

Wide Complex Tachycardia: Diagnosis And Management In The Emergency Department

It's actually a slow day in the ED and you make the mistake of saying so. Suddenly, the EMS radio goes off and reports that they are en route with a dyspneic older male, status post recent syncopal event—ETA 10–12 minutes; an ECG rhythm strip is sent for your review (Figure 1, patient #1). He has a history of myocardial infarction (MI) with congestive heart failure (CHF). His vital signs include a blood pressure (BP) of 100/65 mm Hg, pulse (P) of 170 bpm, respiratory rate (RR) of 28/minute, and oxygen saturation (SAT) of 92% on 4l nasal cannula.

Seconds later, the charge nurse tells you that you should see the elderly lady in room 22 soon. Upon entering the room, you see an awake, alert, elderly female sitting comfortably on the stretcher. She is conversant and in no distress. Her vital signs include a BP of 170/120 mm Hg, pulse 170 bpm, RR of 22/minute, and SAT of 94% on room air; the 12-lead ECG reveals a wide complex tachycardia (WCT, Figure 2, patient #2).

Then, you hear "I need a doctor now" from room 9. You find a young adult male supine on the cot. He is alert and oriented, complaining of extreme dizziness and weakness. He is pale and diaphoretic. The monitor demonstrates a WCT (Figure 3, patient #3). The examination is significant for a BP of 85 mm Hg by palpation, pulse 190 bpm, RR of 34/minute, and SAT of 96% on room air.

Around the same time, you are called to the telephone regarding a patient a local internist is sending in for evaluation. An elderly male, aged 71 years, presented with palpitations. He has a history of stable angina managed with atenolol. His examination is remarkable for normal vital

June 2008

Volume 10, Number 6

Authors

N. Raj Subramanian, MD

Fellow in Cardiovascular Medicine, Division of Cardiology, Department of Medicine, University of Virginia, Charlottesville, VA

William J. Brady, MD

Professor of Emergency Medicine and Medicine Vice Chair of Emergency Medicine, University of Virginia School of Medicine, Charlottesville, VA

Peer Reviewers

Luke K. Herman, MD

Director, Chest Pain Unit; Assistant Professor, Department of Emergency Medicine, Mount Sinai School of Medicine, New York, NY

Keith A. Marill, MD

Assistant Professor, Department of Emergency Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA

Amal Mattu, MD

Program Director, Emergency Medicine Residency, Associate Professor, Department of Emergency Medicine, University of Maryland School of Medicine, College Park, MD

CME Objectives

Upon completion of this article, you should be able to:

1. Describe the common etiologies of wide complex tachycardia (WCT).
2. Identify the commonly encountered etiologies.
3. Describe the key historical questions that suggest a particular etiology for a WCT.
4. Discuss the electrocardiographic algorithm proposed by Griffith and colleagues to distinguish between ventricular tachycardia and supraventricular tachycardia with aberrancy.
5. Summarize the management protocol for monomorphic and polymorphic ventricular tachycardia.

Date of original release: June 1, 2008

Date of most recent review: May 10, 2008

Termination date: June 1, 2011

Time to complete activity: 4 hours

Medium: Print & online

Method of participation: Print or online answer form and evaluation

Prior to beginning this activity, see "Physician CME Information" on the back page.

Editor-in-Chief

Andy Jagoda, MD, FACEP

Professor and Vice-Chair of Academic Affairs, Department of Emergency Medicine; Residency Program Director, Mount Sinai School of Medicine, New York, NY.

Associate Editor

John M. Howell, MD, FACEP

Clinical Professor of Emergency Medicine, George Washington University, Washington, DC; Director of Academic Affairs, Best Practices, Inc, Inova Fairfax Hospital, Falls Church, VA.

Editorial Board

William J. Brady, MD

Professor of Emergency Medicine and Medicine Vice Chair of Emergency Medicine, University of Virginia School of Medicine, Charlottesville, VA

Peter DeBlieux, MD

Professor of Clinical Medicine, LSU Health Science Center, New Orleans, LA.

Wyatt W. Decker, MD

Chair and Associate Professor of EM, Mayo Clinic College of Medicine, Rochester, MN.

Francis M. Fesmire, MD, FACEP

Director, Heart-Stroke Center, Erlanger Medical Center; Assistant Professor, UT College of Medicine, Chattanooga, TN.

Michael J. Gerardi, MD, FAAP, FACEP

Director, Pediatric EM, Children's Medical Center, Atlantic Health System; Department of Emergency Medicine, Morristown Memorial Hospital, Morristown, NJ.

Michael A. Gibbs, MD, FACEP

Chief, Department of Emergency Medicine, Maine Medical Center, Portland, ME.

Steven A. Godwin, MD, FACEP

Assistant Professor and Emergency Medicine Residency Director, University of Florida HSC, Jacksonville, FL.

Gregory L. Henry, MD, FACEP

CEO, Medical Practice Risk Assessment, Inc; Clinical Professor

of Emergency Medicine, University of Michigan, Ann Arbor, MI.

Keith A. Marill, MD

Assistant Professor, Department of Emergency Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA.

Charles V. Pollack, Jr, MA, MD, FACEP

Chairman, Department of Emergency Medicine, Pennsylvania Hospital, University of Pennsylvania Health System, Philadelphia, PA.

Michael S. Radeos, MD, MPH

Assistant Professor of Emergency Medicine, Lincoln Health Center, Bronx, NY.

Robert L. Rogers, MD, FAAEM

Assistant Professor and Residency Director, Combined EM/IM Program, University of Maryland, Baltimore, MD.

Alfred Sacchetti, MD, FACEP

Assistant Clinical Professor, Department of Emergency Medicine, Thomas Jefferson

University, Philadelphia, PA.

Scott Silvers, MD, FACEP

Medical Director, Department of Emergency Medicine, Mayo Clinic, Jacksonville, FL.

Corey M. Slovis, MD, FACP, FACEP

Professor and Chair, Department of Emergency Medicine, Vanderbilt University Medical Center, Nashville, TN.

Jenny Walker, MD, MPH, MSW

Assistant Professor; Division Chief, Family Medicine, Department of Community and Preventive Medicine, Mount Sinai Medical Center, New York, NY.

Ron M. Walls, MD

Chairman, Department of Emergency Medicine, Brigham & Women's Hospital; Associate Professor of Medicine (Emergency), Harvard Medical School, Boston, MA.

Research Editors

Nicholas Genes, MD, PhD

Chief Resident, Mount Sinai Emergency Medicine Residency, New York, NY.

International Editors

Valerio Gai, MD

Senior Editor, Professor and Chair, Department of Emergency Medicine, University of Turin, Turin, Italy.

Peter Cameron, MD

Chair, Emergency Medicine, Monash University; Alfred Hospital, Melbourne, Australia.

Amin Antoine Kazzi, MD, FAAEM

Associate Professor and Vice Chair, Department of Emergency Medicine, University of California, Irvine; American University, Beirut, Lebanon.

Hugo Peralta, MD

Chair of Emergency Services, Hospital Italiano, Buenos Aires, Argentina.

Maarten Simons, MD, PhD

Emergency Medicine Residency Director, OLVG Hospital, Amsterdam, The Netherlands.

Accreditation: This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of Mount Sinai School of Medicine and *Emergency Medicine Practice*. The Mount Sinai School of Medicine is accredited by the ACCME to provide continuing medical education for physicians. **Faculty Disclosure:** Dr. Subramanian, Dr. Herman, Dr. Marill, and Dr. Mattu report no significant financial interest or other relationship with the manufacturer(s) of any commercial product(s) discussed in this educational presentation. Dr. Brady has received consulting fees from Heartscape Tech. **Commercial Support:** *Emergency Medicine Practice* does not accept any commercial support.

signs with the exception of a pulse of 190 bpm. The ECG rhythm strip demonstrates a WCT (Figure 4, patient #4).

When confronted with a wide complex tachycardia (WCT), it is crucial to consider the differential diagnosis, which includes both common and uncommon entities. The common entities include supraventricular tachycardia (SVT) with aberrant ventricular conduction (AVC) and ventricular tachycardia (VT). Less commonly encountered processes include preexcited tachycardias (seen in patients with Wolff-Parkinson-White [WPW] syndrome) as well as toxic- and metabolically-mediated WCTs (sodium channel blocker toxicity, severe hyperkalemia).

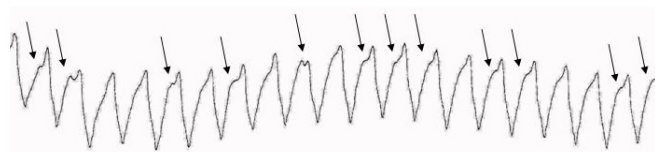
Certain electrocardiographic (ECG) distinctions among these dysrhythmias are of critical importance. For instance, differentiating a WCT resulting from hyperkalemia as opposed to primary VT is vital in the immediate management. While one might be tempted into complacency when faced with a patient with WCT who is hemodynamically stable, distinguishing SVT with AVC from VT is equally important for both the immediate and long-term management.

This issue of *Emergency Medicine Practice* provides a systematic approach to wide complex tachycardia.

Critical Appraisal Of The Literature

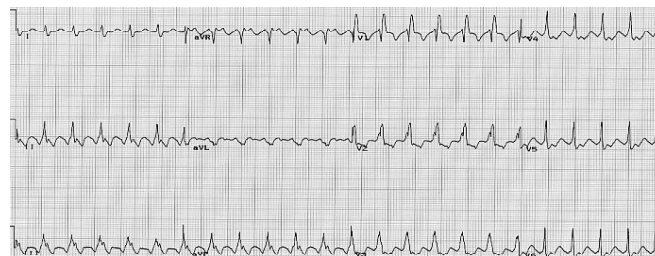
A literature search was performed using PubMed and Ovid MEDLINE. The search included articles on wide (broad) complex tachycardia, supraventricular tachycardia with aberrant ventricular conduction,

Figure 1. Patient #1—Wide Complex Tachycardia Which Is Regular



Note the appearance of notching (arrows) which occurs irregularly—P wave activity consistent with AV dissociation. This AV dissociation supports an electrocardiographic diagnosis of ventricular tachycardia.

Figure 2. Patient #2—Wide Complex Tachycardia With The Classic RBBB Morphology



Note the rate of 150 bpm with typical flutter waves (best seen in leads II, III, and avF). This rhythm is likely an SVT with AVC—probably atrial flutter with RBBB.

ventricular tachycardia, cardiovascular resuscitation, antidromic tachycardias, preexcitation syndromes, Wolff-Parkinson-White syndrome, poisoning and arrhythmias, pacemaker tachycardias, pediatric arrhythmias, pregnancy and arrhythmias, and hyperkalemia. This search provided a large number of studies ranging from case series to well-designed prospective data on a large cohort of patients. There are a limited number of randomized, double-blinded studies available. Further, standard textbooks and other reference materials from both cardiology and emergency medicine were reviewed.

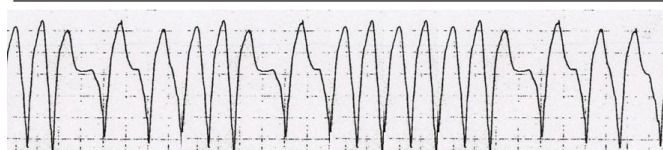
A number of investigators have tried to identify criteria and develop algorithms for the diagnosis of WCT.¹⁻⁶ In general, the major flaw in their methodologies is that their approach excluded the analysis of patients with preexisting ventricular conduction abnormalities, metabolic abnormalities, and drug toxicities.

Regarding algorithms, though they may be helpful in identifying the etiology of a WCT, there is no single criterion or combination of criteria that has been shown to be 100% sensitive. Hence, clinical judgment cannot be replaced by algorithms alone. Despite these limitations, the 12-lead ECG is useful in making a presumptive clinical diagnosis in the vast majority of patients who present with a WCT.

Epidemiology

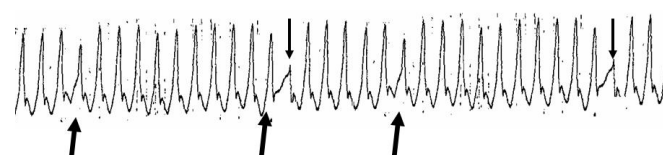
WCT is defined as a tachyarrhythmia with a rate greater than 100 bpm and a QRS complex duration of 0.12 seconds or greater in the adult patient. In the pediatric population, both of these parameters are age dependant.⁷ Age-related rate differences are understandable and readily recognized; QRS complex duration changes with respect to age, however, are less

Figure 3. Patient #3—Wide Complex Tachycardia Which Is Irregular With A Mean Ventricular Rate Of 190 bpm



Note the varying configurations of the QRS complexes across the rhythm strip but no significant QRS amplitude variations.

Figure 4. Patient #4—Wide Complex Tachycardia Which Is Regular With A Mean Ventricular Rate Of 190 bpm



Note the regular appearance of widened QRS complexes. On occasion, 2 different QRS complex configurations appear—fusion (larger arrows) and capture beats (smaller arrows) which strongly suggest ventricular tachycardia.

obvious. As noted, the total width of the QRS complex is a function of the total ventricular depolarization time. In young children with relatively smaller ventricular muscle mass, the QRS complex can appear “narrow” from the adult perspective and yet still be “wide” when interpreted correctly using age-related criteria.

Extrapolating the number of episodes of ventricular tachycardia seen at one urban emergency medicine department with annual ED census of around 52,000 equates to approximately 1.6 cases of wide complex tachycardia seen per month.⁸ In another setting, 178 patients were noted to have WCT among a total of 82,559 ED visits in a 2-year period (7.4 cases per month).⁹ If one considers “all comers” with WCT, including patients with sinus tachycardia and fixed bundle branch block (BBB), then these numbers will only increase, potentially approaching several such instances daily.

The conventional wisdom in the medical literature is that approximately 80% of patients presenting with WCT are ultimately diagnosed with VT; the remaining 20% demonstrate SVT with AVC. Early non-ED-based studies did suggest a high incidence of VT among patients with WCT. In 2 series of patients with WCT referred to an EPS, 80%–85% of patients had VT as the underlying mechanism; the remaining had SVT with aberrancy.^{10,11} Both these cohorts suffer from referral bias. In contrast, in an ED study of 178 consecutive patients with WCT, 49% (88) had sinus tachycardia with preexisting BBB or aberrancy.⁹ Thirty-five percent had irregular WCT; while this may represent atrial fibrillation (AF), this group of patients was not described in the study. Among the remaining patients with regular WCT, the majority (63%) were considered to be VT by at least 2 of the 3 physicians classifying these patients using ECG criteria. Unfortunately, these diagnoses were not confirmed by electrophysiological study (EPS). It is important to note in this study that 84% of all cases were supraventricular in origin, leaving at most only 16% of individuals presenting with a WCT that was ventricular in origin—a complete reversal of the non-ED-based studies.^{10,11} Of note, this

Abbreviations Used In This Article

ACLS: Advanced Cardiac Life Support
 ACS: Acute Coronary Syndrome
 ART: Antidromic Reciprocating Tachycardia
 AV: Atrioventricular
 AVC: Aberrant Ventricular Conduction
 CABG: Coronary Artery Bypass Grafting
 CAD: Coronary Artery Disease
 EPS: Electrophysiological Study
 LBBB: Left Bundle Branch Block
 RBBB: Right Bundle Branch Block
 SA: Sinoatrial
 TCA: Tricyclic Antidepressant
 SVC: Supraventricular Tachycardia
 TdP: Torsades de Pointes
 UA: Unstable Angina
 VT: Ventricular Tachycardia
 WCT: Wide Complex Tachycardia
 WPW: Wolff-Parkinson-White

calculation includes sinus tachycardia with bundle branch block, a subgroup that is underrepresented in the non-ED-based studies.

Etiology, Pathophysiology, And Differential Diagnosis

Although the pathophysiologic mechanisms of the underlying WCTs are quite diverse, the mechanism for the widened QRS complex is easily understood if the clinician recalls that the ECG is simply a graph of voltage (y axis) relative to time (x axis). In sinus rhythm with normal intraventricular conduction, the electrical impulse is rapidly propagated throughout the ventricular myocardium with near-simultaneous depolarization—thus the total time of electrical discharge in the ventricles is brief and the QRS complex is narrow (**Diagram 1**). In situations in which the conduction system is either dysfunctional (either permanently due to underlying pathology or temporarily during the tachycardia) or not utilized for primary conduction, the electrical impulse requires more time to depolarize the ventricle, resulting in a widened QRS complex (**Diagrams 2–6**).

SVT With Aberrant Ventricular Conduction (AVC)

WCTs can be classified into 6 groups, which are described in **Table 1**. SVT with AVC occurs because of abnormal intraventricular conduction delay within the His-Purkinje system (HPS) during the tachycardia. This intraventricular conduction delay may occur either due to preexisting bundle branch block or transient slowing of bundle branch conduction during tachycardic states (**Diagrams 2 and 3**). In either case, the intraventricular conduction delay prevents the

Table 1. Differential Diagnosis Of WCT

Broad Classification	Specific Arrhythmias/ Considerations
Supraventricular tachycardia with aberrant ventricular conduction	SVT with either preexisting BBB or tachycardia-related aberrancy
Preexcited tachycardia (in patients with WPW)	Antidromic tachycardia Atrial fibrillation with preexcitation Any other SVT with preexcitation
Ventricular tachycardia	Monomorphic VT Polymorphic VT
Toxic and metabolic derangement	Acidemia Electrolyte abnormalities <ul style="list-style-type: none"> • Hyperkalemia • Hypomagnesemia Drug toxicity/poisoning <ul style="list-style-type: none"> • Class IC anti-arrhythmic drugs • Tricyclic antidepressants
Pacemaker-related WCT	Runaway pacemaker Sensor-mediated WCT Atrial-tracking-mediated WCT Endless loop tachycardia
Artifact	Need 12-lead ECG to distinguish artifact from a true WCT

rapid and efficient transmission of electrical impulses throughout the ventricular myocardium, slowing the time to depolarize the ventricle and manifesting clinically as a widened QRS.

Ventricular Tachycardia

Ventricular tachycardia most commonly originates in the ventricular myocardium external to the ventricular conduction system. Monomorphic ventricular tachycardia usually necessitates the presence of a scar in the myocardium that contains islands of viable tissue within it.^{88,89} Only a small minority of patients have ventricular tachycardia in the absence of structural heart disease (i.e., myocardial scar). The etiology for the scar in patients with coronary artery disease (CAD) is typically prior myocardial infarction (MI). The ability to induce monomorphic ventricular tachycardia during an electrophysiological study is much higher in patients with CAD and prior MI than in patients with CAD without prior MI.⁸⁸ For unclear reasons, many patients with non-ischemic cardiomyopathy also tend to have scar in their myocardium, which can promote ventricular tachycardia.⁸⁸ Because the electrical impulses during ventricular tachycardia travel from myocyte to myocyte through the ventricular myocardium instead of the His-Purkinje conduction system, a longer time is required for ventricular depolarization, resulting in a widened QRS complex (**Diagram 4**).

Preexcited Tachycardias

Another type of WCT is the preexcited tachycardias, which are pathognomonic for Wolff-Parkinson-White syndrome. While preexcited tachycardias are only seen in patients with WPW syndrome, a patient with WPW can present with any of the WCTs listed in **Table 1**. Preexcited tachycardias include antidromic reciprocating tachycardia (ART), atrial fibrillation with preexcitation, and SVT with preexcitation. Preexcitation causes the electrical impulse to preferentially travel down an accessory pathway (connecting the atria to the ventricle) that is located external to the His-Purkinje system/atrioventricular node (AV Node). Once the electrical impulses reach the ventricle through the accessory pathway, propagation through the ventricular myocardium occurs from ventricular myocyte to myocyte, in a fashion very similar to VTs (**Diagrams 5 and 6**). Because it depolarizes much of the ventricle without using the HPS (similar to VTs), the management of preexcited tachycardias shares many similarities to VT management.

WCT From Toxic And Metabolic Derangement

Yet another category of WCTs are those associated with toxic and metabolic derangement. Classic examples in this category of WCTs include anti-arrhythmic toxicity, tricyclic antidepressant overdose, and severe

hyperkalemia. Even though the specific etiology can be quite varied, the underlying pathology relates to “poisoning” of the HPS through their effect on the ion-channels that are responsible for the action potential of the HPS. With many of the toxic and metabolic abnormalities, the effects are more generalized. They can also affect the action potential of the sinoatrial (SA) node, atria, atrioventricular (AV) node, and the ventricular myocardium. The net result is a wide QRS complex tachycardia. This is an important category to distinguish from the others because the usual management strategies for SVT or VT are less efficacious in this setting unless the underlying toxic and metabolic abnormalities are treated.

Any anti-arrhythmic drug has the potential to be proarrhythmic, particularly the class IC agents which are potent sodium channel blockers.⁹⁹ Overdose or toxicity with class IC agents (e.g., propafenone or flecainide) can cause severe conduction system dysfunction, malignant ventricular arrhythmias, electromechanical dissociation, and asystole (**Figure 5**).³⁴ Flecainide overdose has been reported to result in a mortality rate of 8% compared with other drug overdoses in general that have a mortality rate of less than 1%.³⁴ Other anti-arrhythmic agents, like class Ia and III drugs (e.g., quinidine, sotalol, dofetilide, etc.), tend to be proarrhythmic by their QT interval prolonging effects, which can put patients at risk for torsades de pointes (TdP).¹⁰⁴ This risk of QT interval prolongation is not restricted to anti-arrhythmic drugs but can happen with a whole host of other commonly used medications.¹⁰⁴

Tricyclic antidepressant (TCA) toxicity is well known to cause numerous cardiovascular complications including hypotension and wide complex tachycardias.³⁶ Toxicity is worsened by acidemia, hypotension, and hyperthermia.³⁷ The spectrum of TCA-related cardiac dysrhythmias ranges from sinus tachycardia with a wide QRS complex to ventricular tachycardia and ventricular fibrillation (**Figure 6**).³⁸ Distinguishing sinus tachycardia with a wide QRS from ventricular tachycardia can be difficult in this setting. The presence of an anticholinergic

Figure 5. Wide Complex Tachycardia Due To Sodium Channel Blockade Resulting From Flecainide Toxicity

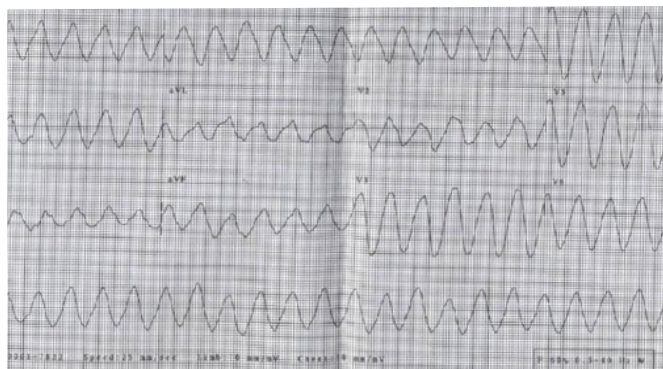
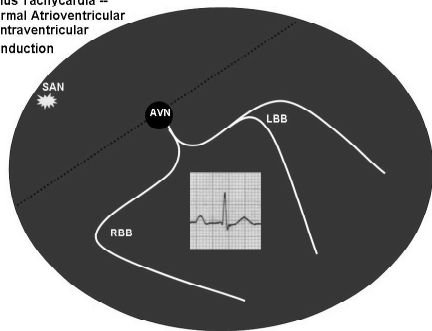


Diagram 1. Normal Intraventricular Conduction In Sinus Rhythm

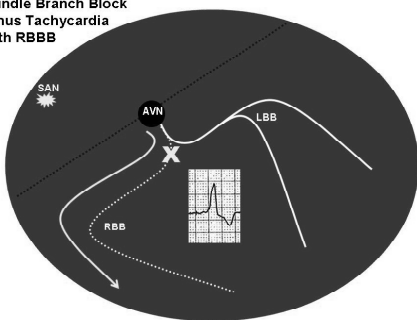
Sinus Tachycardia – Normal Atrioventricular & Intraventricular Conduction



Note the intraventricular conduction system (white lines) and its ability to rapidly spread the depolarization throughout the ventricular system, producing a narrow QRS complex.

Diagram 2. Intraventricular Conduction With Preexisting Bundle Branch Block—Sinus Tachycardia With RBBB

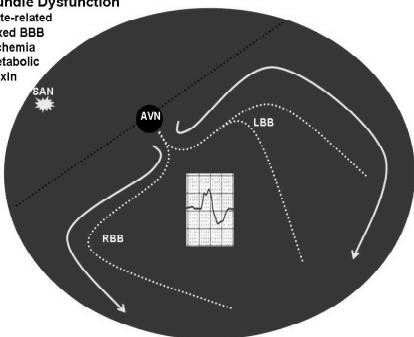
Bundle Branch Block Sinus Tachycardia with RBBB



Note the intraventricular conduction system (white lines) with malfunction of the right bundle branch (i.e., RBBB). The depolarization impulse is then spread throughout the right portion of the ventricular myocardium via cell-to-cell transmission (solid white arrow), a much less-efficient means, resulting in a longer period of depolarization and a widened QRS complex.

Diagram 3. Intraventricular Conduction With Bundle Dysfunction Resulting From Rate-Related, Fixed BBB, Ischemia, Metabolic, Or Toxic Events

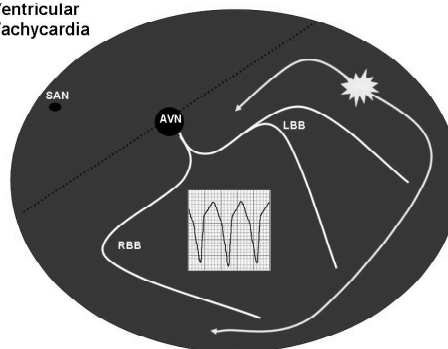
Bundle Dysfunction Rate-related Fixed BBB Ischemia Metabolic Toxin



Note the intraventricular conduction system dysfunction (dotted white lines). The depolarization impulse is then spread throughout the ventricular myocardium via cell-to-cell transmission (solid white arrow), a much less-efficient means, resulting in a longer period of depolarization and a widened QRS complex.

Diagram 4. Ventricular Tachycardia

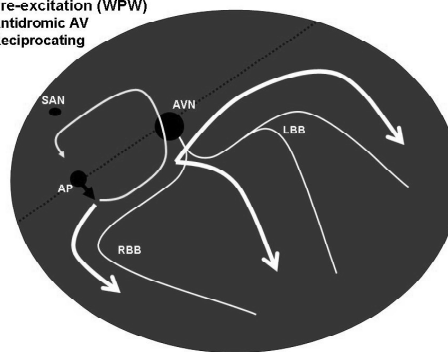
Ventricular Tachycardia



With the focus in the ventricle, the depolarization impulse is spread throughout the ventricular myocardium via cell-to-cell transmission (solid white arrow), a much less-efficient means, resulting in a longer period of depolarization and a widened QRS complex. The intraventricular conduction system is not involved in this process.

Diagram 5. Intraventricular Conduction With Ventricular Preexcitation (WPW)—The Antidromic AV Reciprocating Tachycardia

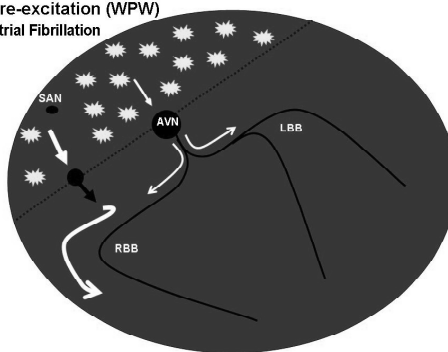
Pre-excitation (WPW) Antidromic AV Reciprocating



Note the retrograde movement of the impulse through the AV node and antegrade movement through the accessory pathway (circular, white arrow). The impulse then reaches the ventricular myocardium external to the conduction system, requiring cell-to-cell transmission (large white arrow), a much less efficient means, resulting in a longer period of depolarization and a widened QRS complex.

Diagram 6. Intraventricular Conduction With Ventricular Preexcitation (WPW)—Atrial Fibrillation

Pre-excitation (WPW) Atrial Fibrillation



The impulse travels to the ventricle via both the accessory pathway and the AV node. The resultant ventricular depolarization is a function of both routes of depolarization—from the accessory pathway and the AV node.

toxidrome supports the diagnosis of TCA overdose and should be sought. TCA toxicity is also supported by the presence of deep S waves in lead I and prominent R waves in lead aVR—which indicates a far rightward deviation of the terminal 40 ms of the QRS complex. This finding is not only suggestive of TCA cardiotoxicity (sodium channel blocking agents in general) but also predictive of dysrhythmia.³⁹ Many other psychiatric drugs have also been reported to cause a WCT (e.g., lithium toxicity).³⁵

Among electrolyte abnormalities, hyperkalemia is a common source of severe conduction abnormalities. As serum concentrations of potassium rise, persistent membrane depolarization impairs sodium channel activity, resulting in a wide QRS complex arrhythmia that can simulate ventricular tachycardia and, if unabated, can result in ventricular fibrillation and asystole (**Figure 7**). Because hyperkalemia causes slowing of atrioventricular and intraventricular conduction, the wide QRS complex arrhythmias that result from hyperkalemia are rarely faster than 140 bpm, usually have extremely wide and bizarre QRS morphologies, and do not demonstrate any rapid deflections within the QRS complex.⁴⁰⁻⁴³

Pacemaker-Related WCT

Pacemaker-related WCT is another important category of WCT. Ventricular pacing usually results in a wide QRS complex. Pacemaker-mediated WCT usually occurs in persons with a dual-chamber pacemaker. It is usually readily distinguished from other WCT by visualization of pacing spikes (**Figure 8**).

WCT due to ventricular pacing is rarely due to true pacemaker hardware malfunction (i.e., runaway pacemaker). A runaway pacemaker is an emergency because it can result in rapid delivery of pacing stimuli to the myocardium and the potential to induce high-grade ventricular arrhythmias. It necessitates emergent intervention to replace the device or cut the lead to interrupt the pacing stimulus.⁴⁵⁻⁴⁷ In pacemaker-dependant patients, cutting the lead can create new challenges (i.e., asystole or slow ventricular escape rhythm) unless a temporary pacing mechanism is provided. As most modern systems incorporate protective circuitry, this malfunction is extremely rare.

More common reasons for WCT due to ventricular pacing include sensor-mediated tachycardia, atrial

tracking, and endless loop tachycardia.⁴⁵ Sensor-mediated tachycardia involves ventricular pacing at rates higher than the programmed lower rate limit of the pacemaker, based on the built-in mechanical sensor that tracks activity level in patients with sick sinus syndrome.⁴⁵ Different types of physiologic and non-physiologic signals can interfere with the mechanical sensor, resulting in pacemaker-related WCT (e.g., Parkinsonian tremor, electrocautery, connecting a patient to cardiac monitor in the ED).^{92,93,95} Sensor-mediated WCTs are usually at or below the programmed upper sensor rate (usually 110–150 bpm). Atrial tracking refers to the attempt by the pacemaker to maintain atrioventricular synchrony usually in patients with some degree of AV block. Atrial tracking can cause WCT during supraventricular tachycardia (e.g., atrial flutter) and is usually at or below the programmed upper tracking rate (typically 110–150 bpm).^{44,45} Endless loop tachycardia is usually seen in dual chamber pacemakers with WCT at the upper tracking rate.^{44,45} Ventricular pacing in these patients causes retrograde transmission of electrical impulse through the HPS/AV node back to the atrium where it is sensed as another native atrial event; this causes the pacemaker to track the atrial signal in an attempt to maintain AV synchrony.⁴⁵ Most modern pacemaker systems have algorithms to terminate endless loop tachycardia.⁴⁵

Figure 6. Wide Complex Tachycardia Due To Sodium Channel Blockade Resulting From Tricyclic Antidepressant Poisoning

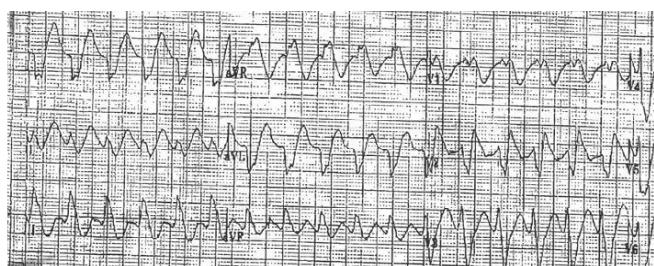
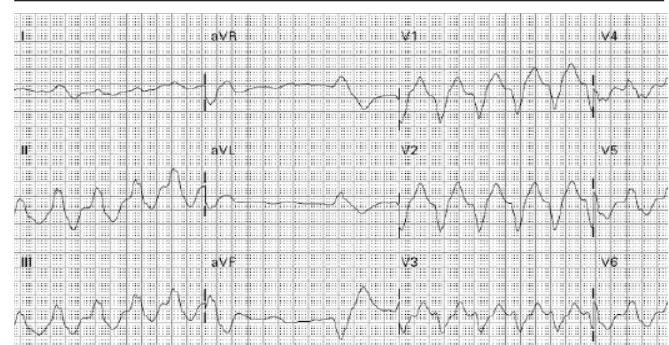
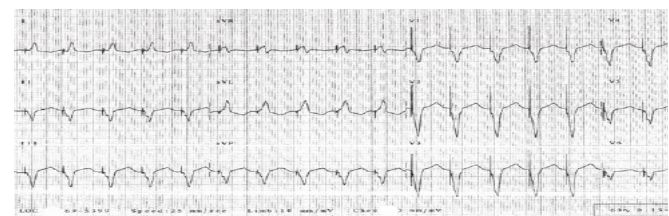


Figure 7: Wide Complex Tachycardia At A Rate Of 115 With Markedly Widened QRS Complexes



This relatively slow rate coupled with the very wide QRS complexes is suggestive of hyperkalemia.

Figure 8. Pacemaker-Mediated Tachycardia



Note the pacer spikes appearing prior to each QRS complex.

WCT From Artifacts

Artifacts are the last category of WCT. ECG artifacts can simulate WCT, especially if the diagnosis is made using a 1–2 lead rhythm strip (Figure 9). For this reason, it is important to acquire a complete 12-lead ECG, if at all possible.

Prehospital Care

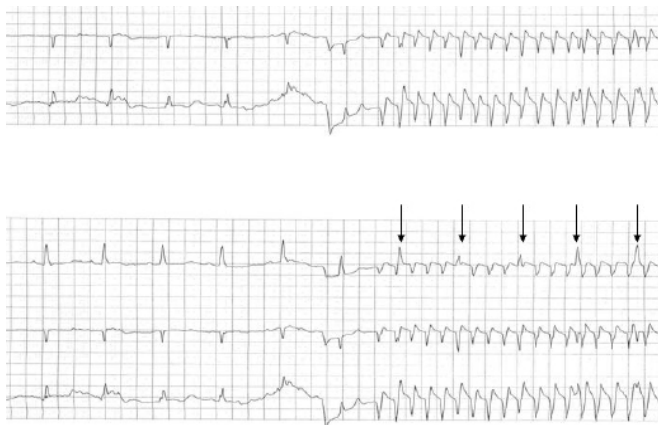
The general goals of prehospital care include recognition of and initiation of therapy for life-threatening conditions while providing rapid transport to the nearest ED. In patients with WCT, prehospital providers should screen for hemodynamic instability and initiate care per basic life support and Advanced Cardiac Life Support (ACLS) protocols consistent with their level of training. As differentiation of WCT can be difficult, additional diagnostic maneuvers or therapeutic intervention is generally not indicated in the hemodynamically stable patient.

ED Evaluation

Vital Signs

It is a common misconception that hemodynamic stability in the setting of WCT suggests a SVT variant while instability confirms VT. Unfortunately, the patient's vital signs and hemodynamic status have little correlation with the etiology of the WCT; therefore, they should not be used to guide management decisions. Of clinical note, pharmacologic interventions effective in the management of VT are not harmful in patients with SVT in most instances, yet the converse is not true. Patients can rapidly decline if "typical SVT medications" are given to the patient with VT (e.g., diltiazem).⁸⁷ Because of this, the clinician should assume a ventricular origin of the wide

Figure 9. ECG Artifact Mimicking VT



Note that the native QRS complexes (arrows) are still evident as it continues to occur within the artifactually-produced wide QRS complexes. This patient was experiencing rigors due to bacteremia related to pneumococcal pneumonia.

complex rhythm in the event of uncertainty. In other words, when choosing a management plan in the setting of undifferentiated WCT, default to a diagnosis of VT and treat accordingly.

Because hemodynamic instability in the setting of WCT does mandate immediate management, continuous telemetry monitoring with frequent blood pressure checks is recommended for all patients.¹²⁻¹⁴ Any of the etiologies listed in Table 1 (except artifact) can result in hemodynamic decompensation.

Focused History

Retrospective univariate analysis suggests that certain historical clues can help distinguish ventricular from supraventricular-based rhythms (Table 2). Additional key questions in the history are noted in Table 3. The historical features listed in both tables have demonstrated statistical significance ($P < 0.01$) for distinguishing between VT and SVT.¹¹ Particularly, a history of MI, a history of CHF, or recent angina pectoris have sufficient predictive value to strongly suggest a diagnosis of VT.¹¹ The definition of "recent angina pectoris" in this study is not clearly defined other than to exclude chest pain at the time of presentation with WCT. In the author's opinion, "recent angina pectoris" probably refers to "recent unstable angina/acute coronary syndrome (UA/ACS)." Given that we

Table 2. Clinical Predictors Of Ventricular Tachycardia

Univariate Predictors	Positive Likelihood Ratio	1/Negative Likelihood Ratio
History of MI	6.6–72	2.2–3.53
History of CHF	Very high*	1.23–1.41
Recent unstable angina	Very high*	1.23–1.41

*The positive likelihood ratios for "History of CHF" and "Recent Unstable Angina" are listed as "very high" because the specificity for both criteria in this small study of 84 patients was 100%. A specificity of 100% results in a positive likelihood ratio of infinity. Adapted from Baerman et al. *Differentiation of Ventricular Tachycardia from Supraventricular Tachycardia with Aberration: Value of the Clinical History.* *Ann Emerg Med.* 1987;16:40-43.

Table 3: Key Questions For History Of Present Illness In The WCT Patient

Key Questions	Implications
History of MI?	Highly favors VT
History of CHF?	Highly favors VT
Recent angina?	Highly favors VT
History of dialysis, end-stage renal disease?	Favors hyperkalemia, digoxin toxicity
History of pacemaker?	Possibly pacemaker-mediated tachycardia
History of ICD?	Favors VT
Ongoing pregnancy?	May change management
Drug ingestions, suicide attempt?	Favors metabolic derangement or VT
Patient's home medications and doses: are they taking any TCAs, anti-arrhythmics, or digoxin?	Favors drug toxicity, though VT should be considered in patients with significant preexisting heart disease

now have very sensitive cardiac enzyme assays (e.g., troponin assays) that can detect very small levels of myocardial necrosis/scarring, “recent UA/ACS” would have better discriminatory power between VT and SVT with AVC than “recent angina” (which can include both chronic stable and unstable angina). Baerman and colleagues also evaluated other clinical variables, including “history of coronary artery bypass grafting (CABG)” and “valvular or congenital heart disease.” Neither of these two criteria had statistical significance in differentiating VT from SVT with AVC.¹¹ While they did not evaluate a “history of percutaneous coronary intervention (PCI),” it is unlikely that this would have been any different than “history of CABG” in its discriminatory power. In the author’s opinion, one possible reason for the lack of predictive capacity for history of coronary revascularization (either CABG or PCI) is that the vast majority of revascularization procedures are performed for chronic angina and not for myocardial infarction/acute coronary syndrome. As elaborated earlier while discussing the pathophysiology of ventricular tachycardia, it is the presence of a myocardial scar (e.g., due to myocardial infarction) that creates the substrate for the vast majority of ventricular tachycardia.^{88,89} Tchou confirmed the utility of a history of MI to predict the correct diagnosis of ventricular tachycardia in a prospective study with electrophysiological study as the gold standard.¹⁵ Unfortunately, the absence of these clinical predictors (listed in **Table 2**) is not very useful (i.e., their absence does not suggest a diagnosis of SVT with AVC).

Unlike the other 3 univariate predictors, age less than 35 years has only mild predictive capacity for SVT with AVC as evidenced by its low likelihood ratios. In the Baerman cohort, the use of age less than 35 years as a diagnostic criteria would have resulted in mislabeling VT as SVT in approximately 10% of cases.¹¹ Since medications that may be safe and effective in SVT can lead to hemodynamic collapse in the setting of VT, younger age by itself should not be considered adequately predictive of SVT to guide management decisions.

Other historical clues can also help in WCT discrimination. These include: 1) history of pacemaker or ICD implantation, 2) prior history of arrhythmia with available ECG, 3) cardiac medications or other medications that are frequently associated with long QT or sodium channel blockade, and 4) history of renal insufficiency and/or dialysis.⁹⁰

Important Examination Findings

Physical examination findings are useful in a minority of patients. The presence of irregular cannon “a” waves in the jugular venous pulse suggests VT.⁷ The irregularity of the cannon “a” waves is a physical representation of the electrocardiographic atrioventricular dissociation that is seen in a minority

of patients with VT.⁷ Similarly, variations in the intensity of the first heart sound (S1) also suggests VT.⁷ Of course, in a hectic ED, these subtle findings may not be readily discernible and therefore of minimal assistance in this distinction.

Other important findings include the presence of a dialysis catheter or arteriovenous grafts. Their presence implies a history of renal insufficiency or failure, which confers a significant risk of hyperkalemia and hypomagnesemia or hypermagnesemia. Similarly, the presence of an implanted device found on examination, might suggest the presence of a pacemaker or ICD and, therefore, pacemaker-mediated tachycardia or VT as the etiology for the WCT.

Laboratory Studies

Rapid evaluation of the patient’s electrolytes, especially the serum potassium and magnesium level, can be very useful. Electrolyte abnormalities can not only trigger ventricular arrhythmias, but can also complicate management by decreasing the success rate of cardioversion/defibrillation. Other laboratory studies that might assist in the diagnosis of WCT are troponin and BNP assays. Because of the urgency involved, laboratory studies that are not available within a 10- to 15-minute turn-around time are generally not that useful in the initial evaluation and management of WCT. Therefore, a venous or arterial blood gas with electrolytes might be the most expedient method of measuring pH and electrolytes (i.e., the potassium) in most institutions.

The Electrocardiogram: Electrocardiographic Rhythm Strips And The 12-Lead Electrocardiogram

While it is ultimately important to differentiate between supraventricular- and ventricular-based WCTs, immediate management decisions should be based on the patient’s hemodynamic status. As previously noted, in patients with hemodynamic instability, the first objective should be immediate electrical cardioversion regardless of the etiology of the WCT with the caveat that WCT due to metabolic or drug toxicities should be considered early as these entities may be resistant to electrical cardioversion.

The First Objective

If the patient is hemodynamically stable, the first objective should be to obtain a paper copy of the 12-lead ECG during the WCT. It can usually exclude artifact as one of the etiologies for the WCT. Many telemetry systems have a high-frequency filter that eliminates the pacing spikes from the rhythms that are displaced on the monitor. A paper copy of the 12-lead ECG can assist in the diagnosis of a pacemaker-related WCT by depicting the pacing spikes before the QRS complex (however, some pacing spikes are very subtle) (**Figure 8**).

The Second Objective

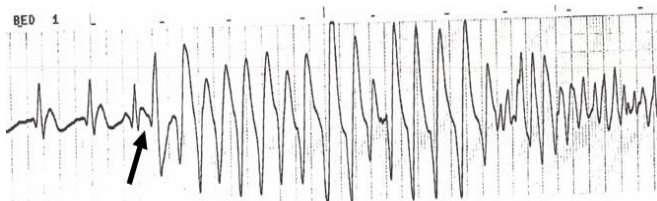
Provided the patient is hemodynamically stable, the second objective should be to locate a prior ECG. If a prior sinus rhythm ECG is immediately available, it helps to discriminate between the different WCT etiologies. A diagnosis of VT is strongly suggested if the wide QRS complexes during WCT have a morphology different from that of the wide QRS complexes during sinus rhythm. If the wide QRS complexes in the WCT are of the same appearance in all 12 leads as seen during sinus rhythm, then a diagnosis of SVT can be cautiously considered (since few exceptions have been reported).¹⁸⁻²⁰ Since none of the studies included enough patients with a baseline ECG, an objective assessment of the discriminatory power of the baseline ECG is unclear. When the QRS complex in the baseline ECG is narrow, it is not as helpful in the diagnosis of WCT.

The Third Objective

The third objective, provided continued hemodynamic stability, is to classify the WCT as either irregular or regular rhythm. An irregular WCT is usually one of 3 arrhythmias: polymorphic VT, AF with AVC, or AF with antegrade conduction down an accessory pathway (AF with preexcitation, AF with WPW). The distinction between polymorphic VT and the other two arrhythmias (both of them being AF) is usually quite straightforward. Apart from being irregular, polymorphic VT typically has the following two characteristics: rate usually > 200 bpm and significant irregularity/variations in the amplitude of the QRS complexes (**Figure 10**).⁹⁶ AF with AVC usually has a rate < 200 bpm and does not have any significant variations in the amplitude of the QRS complexes (**Figure 11**).⁹⁶ Patients with AF with preexcitation can have rates > 200 bpm but usually only have subtle beat-to-beat alterations in the QRS complex morphology and amplitude that are not as varied as polymorphic VT (**Figure 12**). A regular WCT could either be a monomorphic VT, a regular SVT with AVC, regular SVT with preexcitation (antegrade conduction down an accessory pathway), or a pacemaker-related WCT. Toxic- and metabolically-mediated tachycardia can present either as irregular or regular WCT.

Numerous criteria are available to distinguish between the different etiologies of regular and irregular

Figure 10: Sinus Rhythm With ST Segment Elevation In A Patient With Inferior STEMI



Note the appearance of the R-on-T PVC (arrow) and subsequent development of a polymorphic VT.

WCTs using the 12-lead ECG.^{1-4,6,21,22,24} The Brugada criteria might be the most well known algorithm, but it is still infrequently applied by clinicians, likely because it is complex and difficult to remember.⁹¹ Given the need for simplicity, easy recollection, and maximal sensitivity for the diagnosis of VT, especially in the hustle and bustle of emergency care, we recommend the approach taken by Griffith and colleagues.^{3,23} This algorithm has applications in both regular and irregular WCT.

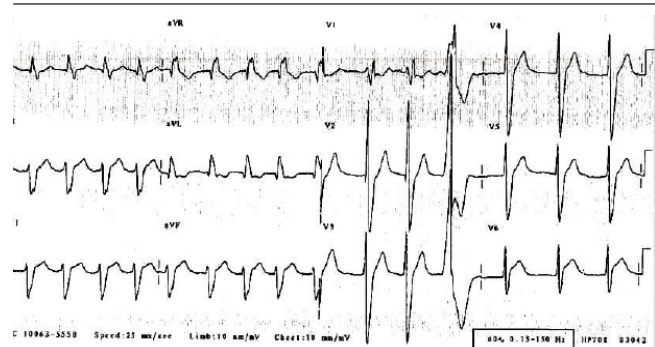
The Fourth & Fifth Objectives

In patients with regular WCT, the fourth and fifth objectives are to apply the Griffith QRS Morphology/Axis criteria to regular WCT and to evaluate for AV dissociation respectively (**Tables 4 and 5**). In patients with irregular WCT, the fourth and fifth objectives are to evaluate if the rhythm is polymorphic VT and apply the Griffith QRS Morphology criteria respectively (**Tables 4 and 6**) (evaluation for QRS axis and AV dissociation is not useful in irregular WCT).

The Griffith QRS Morphology/Axis Criteria (Table 4)

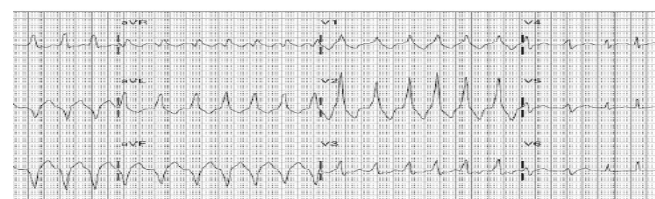
The Griffith QRS morphology criteria has discriminatory power for both regular WCTs and irregular WCTs. Evaluation of the rhythm begins by characterizing the QRS complex as either predominantly positive in V1 or predominantly negative in V1. In WCTs that have a predominantly

Figure 11: Wide Complex Tachycardia With Marked Irregularity



The QRS complex is wide with RBBB morphology. This rhythm is atrial fibrillation with preexisting RBBB.

Figure 12: Wide Complex Tachycardia With A Rapid Rate And Significant Irregularity

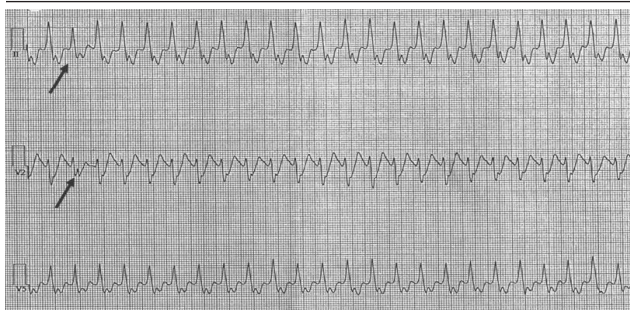


Note the subtle beat-to-beat changes in QRS complex morphology but no significant QRS amplitude variations. This WCT does not have the classic right bundle block appearance which suggests a diagnosis of atrial fibrillation with preexcitation.

positive lead V1, the Griffith approach calls for strict evaluation to determine if the QRS morphology represents a classic right bundle branch block (RBBB, **Table 4**). Based on the work of Wellens et al,¹ the classic RBBB morphology is one in which lead V1 is triphasic with rsR' pattern with ($R' > r$) and (s) representing a negative inflexion below the baseline and V6 demonstrating either an Rs pattern ($R > s$) or a qRs pattern ($q < 40$ ms, $q < 0.2$ mV, and $R > s$). In WCTs that have a predominantly negative V1, Griffith and colleagues would evaluate for strict criteria for left bundle branch block (LBBB). The strict criteria for LBBB include an rS or QS pattern in V1 with time to S wave nadir of less than 70 ms, the r wave that is less than 30 ms ($r < S$), and the V6 lead with an R wave without any Q wave.¹

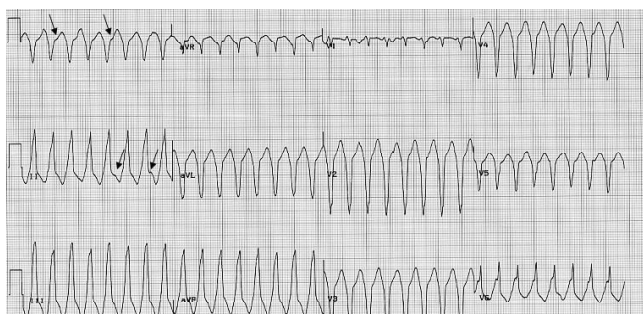
In patients with regular WCT, the next step in the Griffith criteria is analyzing the QRS axis, which is easily done by evaluating leads I and avF. When lead I is more negative than positive, then the axis is either right axis (90–180 degrees) or northwest axis (180–270 degrees). If lead I is more positive than negative, then lead avF should be analyzed. If lead avF is more positive than negative, then the QRS has a right axis; whereas, if it is more negative than positive, then the QRS has a northwest axis. A normal axis (0–90 degrees) or a leftward axis (270–0 degrees) is helpful in the diagnosis of WCT. The QRS axis criteria is not helpful in patients with irregular WCT.

Figure 13: Wide Complex Tachycardia, Likely VT



Note the fusion beat (arrows) suggestive of VT.

Figure 14: Patient #1—Wide Complex Tachycardia With A LBBB Morphology



The morphology in lead V1 is not the classic LBBB appearance and the axis is rightward, both of which support a diagnosis of VT. This is further substantiated by the presence of AV dissociation, best seen here in leads I, II, and V1.


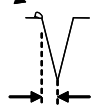


AV Dissociation (Only Useful In Regular WCT)

AV dissociation is characterized by the presence of P waves that “march” through the WCT without a 1:1 association between the P waves and the QRS complex. P waves during WCT are usually best visualized in the inferior leads or lead V1. Rarely, the P waves may conduct through to the ventricle in patients with slow VT resulting in either a capture or a fusion beat.¹⁶ Hence, capture and fusion beats represent sporadic conduction to the ventricle, confirming the presence of AV dissociation (**Figure 13**). According to Wellens et al, the presence of AV dissociation is highly specific for VT.¹ This finding was confirmed by Akhtar et al and Brugada et al.^{4,10} Unfortunately, AV dissociation is only found in 21-36% of VTs.^{4,91} While AV dissociation is essentially pathognomonic for VT, its absence (in the other 69-79% of VT's) does not give any useful information. Not infrequently, AV association (1:1 AV relationship) is noted. Unlike AV dissociation, AV association is not helpful because it can be seen with every etiology for WCT. The finding of AV dissociation is frequently quite subtle and requires careful review of a printed copy of the 12-lead ECG or multi-lead rhythm strip (**Figure 14**).

AV Dissociation: Fusion And Capture Beats

A fusion beat results from a fusion of a supraventricular electrical impulse with a ventricular

Table 4. Summary of Griffith ECG Morphology/Axis Criteria

	Classic RBBB Pattern	Classic LBBB Pattern
	<p>Lead V1: rSR'</p>  <ul style="list-style-type: none"> - $R' > r$ - S cuts baseline 	<p>Lead V1 & V2: QS</p>  <ul style="list-style-type: none"> • Small initial 'r' allowed (< 30 ms width) • Time to S nadir < 70 ms
	<p>Lead V6: RS</p>  <ul style="list-style-type: none"> - $R > S$ - small initial 'q' allowed (< 2 mm depth, < 40 sec width) 	<p>Lead V6: R</p>  <ul style="list-style-type: none"> • NO 'Q' allowed • Either 'RR' or monophasic R
Regular WCT	First, evaluate QRS criteria listed in Step 1. If QRS criteria are met AND axis criteria (in Step 3) are not met AND no AV dissociation is present, then the diagnosis is SVT with AVC. All other WCTs are VT.	
Irregular WCT	First, exclude polymorphic VT. Then, evaluate Step 1 for all other WCTs. If QRS criteria in Step 1 are met, then the diagnosis is AF with AVC. Otherwise, the diagnosis is AF with preexcitation.	
Step 1	Evaluate QRS morphology to see if it meets the above criteria.	
Step 2 (Only if Regular WCT)	If QRS complex meets the above criteria, then evaluate the QRS axis.	
Step 3 (Only if Regular WCT)	If it is classic RBBB pattern, evaluate if it is NW axis (180–270). If a classic LBBB pattern, evaluate if it is RAD or NW axis (90–270).	
Step 4 (Only if Regular WCT)	If Step 3 axis criteria are NOT met, then check for AV dissociation.	

Figures adapted from Lau EW, et al. PACE 2000.²⁴

impulse, producing a QRS complex of variable morphology. A capture beat results in a QRS complex solely from the supraventricular impulse. The resultant QRS complex is usually dissimilar from the other QRS complex structures on the ECG. The finding of either a fusion or a capture beat essentially confirms the diagnosis of VT. Unfortunately, the utility of fusion and/or capture beats is limited because they are rarely seen (0.5% of all cases of VT).⁹¹

Application Of The Griffith QRS Complex Morphology/ Axis Criteria And AV Dissociation To Regular WCT (Tables 4 and 5)

The algorithm proposed by Griffith and colleagues to discriminate between the different etiologies of regular WCT requires the following sequence of analysis: 1) apply the Griffith QRS complex morphology and axis criteria, and 2) evaluate for the presence of AV dissociation.³

A regular WCT that meets the Griffith QRS morphologic criteria for a classic RBBB without a

Table 5. Summary Of ECG Criteria For Regular WCT—The Griffith(I) Algorithm

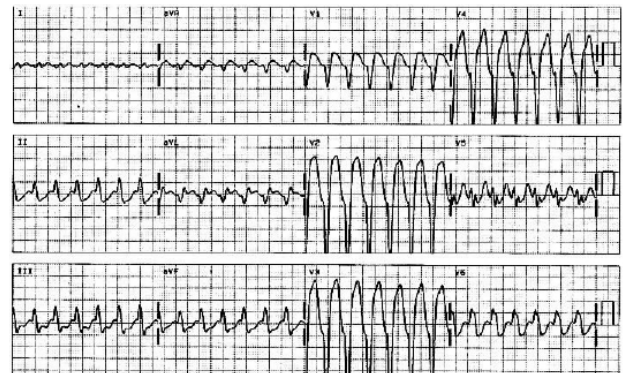
	Step-By-Step Process		Comments
Step 1	The first objective in an unstable patient is to evaluate for hemodynamic instability and treat per ACLS guidelines. The first objective in a stable patient is to acquire a paper copy of the 12-lead ECG and/or 12-lead rhythm strip of WCT. Examine the ECG for pacing spikes at the beginning of every QRS. Consider toxic and metabolic derangement early in the differential diagnosis (obtain arterial or venous blood gas with electrolytes if needed).		A single lead rhythm strip is inadequate for WCT diagnosis.
Step 2	Attempt to retrieve an old/ baseline ECG if possible.		A comparison with an old ECG is very useful.
Step 3	Does the QRS morphology of WCT in leads V1 and V6 match the classic RBBB or LBBB pattern as shown in Table 4 ?		It is unusual for VTs to have QRS morphologies that match typical RBBB or LBBB pattern.
Step 4	Evaluate axis criteria in the WCT with classic RBBB pattern. Is it a NW axis?	For WCTs that match the LBBB pattern, does the QRS have RAD or a NW axis?	If these axis criteria are met, then the WCT is likely VT.
Step 5	Check for AV dissociation or other related phenomenon (fusion, capture beat).	Always check for AV dissociation on paper copy of 12-lead ECG.	The presence of AV dissociation or fusion/capture beats strongly favors VT.
Step 6	If WCT has an exact RBBB pattern or exact LBBB pattern AND does not meet the RBBB axis criteria AND does not meet the AV dissociation criteria → then it is an SVT with aberrancy.		Proceed cautiously.

northwest axis (180–270 degrees) AND that does not demonstrate AV dissociation is highly suggestive of SVT with AVC (**Figure 12**). Similarly, a regular WCT that meets the strict Griffith QRS morphology criteria for a classic LBBB without either a right axis (90–180 degrees) or northwest axis (180–270 degrees) AND does not demonstrate AV dissociation is highly suggestive of SVT with AVC (**Table 5 and Figure 15**).

All other WCTs should be considered VT (**Figures 6 and 14**).³ Hence, any QRS morphology that doesn't meet the classic RBBB or LBBB criteria suggests VT. If V1 demonstrates an equiphasic RS or QR morphology (i.e., equal positive and negative portions of the QRS complex), it strongly suggests VT.¹ When all the precordial leads in a WCT demonstrate concordant QRS morphologies (V1 to V6 being all negative or all positive), it is strongly suggestive of VT (**Figure 16**).^{4,10}

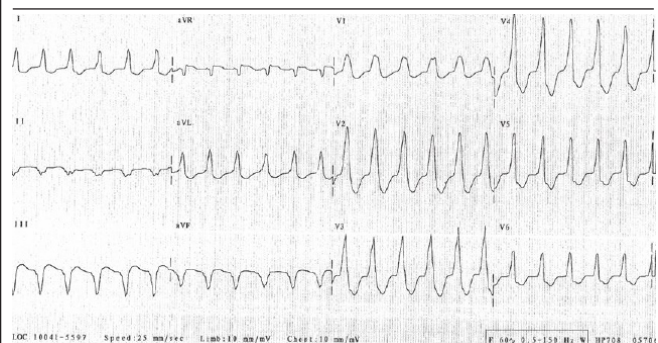
In the authors' opinion, the power of the Griffith approach is its simplicity and its emphasis on making VT the default diagnosis of any WCT during initial management in the ED (**Table 5**). The Griffith approach is simple because only one classic RBBB and one classic LBBB QRS morphology need to be memorized (**Table 4**). By structuring the algorithm so that VT is the

Figure 15. Wide Complex Tachycardia With The Classic LBBB Morphology With A Normal Axis And No Evidence Of AV Dissociation



This rhythm is likely supraventricular in origin—namely atrial flutter with LBBB. Yet, if the clinician is uncertain, therapy aimed at VT is most appropriate.

Figure 16. Wide Complex Tachycardia With A RBBB Branch Block Morphology And Positive QRS Concordance (All QRS Complexes Are Positively Oriented) From Leads V1 To V6



WCT fails the classic RBBB morphology criteria. The patient is diagnosed with VT.

default diagnosis of any WCT, the Griffith approach misclassifies many SVTs with AVC and most SVTs with preexcitation as VTs.³ While this can lead to erroneous long-term care (e.g., candidacy for an implantable defibrillator in patients diagnosed with VT), it promotes safer ED management with drugs that are appropriate in both VT and SVT with AVC. Errors in long-term management can be reduced by obtaining a paper copy of the 12-lead ECG during WCT because it provides a frame of reference for the analysis of an electrophysiologic study in the future. The advantage of misclassifying SVTs with preexcitation as VT is that management for both these categories of WCT is very similar.

When the Griffith approach was applied to a cohort of patients with regular WCT, 4% of patients with VT were misdiagnosed as SVT with AVC.³ While this may seem high, recent analysis of the Brugada approach to regular WCT resulted in a rate of misdiagnosis of VT

that was 9–21% (a much higher rate than the original paper).^{4,91}

Application Of The QRS Morphological Criteria To Irregular WCT (Tables 4 and 6)

The application of the Griffith algorithm for irregular WCT initially requires rhythm evaluation to identify polymorphic VT. Polymorphic VT usually has 2 features allowing prompt identification: rate usually > 200 bpm and significant irregularity/variations in the amplitude of the QRS complexes (Figure 10).⁹⁶ Once polymorphic VT has been excluded, the next step is to distinguish AF with AVC and AF with preexcitation.

Distinguishing between AF with AVC and AF with preexcitation requires evaluation of the QRS morphology.²⁴ No evaluation for QRS axis or AV dissociation is needed. An irregular WCT that meets the Griffith QRS morphologic criteria for a classic RBBB is highly suggestive of AF with AVC (Figure 11). Similarly, an irregular WCT that meets the strict Griffith QRS morphology criteria for a classic LBBB is highly suggestive of AF with AVC (Table 6).²⁴ All other irregular WCTs should be considered AF with preexcitation (Figure 12).

The Griffith QRS morphology approach to irregular WCTs (after the exclusion of polymorphic VT) resulted in 0% rate of misdiagnosis of AF with preexcitation.²⁴ While this approach does lead to a misclassification of a few cases of AF with AVC as AF with preexcitation, this leads to fewer management challenges than the reverse case of misdiagnosis. The distinction between AF with AVC and AF with preexcitation is an important one because AV nodal blocking agents (e.g., beta-adrenergic or calcium channel blockers), which are commonly used in patients with AF with AVC, can lead to hemodynamic deterioration and ventricular fibrillation in patients with AF with preexcitation. On the other hand, agents like procainamide (which are indicated for AF with preexcitation) are also effective in AF with AVC.

Other Diagnostic Modalities for WCT

Advanced Cardiac Life Support guidelines from 1992 recommended the use of adenosine in hemodynamically stable patients with WCT.²⁵ The most recent ACLS guidelines, however, do not support this approach. While adenosine does convert approximately a third of patients with WCT to sinus rhythm, its administration can also lead to angina, bronchospasm, worsening of arrhythmia, and acceleration of accessory pathway conduction as well as degeneration to ventricular fibrillation (VF).²⁶⁻³² Additionally, response to adenosine is also not diagnostically helpful, because it can convert many VTs into sinus rhythm, or it may fail to convert SVTs.⁹⁸ For these reasons, the use of adenosine in the setting of WCT should be limited to patients in whom a presumptive diagnosis of SVT has been made, particularly if vagal maneuvers are not successful in

Table 6. Summary Of ECG Criteria For Irregular WCT—The Griffith(II) Algorithm

	Step-By-Step Process	Comments
Step 1	The first objective in an unstable patient is to evaluate for hemodynamic instability and treat per ACLS guidelines. The first objective in a stable patient is to acquire a paper copy of the 12-lead ECG and/or 12-lead rhythm strip of WCT. Examine the ECG for pacing spikes at the beginning of every QRS. Consider toxic and metabolic derangement early in the differential diagnosis (obtain arterial or venous blood gas with electrolytes if needed).	A single lead rhythm strip is inadequate for WCT diagnosis.
Step 2	Attempt to retrieve an old/baseline ECG, if possible.	A comparison with an old ECG is very useful.
Step 3	Distinguish between polymorphic VT and the other two AF arrhythmias (AF with AVC or AF with preexcitation). <ul style="list-style-type: none"> • PVT: the rate is usually > 200; irregular; pronounced amplitude variation, especially in lead with largest amplitude. • AF: the rate is usually < 200; irregular; no significant amplitude variation. AF with preexcitation can have rates > 200, but it is very unusual to have significant amplitude variation. 	Once PVT has been excluded, use the morphology criteria to distinguish between the two atrial fibrillation arrhythmias (atrial fibrillation with bundle branch block or atrial fibrillation with preexcitation).
Step 3	Does the QRS morphology of WCT in leads V1 and V6 match the classic RBBB or LBBB pattern in Table 4?	It is unusual for atrial fibrillation with preexcitation to have QRS morphologies that match classic RBBB or LBBB.
Step 4	If WCT has an exact RBBB pattern or exact LBBB pattern → then it is atrial fibrillation with preexisting BBB/aberrancy. OTHERWISE → it is a atrial fibrillation with preexcitation.	

termination of the underlying rhythm.

Whenever pacemaker-mediated WCT is considered, a 12-lead ECG before and after placement of a magnet over the device can be very useful.⁴⁴ The 12-lead ECG before magnet application usually demonstrates pacing spikes at the beginning of each QRS complex. Pacemakers are typically programmed to be able to both “sense” (detect native cardiac electrical activity) and “pace” the myocardium. The application of a magnet over a pacemaker prevents it from being able to “sense” native electrical impulses and forces the magnet to pace the heart at a set rate (asynchronous pacing; usually at 60–100 bpm depending on the pacemaker manufacture).^{44,45} The inability to “sense” usually terminates most pacemaker-mediated WCTs as long as the magnet is over the device. This approach is usually successful in sensor-mediated tachycardias, tachycardias with atrial tracking, and endless loop tachycardias. In very rare situations, asynchronous pacing can cause ventricular tachycardia and fibrillation.¹⁰⁰ For this reason, one should have immediate access to the defibrillator and other ACLS interventions while applying a magnet over a device.

General Management Principles

The treatment of WCT varies based on the clinical presentation as well as the rhythm diagnosis. In many instances, a specific rhythm diagnosis is not possible regardless of the diagnostic strategy; thus, the patient’s clinical presentation should direct therapy within the diagnostic realm of the WCT. The first objective of treatment of any patient with WCT (or for that matter any tachycardia) is to evaluate for hemodynamic instability and prepare for interventions (defibrillation, airway management, etc) because any given patient with WCT can deteriorate quickly, and the rhythm can degenerate into VF.

Clinical instability in the WCT patient is manifested by systemic hypoperfusion (hypotension and other evidence of hypoperfusion), pulmonary edema, acute coronary ischemia, altered mentation, or extremely rapid rate. The presence of any of these criteria should prompt consideration for immediate electrical therapy and other recommendations consistent with the current ACLS guidelines;⁴⁸ however, the following caveat applies. Guidelines by their nature are designed for application across a population of patients, and individual patient and system issues may suggest alternative approaches to specific presentations. This is especially true in patients with significant electrolyte or metabolic imbalances, digoxin toxicity, and anti-arrhythmic toxicity where the usual approaches for ventricular tachycardia management are less likely to be successful.^{34,57} Pacemaker-mediated WCT should also be considered early in the course of the ED evaluation

because their management is usually quite different than the other etiologies of WCT.

The early consideration and exclusion of toxic/metabolic causes and pacemaker-mediated WCT leaves SVT with AVC, VT, and preexcited tachycardias as the only remaining etiologies for WCT (other than artifact). The subsequent application of the Griffith approach (**Tables 4 and 5**) to a regular WCT should result in one of two diagnoses: SVT with AVC or VT. The vast majority of cases of preexcited tachycardias will fall in the VT category (which is advantageous because their management is similar). With irregular WCTs, the algorithm described earlier (**Tables 4 and 6**) results in one of three diagnoses: polymorphic VT, AF with AVC, and AF with preexcitation.

Toxic- And Metabolically-Mediated WCT

Ventricular arrhythmias are often resistant to cardioversion or defibrillation in cases of drug toxicity.^{34,57} Because patients can present with hemodynamic instability or frank cardiogenic shock, inotropes, pressors, and an intra-aortic balloon pump are not infrequently required.⁵⁷ In cases of regular WCT secondary to class I anti-arrhythmic toxicity (e.g., flecainide, propafenone), sodium bicarbonate is a very useful medication. Because sodium channel dysfunction is typical of toxicity-related regular WCT, administration of sodium bicarbonate can cause narrowing of the QRS complex and termination of the dysrhythmia.^{58,59} The management of toxic- or electrolyte-induced polymorphic VT is similar to other etiologies of polymorphic VT (described later).

Reported effective dosage of sodium bicarbonate is variable, ranging between 200 and 450 meq, approximately 4–9 “ampules” of sodium bicarbonate.^{58,59} Animal data would suggest that to obtain adequate clinical effect in cases of flecainide or encainide toxicity, more than 3 meq/kg per dose of sodium bicarbonate may be required.^{58,60} Both lidocaine and amiodarone have been reported as effective alternatives to sodium bicarbonate in cases of flecainide toxicity.^{61,62} The usual lidocaine dose is 1–1.5 mg/kg IV bolus with repeat bolus of 0.5–1 mg/kg IV in 5–10 minutes up to a total of 3 mg/kg; a continuous infusion of 1–4 mg/min is then started. The dosage of amiodarone used in cases of flecainide toxicity has been the pulseless VT/VF dose of amiodarone: 300 mg IV bolus in 20–30 mL of NS or D5W with repeat bolus of 150 mg IV as needed, and a continuous infusion of 1 mg/min for 6 hours, then 0.5 mg/min.

Sodium bicarbonate is also valuable in the treatment of TCA toxicity.^{37,101} The initial dose is 1–2 meq/kg (2–3 100 mL ampules of 8.4% sodium bicarbonate) given as a rapid IV bolus. It is useful to run a continuous 12-lead ECG during the infusion, to demonstrate any changes in the dysrhythmia or narrowing of the QRS complex. Supplemental boluses are frequently useful to achieve a pH of 7.5–7.55. The use of sodium bicarbonate should not detract from the

need for volume replacement in patients with TCA toxicity and hypotension.³⁷

The treatment of hyperkalemia involves the use of membrane-stabilizing agents (calcium), transient-shifting agents (sodium bicarbonate, albuterol, insulin, dextrose, magnesium sulfate), and removal agents (polystyrene binding resins and hemodialysis).

Pacemaker-Mediated WCT

The management and diagnostic strategy when faced with a WCT that is likely pacemaker mediated is to obtain a 12-lead ECG before and after the magnet placement. The application of a magnet will terminate most pacemaker-mediated WCTs with the exception of the runaway pacemaker. Fortunately, runaway pacemakers are extremely rare in the current generation of pacing systems. Comprehensive management of a pacemaker-mediated WCT involves interrogation and reprogramming of the device.⁴⁴ This usually necessitates contacting either the on-call technical representative of the device manufacturer or the cardiologist or electrophysiologist on call.

Regular WCT: VT And Preexcited Tachycardia

The Unstable Patient

The first objective in the management of these WCTs is the evaluation for hemodynamic instability. Synchronized electrical cardioversion is the treatment of choice in the unstable patient.⁴⁸ If recurrent ventricular arrhythmias are noted in the unstable patient, additional electrical cardioversion is indicated. Patients with shock-resistant or recurrent pulseless VT (VF or cardiac arrest) should receive an amiodarone bolus of 300 mg IV with an additional bolus of 150 mg IV as needed and a continuous infusion at 1 mg/min for 6 hours and then 0.5 mg/min for 18 hours. In patients who have recurrent VT or preexcited tachycardia with signs of hypoperfusion (but do have a pulse), amiodarone should be given at a dose of 150 mg IV; an additional bolus of 150 mg IV can be given followed by a continuous infusion at 1 mg/min for 6 hours and then 0.5 mg/min for 18 hours. Lidocaine is an alternative to amiodarone and is dosed at 1–1.5 mg/kg IV, with supplemental doses of 0.5–0.75 mg/kg IV (max dose of 3 mg/kg) in cases of recurrent pulseless VT (VF or cardiac arrest). In cases of recurrent unstable VT or preexcited tachycardia (with a pulse), the dose of lidocaine is 0.5–0.75 mg/kg IV with supplemental doses as needed. Lidocaine is then given as a continuous infusion at a dose of 1–4 mg/min intravenously.

The Stable Patient

Even in stable patients with VT or preexcited tachycardia, synchronized cardioversion should be immediately available because anti-arrhythmic failures and hemodynamic decompensation can occur. In these patients, procainamide is the drug of choice.⁴⁹ Procainamide will terminate the rhythm in the vast majority of cases (77% termination rate in stable VTs),⁵¹

and it is superior to amiodarone (30% termination rate in stable VTs)⁵² and lidocaine (27% termination rate in stable VT)⁵¹ in patients with stable VT.^{52,53} Not only is it more effective in the termination of stable VT, but procainamide also blocks accessory pathway conduction which terminates preexcited tachycardias.

Procainamide infusion requires frequent blood pressure monitoring and continuous ECG monitoring. Prior to administering procainamide, the QRS complex duration of the WCT should be measured in the lead with the widest QRS. Procainamide should be given intravenously until either the arrhythmia terminates or one of the following criteria is met: hypotension (SBP < 90), prolongation of the QRS complex duration by 50% compared with baseline duration, acceleration of the tachycardia, or a total of 1 gm administered. We recommend the procainamide dosing protocol used by Gorgels and colleagues that requires only 10 minutes for their maximum dose of 10 mg/kg.⁵¹ These investigators administered procainamide at 100 mg/min until a maximum of 10 mg/kg or one of the previously mentioned criteria are reached. This approach is attractive because it allows a more rapid loading of procainamide, although the accelerated regimen (compared with the ACLS regimen) may increase the risk of adverse effects. The ACLS dosing recommendation is an infusion of 20 mg/min (until a maximum dose of 17 mg/kg) until either the arrhythmia terminates or one of the previously mentioned criteria is met.⁴⁸ The dosing of the maintenance infusion is 1–4 mg/min diluted in normal saline or D5W with dosing at the lower end of the range in patients with renal insufficiency.

In patients with stable VT or preexcited tachycardia, amiodarone is given as an intravenous dose of 150 mg over 10 minutes with a maintenance infusion of 1 mg/min for 6 hours and 0.5 mg/min thereafter. Supplemental doses of 150 mg over 10 minutes may be given as needed. Lidocaine is less efficacious than either procainamide or amiodarone.^{51-53,55,56} In addition, lidocaine is not significantly effective in the treatment of preexcited tachycardias. When used for the treatment of stable VT, the dosing for lidocaine is an IV bolus of 0.5–1.5 mg/kg over 2 minutes with an infusion of 1–4 mg/min. Supplemental doses of 0.5–0.75 mg/kg may be given every 5–10 minutes to a maximum total dose of 3 mg/kg.^{48,49,51}

Careful cardiovascular monitoring is recommended in all patients receiving anti-arrhythmic medications, especially in those with heart failure symptoms or known left ventricular dysfunction.⁴⁸ Synchronized cardioversion is the next treatment of choice in the event of anti-arrhythmic failure or if the patient becomes unstable.

The management of a patient with a hemodynamically stable WCT in the setting of ACS is similar to patients without ACS; the only caveat being that the threshold for using cardioversion to terminate arrhythmia should be lower. Urgent cardiology

consultation is recommended so that early coronary angiography and revascularization can be facilitated.

Regular WCT: Supraventricular Tachycardia With Aberrant Ventricular Conduction

As with all WCT patients, initial evaluation and management decisions should be based on the patient's hemodynamic status. As described earlier, this group of patients includes those with SVT with either preexisting bundle branch block or transient functional bundle branch aberrancy. If instability is noted, immediate electrical cardioversion should be initiated.

Patients with SVT with AVC should be distinguished from those with preexcited tachycardias because the latter group of patients shares many features with ventricular tachycardia and should be treated as such (as described in the section titled: "Regular WCT: VT And Preexcited Tachycardia"). Patients with preexcitation should not receive AV nodal agents because this can precipitate ventricular tachycardia or ventricular fibrillation. The benefit of the Griffith algorithm is that most preexcited tachycardias get classified as ventricular tachycardia, which decreases the risk of inadvertent administration of AV nodal blocking agents to these patients.

In patients with SVT with AVC, AV nodal blocking agents are the drugs of choice after attempts at vagal maneuvers (either carotid sinus massage or the Valsalva maneuver). In this setting, intravenous adenosine is the treatment of choice. The initial dose is 6 mg bolus, followed by saline IV flush, which can be followed by a 12 mg bolus dose if no clinical effect is seen within a few minutes (a second 12 mg bolus dose can be given if needed). If a clinical effect is seen with adenosine, it is either a conversion to sinus rhythm or a continuation of the SVT with a transient AV nodal block. If adenosine is unsuccessful in converting the rhythm, the next drugs to consider are calcium channel or beta-adrenergic blocking agents. Diltiazem is usually given as a bolus dose of 10–20 mg (0.25 mg/kg) IV over 2 minutes; a repeat bolus intravenous dose can be given in 15-minute increments of 20–25 mg (0.35 mg/kg). A maintenance infusion of 5–15 mg/hr can then be started as needed or, alternatively, the patient may be loaded with oral diltiazem if the initial bolus dose effectively controls the heart rate. The usual beta-blocking agents used are atenolol, metoprolol, and esmolol. Atenolol is given as a slow 5 mg IV dose (over 5 minutes); a second dose of 5 mg slow IV (over 5 minutes) can be given after 10 minutes if the first dose is well tolerated. The metoprolol dose is 5 mg slow IV push at 5-minute intervals to a total of 15 mg. Intravenous esmolol is given as an IV loading dose of 0.5 mg/kg over 1 minute, followed by a 4-minute infusion of 0.05 mg/kg/min for a total of 0.2 mg/kg. If the effect is inadequate, a second loading dose can be given, with a subsequent increase in the maintenance infusion to 0.1 mg/kg/min (maximum

infusion rate of 0.3 mg/kg/min). Side effects of diltiazem and the aforementioned beta-blocking agents include bradycardias, hypotension, and pulmonary edema. Therefore, careful cardiovascular monitoring is important, especially when bolus doses are given.

Irregular WCT: Polymorphic Ventricular Tachycardia

Sustained polymorphic VT (PVT) typically degenerates into VF. Therefore, the provider should be prepared to treat VF. According to ACLS guidelines, the immediate goals are to provide oxygen, obtain a 12-lead ECG, and prepare for more aggressive resuscitation.

Recurrent salvos of nonsustained polymorphic VT is not an infrequent presentation. These patients should be treated with direct current (DC) cardioversion if unstable. The management of hemodynamically stable patients with single or recurrent nonsustained polymorphic VT should begin with the evaluation of the QT interval on the 12-lead ECG.⁴⁹ This QT interval distinguishes between torsades de pointes (i.e., polymorphic VT with a prolonged QT interval) and polymorphic VT with normal QT interval.

In patients with a normal QT interval ($QTc < 450$ ms), ongoing myocardial ischemia is the most frequent etiology.^{49,102} Hence, the treatment in patients with a single or recurrent nonsustained polymorphic VT with a normal QT interval usually involves the use of beta blockers, amiodarone, and early cardiac catheterization.⁴⁹ Lidocaine is considered a second line anti-arrhythmic agent.⁴⁹ As with torsades de pointes, these patients should have rapid assessment of serum electrolytes with particular attention to repletion of potassium and magnesium. Beta-blockade can be accomplished with the following: 1) IV propranolol 0.15 mg/kg over 10 minutes and then 3–5 mg q6hours, 2) esmolol 300–500 mg/kg load over 1 minute and then 25–50 mg/kg/min, and 3) metoprolol 5 mg IV every 5 minutes for 3 doses and then 50 mg orally every 6 hours.⁵⁴ Intravenous amiodarone is administered as a 150 mg intravenous bolus with a maintenance infusion of 1 mg/min for 6 hours and 0.5 mg/min thereafter. Supplemental doses of 150 mg amiodarone can be given as needed. Lidocaine is dosed as an IV bolus of 0.5–1.5 mg/kg over 2 minutes with an infusion of 1–4 mg/min. Supplemental doses of 0.5–0.75 mg/kg may be given every 5–10 minutes to a maximum total dose of 3 mg/kg.

Patients who have polymorphic VT with a long QT interval are deemed to have torsades de pointes. Patients with single or recurrent salvos of nonsustained torsades de pointes should be treated with a 2 gm bolus dose of IV magnesium sulfate with repeat boluses and infusion as needed. These patients should have a rapid evaluation of their electrolytes. Since the QT interval shortens with faster heart rates in most patients, treatments that increase the heart rate can suppress recurrent episodes of torsades de pointes. Hence, urgent transvenous

pacing can be very useful in these patients, especially in patients with significant bradycardia (e.g., sinus bradycardia, heart block); the goal ventricular rate is 100 bpm.¹⁰² Since transcutaneous pacing can be quite painful, it should be performed with the patient sedated as a bridge to transvenous pacing. If pacing is not immediately available, isoproterenol at 1–4 mcg/min can be considered to achieve a heart rate of 100 bpm. Isoproterenol should be used with the caveat that it be avoided in patients with significant hypertension, suspicion of myocardial ischemia, or history of congenital long QT syndrome. By increasing the sympathetic activity, isoproterenol can potentiate life-threatening arrhythmias in patients with myocardial ischemia and congenital long QT syndrome. Intravenous lidocaine is another useful option in patients with torsades de pointes.^{49,102} It is dosed as an IV bolus of 1–1.5 mg/kg over 2 minutes with an infusion of 1–4 mg/min. Supplemental doses of 0.5–0.75 mg/kg may be given every 5–10 minutes to a maximum total dose of 3 mg/kg.¹⁰² Unlike patients with monomorphic VT and polymorphic VT with normal QT interval, amiodarone should be avoided in patients with torsades de pointes because it can further prolong the QT interval and worsen the situation.

Irregular WCT: AF With Preexcitation

One arrives at the diagnosis of AF with preexcitation by excluding polymorphic VT as a potential diagnosis and then applying the Griffith algorithm (**Tables 4 and 6**) to irregular WCTs to distinguish it from AF with AVC. The treatment options for these patients include DC cardioversion, procainamide, and amiodarone (see “Regular WCT: VT And Preexcited Tachycardias”).

Irregular WCT: AF With AVC

These patients are treated similar to other patients with SVT with AVC (see “Regular WCT: SVT With AVC”).

Special Circumstances

Wide Complex Tachycardia In The Pediatric Patient

The process of diagnosis and acute management of WCT in the pediatric patient is similar to the adult patient. Diagnostically, age-related differences in rate and QRS complex duration must be considered. Therapeutically, the primary differences between the two populations includes alterations in the drug dosages of anti-arrhythmic drugs and energy required for synchronized cardioversion (0.5–1 joules/kg) or, if pulseless, defibrillation (2–4 joules/kg).⁶³

Acute management of WCT in a pediatric patient depends on the patient’s age and hemodynamic status.⁶⁴ When faced with shock or cardiovascular collapse, immediate synchronized cardioversion with 0.5–1 joule/kg is necessary. In more stable situations and if a diagnosis of SVT is made, 0.1–0.2 mg/kg of adenosine can be given in rapid boluses to induce

transient AV block.⁶³ If VT is the diagnosis, amiodarone, procainamide, or lidocaine are all acceptable therapeutic agents.

The Adult Patient With Congenital Heart Disease

The evaluation and management of WCT in patients with ‘simple’ adult congenital heart diseases, such as atrial septal defects and un-operated small ventricular septal defects, are not significantly different from the standard approach. Complex adult congenital heart disease, however, does merit additional consideration. The largest body of information is available for post-operative tetralogy of Fallot.

Supraventricular arrhythmias are a frequent complication in patients who have a surgical correction of their tetralogy of Fallot.⁶⁵ The supraventricular rhythms in these patients are typically RBBB in morphology.⁶⁵ Therefore, if a post-operative tetralogy of Fallot patient has a WCT with an LBBB morphology, the dysrhythmia is likely VT.⁶⁵

The Pregnant Patient With Wide Complex Tachycardia

The major concern regarding anti-arrhythmic drug therapy during pregnancy is the potential adverse effects on the fetus.⁶⁶ None of the anti-arrhythmic drugs available are FDA category A for use in pregnancy. Among the anti-arrhythmics commonly used, only lidocaine and sotalol are category B. Of note, amiodarone, which is the most commonly used anti-arrhythmic in the treatment of WCT in non-pregnant patients, is a category D drug in pregnancy. Other than phenytoin (FDA category X), the vast majority of the remaining anti-arrhythmics are FDA category C.

SVT and VT can arise for the first time during pregnancy or become more frequent during this period.⁶⁷ At least two groups of investigators have noted an increased incidence of arrhythmias associated with an accessory pathway during pregnancy.^{68,69} Similar to non-pregnant patients, the 12-lead ECG is an important tool in the diagnosis of WCT. The diagnostic strategies used to discriminate between the different etiologies of WCT are similar to the non-pregnant patient. When a pregnant patient presents with new onset WCT in the last few weeks of pregnancy or within 6 months of delivery, the possibility of peripartum cardiomyopathy should be considered.

Regardless of the etiology, if a pregnancy-associated WCT becomes hemodynamically unstable, DC cardioversion of 50–100 J should be considered. If unsuccessful, higher energies should be used (100–360 J). DC cardioversion is considered safe in all stages of pregnancy with no significant fetal complication.⁷⁰⁻⁷² As with other pregnancy-associated disease processes, “stabilize the mother and you will stabilize the fetus.” It is necessary, however, to monitor the fetal rhythm if possible, because transient fetal arrhythmia has been reported during DC cardioversion in pregnancy.⁷³ As fetal monitoring is not possible in most EDs, specialty consultation is advised, but management decisions

should not be delayed if the patient becomes unstable. When necessary, cardiopulmonary resuscitation (CPR) should be performed with the pregnant patient tilted on her side, with either a wedge or another rescuer's knees for support.⁷⁴

In the hemodynamically stable pregnant patient with undifferentiated monomorphic WCT, diagnosed VT, or preexcited tachycardia, initial therapy should be intravenous procainamide (FDA category C).^{75,76} Procainamide has been used with no evidence of teratogenicity.⁷⁷ The next drug of choice is lidocaine (FDA category B) in patients with VT or undifferentiated WCT (it is not effective in preexcited tachycardias). Given the category B status of lidocaine, one could consider using lidocaine before procainamide in a pregnant patient with VT. Data from non-pregnant patients, however, does demonstrate a marked superiority of procainamide over lidocaine in the acute termination of hemodynamically stable VT (80% vs. 21%).⁵¹ Use of lidocaine in the early stages of pregnancy is not teratogenic.⁷⁵ Amiodarone (FDA category D) is of limited value in pregnancy; it is associated with many serious side effects for the fetus, including hypothyroidism, growth retardation, and premature delivery.⁷⁸ Hence, it should be reserved for life-threatening and refractory conditions.⁷⁹

In the pregnant patient with polymorphic VT, intravenous magnesium can be very useful, similar to its use in the non-pregnant patient. It is particularly effective in patients with torsades de pointes. The dosage is 2 g intravenously with supplemental doses and an infusion as needed. Adverse impacts are rare and include maternal hypothermia, fetal bradycardia, respiratory depression, and hypotonia in the newborn which may require aggressive measures.⁸⁰

Initial treatment of the stable patient with SVT with AVC should start with vagal maneuvers to terminate the arrhythmia. If this fails, adenosine (FDA category C) should be used. It is not known to be teratogenic and it is as effective in terminating SVT (> 90% successful) in pregnant patients as it is in those not pregnant.^{76,82} While information about the use of diltiazem (FDA category C) in pregnancy is limited, verapamil (FDA category C) has a history of safe use in pregnancy.¹⁰³ One retrospective analysis suggested a risk of birth defects associated with its use in the first trimester.⁸³ Beta blockers have been widely used in pregnancy for a variety of indications. Propranolol (FDA category C) has been extensively used but has been associated with a small risk to the fetus.⁷³ Labetolol and metoprolol are also frequently used, but both are category C. While atenolol is category D, two new beta blockers (pindolol and acebutolol) are category B.¹⁰³

Stable pregnant patients with AF with AVC should be managed with beta-blockers, diltiazem, or verapamil to achieve rate control similar to non-pregnant patients. Early DC cardioversion or chemical cardioversion should be considered (within 48

hours) to avoid the need for anticoagulation.¹⁰³ While quinidine (FDA category C) has a long history of safe use in pregnancy, chemical cardioversion with other anti-arrhythmic drugs (e.g., flecainide, propafenone, ibutilide, procainamide) has also been reported in pregnancy patients.^{81,103}

Disposition

The ECG is only a test and in isolation does not provide adequate information for the definitive management of all WCTs. As is true of most syndromes in clinical medicine, WCTs present across a spectrum of severity, ranging from non-worrisome to life threatening. Generally, the two extremes of this spectrum are easily identified: 1) the patient with sinus tachycardia and preexisting BBB demonstrates a WCT in the setting of mild volume depletion from gastroenteritis, and 2) the patient with ventricular tachycardia with hemodynamic compromise manifested by hypotension and pulmonary congestion. While the ECG provides little clinically relevant data in the first scenario (other than confirming sinus tachycardia), in the second case, the ECG can be very helpful in long-term management, even though it doesn't change short-term management (electrical cardioversion). Again, as is true in much of clinical medicine, the middle ground along this spectrum of severity can be difficult to sort out.

In general, most instances of ventricular tachycardia should be admitted to the hospital. Similarly, patients with preexcited tachycardias likely require hospital admission for prompt evaluation and management because these patients are at risk for sudden death. Cases of SVT with AVC or AF with AVC must be judged on an individual basis. For instance, the patient with recurrent AF and BBB may only require rate control. Assuming other underlying medical issues do not exist, initial management would focus on rate control which, if successful, may enable the patient to be discharged from the ED. In summary, disposition of the WCT patient should be driven by consideration of the underlying medical issues specific to each case and not by the presence of a WCT alone.

Case Conclusions

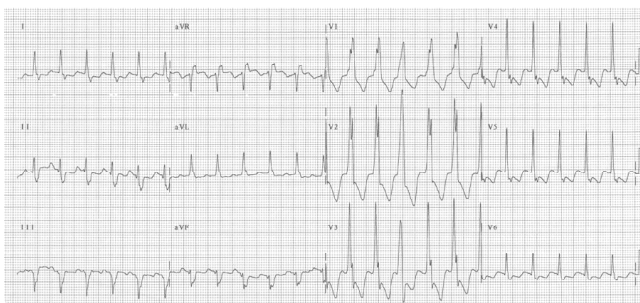
Patient #1, a 67-year-old male with MI and CHF, demonstrates a WCT. In the prehospital setting, the patient was stable or, more appropriately phrased, not unstable. Unfortunately, he has deteriorated upon arrival. He is lethargic with a BP of 70 mmHg by palpation. The monitor demonstrates the WCT as seen in Figure 1; note the appearance of deflections and irregularities in the QRS complexes—likely representing AV dissociation. A 12-lead ECG of this WCT is shown in Figure 14. The Griffith algorithm reveals a right axis deviation with a left bundle branch morphology WCT and AV dissociation, all of which lead to the diagnosis of VT. You rapidly assess him, noting his significant instability. He is sedated with etomidate

and cardioverted at 100 joules to sinus tachycardia and an improved hemodynamic picture. The 12-lead ECG subsequently performed does not demonstrate ACS-related abnormalities; the serum marker analysis is normal. He is admitted to the CCU.

Patient #2, the elderly female with a hemodynamically stable WCT, demonstrates a wide complex rhythm. Her examination is remarkable only for the rapid rate. The 12-lead ECG (Figure 17) reveals a WCT with RBBB morphology at a rate of 150 bpm that does not meet the Griffith RBBB criteria (Table 4). This leads you to a presumptive diagnosis of VT. During your history, the patient notes a history of atrial flutter. You review her prior ECGs, one of which demonstrates sinus rhythm with an identical RBBB morphology as the one today. The other ECG demonstrates atrial flutter with 4:1 conduction (a rate of 75 bpm) with an identical RBBB morphology as the WCT today. She is cautiously diagnosed with atrial flutter with rapid ventricular response (with 2:1 AV conduction) and preexisting bundle branch block. She receives intravenous diltiazem and is admitted to the telemetry unit for further monitoring.

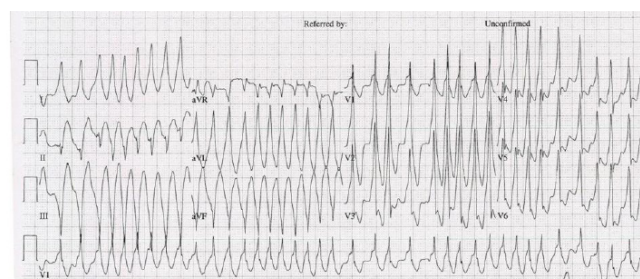
Patient #3, a 19-year-old male with palpitations and weakness, is more problematic. He is somewhat ill-appearing, yet his BP has improved spontaneously to 105/79 mm Hg. The monitor continues to demonstrate an irregular WCT (Figure 3) with the 12-lead ECG revealing a similar finding (Figure 18). Review of the rhythm lead V1 (below the 12-lead ECG in Figure 18) demonstrates no significant

Figure 17. Patient #2—Wide Complex Tachycardia Which Is Regular At A Rate Of 150 bpm



Note the RBBB morphology with the rR' QRS complex configuration in lead V1.

Figure 18. Patient #3—Irregular Wide Complex Tachycardia With A Ventricular Rate Of 230 bpm

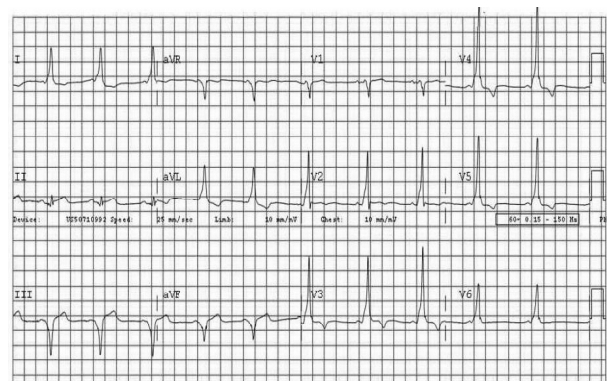


Note the varying configurations of the QRS complexes in any single lead but no significant variation in the amplitude of the QRS complexes. The WCT does not meet the classic RBBB criteria, and the likely diagnosis is atrial fibrillation with preexcitation.

QRS amplitude variation, making polymorphic VT quite unlikely. The Griffith criteria (Tables 4 and 6) lead you to the conclusion that this irregular WCT does not meet the classic RBBB morphology criteria. Hence, you diagnose this patient with atrial fibrillation with preexcitation (WPW syndrome). Due to his apparent stability, the patient receives intravenous procainamide over 40 minutes. He is monitored very closely during this infusion. Despite a full loading dose of procainamide, he continues to demonstrate the WCT. Thus, you administer etomidate and cardiovert this gentleman at 100 J to sinus rhythm with evidence of ventricular preexcitation (Figure 19). He experiences no further issue other than a brief period of myoclonic movement resulting from the etomidate administration. He is admitted to the hospital for further evaluation, including electrophysiologic study.

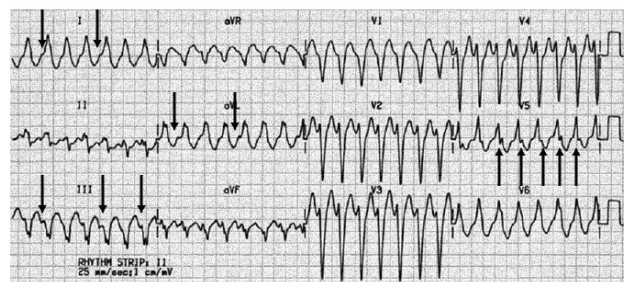
Patient #4, the 71-year-old male transfer patient from the primary physician's office with WCT, arrives as described. He has normal vital signs with the exception of the pulse, which remains in the 180 bpm range. He notes only palpitation and denies chest pain, dyspnea, or other complaint; his medical history is significant for angina, myocardial infarction, and hypertension. The ECG rhythm strip (Figure 4) and 12-lead ECG demonstrate a WCT (Figure 20). Because it demonstrates AV dissociation

Figure 19. Patient #3—Normal Sinus Rhythm With PR Interval Shortening



The initial slurring of the QRS complex (delta wave) and QRS complex widening is consistent with preexcitation seen in Wolff-Parkinson-White syndrome.

Figure 20. Patient #4—Regular Wide Complex Tachycardia With A Ventricular Rate Of 170 bpm



It has a LBBB morphology that fails the Griffith criteria (it has an initial r in V2 that is at least 40 ms in width). It also demonstrates AV dissociation (seen in leads I, III, avF, avL, V5) (arrows)—this finding is suggestive of VT.

Risk Management Pitfalls For Wide Complex Tachycardia

- 1. "Look at this rhythm strip. It's a narrow complex tachycardia. We have an SVT on our hands."**
Avoid making a diagnosis of narrow complex tachycardia by one or two lead rhythm strips alone. Only when the QRS is narrow in **all** 12-leads can a classification of narrow complex tachycardia be used. Many wide QRS tachycardias have a narrow QRS complex in a few leads (because a portion of the QRS is isoelectric in those leads). In the unstable patient with WCT, DC cardioversion is the treatment of choice. The management of stable patients with WCT will be greatly facilitated by a 12-lead ECG that demonstrates the tachycardia.
- 2. "That looks like VT on the telemetry monitor. Get the paddles."**
Avoid making a diagnosis of VT vs. SVT with only a rhythm strip or by monitoring the rhythm on a telemetry screen. If the patient is unstable, then "getting the paddles" is the correct next step (regardless of whether the rhythm is a VT or SVT with AVC). A stable patient will benefit from a paper copy of the 12-lead. Many of the specific signs for VT are quite subtle (e.g., AV dissociation) and need a few minutes of your time with the paper copy of the ECG. Since the specific diagnosis (SVT with AVC or VT) can never be made with 100% certainty in every case, a paper copy of the tachycardia is very useful in the long-term management of the patient.
- 3. "He looks very good. It's probably an SVT."**
Avoid the assumption that a patient with stable vital signs or minimal symptoms could only have SVT. The literature is also littered with cases of wide complex tachycardias where the incorrect diagnosis of SVT was made partly because of "how well the patient looked."
- 4. "This is SVT with aberrancy. See, it meets all the QRS morphological criteria on the 12-lead ECG."**
No single electrocardiographic criterion or combination of criteria are adequate to distinguish between SVT and VT. While the Griffith or Brugada algorithms are quite accurate, neither of them is 100% accurate. The patient's history, physical examination, and laboratory data can be very helpful. Therefore, ask a stable patient regarding any history of MI, CHF, recent unstable angina, complex congenital heart disease, and allergies.
- 5. "This is VT. SVT with aberrancy never looks this bizarre."**
Do consider electrolyte abnormalities and drug toxicity in the differential. While the timely treatment of VT with cardioversion is quite important, it can sometimes be ineffective in patients with metabolic derangement, especially hyperkalemia or toxic ingestion. If possible, question patients and their families regarding drug ingestion, and check laboratory studies for electrolyte levels.
- 6. "She has had an SVT before. This wide complex tachycardia must be SVT with AVC. See if we can slow it down with some diltiazem or verapamil."**
Avoid the use of calcium channel blockers in patients with wide QRS tachycardia unless you are reasonably confident that the current presentation (and today's 12-lead ECG) demonstrates SVT with AVC. Calcium channel blockers can precipitate cardiovascular collapse if given to a patient with VT.
- 7. "That's a WCT. See if adenosine works."**
Avoid the use of adenosine as a tool to distinguish SVT from VT. It is not very useful as a diagnostic tool because adenosine can terminate many VTs and have little impact on many SVTs. Moreover, its use is not without risk. There are multiple reports of adenosine-induced coronary steal and conversion of WCT to ventricular fibrillation.
- 8. "He just had CABG two-months ago. He has significant coronary disease. This must be VT."**
The presence of coronary disease or history of revascularization (percutaneous or surgical) is not a predictor of VT. The vast majority of revascularization procedures in the United States are performed for stable angina (not unstable angina/myocardial infarction). Only a history of myocardial infarction, known left ventricular dysfunction, or recent unstable angina is predictive of VT.
- 9. "I think this is VT, but I can't believe that she is only 36-years old. Since she's stable, let's give her some amiodarone and stop the VT."**
As with most clinical syndromes in medicine, the diagnosis of pregnancy has the potential to significantly alter management. Do remember to check a pregnancy test in women of child-bearing potential. In a stable patient (even if she was pregnant), procainamide should be considered the anti-arrhythmic of first choice in a patient with stable monomorphic VT. This is especially true in pregnancy because of the need to avoid amiodarone exposure to the fetus (FDA category D).
- 10. "He has had a surgical repair for his tetralogy of Fallot. His WCT is most likely VT."**
Supraventricular arrhythmias with preexisting bundle branch block are quite common in patients with complex congenital heart disease. A history of complex congenital heart disease is not a predictor for VT.

(including capture/fusion beats) and doesn't meet the Griffith criteria for classic LBBB (the "initial r" in V2 is at least 40 ms in width), you diagnose the patient with VT. His history of MI further supports your diagnosis. Due to his stability, he receives intravenous amiodarone over 10 minutes without effect; a repeat infusion of amiodarone results in a conversion to sinus tachycardia (procainamide would also have been a very reasonable choice). His subsequent ED evaluation is unremarkable; he is admitted to a monitored unit for further care.

Summary

In most instances, a wide complex tachycardia represents either VT, SVT with aberrancy, preexcited tachycardias, pacemaker-related WCT, or WCT associated with toxic and metabolic abnormalities. Prompt evaluation and management of the varied etiologies requires rapid assessment of hemodynamic stability. The unstable patient should be treated with urgent electrical cardioversion. The management of a stable patient is significantly facilitated by a 12-lead ECG and a few historical and laboratory data. In some cases, a specific rhythm diagnosis may not be possible and patient treatment should focus on the clinical presentation.

Key Points

- Do entertain the diagnosis of electrolyte abnormalities and drug toxicities as a cause of WCT. The usual approach to treatment of VT or SVT with AVC can result in significant delays in treatment of underlying metabolic abnormalities (e.g., hyperkalemia).
- When in doubt, treat a patient with wide QRS tachycardia as VT. While the 12-lead ECG algorithms (Griffith, Brugada) have high degrees of accuracy, they are not 100% accurate in their ability to diagnose VT.
- Attempt to obtain a paper copy of the 12-lead ECG (unless the patient is *in extremis* immediately on arrival). Arrhythmias frequently recur. The ability to compare all future tachycardias (either spontaneous or induced during an electrophysiologic study) with the index spontaneous tachycardia is invaluable in the long-term management of the patient; further, a "hard" copy allows for a more intensive review of the ECG and the potential to detect subtle findings such as AV dissociation and fusion/capture beats.
- Other than metabolic derangement (electrolyte abnormalities, drug toxicities), the differential diagnosis for a WCT includes VT, SVT with AVC, preexcited tachycardias, pacemaker-related WCT, and artifact. Artifact can be easily excluded with a 12-lead ECG.
- Do not assume that the diagnosis of WCT in a

minimally symptomatic stable patient is SVT with AVC. The presence (or absence) of hemodynamic stability has little correlation with the etiology of the WCT; similarly, the absence (or presence) of symptoms adds little to the diagnosis of the dysrhythmia.

- There are many WCT algorithms that are available in the literature that assist in distinguishing VT from SVT with aberrancy/preexisting bundle branch block. No algorithm is 100% accurate. Thus, these decision rules should be used with caution.

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, such as the type of study and the number of patients in 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.

1. Wellens HJ, Bar FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex. *Am J Med.* 1978;64:27-33. **(Retrospective; 62 patients)**
2. Kindwall KE, Brown J, Josephson ME. Electrocardiographic criteria for ventricular tachycardia in wide complex left bundle branch block morphology tachycardias. *Am J Cardiol.* 1988;61:1279-1283. **(Retrospective; 128 patients)**
- *3. Griffith MJ, Garrett CJ, Mounsey P, et al. Ventricular tachycardia as default diagnosis in broad complex tachycardia. *Lancet.* 1994;343:386-388. **(Prospective; 102 patients)**
- *4. Brugada P, Brugada J, Mont L, et al. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation.* 1991;83:1649-1659. **(Prospective; 554 patients)**
5. Alberca T, Almendral J, Sanz P, et al. Evaluation of the specificity of morphological electrocardiographic criteria for the differential diagnosis of wide QRS complex tachycardia in patients with intraventricular conduction defects. *Circulation.* 1997;96(10):3527-3533. **(Prospective; 232 patients)**
- *6. Vereckei A, Duray G, Szenasi G, et al. Application of a new algorithm in the differential diagnosis of wide QRS complex tachycardia. *Eur Heart J.* 2007;28:589-600 **(Prospective; 453 patients)**
7. Gupta AK, Thakur RK. Wide QRS complex tachycardias. *Med Clin North Am.* 2001;85:245-266. **(Review)**
8. Domanovits H, Paulis M, Nikfardjam M, et al. Sustained ventricular tachycardia in the emergency department. *Resuscitation.* 1999;42(1):19-25. **(Retrospective; 78 patients)**
9. Herbert ME, Votey SR, Morgan MT, et al. Failure to agree on the electrocardiographic diagnosis of ventricular tachycardia. *Ann Emerg Med.* 1996;27:1:35-38. **(Retrospective; 178 patients)**
10. Akhtar M, Shenasa M, Jazayeri M, et al. Wide QRS complex tachycardia. Reappraisal of a common clinical problem. *Ann Intern Med.* 1988;109:905-912. **(Retrospective; 150 patients)**
11. Baerman JM, Morady F, DiCarlo LA, et al. Differentiation of ventricular tachycardia from supraventricular tachycardia with aberration: Value of the clinical history. *Ann Emerg Med.* 1987;16:40-43. **(Retrospective; 84 patients)**

12. Steinman RT, Herrera C, Schluger CD, et al. Wide QRS tachycardia in the conscious adult: Ventricular tachycardia is the most frequent cause. *JAMA*. 1989;261:1013-1016. **(Retrospective; 20 patients)**
13. Delbridge TR, Yealy DM. Wide complex tachycardia. *Emerg Med Clin North Am*. 1996;13:902-924. **(Review)**
14. Wrenn K. Management strategies in wide QRS complex tachycardia. *Am J Emerg Med*. 1991;9:592-597. **(Review)**
15. Tchou P, Young P, Mahmud R, et al. Useful clinical criteria for the diagnosis of ventricular tachycardia. *Am J Med*. 1988;84(1):53-56. **(Retrospective; 31 patients)**
16. Wellens HJ. Electrophysiology: Ventricular Tachycardia: diagnosis of broad QRS complex tachycardia. *Heart*. 2001;86(5):579-585. **(Review)**
- *17. Brady WJ, Skiles J. Wide QRS complex tachycardia: ECG differential diagnosis. *Am J Emerg Med*. 1999;17(4):376-381. **(Review)**
18. Dongas J, Lehmann MH, Mahmud R, et al. Value of preexisting bundle branch block in the electrocardiographic differentiation of supraventricular from ventricular origin of wide QRS tachycardia. *Am J Cardiol*. 1985;55(6):717-721. **(Prospective; 18 patients)**
19. Guo H, Hecker S, Levy S, et al. Ventricular tachycardia with QRS configuration similar to that in sinus rhythm and a myocardial origin: differential diagnosis with bundle branch reentry. *Europace*. 2001;3(2):115-123. **(Prospective; 5 patients)**
20. Oretto G, Smeets JL, Rodriguez LM, et al. Wide complex tachycardia with atrioventricular dissociation and QRS morphology identical to that of sinus rhythm: a manifestation of bundle branch reentry. *Heart*. 1996;76(6):541-547. **(Prospective; 3 patients)**
21. Marriott HJ. Differential diagnosis of supraventricular and ventricular tachycardia. *Cardiology*. 1990;77(3):209-220. **(Review)**
22. Antunes E, Brugada J, Steurer G, et al. The differential diagnosis of a regular tachycardia with a wide QRS complex on the 12-lead ECG: ventricular tachycardia, supraventricular tachycardia with aberrant. *PACE*. 1994;17(9):1515-1524. **(Review)**
23. Lau EW, Ng GA. Comparison of two diagnostic algorithms for regular broad complex tachycardia by decision theory analysis. *PACE*. 2001;24(7):1118-1125. **(Review)**
- *24. Lau EW, Pathamanathan RK, Ng GA, et al. Electrocardiographic criteria for diagnosis of irregular broad complex tachycardia with a high sensitivity for preexcited atrial fibrillation. *PACE*. 2000;23(12):2040-2045. **(Prospective; 75 patients)**
25. American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part I. Introduction. *JAMA*. 1992;268:2171-2241. **(Guideline)**
26. Griffith MJ, Linker NJ, Ward DE, et al. Adenosine in the diagnosis of broad complex tachycardia. *Lancet*. 1988;1(8587):672-675. **(Retrospective; 26 patients)**
27. Sharma AD, Klein GJ, Yee R. Intravenous adenosine triphosphate during wide QRS complex tachycardia: safety, therapeutic efficacy, and diagnostic utility. *Am J Med*. 1990;88(4):337-343. **(Retrospective; 34 patients)**
28. Rankin AC, Oldroyd KG, Chong E, et al. Value and limitations of adenosine in the diagnosis and treatment of narrow and broad complex tachycardias. *Br Heart J*. 1989;62(3):195-203. **(Retrospective; 64 patients)**
29. Camm AJ, Garratt CJ. Adenosine and supraventricular tachycardia. *N Engl J Med*. 1991;325(23):1621-1629. **(Review)**
30. Exner DV, Muzyka T, Gillis AM. Proarrhythmia in patients with Wolff-Parkinson-White syndrome after standard doses of intravenous adenosine. *Ann Intern Med*. 1995;122(5):351-352. **(Case Report)**
31. Parham WA, Mehdirad AA, Biermann KM, et al. Case report: adenosine induced ventricular fibrillation in a patient with stable ventricular tachycardia. *J Interv Card Electrophysiol*. 2001;5(1):71-74. **(Case report)**
32. Mallet ML. Proarrhythmic effects of adenosine: a review of the literature. *Emerg Med J*. 2004;21(4):408-410. **(Review)**
33. Shaw M, Niemann JT, Haskell RJ, et al. Esophageal electrocardiography in acute cardiac care. Efficacy and diagnostic value of a new technique. *Am J Med*. 1987;82(4):689-696. **(Case Report)**
34. Koppel C, Oberdisse U, Heinemeyer G. Clinical course and outcome in class IC antiarrhythmic overdose. *J Toxicol Clin Toxicol*. 1990;28(4):433-444. **(Retrospective; 120 patients)**
35. Francis J, Hamzeh RK, Lantin-Hermoso MR. Lithium toxicity-induced wide-complex tachycardia in a pediatric patient. *J Pediatr*. 2004;145(2):235-240. **(Case Report)**
36. Thanacoody HK, Thomas SH. Tricyclic antidepressant poisoning: cardiovascular toxicity. *Toxicol Rev*. 2005;24(3):205-214. **(Review)**
37. Bradberry SM, Thanacoody HK, Watt BE, Thomas SH, Vale JA. Management of cardiovascular complications of tricyclic antidepressant poisoning: role of sodium bicarbonate. *Toxicol Rev*. 2005;24(3):195-204. **(Review)**
38. Harrigan RA, Brady WJ. ECG abnormalities in tricyclic antidepressant ingestion. *Am J Emerg Med*. 1999;17(4):387-393. **(Case Series)**
39. Liebelt EL, Francis PD, Woolf AD. ECG lead aVR versus QRS interval in predicting seizures and arrhythmias in acute tricyclic antidepressant toxicity. *Ann Emerg Med*. 1995;26(2):195-201. **(Retrospective; 79 patients)**
40. Dittrich KL, Walls RM. Hyperkalemia: ECG manifestations and clinical considerations. *J Emerg Med*. 1986;4(6):449-455. **(Review)**
41. Mattu A, Brady WJ, Robinson DA. Electrocardiographic manifestations of hyperkalemia. *Am J Emerg Med*. 2000;18(6):721-729. **(Case Series)**
42. Fisch C. Relation of electrolyte disturbances to cardiac arrhythmias. *Circulation*. 1973;47(2):408-419. **(Review)**
43. Dananberg J. Electrolyte abnormalities affecting the heart. In: Schwartz GR, editor. Principles and practice of emergency medicine. 4th ed. Baltimore. Williams & Wilkins; 1999. p. 425-427. **(Textbook)**
44. Sobel RM, Donaldson PR, Dhruva N. Pacemaker-mediated tachycardia: management by pacemaker interrogation/reprogramming in the ED. *Am J Emerg Med*. 2002;20(4):336-339. **(Review)**
45. Love CJ. Pacemaker Troubleshooting and Follow-up. In: Ellenbogen KA, Kay GN, Lau C, et al. editor. Clinical Cardiac pacing, Defibrillation, and Resynchronization Therapy. 3rd edition. Philadelphia. Saunders; 2007. p. 1047-1053. **(Textbook)**
46. Griffin J, Smithline H, Cook J. Runaway Pacemaker: a case report and review. *J Emerg Med*. 2000;19(2):177-181. **(Case report)**
47. Makaryus AN, Patrick C, Maccaro P. A rare case of "runaway" pacemaker in a modern CPU-controlled pacemaker. *PACE*. 2005;28(9):993-996. **(Case report)**
48. American Heart Association. AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: Management of Symptomatic Bradycardia and Tachycardia. *Circulation*. 2005;112[Suppl I]:IV-67-IV-77. **(Guideline)**
- *49. Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (writing committee to develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Circulation*. 2006;114(10):e385-484. **(Guideline)**
50. Tomlinson DR, Cherian P, Betts TR, et al. Should intravenous amiodarone be utilized for haemodynamically tolerated sustained monomorphic ventricular tachycardia? *J Am Coll Cardiol*. 2007;49(9):19A. Suppl A. **(Abstract)**
- *51. Gorgels AP, van den Dool A, Hofs A, et al. Comparison of procainamide and lidocaine in terminating sustained monomorphic ventricular tachycardia. *Am J Cardiol*. 1996;78(1):43-46. **(Prospective; 29 patients)**
- *52. Marill KA, deSouza IS, Nishijima DK, et al. Amiodarone is poorly effective for the acute termination of ventricular tachycardia. *Ann Emerg Med*. 2006;47(3):217-224. **(Retrospective; 33 patients)**
53. Ho DS, Zecchin RP, Richards DA, et al. Double-blind trial of

- lidocaine versus sotalol for acute termination of spontaneous sustained ventricular tachycardia. *Lancet*. 1994;344(8914):18-23. **(Prospective; XX patients)**
54. Nademanee K, Taylor R, Bailey WE, et al. Treating electrical storm:sympathetic blockade versus advanced cardiac life support-guided therapy. *Circulation*. 2000;102(7):742-747. **(Retrospective; 49 patients)**
- *55. Dorian P, Cass D, Schwartz B, et al. Amiodarone as compared to lidocaine for shock-resistant ventricular fibrillation. *N Engl J Med*. 2002;346:884-890. **(Prospective; 347 patients)**
56. Somberg JC, Bailin SJ, Haffajee CI, et al. Intravenous lidocaine versus intravenous amiodarone (in a new aqueous formulation) for incessant ventricular tachycardia. *Am J Cardiol*. 2002;90(8):853-859. **(Prospective; 29 patients)**
57. Timperley J, Mitchell AR, Brown PD, et al. Flecainide overdose - support using an intra-aortic balloon pump. *BMC Emerg Med*. 2005;5:10. **(Case report)**
58. Goldman MJ, Mowry JB, Kirk MA. Sodium bicarbonate to correct widened QRS in a case of flecainide overdose. *J Emerg Med*. 1997;15(2):183-186. **(Case report)**
59. Brubacher J. Bicarbonate therapy for unstable propafenone-induced wide complex tachycardia. *CJEM*. 2004;6(5):349-356. **(Case report)**
60. Salerno DM, Murakami MM, Johnson RB, et al. Reversal of flecainide-induced ventricular arrhythmia by hypertonic sodium bicarbonate in dogs. *Am J Emerg Med*. 1995;13(3):285-293. **(Basic science / animal research)**
61. Bauman JL, Gallastequi J, Tanenbaum SR, et al. Flecainide-induced sustained ventricular arrhythmia successfully treated with lidocaine. *Chest*. 1987;92(3):573-575. **(Case report)**
62. Siegers A, Board PN. Amiodarone used in successful resuscitation after near-fatal flecainide overdose. *Resuscitation*. 2002;53(1):105-108. **(Case report)**
63. American Heart Association. AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: Pediatric Advanced Life Support. *Circulation*. 2005;112[Suppl I]:IV-167-IV-187. **(Guideline)**
64. Hanisch D. Pediatric arrhythmias. *J Pediatr Nurs*. 2001;16(5):351-362. **(Review)**
65. Wiseman MN, Tavel ME. Wide-complex tachycardia: management in a patient with history of congenital heart disease. *Chest*. 2000;117(1):268-271. **(Case report with commentary)**
66. Oktay C, Kesapli M, Altekin E. Wide-QRS complex tachycardia during pregnancy: treatment with cardioversion and review. *Am J Emerg Med*. 2002;20(5):492-493. **(Case report)**
67. Tan HL, Lie KI. Treatment of tachyarrhythmias during pregnancy and lactation. *Eur Heart J*. 2001;22(6):458-464. **(Review)**
68. Widerhorn J, Widerhorn AL, Rahimtoola SH, et al. WPW syndrome during pregnancy: increased incidence of supraventricular arrhythmias. *Am Heart J*. 1992;123(3):796-798. **(Case series)**
69. Lee SH, Chen SA, Wu TJ, et al. Effects of pregnancy on first onset and symptoms of paroxysmal supraventricular tachycardia. *Am J Cardiol*. 1995;76(10):675-678. **(Retrospective; 207 patients)**
70. Finlay AY, Edmunds V. D.C. cardioversion in pregnancy. *Br J Clin Pract*. 1979;33(3):88-94. **(Review)**
71. Lee RV, Rodgers BD, White LM, et al. Cardiopulmonary resuscitation of pregnancy women. *Am J Med*. 1986;81(2):311-318. **(Review)**
72. Schroeder JS, Harrison DC. Repeated cardioversion during pregnancy. Treatment of refractory paroxysmal atrial tachycardia during 3 successive pregnancies. *Am J Cardiol*. 1971;27(4):445-446. **(Case series)**
73. Ferrero S, Colombo BM, Ragni N. Maternal arrhythmias during pregnancy. *Arch Gynecol Obstet*. 2004;269(4):244-253. **(Review)**
74. Goodwin AP, Pearce AJ. The human wedge. An manoeuvre to relieve aorticaval compression during resuscitation in late pregnancy. *Anaesthesia*. 1992;47(5):433-434. **(Case report)**
75. Page RL. Treatment of arrhythmias during pregnancy. *Am Heart J*. 1995;130(4):871-876. **(Review)**
76. Trappe HJ. Acute therapy of maternal and fetal arrhythmias during pregnancy. *J Intensive Care Med*. 2006;21(5):305-315. **(Review)**
77. Allen NM, Page RL. Procainamide administration during pregnancy. *Clin Pharm*. 1993;12(1):58-60. **(Review)**
78. Khositseth A, Ramin KD, O'Leary PW, et al. Role of amiodarone in the treatment of fetal supraventricular tachyarrhythmias and hydrops fetalis. *Pediatr Cardiol*. 2003;24(5):454-456. **(Case series)**
79. Magee LA, Downar E, Sermer M, et al. Pregnancy outcome after gestational exposure to amiodarone in Canada. *Am J Obstet Gynecol*. 1995;172(4 Pt 1):1307-1311. **(Retrospective; 12 patients)**
80. Cardosi RJ, Chez RA. Magnesium sulfate, maternal hypothermia, and fetal bradycardia with loss of heart rate variability. *Obstet Gynecol*. 1998;92(4 Pt 2):691-693. **(Case report)**
81. Rotmensch HH, Elkayam U, Frishman W. Antiarrhythmic drug therapy during pregnancy. *Ann Intern Med*. 1983;98(4):487-497. **(Review)**
82. Elkayam U, Goodwin TM. Adenosine therapy for supraventricular tachycardia during pregnancy. *Am J Cardiol*. 1995;75(7):521-523. **(Retrospective; 33 patients)**
83. Briggs G, Freeman RK, Yaffe SJ. Drugs in pregnancy and lactation. A reference guide to fetal and neonatal risk, 5th ed. Williams and Wilkins, Baltimore 1998. **(Textbook)**
84. Frishman WH, Chesner M. Beta-adrenergic blockers in pregnancy. *Am Heart J*. 1988;115(1 Pt 1):147-152. **(Review)**
85. Gowda et al. Cardiac arrhythmias in pregnancy: clinical and therapeutic considerations. *Int J Cardiol*. 2003;88(2-3):129-133. **(Review)**
86. Natale A, Davidson T, Geiger MJ, et al. Implantable cardioverter-defibrillators and pregnancy: a safe combination? *Circulation*. 1997;96(9):2808-2812. **(Retrospective; 44 patients)**
87. Stewart RB, Bardy GH, Greene HL. Wide complex tachycardia: misdiagnosis and outcome after emergent therapy. *Ann Intern Med*. 1986;104(6):766-771. **(Retrospective; 46 patients)**
88. Josephson ME. Electrophysiology of Ventricular Tachycardia: An Historical Perspective. *J Cardiovasc Electrophysiol*. 2003;14:1134-1148. **(Review)**
89. Stevenson WG. Catheter ablation of monomorphic ventricular tachycardia. *Curr Opin Cardiol*. 2005;20(1):42-47. **(Review)**
90. Hollowell H, Mattu A, Perron AD, Holstege C, Brady WJ. Wide-Complex tachycardia: beyond the traditional differential diagnosis of ventricular tachycardia vs supraventricular tachycardia with aberrant conduction. *Am J Emerg Med*. 2005;23(7):876-879. **(Case review; 3 patients)**
- *91. Miller JM, Das MK, Yadav AV, Bhakta D, Nair G, Alberte C. Value of the 12-lead ECG in wide QRS tachycardia. *Cardiol Clin*. 2006;24(3):439-451. **(Retrospective; 656 patients)**
92. Wills AM, Plante DT, Dukkupati SR, Corcoran CP, Standaert DG. Pacemaker-induced tachycardia caused by inappropriate response to parkinsonian tremor. *Neurology*. 2005;65(10):1676-1677. **(Case report)**
93. Wong DT, Middleton W. Electrocautery-induced tachycardia in a rate responsive pacemaker. *Anesthesiology*. 2001;94(4):710-711. **(Case report)**
94. Lau W, Corcoran SJ, Mond HG. Pacemaker tachycardia in a minute ventilation rate-adaptive pacemaker induced by electrocardiographic monitoring. *Pacing Clin Electrophysiol*. 2006;29(4):438-440. **(Case report)**
95. Passman R, Kadish A. Polymorphic ventricular tachycardia, long Q-T syndrome, and torsades de pointes. *Med Clin North Am*. 2001;85(2):321-341. **(Review)**
96. Chou TC. Electrocardiography in clinical practice: Adult and Pediatric. 4th Edition. W.B. Saunders Company, Philadelphia. 1996. **(Textbook)**
97. Lau EW, Ng GA. The reliable electrocardiographic diagnosis of regular broad complex tachycardia: a holy grail that will forever elude the clinician's grasp? *Pacing Clin Electrophysiol*. 2002;25(12):1756-1761. **(Review)**
98. Lerman BB, Stein KM, Markowitz SM. Adenosine-sensitive ventricular tachycardia: a conceptual approach. *J Cardiovasc Electrophysiol*. 1996;7(6):559-569. **(Review)**

99. Roden DM. Antiarrhythmic drugs: past, present, and future. *J Cardiovasc Electrophysiol.* 2003;14(12):1389-1396. (Review)
100. Tommaso C, Belic N, Brandfonbrener M. Asynchronous ventricular pacing: a rare cause of ventricular tachycardia. *Pacing Clin Electrophysiol.* 1982;5(4):561-563. (Case report)
101. Liebelt EL, Francis PD. Chapter 57: Cyclic Antidepressants. In Goldfrank LR et al, *Goldfrank's Toxicologic Emergencies* (7th Ed). New York, McGraw-Hill, 2002. (Textbook)
102. Passman R, Kadish A. Polymorphic ventricular tachycardia, long Q-T syndrome, and torsades de pointes. *Med Clin North Am.* 2001;85(2):321-341. (Review)
103. Kron J, Conti JB. Arrhythmias in the pregnant patient: current concepts in evaluation and management. *J Interv Card Electrophysiol.* 2007;19(2):95-107. (Review)
104. Al-Khatib SM, LaPointe NM, Kramer JM, Califf RM. What clinicians should know about the QT interval. *JAMA.* 2003;289(16):2120-2127. (Review)

CME Questions

1. **What is the most common etiology of regular WCTs referred to cardiac electrophysiologists?**
 - a. Ventricular tachycardia
 - b. Supraventricular tachycardia with preexisting bundle branch block
 - c. Supraventricular tachycardia with antegrade conduction over accessory pathway
 - d. Atrial fibrillation with underlying bundle branch block/aberrancy
 - e. Pacemaker-mediated WCT
2. **In contrast to electrophysiologists' experience and conventional wisdom, what was the most common etiology of a WCT in a recent unselective, consecutive series of ED presentations?**
 - a. Ventricular tachycardia
 - b. Supraventricular tachycardia with preexisting bundle branch block
 - c. Supraventricular tachycardia with antegrade conduction over accessory pathway
 - d. Atrial fibrillation with underlying bundle branch block/aberrancy
 - e. Sinus tachycardia with underlying bundle branch block/aberrancy
3. **In the evaluation and management of a WCT, which of the following etiologies can be resistant to DC cardioversion and tends to get missed?**
 - a. Ventricular tachycardia
 - b. Supraventricular tachycardia with preexisting bundle branch block
 - c. Supraventricular tachycardia with antegrade conduction over accessory pathway
 - d. Atrial fibrillation with underlying bundle branch block/aberrancy
 - e. Metabolic derangement (electrolyte abnormalities and drug toxicities)
4. **Which historical clue does not suggest a diagnosis of ventricular tachycardia?**
 - a. Chest pain before arrhythmia
 - b. History of myocardial infarction
 - c. History of coronary stent placement 6 months ago
 - d. History of CHF
 - e. Patient has had an intracardiac defibrillator placed
5. **Which of the following physical examination clues provides the least assistance in the diagnosis of the WCT?**
 - a. The presence of irregular cannon "a" waves in the jugular venous pulse
 - b. Auscultation of variable intensities of the first heart sound (S1)
 - c. The presence of an AV fistula in the arm
 - d. The presence of a midline thoracotomy scar
 - e. The palpation of an implanted device in the subcutaneous tissue of the left upper thorax
6. **Which of the following laboratory tests provides the most useful information in the most expeditious manner in the evaluation and management of a patient with WCT?**
 - a. Troponin
 - b. BNP
 - c. Comprehensive chemistry panel (including LFTs)
 - d. CBC
 - e. Venous or arterial blood gas with electrolytes (especially potassium)
7. **The Griffith algorithm incorporates the following sequence of steps in the evaluation of a regular WCT in a stable patient.**
 - a. Check for AV dissociation, then QRS morphology, then QRS axis
 - b. Check for AV dissociation, then QRS axis, then QRS morphology
 - c. Check for QRS morphology, then AV dissociation, then QRS axis
 - d. Check for QRS morphology, then QRS axis, then AV dissociation
 - e. Check for QRS axis, then QRS morphology, then AV dissociation
8. **During your analysis of a 12-lead ECG of a patient with WCT, you make the following observations: a rate of 185, left axis deviation, all the QRS complexes in the precordial leads are positive, and no visible P waves on the ECG. The diagnosis is:**
 - a. SVT with aberrancy
 - b. SVT with antegrade conduction over accessory pathway
 - c. Ventricular tachycardia
 - d. Hyperkalemia
9. **During your analysis of a 12-lead ECG of a patient with WCT, you make the following observations: a rate of 200, left axis deviation, QRS duration of 140 ms, and no visible P waves on the ECG. The diagnosis is:**
 - a. SVT with preexisting bundle branch block.
 - b. SVT with antegrade conduction over accessory pathway.
 - c. Ventricular tachycardia.
 - d. Hyperkalemia.
 - e. Cannot make diagnosis just yet. Apply the Griffith algorithm to the 12-lead ECG to evaluate the QRS morphology.

10. During your analysis of a 12-lead ECG of a patient with WCT, you make the following observations: a rate of 156, left axis, and the QRS complex in V1 is predominantly positive (Figure 18). You obtain a 12-lead rhythm strip, and you think that there is one P wave for every QRS complex. The patient is symptomatic with moderate sensation of palpitations. He has no prior medical history. His BP is 120/75 and he has normal mental status and physical examination. You should do which of the following?

- Retrieve an old ECG
- Vagal maneuvers to treat SVT with BBB
- Adenosine to treat SVT
- Adenosine to distinguish SVT from VT
- Procainamide to treat VT

Coming In Future Issues

COPD

Adult Ventilators

Class Of Evidence 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

- Case series, animal studies, consensus panels
- Occasionally positive results

Class 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

Class II

- Safe, acceptable
 - Probably useful
- Level of Evidence:
- Generally higher levels of evidence
 - Non-randomized or retrospective studies: historic, cohort, or case-control studies
 - Less robust RCTs
 - Results consistently positive

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. JAMA1992;268(16):2289-2295.

Class III

- May be acceptable
 - Possibly useful
 - Considered optional or alternative treatments
- Level of Evidence:
- Generally lower or intermediate levels of evidence

Physician CME Information

Date of Original Release: June 1, 2008. Date of most recent review: May 10, 2008. Termination date: June 1, 2011.

Accreditation: This activity has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education (ACCME) through the joint sponsorship of Mount Sinai School of Medicine and *Emergency Medicine Practice*. The Mount Sinai School of Medicine is accredited by the ACCME to provide continuing medical education for physicians.

Credit Designation: The Mount Sinai School of Medicine designates this educational activity for a maximum of 48 AMA PRA Category 1 Credit(s)[™] per year. Physicians should only claim credit commensurate with the extent of their participation in the activity.

ACEP Accreditation: *Emergency Medicine Practice* is approved by the American College of Emergency Physicians for 48 hours of ACEP Category 1 credit per annual subscription.

AAFP Accreditation: *Emergency Medicine Practice* has been reviewed and is acceptable for up to 48 Prescribed credits per year by the American Academy of Family Physicians. AAFP Accreditation begins August 1, 2006. Term of approval is for two years from this date. Each issue is approved for 4 Prescribed credits. Credits may be claimed for two years from the date of this issue.

AOA Accreditation: *Emergency Medicine Practice* has been approved for 48 Category 2B credit hours per year by the American Osteopathic Association.

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 & Objectives: Upon completion of this article, 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 ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

Discussion of Investigational Information: As part of the newsletter, faculty may be presenting investigational information about pharmaceutical products that is outside Food and Drug Administration-approved labeling. Information presented as part of this activity is intended solely as continuing medical education and is not intended to promote off-label use of any pharmaceutical product.

Disclosure of Off-Label Usage: This issue of *Emergency Medicine Practice* discusses no off-label use of any pharmaceutical product.

Faculty Disclosure: It is the policy of Mount Sinai School of Medicine to ensure objectivity, balance, independence, transparency, and scientific rigor in all CME-sponsored educational activities. All faculty participating in the planning or implementation of a sponsored activity are expected to disclose to the audience any relevant financial relationships and to assist in resolving any conflict of interest that may arise from the relationship. Presenters must also make a meaningful disclosure to the audience of their discussions of unlabeled or unapproved drugs or devices.

In compliance with all ACCME Essentials, Standards, and Guidelines, all faculty for this CME activity were asked to complete a full disclosure statement. The information received is as follows: **Dr. Subramanian, Dr. Herman, Dr. Marill, and Dr. Mattu report no significant financial interest or other relationship with the manufacturer(s) of any commercial product(s) discussed in this educational presentation. Dr. Brady has received consulting fees from Heartscape Tech.**

Method of Participation:

- **Print Subscription Semester Program:** Paid subscribers who read all CME articles during each *Emergency Medicine Practice* six-month testing period, complete the post-test and the CME Evaluation Form distributed with the June and December issues, and return it according to the published instructions are eligible for up to 4 hours of CME credit for each issue. You must complete both the post test and CME Evaluation Form to receive credit. Results will be kept confidential. CME certificates will be delivered to each participant scoring higher than 70%.
- **Online Single-Issue Program:** Current, paid subscribers who read this *Emergency Medicine Practice* CME article and complete the online post-test and CME Evaluation Form at ebmedicine.net are eligible for up to 4 hours of Category 1 credit toward the AMA Physician's Recognition Award (PRA). You must complete both the post-test and CME Evaluation Form to receive credit. Results will be kept confidential. CME certificates may be printed directly from the web site to each participant scoring higher than 70%.

Hardware/Software Requirements: You will need a Macintosh or PC to access the online archived articles and CME testing. Adobe Reader is required to view the PDFs of the archived articles. Adobe Reader is available as a free download at www.adobe.com.

Emergency Medicine Practice is not affiliated with any pharmaceutical firm or medical device manufacturer.

CEO: Robert Williford

President and Publisher: Stephanie Williford Associate Editor & CME Director: Jennifer Pai
Director of Member Services: Liz Alvarez

Direct all editorial, subscription-related, customer service, or copyright/reprint questions to:

EB Medicine

1-800-249-5770

Outside the U.S.: 1-678-366-7933

Fax: 1-770-500-1316

5550 Triangle Parkway, Suite 150

Norcross, GA 30092

E-mail: ebm@ebmedicine.net

Web Site: ebmedicine.net

Subscription Information:

1 year (12 issues) including evidence-based print issues, 48 AMA/ACEP Category 1, AAFP Prescribed, or AOA Category 2B CME credits, and full online access to searchable archives and additional CME: \$329

1 year institutional/hospital/library rate: \$899

Individual issues, including 4 CME credits: \$30

(Call 1-800-249-5770 or go to www.empractice.com to order)

Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. This publication is intended as a general guide and is intended to supplement, rather than substitute, professional judgment. It covers a highly technical and complex subject and should not be used for making specific medical decisions. The materials contained herein are not intended to establish policy, procedure, or standard of care. *Emergency Medicine Practice* is a trademark of EB Practice, LLC. Copyright © 2008 EB Practice, LLC. All rights reserved. No part of this publication may be reproduced in any format without written consent of EB Practice, LLC. This publication is intended for the use of the individual subscriber only, and may not be copied in whole or part or redistributed in any way without the publisher's prior written permission—including reproduction for educational purposes or for internal distribution within your hospital, library, group practice, or other entity.