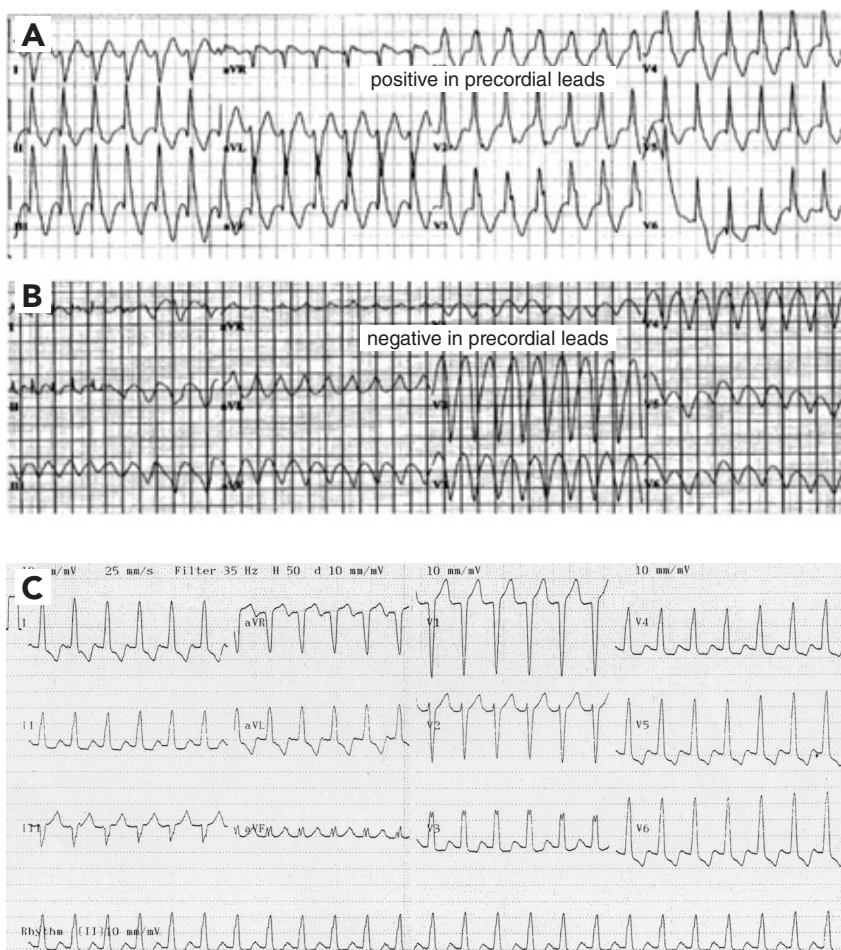
A lack of response to adenosine can also be diagnostic. In a retrospective study of 197 patients with WCT, nonresponse to adenosine increased the odds of VT by a factor of 9.4, while any response to adenosine increased the odds of SVT by a factor of 36.3.⁴⁰ In rare cases, a response to adenosine will suggest VT, as it may transiently induce retrograde ventriculoatrial block to support the diagnosis.⁷²

Although studies have reported the safety of adenosine administration in patients with WCT,^{40,73,74} there are situations in which adenosine is contraindicated. The quintessential WCT rhythm for which adenosine is contraindicated is atrial fibrillation in the setting of WPW syndrome. Approximately 39% of WPW cases may be associated with atrial fibrillation.²³ Although the actual risk may be somewhat overstated,⁷⁵ adenosine (through induced AV nodal blockade) may accelerate conduction of atrial fibrillatory impulses through the accessory pathway. This may lead to very rapid ventricular rates and degeneration to VF.^{22,40,73,74} There are also reports in which adenosine administration in other WCTs may have precipitated VT and VF.^{76,77}

Laboratory Testing

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

Figure 6. Precordial Concordance and Discordance



A: Electrocardiogram demonstrating positive concordance. Image courtesy of Ian deSouza, MD.
B: Electrocardiogram demonstrating negative concordance. Image courtesy of Ian deSouza, MD.
C: Electrocardiogram demonstrating left bundle branch block with discordance. Used with permission of Life in the Fast Lane (<https://litfl.com>). Available at <https://litfl.com/vt-versus-svt-ecg-library/>

Electrolytes

Measurement of potassium and magnesium levels may help identify electrolyte abnormalities that can precipitate VT. Correction of both hypokalemia and hypomagnesemia may prevent VT in susceptible patients or aid in the suppression of recurrent VT.⁷⁸⁻⁸⁰

Cardiac Biomarkers

Measurement of cardiac biomarkers (specifically troponin levels) may be reasonable for patients with WCT, as VT may be related to acute MI. However, when WCT is associated with minor elevations of troponin, it should not be assumed that the underlying etiology is occlusion MI.⁶³ Troponin elevations are of limited diagnostic value and are likely due to nonocclusion MI (ie, ischemia from a mismatch in supply and demand) or other causes of myocardial injury. Patients with WCT with elevated troponin should be treated for VT in the same manner as patients without elevated troponin levels. For patients with WCT confirmed as SVT with aberrancy following termination with adenosine, troponin testing is not routinely required unless symptoms are suggestive of acute coronary syndrome or the rhythm diagnosis remains uncertain.⁸¹

Treatment

Unstable Wide Complex Tachycardia

Pulselessness should be self-evident, but determining hemodynamic stability in a patient with a WCT may be more subjective and involves an assessment of end-organ (eg, heart, brain, extremities) perfusion. Clinicians should not rely solely on the presence or absence of hypotension and must consider other signs and symptoms of hypoperfusion, such as chest discomfort, altered mental status, pallor, and cool extremities.

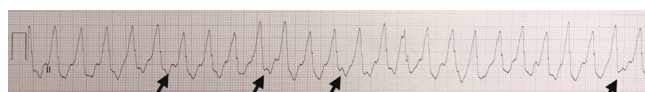
Unstable WCT, whether ventricular or supraventricular in origin, regular or irregular, should be treated initially with electrical therapy.^{9,82,83} If the patient is conscious and the situation allows, procedural analgesedation should be administered beforehand. Defibrillator pads may be placed on the chest in an anterior-lateral or anterior-posterior configuration.³⁰ If a patient has an implantable medical device, pad placement should ideally be at least 8 cm away from the device.⁸² The defibrillator should be set to synchronize shock delivery with the R wave of the cardiac cycle for direct current

cardioversion (DCCV), except in cases of polymorphic VT. This is recommended in order to avoid shock delivery during the repolarization phase of the myocardial action potential (upslope of the T wave), which may precipitate VF.⁹ If synchronization fails, defibrillation is acceptable, as delay in cardioversion can result in hemodynamic compromise.⁸² The 2025 AHA guidelines for cardiopulmonary resuscitation and emergency cardiovascular care suggest synchronized DCCV at an initial dose of 100 J (biphasic), with escalation, if necessary, when there is lack of response.⁹ The 2025 ERC adult advanced life support guidelines recommend a higher initial DCCV dose range of 120 J to 150 J.⁸²

The 2025 AHA guidelines specify that all forms of polymorphic VT should be considered unstable. Synchronization may not be possible due to varying QRS morphologies;⁹ therefore, the recommended initial therapy for polymorphic VT is defibrillation at the manufacturer's maximum setting.^{9,82}

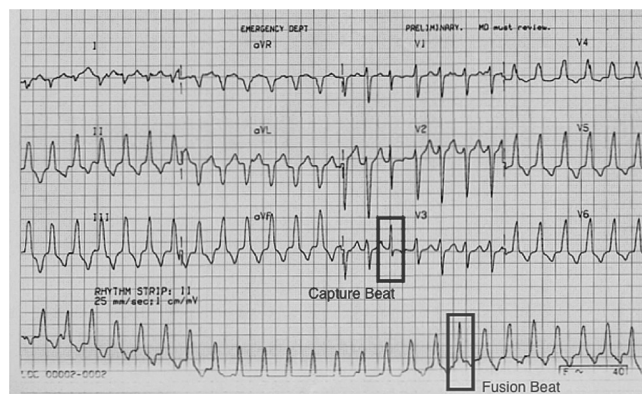
When polymorphic VT occurs in the setting of a prolonged baseline QTc, it is considered torsades de pointes. The clinician should administer IV magnesium (2 g over 15 min, with a repeat dose if needed) to suppress recurrence of torsades de pointes. Magnesium acts by suppressing the afterdepolarizations that can precipitate torsades de pointes. Hypokalemia should be promptly treated with IV potassium chloride, if indicated.⁹ Antidysrhythmic medications will be ineffective, and due to QT-prolonging effect, some agents can theoretically promote torsades de pointes. All potentially offending QT-prolonging medications should be discontinued.⁸⁴ In cases of torsades de pointes preceded by bradycardia or pauses, electrical pacing (transcutaneous or transvenous) or an IV isoproterenol infusion (2-10 mcg/min) may be used to maintain a normal ventricular rate and shorten the QT interval, thereby reducing the risk for recur-

Figure 7. Atrioventricular Dissociation



Electrocardiogram demonstrating P waves (arrows) that do not appear to be associated with the QRS complexes, suggesting that the atria and ventricles are depolarizing independently of each other. Image courtesy of Ian deSouza, MD.

Figure 8. Capture and Fusion Complexes



Electrocardiogram demonstrating capture and fusion complexes. Used with permission of Elsevier Science & Technology Journals, from Richard D. Jenkins, Stephen J. Gerred, *ECGs by Example*, 1997; permission conveyed through Copyright Clearance Center, Inc.

rent bradycardia- or pause-dependent torsades de pointes.^{63,82,84} Polymorphic VT associated with normal baseline QTc is often secondary to MI and should be treated with defibrillation, followed by beta-adrenergic receptor antagonists, amiodarone, or lidocaine, while preparing for emergent reperfusion therapy.^{9,33,63} In the absence of prolonged baseline QTc, magnesium is ineffective in suppressing VT and is not recommended.⁹

Stable Wide Complex Tachycardia

Management of patients with stable WCT begins with assessing for and treating any medical condition (eg, sepsis, acute blood loss) that may have prompted the tachydysrhythmia.²⁸ If a regular WCT is suspected to be paroxysmal SVT, vagal maneuvers can be considered before initiating pharmacological therapy. If vagal maneuvers are unsuccessful, IV adenosine at escalating doses (6 mg followed by 12 mg) may terminate many SVTs with aberrancy.⁹ The 2025 ERC guidelines suggest considering 18 mg if 12 mg is ineffective.⁸² Adenosine may also terminate an idiopathic VT due to triggered activity, albeit transiently.^{63,84} This approach to regular WCT is supported by the 2025 AHA,⁹ 2025 ERC,⁸² and 2022 ESC⁸⁴ guidelines. All patients receiving adenosine should be attached to a defibrillator in case a malignant tachydysrhythmia develops and immediate electrical therapy is required. In addition to prompt treatment, electrolyte levels should be measured, with liberal supplementation of magnesium and potassium when needed.⁶³

Stable VT should be terminated promptly to avoid the development of hemodynamic instability.⁸⁴ Stable VT can be managed with either synchronized DCCV or antidysrhythmic medications. Synchronized DCCV is the most effective treatment for stable VT; however, the risks of procedural analgesedation should be considered.^{20,84} Synchronized DCCV terminates VT in approximately 90% of cases.⁸³ DCCV failure may occur for several reasons, including correctable variables such as poor conduction at the interface of the skin and electrical pad or paddle, poor internal conduction due to pneumothorax, electrolyte abnormality, hypoxemia, severe acidemia, or an insufficient electrical therapy energy setting. Some variables (eg, large body mass index) are not rapidly correctable. Additionally, synchronized DCCV may fail to terminate automatic tachycardias (eg, atrial tachycardia) or prevent VT recurrence.⁸⁵

Pharmacologic options for stable VT include Vaughn-Williams class I antidysrhythmics (eg, lidocaine, procainamide) and class III medications (eg, amiodarone, sotalol). Procainamide will prolong the ventricular myocardial refractory period to terminate re-entry. The PROCAMIO study, a multicenter randomized controlled trial including patients with stable monomorphic WCT, found

that IV procainamide (10 mg/kg over 20 min) was associated with fewer adverse cardiac events and may be more effective for rhythm conversion than IV amiodarone (5 mg/kg over 20 min).⁸⁶ While this is the only randomized controlled trial to date comparing 2 medications for conversion of VT, the PROCAMIO study included only 74 patients, falling significantly short of its target enrollment of 302 patients, and it was underpowered to detect differences in conversion as its secondary outcome. Additionally, the PROCAMIO study excluded patients with anginal symptoms and WCTs that were terminated by adenosine,⁸⁶ so these results may not apply to ischemia-related and adenosine-responsive VT.⁸⁷ Earlier nonrandomized study data were also consistent with the PROCAMIO study,^{88,89} suggesting that amiodarone may be relatively less effective than procainamide for stable VT. IV amiodarone has no significant acute effect on ventricular refractoriness and repolarization. Its antidysrhythmic effect is largely time-dependent and due to accumulation of its active metabolite.^{90,91}

For patients with stable monomorphic WCT of unknown origin, both the 2017 AHA/ACC/Heart Rhythm Society and 2022 ESC guidelines for management of patients with ventricular arrhythmias recommend procainamide (class IIA) and amiodarone (class IIB), but note that procainamide is contraindicated in patients with acute MI, severe heart failure, and end-stage renal disease.^{63,84} The 2025 AHA guidelines suggest using an antidysrhythmic agent other than lidocaine,⁹ as the kinetic properties of lidocaine are relatively less effective for VT at hemodynamically tolerated rates,⁹² and it is unclear if lidocaine is more effective than placebo.^{30,33,63,84,92,93}

If the WCT is irregular and 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 atrial fibrillation or atrial flutter with variable AV conduction and bundle branch block. After considering treatment of an underlying condition, the clinician may attempt rate control, if necessary, with calcium-channel antagonists or beta-adrenergic receptor antagonists.⁵

Treatment of Extrinsic Causes

Extrinsic causes of WCT may include hyperkalemia and toxic ingestions, which should be treated in a case-specific manner. Initial treatment of hyperkalemia should include IV calcium (gluconate or chloride). For more information on the evaluation and management of hyperkalemia, see the November 2016 issue of *Emergency Medicine Practice* titled, "Evidence-Based Management of Potassium Disorders in the Emergency Department," available at www.ebmedicine.net/potassium-disorders

WCT related to toxic ingestion is often associated with TCAs. A rare ECG rhythm known as bidirec-

tional VT should raise suspicion for digoxin toxicity.⁶³ (See Figure 4, page 7.) Emergency clinicians should consider antidote-specific treatment for toxic ingestions (eg, sodium bicarbonate for TCA and digoxin immune Fab for digoxin). Extracorporeal membrane oxygenation may be beneficial for patients with unstable WCT caused by with toxic ingestions.⁹⁴ WCT that is SVT with aberrancy may be due to an underlying acute medical illness, which should be addressed earlier on in management, with the rhythm 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 for adverse events.⁵ If a toxic ingestion is suspected, the local poison control center should be contacted.

■ Special Circumstances

Irregular Wide Complex Tachycardia With Wolff-Parkinson-White Syndrome

The diagnosis of atrial fibrillation in conjunction with WPW syndrome is critical, as it will dictate therapy, but the recognition of this tachydysrhythmia may be difficult. Rates are often rapid, and the rhythm may appear regular to the undiscerning eye. The evidence on ECG that it is atrial fibrillation with WPW may be the presence of beat-to-beat variability in QRS width.²³ 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 together with the additional potential for rate-related aberrancy, this will give rise to QRS complexes of varying duration. (See Figure 9.)

In cases of pre-excited atrial fibrillation (eg, atrial fibrillation with WPW syndrome), current

Figure 9. Atrial Fibrillation With Wolff-Parkinson-White Syndrome



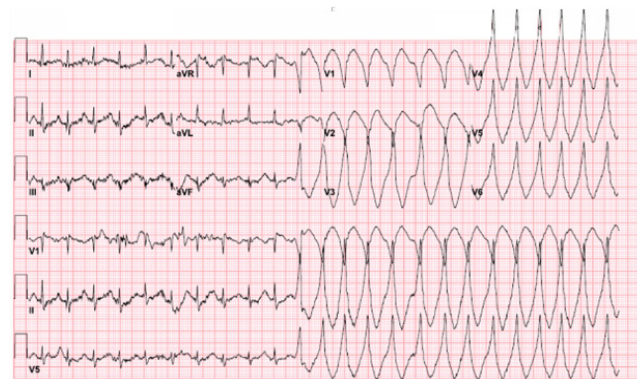
This electrocardiogram illustrates atrial fibrillation with Wolff-Parkinson-White syndrome. Note the extremely rapid ventricular response and beat-to-beat variability in QRS width. Image courtesy of Ian deSouza, MD.

guidelines recommend avoiding agents that slow conduction through the AV node (eg, adenosine, beta-adrenergic receptor antagonists, calcium channel antagonists, amiodarone), as conduction of rapid atrial impulses may then occur preferentially through the accessory tract to yield rapid, hemodynamically intolerable ventricular rates.^{9,82} Medications that slow conduction through the accessory pathway are preferred, and ibutilide and procainamide have both demonstrated success.^{23,95,96}

Outflow Tract Ventricular Tachycardia

Outflow tract VT, an idiopathic VT, is rare and unique in that it may occur in the absence of structural heart disease. Through the use of programmed stimulation and measured response to medications, outflow tract VT has been postulated to arise from triggered activity mediated by cyclic adenosine monophosphate.⁹⁷ Triggered activity refers to an extrasystole due to a premature depolarization that occurs when the amplitude of an early or delayed afterdepolarization brings the cardiac membrane to its threshold potential. A spontaneous action potential may be generated at a consequential point in the cardiac cycle, such as during repolarization ("R-on-T phenomenon"). Outflow tract VT is also known as *adenosine-sensitive, catecholamine-dependent VT*. Unlike myocardial scar-related VTs, adenosine terminates outflow tract VT through its antiadrenergic effect in the ventricular myocardium.⁹⁸ Tachydysrhythmia termination by adenosine may be transient, and the 2022 ESC guidelines recommend beta-adrenergic receptor antagonists to reduce the generation of premature ventricular complexes from the outflow tract and suppress VT recurrence.⁸⁴ (See Figure 10.)

Figure 10. Outflow Tract Ventricular Tachycardia



This electrocardiogram demonstrates sinus tachycardia with prolonged QTc followed by premature ventricular contraction-initiated regular, monomorphic ventricular tachycardia (triggered activity). This rhythm was confirmed to be a subtype of outflow tract tachycardia. Image 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 11.) It is a re-entrant tachydysrhythmia in which the pacemaker forms part of the re-entry circuit with ventriculoatrial (retrograde) conduction through the AV node. 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.⁹⁹ Placing a magnet over the pacemaker will terminate this re-entrant tachydysrhythmia by initiating asynchronous pacing. In contemporary devices, each manufacturer has a proprietary algorithm to detect and terminate pacemaker-mediated tachycardia.¹⁰⁰ After a pacemaker-mediated tachycardia event is successfully treated, the device may need to undergo urgent reprogramming.

Slow VT may be seen in up to 30% of patients with an ICD.¹⁰¹ Slow VT is defined as VT with a rate of 101 to 148 beats/min. The tachydysrhythmia may persist without ICD-delivered therapy, because the rate may lie below the device's tachycardia detection rate. When the WCT exceeds the programmed tachycardia detection rate, electrical therapy is delivered. Patients with slow VT, as in other tachycardias,

may exhibit signs and symptoms of hypoperfusion, but usually without clinical consequences, as the tachydysrhythmia is typically short-lasting.¹⁰¹ If necessary, medication therapy may be instituted in order to suppress further tachycardia episodes. An electrophysiologist may decide to reprogram the ICD to deliver antitachycardia pacing or internal defibrillation therapy to terminate the WCT. If an electrophysiologist is not immediately available, synchronized cardioversion may be attempted.

For patients with an ICD, overdrive pacing can terminate VT.⁸⁴ ICDs can be programmed to provide different types of electrical therapy (antitachycardia pacing, low-energy cardioversion, high-energy defibrillation) based on preset rate thresholds for VT capture.¹⁰² Antitachycardia (overdrive) pacing involves the ICD inducing a surge of ventricular paced impulses that are more rapid than the VT rate or a quickly decelerating sequence of ventricular paced impulses.¹⁰² Overdrive pacing is an advantageous initial therapy because it causes less patient discomfort and battery drain than cardioversion or defibrillation.¹⁰³

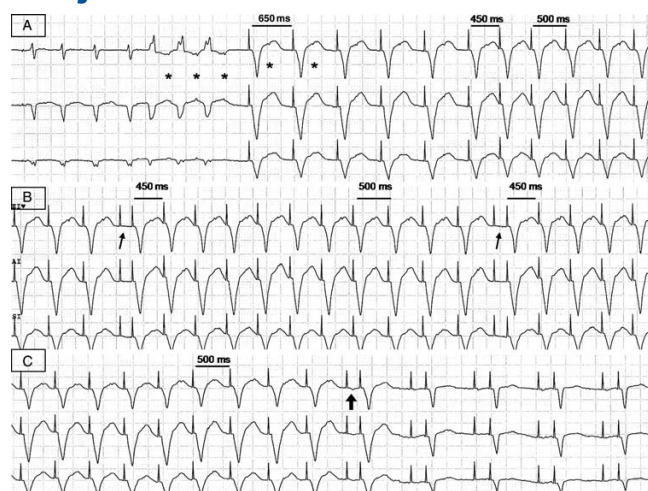
Electrical Storm

Electrical storm is defined as ≥ 3 episodes of sustained ventricular tachydysrhythmia or appropriate shocks within 24 hours.⁶⁴ Management of electrical storm should ideally be directed by the identified etiology, if known. For example, electrical storm secondary to acute MI (typically polymorphic VT), should receive prompt revascularization.^{84,104} If a patient has an existing ICD, magnet placement to disable tachydysrhythmia therapies may be considered, and programming optimization of the ICD should be performed, if possible.⁸⁴ For patients with electrical storm secondary to monomorphic VT, beta-adrenergic receptor antagonists and amiodarone may be considered as initial antidysrhythmic therapy and may reduce further episodes of VT.^{84,104} Electrical storm is theorized to be due, in part, to sympathetic surge, which is the reason beta-adrenergic receptor antagonists such as esmolol may be beneficial.¹⁰⁴ Sedation with propofol has been reported to convert antidysrhythmic-refractory VT electrical storm, likely via sympathetic blockade.^{105,106} Ultrasonography-guided stellate ganglion block reduces sympathetic innervation to the myocardium by injecting local anesthetic at the left C6–C7 level. Systematic reviews of stellate ganglion block found that it may reduce the number of VT episodes and need for ICD-delivered electrical therapy.^{107,108}

Controversies and Cutting Edge

Artificial intelligence (AI) has emerged as a potential tool to assist with differentiating WCT. Broadly speaking, AI encompasses computer algorithms that follow predetermined rules (machine-learning models)

Figure 11. Pacemaker-Mediated Tachycardia



A: The first 3 asterisks indicate premature ventricular contractions with retrograde ventriculoatrial conduction that then triggered the onset of pacemaker-mediated tachycardia.

B: The arrow shows lack of atrial capture, a failed attempt at termination by the device.

C: Arrow indicates successful atrial capture and termination of pacemaker-mediated tachycardia.

Anton Verrijcken, Wim Huybrechts and Rik Willems, Pacemaker-mediated tachycardia with varying cycling length: what is the mechanism? *Europace*, 2009, Volume 11, Number 10, pages 1400-1402, by permission of Oxford University Press.



Case Conclusions

CASE 1

For the 72-year-old man with VT who asked not to be shocked...

You reviewed the chart and saw that this patient had a history of ischemic cardiomyopathy with mildly reduced ejection fraction and prior episodes of VT. Using shared decision-making with the patient, you decided to defer electrical cardioversion and trial pharmacologic cardioversion. Having recently reviewed the PROCAMIO study, you decided to administer IV procainamide, which converted the patient back to sinus rhythm. You called the electrophysiologist, who recommended observing the patient overnight in the cardiac ICU.

CASE 2

For the 28-year-old woman with polymorphic VT following repeated episodes of vomiting...

The nurses promptly applied defibrillator pads in the anterolateral position on the patient, who was now unconscious. You charged the defibrillator to the maximum setting of 200 J. You recalled that the defibrillator might not be able to synchronize, so you defibrillated the patient. The repeat ECG showed sinus tachycardia, with a rate of 115 beats/min and a QTc of 558 milliseconds. You ordered IV magnesium. The chemistry laboratory called with a critical potassium level of 2.4 mEq/L, so you also ordered IV potassium repletion. The patient was admitted to the ICU. On follow-up the next day, her QTc had normalized after repletion of magnesium and potassium.

or learn to solve complicated problems from large datasets (deep-learning models).¹⁰⁹ There is great variability in the number of ECG parameters analyzed by an AI algorithm, with one incorporating as many as 40 characteristics.¹¹⁰ Researchers are testing AI models on WCT ECGs alone^{110,111} or paired with the baseline ECG to assess for changes in predetermined ECG characteristics.¹¹² While this research is probably years away from clinical application, the results are promising. One study showed superior performance of an AI model compared to Brugada, aVR, lead II R-wave-peak-time, and limb lead algorithms at differentiating WCT.¹¹⁰ AI models have also been shown to differentiate VT from SVT with 91.9% sensitivity and 93% accuracy, performing similarly to electrophysiologists and superior to nonelectrophysiologists.¹¹¹ Future prospective studies are needed prior to clinical implementation.

■ Disposition

Patients who present with new-onset WCT, regardless of the outcome of ED management, should be observed and considered for hospitalization. Because it can be difficult to differentiate between VT and SVT, emergency clinicians should maintain a high suspicion for VT. Patients with suspected VT should be admitted to a step-down or intensive care unit. Patients with WCT secondary to extrinsic causes (eg, toxic ingestions, hyperkalemia) should be evaluated by the appropriate consulting service. Some VTs may be terminated with adenosine, so the response to adenosine should not necessarily be considered reassurance for a safe discharge. If SVT with aberrancy

is apparent, discharge may be considered after consultation with an electrophysiologist or cardiologist and shared decision-making with the patient.



5 Things That Will Change Your Practice

1. Consider procainamide for first-line antidysrhythmic therapy for stable, monomorphic VT, as it may be safer and more effective than amiodarone.
2. While adenosine may terminate many SVTs, it may also terminate outflow tract VT, which will necessitate hospitalization for electrophysiology evaluation.
3. Bradydysrhythmia that is associated with torsades de pointes should be managed with pacing to maintain a normal ventricular rate.
4. Electrical storm may be treated with sympatholytic agents such as esmolol and stellate ganglion block.
5. For patients with irregular WCT and an ECG that suggests atrial fibrillation with WPW, avoid use of AV nodal antagonists, which may precipitate hemodynamic collapse.



Risk Management Pitfalls in the Emergency Department Management of Wide Complex Tachycardia

1. **"There's no way it could be VT. The patient was completely stable."** A patient's hemodynamic status is a poor predictor of VT. The diagnosis should be based on examination of the 12-lead ECG and use of adenosine (if it is a stable, regular WCT), and treatment should be tailored appropriately.
2. **"I thought if I just tried to shock him again at 200 J, it would work."** If the patient is not responding as expected, be cautious repeating treatments, including electrical cardioversion. Hyperkalemia and toxic ingestions can mimic VT, as can sinus tachycardia with a bundle branch block. All such cases will be refractory to synchronized DCCV.
3. **"She's had a pacemaker, so I thought it was 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. **"The kid was barely 18 years old, so I thought the irregular WCT had to be atrial fibrillation with WPW syndrome."** For young adults, be sure to consider the risk of VT before assuming that the tachydysrhythmia is SVT. Long-QT syndrome, congenital heart defects, and illicit substance use can increase the risk for ventricular tachydysrhythmias.
5. **"The QRS complex didn't look that different from the bundle branch block seen on prior ECGs, so I was sure it was just SVT with aberrancy."** In patients with underlying bundle branch block, VT 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 knew it had to be torsades de pointes, so I gave 2 g of IV magnesium."** Torsades de pointes is defined by prolonged baseline QTc and is only one subtype of polymorphic VT, so consider occlusion MI when a patient presents with polymorphic VT.
7. **"He was stable, so I wanted to avoid electrical cardioversion and just use amiodarone."** The PROCAMIO study demonstrated that IV procainamide may be safer and more effective than IV amiodarone for cardioverting VT.
8. **"He didn't respond to adenosine, so it must be VT."** Although the majority of SVTs will demonstrate some response to adenosine, some SVTs will not respond. Furthermore, outflow tract VT may be terminated by adenosine.
9. **"The patient's WCT could be related to hyperkalemia, but she's pretty stable, so I started procainamide and waited for her potassium level to come back from the lab."** WCT secondary to hyperkalemia may not respond to antidysrhythmics, and this situation carries a risk for hemodynamic decompensation and cardiac arrest. If hyperkalemia is suspected, IV calcium gluconate can be administered.
10. **"This was new-onset SVT with aberrancy, so the patient had to be admitted."** Young adult patients with SVT who are stable throughout the ED course after conversion to sinus rhythm and do not have an underlying, life-threatening medical illness may be safely discharged home after cardiology consultation and shared decision-making with the patient.

■ Summary

WCT is a potentially life-threatening condition that can be difficult to treat. The management of unstable WCT focuses on ensuring adequate perfusion, with prompt electrical therapy if necessary. However, in the setting of hemodynamic stability, appropriate medication therapy may be an effective management strategy. The most important tool in assessing patients with WCT is the ECG, while the history, physical examination, and response to adenosine serve as important adjuncts to guide diagnosis and management.

■ Time- and Cost-Effective Strategies

- The simplest WCT diagnostic algorithm assumes that the rhythm is VT unless proven otherwise.
- Adenosine may be both diagnostic and therapeutic in patients with stable, regular, monomorphic WCT.
- Although antidysrhythmic agents may be effective, synchronized DCCV is the most effective therapy for terminating monomorphic VT.
- Magnesium and potassium levels should be checked and liberally repleted in patients with suspected VT, as this may suppress further episodes.
- Patients with suspected SVT with aberrancy may not require a costly hospitalization if the cardiology consultant can expedite a follow-up outpatient re-evaluation.

■ 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, are noted by an asterisk (*) next to the number of the reference.

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CME Questions



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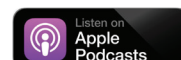
1. The characteristic that is most suggestive of ventricular tachycardia (VT) in an electrocardiogram (ECG) demonstrating wide complex tachycardia (WCT) is _____:
 - a. Upward right QRS axis
 - b. QRS duration >160 milliseconds
 - c. Positive concordance of the ECG chest leads
 - d. Atrioventricular (AV) dissociation

2. A 60-year-old woman with hypertension presents with nausea and acute chest pain radiating to the left shoulder. The pain improves with sublingual nitroglycerin. Her vital signs are: temperature, 36.5°C; heart rate, 78 beats/min; blood pressure, 160/100 mm Hg; and respiratory rate, 20 breaths/min. She is mildly diaphoretic. Cardiac examination reveals a regular rhythm. His lungs are clear to auscultation. The ECG demonstrates normal sinus rhythm with newly inverted T waves in the V4-V6, but no ST-segment elevation, and a normal corrected QT interval. Continuous cardiac monitoring shows nonsustained polymorphic VT at 150 beats/min. She remains alert without recurrent chest pain. While preparing her for cardiac catheterization, which of the following is the most appropriate management?
 - a. Adenosine
 - b. Ibutilide
 - c. Amiodarone
 - d. Defibrillation
3. A 57-year-old man presents to the ED with chest pain, palpitations, and confusion. His vital signs are: temperature, 36.1°C; heart rate, 172 beats/min; blood pressure, 89/50 mm Hg; and respiratory rate, 20 breaths/min. He has normal respirations and a rapid, regular pulse. The ECG shows a WCT with QRS duration of 162 milliseconds and left bundle branch block pattern. Which of the following is the most appropriate initial management?
 - a. Procainamide
 - b. Amiodarone
 - c. Synchronized direct current cardioversion (DCCV)
 - d. Defibrillation
4. A 69-year-old woman with a history of myocardial infarction (MI), heart failure with severely reduced ejection fraction, and VT presents with palpitations and mild dyspnea on exertion. Her vital signs are: temperature, 37.2°C; heart rate, 165 beats/min; blood pressure, 102/75 mm Hg; and respiratory rate, 18 breaths/min. Examination reveals jugular venous distension, rapid regular heart sounds, bibasilar rales, and 1+ bilateral lower extremity edema. The ECG shows a regular WCT at 165 beats/min with QRS duration of 170 milliseconds and QRS with an upward right axis. Which of the following is the most appropriate initial management?
 - a. Adenosine
 - b. Procainamide
 - c. Amiodarone
 - d. Defibrillation
5. A 40-year-old woman presents with palpitations and mild lightheadedness while driving today. She reports several similar prior episodes without syncope and denies chest pain or shortness of breath. She has no history of cardiac or metabolic disease and takes amitriptyline for depression and insomnia. There is no family history of heart disease or sudden death. Her vital signs are: temperature, 37.3°C; heart rate, 160 beats/min; blood pressure, 110/80 mm Hg; and respiratory rate, 22 breaths/min. She is mildly anxious but alert and oriented. Cardiac examination reveals a regular tachycardia. Her lungs are clear and her extremities are normal. The ECG shows a regular WCT at 160 beats/min with right bundle branch block morphology, a QRS duration of 126 milliseconds, and no visible P waves. Which of the following is the most appropriate initial management?
 - a. Adenosine
 - b. Procainamide
 - c. Amiodarone
 - d. Synchronized DCCV
6. A 35-year-old man with hypertension, diabetes mellitus, and chronic renal failure on hemodialysis presents with lightheadedness and mild dyspnea. He denies chest pain and has no known history of heart disease. His vital signs are: temperature, 37.5°C; heart rate, 130 beats/min; blood pressure, 110/70 mm Hg; and respiratory rate, 24 breaths/min. He is alert and cooperative, with a normal neurologic examination. Cardiac examination reveals a regular rhythm. His lungs have mild bibasilar rales without peripheral edema. An arteriovenous fistula is present in the left upper extremity. The ECG shows a WCT at 130 beats/min with a QRS duration of 160 milliseconds and a rounded morphology. Which of the following is the most appropriate initial management?
 - a. Adenosine
 - b. Amiodarone
 - c. Synchronized DCCV
 - d. Calcium gluconate

7. A 33-year-old man with a history of Wolff-Parkinson-White syndrome and paroxysmal atrial fibrillation presents with palpitations and nausea. His vital signs are: temperature, 37.0°C; heart rate, 220 beats/min; blood pressure, 130/90 mm Hg; and respiratory rate, 24 breaths/min. He is alert and oriented. Cardiac examination reveals an irregular tachycardia. His lungs are clear and his extremities are warm. Which of the following is the most appropriate initial management?
 - a. Adenosine
 - b. Procainamide
 - c. Amiodarone
 - d. Calcium chloride
8. A 68-year-old man with a history of prior MI and an implantable cardioverter-defibrillator (ICD) placement presents after experiencing 3 ICD shocks in the past 12 hours. While he is being placed on the monitor, he develops a WCT and cries out as he receives another shock. Which of the following is NOT an appropriate next step in management?
 - a. Cardiology consultation for catheter ablation
 - b. Propofol in preparation for synchronized DCCV
 - c. Isoproterenol
 - d. Esmolol
9. Emergency medical services bring a patient to the ED after a syncopal episode. The patient was recently started on ondansetron for vomiting. The initial ECG shows a QTc of 530 milliseconds. Shortly thereafter, the patient develops polymorphic VT and is promptly defibrillated. Which of the patient's home medications most likely contributed to this presentation through an additive effect with ondansetron?
 - a. Amlodipine
 - b. Quetiapine
 - c. Atorvastatin
 - d. Aspirin
10. You are called to evaluate a patient admitted for syncope. Cardiac monitoring shows a WCT at 180 beats/min. The patient's blood pressure is 100/60 mm Hg. The patient appears diaphoretic and reports chest pain and shortness of breath. Which of the following is the most appropriate next step in management?
 - a. Adenosine
 - b. Procainamide
 - c. Amiodarone
 - d. Synchronized DCCV



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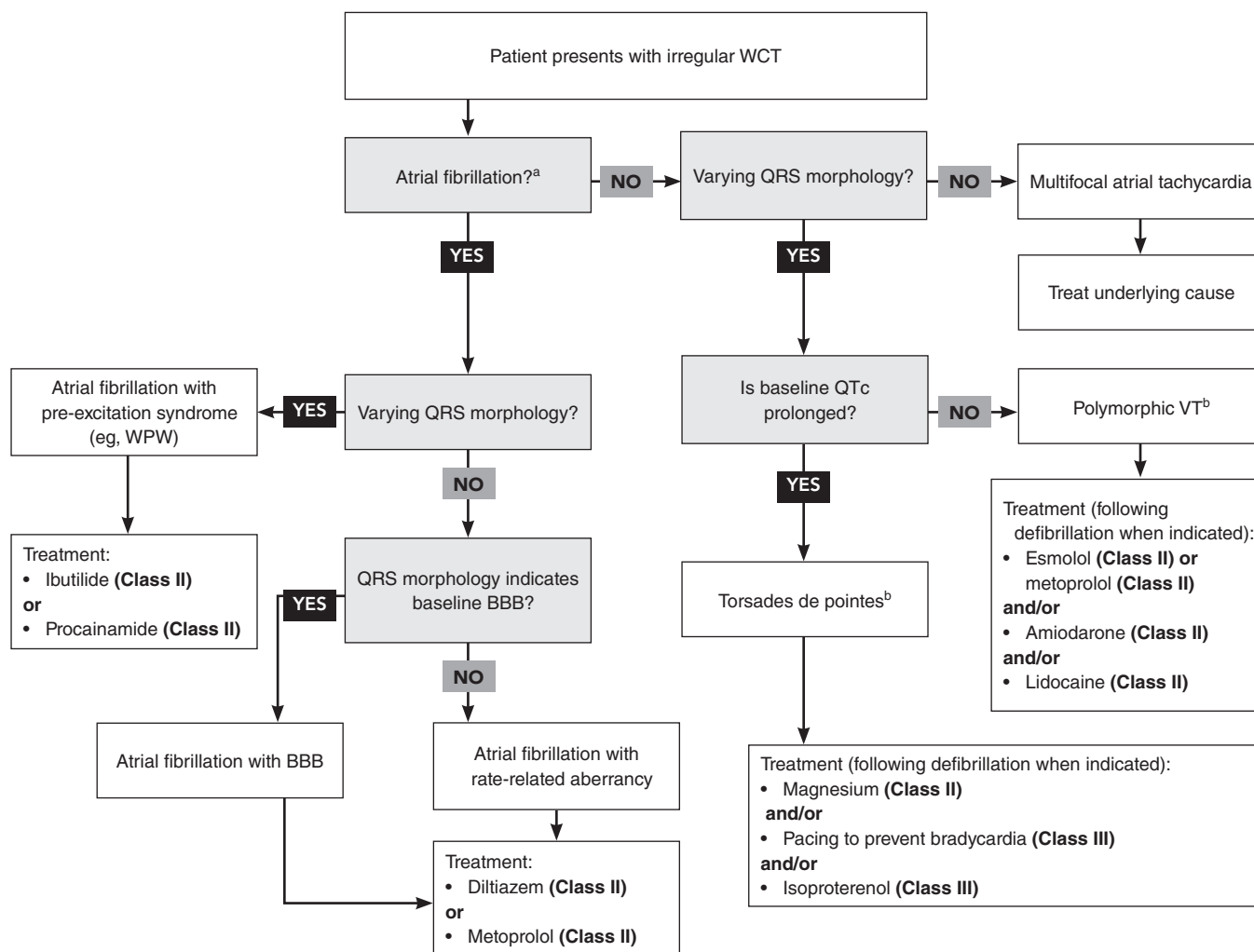




Clinical Pathway for Medical Management of Stable, Irregular Wide Complex Tachycardia



Click here or scan for interactive pathway



^aThe approach to atrial fibrillation can also be applied to atrial flutter with variable atrioventricular conduction.

^bThis tachyarrhythmia should be considered unstable and treated with immediate defibrillation.

Abbreviations: BBB, bundle branch block; ECG, electrocardiogram; VT, ventricular tachycardia; WCT, wide complex tachycardia; WPW, Wolff-Parkinson-White syndrome.

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

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

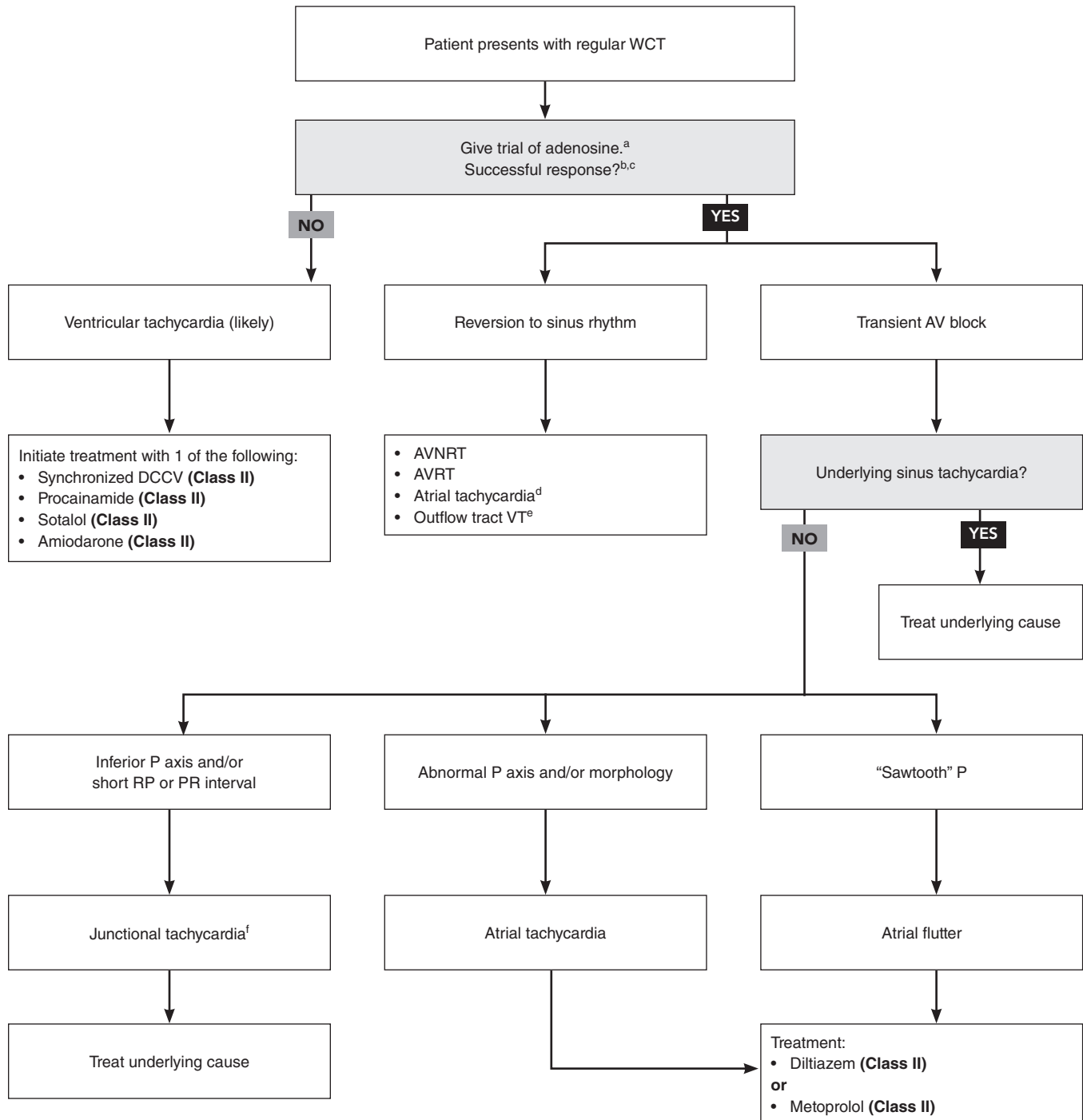
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Clinical Pathway for Initial Management of Stable, Regular Wide Complex Tachycardia



[Click here](#) or scan for interactive pathway



Abbreviations: AV, atrioventricular; AVNRT, atrioventricular nodal re-entrant tachycardia; AVRT, atrioventricular re-entrant tachycardia; DCCV, direct current cardioversion; VT, ventricular tachycardia; WCT, wide complex tachycardia.

^aAvoid use of adenosine in cases in which VT is probable (eg, evidence of AV dissociation).

^bVT may respond to adenosine with transient ventriculoatrial block.

^cThe character of response will help determine the underlying rhythm.

^dAdenosine will terminate some atrial tachycardias.

^eThis may require beta-adrenergic receptor antagonists to suppress recurrent episodes.

^fThis may be seen in patients with enhanced AV nodal automaticity, digoxin toxicity, or myocarditis.

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Emergency Department Management of Patients With Low Back Pain: A Review of Current Evidence



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Action

- Patient presents to the ED with low back pain
- Assess vital signs

Decision

What are the patient's vital signs?

☒ Normal

☐ ↑ Febrile

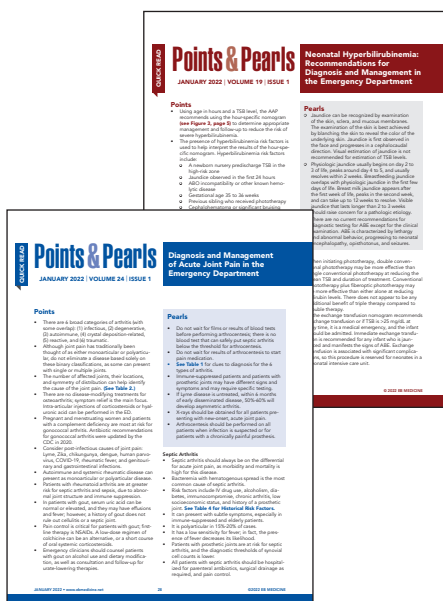
☐ ↓ Hypotensive, afebrile



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Points & Pearls

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Wide Complex Tachycardia in the Emergency Department: An Updated Approach to Diagnosis and Management

Points

- *Wide complex tachycardia* (WCT) is defined as a tachydysrhythmia with QRS duration ≥ 120 milliseconds. The primary diagnostic distinction is ventricular tachycardia (VT) versus supraventricular tachycardia (SVT) with aberrancy.
- WCT may result from metabolic or toxicologic causes, including hyperkalemia (see Figure 1) and sodium-channel blocker toxicity; these etiologies should be considered during initial evaluation.
- VT is the most common cause of WCT in adults and should be strongly considered in patients with structural heart disease or prior myocardial infarction unless an alternative diagnosis is clearly established.
- Evaluation of WCT requires integration of clinical context, rhythm regularity, and electrocardiogram (ECG) features to narrow the differential diagnosis. (See Table 1.)
- Antidromic atrioventricular (AV) re-entrant tachycardia in Wolff-Parkinson-White syndrome presents as a WCT due to anterograde conduction over an accessory pathway and may closely mimic monomorphic VT. (See Figure 3.)
- ECG findings that favor VT include extreme axis deviation (see Figure 5), precordial concordance (see Figure 6), AV dissociation (see Figure 7), and capture and fusion complexes (see Figure 8), which increase diagnostic specificity.
- For patients with baseline bundle branch block, SVT may present as WCT, and rate-related QRS morphology changes can complicate distinction from VT.
- Pre-excited atrial fibrillation presents as an irregular WCT with very rapid ventricular rates and beat-to-beat variability in QRS width, and may be difficult to identify.
- Adenosine may assist in clarifying rhythm diagnosis in undifferentiated stable, regular WCTs, by terminating or revealing an SVT with aberrancy.
- ECG-based diagnostic algorithms for WCT demonstrate variable accuracy, reinforcing the importance of systematic interpretation rather than reliance on a single rule.

Pearls

- Adenosine is safe and may be therapeutic in patients with stable, regular WCT.
 - Physical examination findings such as cannon A waves may support AV dissociation, but examination on its own is not sufficiently sensitive to exclude VT.
 - Avoid AV-nodal-blocking agents in atrial fibrillation with pre-excitation, as preferential conduction over an accessory pathway may result in rapid ventricular rates and hemodynamic compromise.
 - For patients with implantable cardiac devices, device interrogation may help clarify the tachydysrhythmia mechanism after stabilization.
 - Artificial intelligence-assisted ECG interpretation is an emerging tool that may support rhythm classification in WCT, but it does not replace clinical judgment and systematic ECG analysis.
- Pacemaker-mediated tachycardia occurs when a dual-chamber pacemaker forms part of a re-entry circuit, resulting in a paced WCT generated by retrograde conduction through the AV node and repeated ventricular pacing.
 - For stable monomorphic WCT presumed to be VT, procainamide may be associated with higher rates of tachydysrhythmia termination and fewer adverse events compared with amiodarone in appropriate patients.
 - Electrical therapy (eg, synchronized cardioversion or defibrillation) is appropriate when WCT is refractory to pharmacologic therapy or when clinical status deteriorates.
 - Synchronized cardioversion is indicated for patients with WCT and hemodynamic instability (eg, hypotension, altered mental status, ischemic chest pain, shock, or acute heart failure).
 - Hospitalization is appropriate for patients with VT or diagnostic uncertainty, while selected patients with clearly identified SVT and reassuring evaluation may be considered for discharge with follow-up.