Wide QRS complex tachycardias: Approach to the diagnosis

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INTRODUCTION

Tachycardias are broadly categorized (algorithm 1) based upon the width of the QRS complex on the electrocardiogram (ECG).

- A narrow QRS complex (<120 milliseconds) reflects rapid activation of the ventricles via the normal His-Purkinje system, which in turn suggests that the arrhythmia originates above or within the atrioventricular (AV) node (ie, a supraventricular tachycardia [SVT]).
- A widened QRS complex (≥120 milliseconds) occurs when ventricular activation is abnormally slow for one of the following reasons (see "Wide QRS complex tachycardias: Causes, epidemiology, and clinical manifestations", section on 'Differential diagnosis of WCT'):
  - The arrhythmia originates outside of the normal conduction system and below the AV node (ie, ventricular tachycardia [VT])
  - Abnormalities within the His-Purkinje system (ie, SVT with aberrancy)
  - Pre-excitation with an SVT conducting antegrade over an accessory pathway, resulting in direct activation of the ventricular myocardium

A wide QRS complex tachycardia (WCT) represents a unique clinical challenge for two reasons:

- Diagnosing the arrhythmia is difficult – Although most WCTs are due to VT, the differential diagnosis includes a variety of SVTs. Diagnostic algorithms to differentiate these two etiologies are complex and imperfect. (See "Wide QRS complex tachycardias: Causes, epidemiology, and clinical manifestations", section on 'Differential diagnosis of WCT' and 'Diagnosis' below.)

- Urgent therapy is often required – Patients may be unstable at the onset of the arrhythmia or deteriorate rapidly at any time, particularly if the WCT is VT or SVT at an extremely rapid rate (eg, >200 beats per minute).

The initial evaluation and approach to diagnosis of patients with a WCT will be discussed here. The causes, epidemiology, clinical manifestations, and management of WCTs, as well as discussion of narrow QRS complex tachycardias, are presented separately. (See "Wide QRS complex tachycardias: Causes, epidemiology, and clinical manifestations" and "Wide QRS complex tachycardias: Approach to management" and "Overview of the acute management of tachyarrhythmias" and "Secondary prevention of sudden cardiac death in heart failure and cardiomyopathy" and "Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation".)

INITIAL APPROACH

The first priority when evaluating a patient with a WCT is an assessment of patient stability. Unstable patients (ie, pulseless patients in cardiac arrest or patients who are hemodynamically unstable) should be treated immediately, before an extensive diagnostic evaluation. (See 'Assessment of hemodynamic stability' below and "Wide QRS complex tachycardias: Approach to management", section on 'Unstable patients'.)

Stable patients (ie, patients with no or minimal symptoms and who are hemodynamically stable) will most often have already had an ECG showing the presence of a WCT. In a stable patient, the ECG should be thoroughly and systematically reviewed in an effort to determine the etiology of the WCT. (See 'Evaluation of the electrocardiogram' below.)

Assessment of hemodynamic stability — Immediate assessment of the patient's symptoms, vital signs, and the level of consciousness are of primary importance. In the discussions that follow, patients are categorized as follows:

- Unstable – An unstable patient with WCT has evidence of hemodynamic compromise, such as hypotension, altered mental status, chest pain, or HF, but generally remains awake with a discernible pulse. In this setting, emergency synchronized cardioversion (after

intravenous sedation, whenever possible) is the treatment of choice regardless of the mechanism of the arrhythmia. (See "Wide QRS complex tachycardias: Approach to management", section on 'Unstable patients'.)

Patients who become unresponsive or pulseless are considered to have a cardiac arrest and are treated according to standard resuscitation algorithms. (See "Advanced cardiac life support (ACLS) in adults" and "Adult basic life support (BLS) for health care providers".)

- Stable – A stable patient with WCT shows no evidence of hemodynamic compromise despite a sustained rapid heart rate. Such patients should have continuous monitoring (if hospitalized) and frequent reevaluations (in either the inpatient or outpatient setting) due to the potential for rapid deterioration as long as the WCT persists.

  The presence of hemodynamic stability should **not** be regarded as diagnostic of SVT [1,2]. Misdiagnosis of VT as SVT based upon hemodynamic stability is a common error that can lead to inappropriate and potentially dangerous therapy [1,3]. (See "Wide QRS complex tachycardias: Approach to management", section on 'Pharmacologic interventions'.)

Immediate cardioversion in unstable patients — Hemodynamic compromise may occur with any WCT, regardless of the etiology, but is more likely in patients with ventricular tachycardia (VT). Patients who are felt to be hemodynamically unstable require prompt treatment with electrical cardioversion/defibrillation to prevent further clinical deterioration or sudden cardiac arrest (SCA). The approach to treatment in these patients is discussed in detail separately. (See "Wide QRS complex tachycardias: Approach to management", section on 'Unstable patients'.)

Initial evaluation in stable patients — In a stable patient, a focused clinical evaluation should include the following:

- History (see 'History' below)
- Physical examination (see 'Physical examination' below and "Wide QRS complex tachycardias: Causes, epidemiology, and clinical manifestations", section on 'Physical examination findings')
- Laboratory testing (see 'Ancillary testing' below)
- Diagnostic maneuvers in selected patients

The primary goals of the initial evaluation of a stable patient with WCT are to determine the etiology of the WCT (through evaluation of the ECG) and to elucidate any underlying conditions related to the event (eg, heart failure, myocardial ischemia, drug reaction, or electrolyte abnormalities). (See 'Evaluation of the electrocardiogram' below.)

Among patients with WCT, VT is much more common than supraventricular tachycardia (SVT) with aberrant conduction. In addition, inadvertently treating VT as though it were SVT could precipitate hemodynamic collapse and/or cardiac arrest, while "mistreating" SVT as though it were VT will rarely cause a clinically significant adverse effect. Thus, if there is any uncertainty of the diagnosis, it is generally preferable to be conservative and presume VT in the case of WCT. The approach to treatment of WCT is discussed in detail separately. (See "Wide QRS complex tachycardias: Approach to management".)

History — When evaluating the stable patient with a WCT, the following historical features may help determine the likely etiology and/or guide therapy:

- History of heart disease – The presence of structural heart disease, especially coronary heart disease and/or a previous myocardial infarction (MI), strongly suggests VT as an etiology [1,4]. It is also important to establish whether a cardiac arrhythmia has occurred in the past and, if so, whether the patient is aware of the etiology. (See "Wide QRS complex tachycardias: Causes, epidemiology, and clinical manifestations", section on 'Epidemiology'.)

- Presence of an implantable cardioverter-defibrillator (ICD) – The presence of an ICD implies a known increased risk of VTs and suggests strongly (but does not prove) that the WCT is VT. (See "Implantable cardioverter-defibrillators: Overview of indications, components, and functions".)

- Presence of a pacemaker – The patient should be asked about the presence of pacemaker, which raises the possibility of a device-associated WCT. (See "Wide QRS complex tachycardias: Causes, epidemiology, and clinical manifestations", section on 'Supraventricular tachycardia'.)

- Atrial arrhythmias – In a patient known to have persistent atrial fibrillation (AF), a **regular** WCT is likely VT, as aberrant conduction during AF would create an **irregular** rhythm. An exception is when AF "organizes" into atrial flutter; this can occur spontaneously but occurs much more commonly in the setting of antiarrhythmic drugs (especially class IC agents, amiodarone, or dronedarone).

- Age – For patients over the age of 35 years presenting to an emergency department, a WCT is likely to be VT (positive predictive value 85 percent in one series) [5]. SVT is more likely in younger patients (positive predictive value 70 percent). However, VT must be considered in younger patients, particularly those with a family history of ventricular arrhythmias or premature sudden cardiac death.

- Symptoms – Symptoms are **not** useful in determining the diagnosis, but they may be important as an indicator of the severity of hemodynamic compromise. Symptoms are primarily due to the elevated heart rate, associated heart disease, and the presence of left ventricular dysfunction [1,4]. Some patients with a WCT have no or relatively minor symptoms (palpitations, lightheadedness, diaphoresis), while others have severe manifestations including chest pain or angina, syncope, shock, seizures, and cardiac arrest.

Medications — Many medications have proarrhythmic effects, either directly by prolonging the QT interval or indirectly via alterations in electrolyte levels, and obtaining a medication history is among the first priorities in the evaluation of a patient with a WCT. The most common drug-induced WCT is a form of polymorphic VT called torsades de pointes (TdP). This arrhythmia is associated with QT interval prolongation when the patient is in sinus rhythm. Many medications (eg, antiarrhythmic drugs, anti-infective drugs, psychotropic drugs, etc) are known to prolong the QT interval (table 1) and are associated with a risk of polymorphic VT. An internet resource with updated lists of specific drugs that cause TdP is available at the Credible Meds website. (See "Acquired long QT syndrome: Definitions, pathophysiology, and causes".)

Class IC antiarrhythmic drugs can sometimes organize AF into a relatively slow atrial flutter, which may conduct 1:1 with a very wide QRS complex, mimicking VT.

Physical examination — As with the history, the initial physical examination should search for evidence of underlying cardiovascular disease, which can impact the likelihood that the WCT is VT.

Findings suggestive of cardiovascular disease include:

- Signs of acute or chronic HF (eg, hypoxia, lung crackles, etc). (See "Treatment of acute decompensated heart failure: General considerations" and "Heart failure: Clinical manifestations and diagnosis in adults".)
- A healed sternal incision as evidence of previous cardiothoracic surgery.
- The sequelae of peripheral artery disease or stroke (eg, surgical scars consistent with vascular surgery, residual neurologic deficits, etc).
- A pacemaker or ICD. These devices are usually palpable and are in the left or, less commonly, right pectoral area below the clavicle; rarely, ICDs are found in the anterior abdominal wall.

ECG initial impressions — Once a WCT has been identified on the ECG, prompt diagnosis and management is important. Unstable patients should be presumed to have VT and treated as such. While the Brugada criteria are the most widely known and applied criteria for distinguishing VT from SVT, time and/or technical limitations may not allow for immediate review of the ECG in such detail, particularly in a symptomatic or borderline unstable patient. (See 'Brugada criteria' below.)

The absence of the historical or ECG features of VT does not confirm a diagnosis of SVT. The diagnosis of SVT should be considered primarily in young patients, whose hearts are structurally normal, in whom none of the historical (eg, family history of sudden cardiac death), physical, or ECG criteria supporting VT are present, or in patients with a history of SVT with a similar presentation. When the diagnosis of a WCT remains uncertain, we recommend that the patient be treated as if the rhythm is VT until definitively proven otherwise.

Our approach emphasizes immediate ECG review for key high-yield features in parallel with a brief history that can guide the management of most patients with a WCT.

- Is the rhythm regular or irregular? – VT and most SVTs are generally regular, though slight variation of the RR interval can be seen in VT. An irregular WCT usually represents AF with aberrant conduction, although polymorphic VT should also be considered. (See 'Basic features' below.)

- What is the QRS axis (figure 1)? – Extreme right axis deviation (ie, a right superior axis [axis from -90 to ±180 degrees]) strongly favors VT, as does any axis shift of greater than 40 degrees when compared with baseline or concordance of the QRS complexes. (See 'Basic features' below and 'Concordance' below.)

- Is there AV dissociation? – If AV dissociation can be quickly identified, an atrial rate slower than the ventricular rate, along with any fusion or capture beats, strongly suggests VT. (See 'AV dissociation' below.)

- Is there a history of heart disease or arrhythmias? – A quick history focusing on structural heart disease, particularly coronary heart disease and/or prior MI as well as any known arrhythmias or cardiac implantable electronic devices (eg, pacemaker or ICD), can aid in identifying the most likely etiology of WCT. The WCT is far more likely to be VT in patients over 35 years of age with known coronary heart disease or prior MI and in those with an ICD. (See 'History' above.)

Ancillary testing — A number of additional tests may provide further insight to the mechanism of the tachycardia and the presence of associated conditions.

- Laboratory testing – Initial laboratory testing in all patients with a WCT should include electrolytes and cardiac troponin, with additional testing for elevated serum drug levels in patients taking certain medications.

  - The plasma potassium and magnesium concentrations should be measured as part of the initial evaluation since hypokalemia and hypomagnesemia both predispose to the development of VT. Hyperkalemia can cause a wide QRS complex rhythm with the loss of a detectable P wave, although this usually is a supraventricular rhythm with a slow rate (so-called "sinoventricular rhythm"). (See "Causes and evaluation of hyperkalemia in adults" and "ECG tutorial: Miscellaneous diagnoses", section on 'Hyperkalemia'.)

  - Cardiac troponin testing should be performed in patients with WCT when the patient is having ongoing chest pain or hemodynamic instability. Cardiac troponin can be elevated in the setting of myocardial ischemia (as the result of myocardial oxygen supply and demand mismatch) or MI (as a possible cause of the arrhythmia). Elevated cardiac troponin is associated with an increased risk of adverse outcomes and may guide hospital admission decisions, cardiac testing, and/or anti-ischemic therapy. (See "Troponin testing: Clinical use".)

  - In patients taking digoxin, quinidine, or procainamide, plasma concentrations of these drugs (and, in the case of procainamide, the metabolite N-acetylprocainamide) should be measured to assist in evaluating possible toxicity. (See "Major side effects of class I antiarrhythmic drugs", section on 'Procainamide'.)

- Chest radiograph – A chest radiograph can provide evidence suggestive of structural heart disease, such as cardiomegaly, or evidence of HF with pulmonary congestion. In general, a chest radiograph should be ordered when there is concern for HF or a thoracic or pulmonary process, or to assess for underlying structural heart disease when the patient is unable to provide a history.

Early diagnostic/therapeutic interventions — In patients with a WCT who are hemodynamically stable, certain vagal maneuvers (eg, carotid sinus massage) or the administration of certain intravenous medications (eg, adenosine, beta blockers, verapamil) may be both helpful for establishing the diagnosis and potentially therapeutic, although they do carry a risk of hemodynamic deterioration and should only be performed in closely monitored settings by experienced clinicians. These potential interventions are discussed in detail separately. (See "Wide QRS complex tachycardias: Approach to management", section on 'Vagal maneuvers'.)

EVALUATION OF THE ELECTROCARDIOGRAM

In most patients, a probable diagnosis (ventricular tachycardia [VT] or supraventricular tachycardia [SVT]) may be made by closely reviewing the ECG, although definitive diagnosis is not always possible. For optimal ECG analysis, both a 12-lead ECG and a rhythm strip should be obtained. However, if there are any questions regarding the patient's stability, detailed ECG evaluation should be deferred in favor of urgent therapy. (See "Wide QRS complex tachycardias: Approach to management", section on 'Unstable patients'.)

Basic features — As with the interpretation of any ECG, the standard initial approach includes an assessment of rate, regularity, axis, QRS duration, and QRS morphology. If available, comparison with a baseline ECG tracing in sinus rhythm (or atrial fibrillation [AF]) can be very helpful. Helpful clues on a baseline ECG may include fascicular and/or bundle branch block, signs of prior infarction, or ventricular pre-excitation. Patients with aberrant conduction at baseline will virtually always have aberrant conduction during SVT. A QRS complex during WCT identical to that during SR usually implies SVT, though an unusual form of VT called bundle branch reentrant VT may be possible. If the patient is known to have persistent AF at baseline, a regular WCT usually implies VT.

- Rate – The rate of the WCT is of limited use in distinguishing VT from SVT. When the rate is approximately 150 beats per minute, atrial flutter with 2:1 AV block and with aberrant conduction should be considered, although this diagnosis should not be accepted without other supporting evidence.

- Regularity – VT is generally regular. Slight variation in the RR intervals is sometimes seen and suggests VT as opposed to most SVTs, which are characterized by uniformity of the RR intervals. When the onset of the arrhythmia is available for analysis, a period of some irregularity and acceleration ("warm-up phenomenon") suggests VT. More marked irregularity of RR intervals occurs in polymorphic VT and in AF with aberrant conduction.

- Morphology – For distinguishing between VT and SVT, there are no universal morphologic criteria that would allow for the distinction. However, when describing WCTs, the QRS morphology in lead V1 is the key. If the complex is primarily negative in lead V1, the WCT is said to be left bundle branch block (LBBB)-like. If the QRS is primarily positive in lead V1, the WCT is said to be right bundle branch block (RBBB)-like. (See 'QRS morphology' below.)

- Axis – The QRS axis in the frontal plane can be useful in distinguishing SVT from VT. The presence of one or more of the following is highly suggestive of VT: right superior axis, shift in axis >40 degrees from a prior baseline, and a specific QRS axis in association with

other QRS morphologies. (See "Basic principles of electrocardiographic interpretation", section on 'QRS axis'.)

- A right superior axis (axis from -90 to ±180 degrees) (figure 1), sometimes called an extreme axis deviation, indeterminate axis, or "northwest" axis, is rare in SVT and strongly suggests VT. There are two exceptions to this rule. The first is an antidromic AV reentrant tachycardia (AVRT) seen with ventricular pre-excitation. In this situation, there is direct activation of the ventricular myocardium, bypassing the normal His-Purkinje system, and the QRS complex may have an indeterminate axis. The second is a biventricular pacemaker in which the axis is often indeterminate with an initial Q wave in lead I.

- Compared with the axis during sinus rhythm (when an old ECG is available for review), an axis shift during the WCT of more than 40 degrees suggests VT [6].

- In a patient with an RBBB-like WCT, a QRS axis to the left of -30 degrees suggests VT. (See 'QRS morphology' below.)

- In a patient with an LBBB-like WCT, a QRS axis to the right of +90 degrees suggests VT [7]. (See 'QRS morphology' below.)

- QRS duration – By definition, the QRS duration is at least 120 milliseconds in a WCT. In general, a wider QRS favors VT. In an RBBB-like WCT, a QRS duration >140 milliseconds suggests VT; while in an LBBB-like WCT, a QRS duration >160 milliseconds suggests VT [7]. (See 'QRS morphology' below.)

  In an analysis of several studies, a QRS duration >160 milliseconds was a strong predictor of VT (likelihood ratio >20:1) [8]. However, a QRS duration >160 milliseconds is not helpful in some settings, including SVT with an AV accessory pathway; the presence of drugs capable of slowing intraventricular conduction, such as class I antiarrhythmic drugs; and in association with hyperkalemia [7,9,10]. A very wide QRS complex may also be seen with a dilated cardiomyopathy in which diffuse fibrosis may produce a marked slowing of impulse conduction through the ventricular myocardium. (See 'VT versus AVRT' below and 'Medications' above.)

  A QRS duration <140 milliseconds does not exclude VT since VT originating from the septum or within the His-Purkinje system (as opposed to the myocardium) may be associated with a relatively narrow QRS complex. (See "Ventricular tachycardia in the absence of apparent structural heart disease", section on 'Idiopathic left ventricular tachycardia'.)

Concordance — Concordance is present when the QRS complexes in all six precordial leads (V1 through V6) are monophasic with the same polarity. When present, concordance is frequently associated with VT but by itself is not diagnostic for VT (waveform 1). If any of the six leads have a biphasic QRS (qR or RS complexes), concordance is not present.

- Negative concordance (ie, V1 through V6 entirely negative with deep, monophasic QS complexes) is strongly suggestive of VT but is not definitive. Rarely, SVT with LBBB aberrancy will demonstrate negative concordance, but there is almost always some evidence of an R wave in the lateral precordial leads (waveform 2).

- Positive concordance (ie, V1 through V6 entirely positive with tall, monophasic R waves) is most often due to VT but can also occur in the relatively rare case of antidromic AVRT with a left posterior accessory pathway (waveform 3) [7]. (See "Anatomy, pathophysiology, and localization of accessory pathways in the preexcitation syndrome".)

While the presence of concordance strongly suggests VT (>90 percent specificity), its absence is not helpful diagnostically (approximately 20 percent sensitivity) [11].

AV dissociation — When identified on the ECG, the presence of AV dissociation largely establishes VT as the diagnosis. AV dissociation is characterized by atrial activity that is independent of ventricular activity, usually with the ventricular rate exceeding the atrial rate (except in the instance of a "double tachycardia" with, for example, concurrent atrial flutter and VT) (waveform 4). Though not always present, fusion beats and capture beats are readily recognized indicators of AV dissociation.

In a WCT with AV dissociation, an atrial rate slower than the ventricular rate strongly suggests VT. An atrial rate that is faster than the ventricular rate is seen with some SVTs, such as atrial flutter or an atrial tachycardia with 2:1 AV conduction associated with blocked atrial impulses (due to refractoriness of the AV node). In these settings, however, there is a consistent relationship between the P waves and the QRS complexes, so there is not true AV dissociation.

While AV dissociation largely establishes the diagnosis, its absence is not as helpful for two reasons:

- AV dissociation may be present but not obvious on the ECG.

- In some cases of VT, the ventricular impulses conduct backward through the AV node and capture the atrium (referred to as 1:1 retrograde conduction), so in fact, there is AV association rather than AV dissociation [12]. This may be seen more commonly when the rate of VT is slower. VT with retrograde Wenckebach conduction, or with 2:1

ventriculoatrial (VA) conduction, technically does not exhibit AV dissociation, though the ventricular rate exceeds the atrial rate. In all cases in which there is retrograde VA conduction, the P waves will be inverted in the inferior leads.

Dissociated P waves — P waves are said to be dissociated if they are not consistently coupled to the QRS complexes, as evidenced by the following:

- PP and RR intervals are different
- PR intervals are variable
- There is no association between P and QRS complexes
- The presence of a P wave with one or more, but not all, QRS complexes

During a WCT, P waves are often difficult to identify. If P waves are not evident on the surface ECG, direct recordings of atrial activity (eg, via an implanted pacemaker or implantable cardioverter-defibrillator, or via an esophageal electrode or temporary pacing catheter or epicardial pacing wires after heart surgery) can reveal AV dissociation [13].

Fusion and capture beats — Fusion and/or capture beats, when identified on the surface ECG in a patient with WCT, are diagnostic for VT.

- Fusion beats occur when one impulse originating from the ventricle and a second supraventricular impulse simultaneously activate the ventricular myocardium. The resulting QRS complex has a morphology intermediate between that of a sinus beat and a purely ventricular complex (waveform 5). Intermittent fusion beats during a WCT are diagnostic of AV dissociation and therefore of VT.

- Capture beats, or Dressler beats, are QRS complexes during a WCT that are identical to the sinus QRS complex (waveform 5). The term "capture beat" implies that the normal conduction system has momentarily "captured" control of ventricular activation from the VT focus.

Fusion beats and capture beats are more commonly seen when the tachycardia rate is slower.

QRS morphology — A definitive diagnosis of VT or SVT cannot be made based upon QRS morphology alone. Further ECG evaluation involves assessment of the morphology of the QRS complex. (See 'Diagnosis' below.)

Analysis of QRS morphology is based upon an understanding of the relationships between the sites of tachycardia origin, ventricular activation patterns, and the resulting morphologies of the QRS complex in the 12 standard ECG leads.

Subtle, non-rate-related fluctuations or variations in QRS and ST-T wave configuration suggest VT and may reflect variations in the VT reentrant circuit within the myocardium as well as a subtle difference in the activation sequence of the myocardium reflecting activation that bypasses the normal conduction system. AV dissociation can cause variability in the ST segment and T wave morphology. By contrast, SVT, because it follows a fixed conduction pathway to and through the ventricular myocardium, is characterized by uniformity of QRS and ST-T shape unless the rate changes.

DIAGNOSIS

Most patients with WCT will have some, but not all, of the ECG features favoring ventricular tachycardia (VT). There is no single criterion or combination of criteria that provides complete diagnostic accuracy in evaluating a WCT . It is typically necessary, therefore, to integrate multiple ECG findings into a diagnostic strategy. Of the several strategies that have been proposed, the Brugada criteria are the most widely known and reflect the initial approach that we generally use [8,14,15]. (See 'Brugada criteria' below.)

However, because the diagnosis of a WCT cannot always be made with complete certainty even when using multiple ECG criteria, an unknown or uncertain rhythm should be presumed to be VT in the absence of contrary evidence [16]. VT is far more common than supraventricular tachycardia (SVT), by a factor of four in unselected populations and by as much as 10-fold in patients with prior myocardial infarction. Additionally, presuming VT guards against inappropriate and potentially dangerous therapies directed at an SVT, which can precipitate hemodynamic deterioration in patients with VT, and treatment of SVT as if it were VT is safe and frequently effective in restoring sinus rhythm. (See "Wide QRS complex tachycardias: Approach to management", section on 'Management'.)

An algorithmic approach to the diagnosis of a WCT will be reviewed here, with emphasis on the distinction between VT and SVT. The approach to a narrow QRS complex tachycardia is discussed separately. (See "Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation".)

Brugada criteria — The Brugada criteria, the most commonly used ECG algorithm to differentiate VT from SVT, are a stepwise approach in which four criteria for VT are sequentially assessed (algorithm 2) [14]. If any of the four criteria are satisfied, the diagnosis of VT is made, and if none are fulfilled, an SVT is diagnosed. An exception is an antidromic AV reentrant tachycardia (AVRT) in Wolff-Parkinson-White syndrome.

The steps are as follows:

- Leads V1-V6 are inspected to detect an RS complex (ie, a biphasic QRS complex with both positive R and negative S wave components). If there are no RS complexes, concordance is present, and the diagnosis of VT can be made (waveform 2). (See 'Concordance' above.)

- If an RS complex is present, measure the interval between the onset of the R wave and the nadir of the S wave (RS interval). If the longest RS interval in any lead is >100 milliseconds and the R wave is wider than the S wave, the diagnosis of VT can be made. This reflects that with VT, the entire QRS complex is wide and abnormal (even the initial R wave portion), while aberration is due to a terminal delay resulting in a wider terminal portion of the QRS complex (ie, S wave).

  This criterion, however, may be limited in the presence of an underlying diffuse cardiomyopathy, as in this situation, even initial ventricular activation may be slow and abnormal.

- If the longest RS interval is <100 milliseconds, the presence or absence of AV dissociation is assessed. If AV dissociation is seen, the diagnosis of VT is made. (See 'AV dissociation' above.)

- If AV dissociation cannot clearly be demonstrated, the QRS morphology criteria for V1-positive and V1-negative wide QRS complex tachycardias are considered. QRS morphology criteria consistent with VT must be present in leads V1 or V2 and in lead V6 to diagnose VT. If either the V1-V2 or the V6 criteria are not consistent with VT, an SVT is assumed. (See 'QRS morphology' above.)

Alternative approaches — Several alternative approaches have been proposed in the literature, although none are as commonly used as the Brugada criteria. These approaches are generally best performed in consultation with an electrophysiologist with expertise in the diagnosis of WCT.

- An alternative algorithm (Vereckei approach) uses a stepwise approach similar to the Brugada criteria but includes different ECG features (algorithm 3) [15]. Two unique features of this algorithm include:

  - An initial R wave in aVR (diagnostic of VT).

  - Vi:Vt ratio. Vi and Vt are the magnitude of voltage change in the initial and terminal 40 milliseconds of a QRS, respectively. Vi and Vt should be measured from the same biphasic or multiphasic QRS complex. A Vi:Vt ratio ≤1 is diagnostic of VT.

One study of 51 patients with WCT induced during electrophysiology study showed equivalent sensitivity for diagnosis between the Vereckei and Brugada approaches (89 versus 90 percent), but the Vereckei approach yielded significantly fewer incorrect diagnoses following the first step (2 versus 27 percent) and was slightly faster to perform (9.1 versus 9.9 seconds) [17].

- An additional algorithm (limb leads algorithm) diagnoses VT in the presence of at least one of the following criteria:
  - Monophasic R wave in lead aVR
  - Predominantly negative QRS complex in leads I, II, III
  - Opposing QRS complex in the limb leads (concordant monophasic QRS complex in all inferior leads [II, III, aVF] and concordant monophasic QRS complex in two or three of the remaining limb leads [I, aVR, aVL] with opposite polarity of the inferior leads)

  Among a series of 528 wide QRS complex tachycardias (397 VT, 131 SVT), the limb leads algorithm had similar overall accuracy compared to other algorithms (88 percent versus 85 percent with Brugada criteria and 88 percent with Vereckei approach); in general, the limb leads algorithm had lower sensitivity but higher specificity for diagnosing VT [18].

- A Bayesian approach utilizes likelihood ratios (LR) for six ECG criteria [8]. Patients are presumed to start with a "prior odds ratio" of 4.0 (4:1) in favor of VT. As each criterion is evaluated sequentially, the associated LR is multiplied by the prior odds ratio to calculate the new probability of VT. The final odds ratio (posterior probability) is considered consistent with VT if the value is ≥1.0 and SVT if the value is <1.0.

- Intravenous adenosine may be administered as both a diagnostic and potentially therapeutic agent. (See "Wide QRS complex tachycardias: Approach to management", section on 'Pharmacologic interventions'.)

VT versus AVRT — Differentiation between VT and antidromic AVRT is particularly difficult. Because there is direct myocardial activation (since ventricular activation begins outside of the normal conduction system) in both VT and antidromic AVRT, many of the standard criteria are not able to discriminate antidromic AVRT from VT. The clinical significance of this problem is often limited, however, because pre-excitation is an uncommon cause of WCT (6 percent in one series), particularly if other clinical factors (eg, age, underlying heart disease) suggest VT [11].

For cases in which pre-excitation is thought to be likely (such as a young patient without structural heart disease, or a patient with a known accessory pathway), a separate algorithm

was developed that consists of the following three steps (algorithm 4) [14]:

- The predominant polarity of the QRS complex in leads V4 through V6 is defined either as positive or negative. If predominantly negative, the diagnosis of VT can be made.
- If the polarity of the QRS complex is predominantly positive in V4 through V6, the ECG should be examined for the presence of a qR complex in one or more of the precordial leads V2 through V6. If a qR complex can be identified, VT can be diagnosed.
- If a qR wave in leads V2 through V6 is absent, the AV relationship is then evaluated (AV dissociation). If a 1:1 AV relationship is not present and there are more QRS complexes present than P waves, VT can be diagnosed.

When any of these criteria are met, VT is likely. However, if the ECG does not display any of the three morphologic characteristics diagnostic of VT in this algorithm, the diagnosis of antidromic AVRT must be considered. Importantly, the QRS complex morphology and ST-T waves are uniform with AVRT as every impulse to the ventricle is conducted through the same accessory pathway.

Algorithm performance — Algorithms often perform well in initial reports. However, such studies include selected populations and experienced ECG analysts.

- In its initial description, the reported sensitivity and specificity of the Brugada criteria were 98.7 and 96.5 percent, respectively [14]. However, in two subsequent reports in which a total of nine clinicians (two cardiologists, two emergency department clinicians, and five internists) used these criteria in interpreting a total of 168 WCTs that had been diagnosed with electrophysiologic testing, the sensitivity ranged from 79 to 92 percent and specificity ranged from 43 to 70 percent [19,20].
- In a comparison of the Brugada criteria and the Bayesian approach, the two approaches performed similarly, with sensitivities of 92 and 97 percent and specificities of 44 and 56 percent, respectively [20].
- In the initial description of the alternative algorithm (Vereckei approach), it was significantly more accurate than the Brugada criteria [15]. Further study is necessary to confirm both the overall accuracy of this approach and its superiority to the Brugada criteria.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Arrhythmias in adults" and "Society guideline links: Ventricular arrhythmias" and "Society guideline links: Supraventricular arrhythmias".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Ventricular tachycardia (The Basics)")

SUMMARY AND RECOMMENDATIONS

- The first priority when evaluating a patient with a wide QRS complex tachycardia (WCT) is an immediate assessment of patient stability, including the patient's symptoms, vital signs, and the level of consciousness. An unstable patient will have evidence of hemodynamic compromise, such as hypotension, altered mental status, chest pain, or heart failure.
  - A patient who is unresponsive or pulseless should be treated according to standard advanced cardiac life support (ACLS) algorithms (algorithm 5). (See 'Assessment of hemodynamic stability' above and "Advanced cardiac life support (ACLS) in adults".)
  - For a patient who is unstable, with evidence of hemodynamic compromise but who remains conscious, emergency synchronized cardioversion (after intravenous sedation, whenever possible) is the treatment of choice regardless of the mechanism of the arrhythmia. (See 'Assessment of hemodynamic stability' above and "Wide QRS complex tachycardias: Approach to management", section on 'Unstable patients'.)

- In a stable patient, or following cardioversion to stabilize an unstable patient, our initial approach includes the following:

  - A succinct history and physical examination, focusing on the presence of structural heart disease, especially coronary heart disease and/or a previous myocardial infarction, as well as the history of any arrhythmias and the presence of a pacemaker or implantable cardioverter-defibrillator. (See 'History' above and 'Physical examination' above.)

  - Review of the patient's medications for drugs that may be proarrhythmic (by prolonging the QT interval or promoting electrolyte disturbances). (See 'Medications' above.)

  - Initial review of the ECG, with a focus on whether the rhythm is regular or irregular, the width of the QRS complex, the presence or absence of AV dissociation, and any preexisting arrhythmias. (See 'ECG initial impressions' above.)

  - Ancillary testing including serum electrolyte levels, cardiac troponin, and a chest radiograph in all patients. (See 'Ancillary testing' above.)

- In most patients with WCT, a probable diagnosis (ventricular tachycardia [VT] or supraventricular tachycardia [SVT]) may be made by closely reviewing the ECG, although definitive diagnosis is not always possible and may be time consuming. The standard initial approach to ECG interpretation includes an assessment of rate, regularity, axis, QRS duration, and QRS morphology. (See 'Evaluation of the electrocardiogram' above.)

- Most patients with WCT will have some, but not all, of the ECG features favoring VT. There is no single criterion or combination of criteria that provides complete diagnostic accuracy in evaluating a WCT, even when employing an algorithmic approach to the diagnosis of a WCT. (See 'Diagnosis' above.)

  - We prefer using the Brugada algorithm to evaluate for VT versus SVT in patients with WCT when the diagnosis is uncertain and when patient hemodynamic stability allows.

  - ECG features consistent with VT include concordance (waveform 2), AV dissociation, fusion/capture beats, and specific QRS morphologies.

  - The absence of the historical or ECG features of VT does not confirm a diagnosis of SVT. The diagnosis of SVT should be considered primarily in young patients, whose hearts are structurally normal, in whom none of the historical (eg, family history of sudden

cardiac death), physical, or ECG criteria supporting VT are present, or in patients with a history of SVT with a similar presentation.

- When the diagnosis of a WCT is uncertain despite a careful initial evaluation, the patient should be treated as if the rhythm is VT. (See "Wide QRS complex tachycardias: Approach to management".)

ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Philip Podrid, MD, and Leonard Ganz, MD, FHRS, FACC, who contributed to an earlier version of this topic review.

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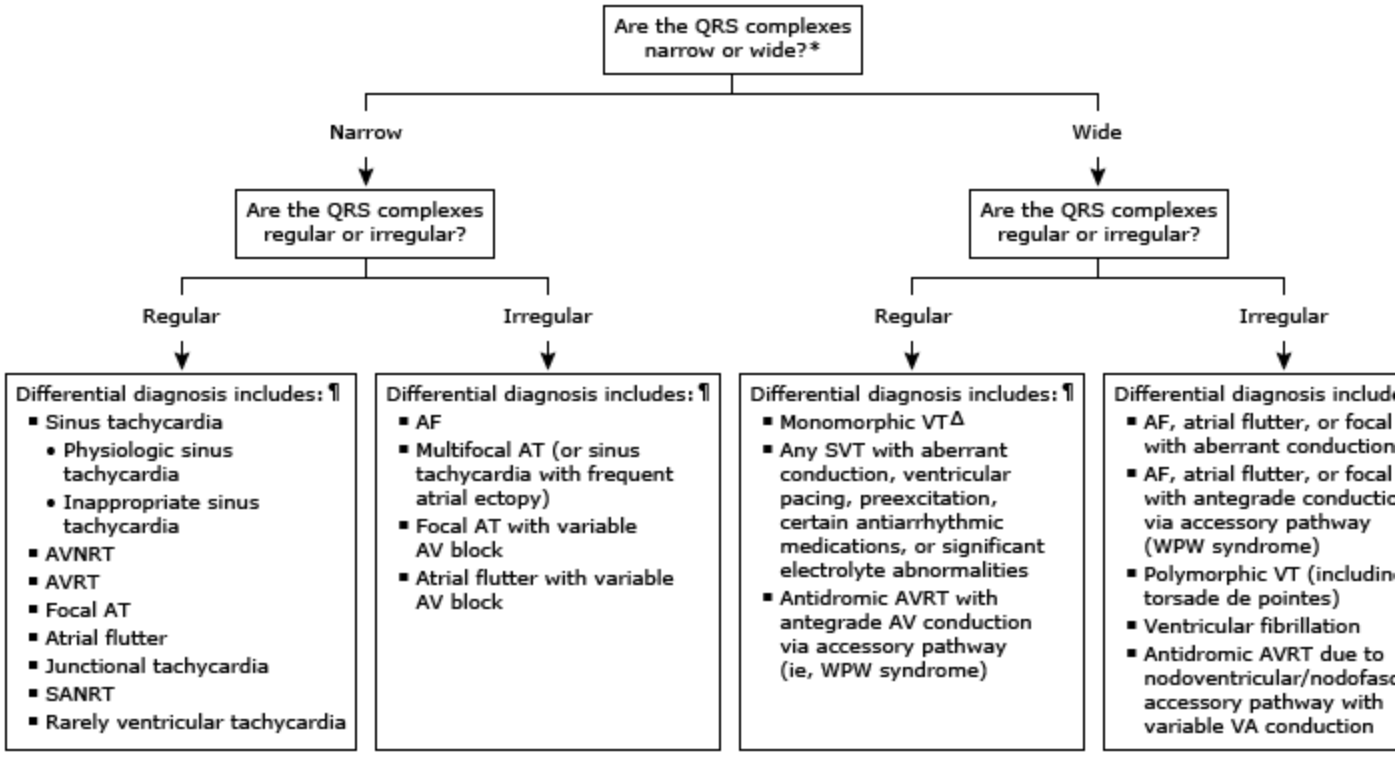
Topic 920 Version 47.0

GRAPHICS

Algorithm for the initial ECG review and differential diagnosis of tachycardia



ECG: electrocardiogram; AVNRT: atrioventricular nodal reentrant tachycardia; AVRT: atrioventricular reciprocating (bypass-tract mediated) tachycardia; AT: atrial tachycardia; SANRT: sinoatrial nodal reentrant tachycardia; AF: atrial fibrillation; AV: atrioventricular; VT: ventricular tachycardia; SVT: supraventricular tachycardia; WPW: Wolff-Parkinson-White.

* A narrow QRS complex is <120 milliseconds in duration, whereas a wide QRS complex is ≥120 milliseconds in duration.

¶ Refer to UpToDate topic reviews for additional details on specific ECG findings and management of individual arrhythmias.

Δ Monomorphic VT accounts for 80% of wide QRS complex tachycardias; refer to UpToDate topic on diagnosis of wide QRS complex tachycardias for additional information on discriminating VT from SVT.

Graphic 117571 Version 3.0

Some reported causes and potentiators of the long QT syndrome

Congenital

- Jervell and Lange-Nielsen syndrome (including "channelopathies")
- Romano-Ward syndrome
- Idiopathic

Acquired

Metabolic disorders

- Hypokalemia
- Hypomagnesemia
- Hypocalcemia
- Starvation
- Anorexia nervosa
- Liquid protein diets
- Hypothyroidism

Bradyarrhythmias

- Sinus node dysfunction
- AV block: Second or third degree

Other factors

- Myocardial ischemia or infarction, especially with prominent T-wave inversions
- Intracranial disease
- HIV infection
- Hypothermia
- Toxic exposure: Organophosphate insecticides

Androgen deprivation therapy

- GnRH agonist/antagonist therapy
- Bilateral surgical orchiectomy

Diuretic therapy via electrolyte disorders particularly hypokalemia and hypomagnesemia

Herbs

- Cinchona (contains quinine), iboga (ibogaine), licorice extract in overuse via electrolyte disturbances

Medications*

High risk

- Adagrasib
- Ajmaline¶
- Amiodarone△
- Arsenic trioxide
- Astemizole◇
- Bedaquline
- Bepridil◇
- Chlorpromazine
- Cisaparide (restricted availability)
- Delamanid¶
- Disopyramide△
- Dofetilide
- Dronedarone
- Haloperidol (IV)
- Ibutilide
- Ivosidenib
- Lenvatinib
- Levoketoconazole
- Methadone
- Mobocertinib
- Papavirine (intracoronary)
- Procainamide
- Quinidine
- Quinine
- Selpercatinib
- Sertindole¶
- Sotalol
- Terfenadine◇
- Vandetanib
- Vernakalant¶
- Ziprasidone

Moderate risk

- Amisulpride¶ (oral)§
- Azithromycin
- Capecitabine
- Carbetocin¶
- Certinib
- Encorafenib
- Entrectinib
- Erythromycin
- Escitalopram
- Etelcalcetide
- Levetiracetam
- Levofloxacin (systemic)
- Lofexidine
- Quetiapine
- Quizartinib
- Ribociclib
- Risperidone
- Saquinavir

- Chloroquine
- Citalopram
- Clarithromycin
- Clofazimine
- Clomipramine[¥]
- Clozapine
- Crizotinib
- Dabrafenib
- Dasatinib
- Deslurane
- Domperidone[¶]
- Doxepin[¥]
- Doxifluridine[¶]
- Droperidol
- Fexinidazole
- Flecainide
- Floxuridine
- Fluconazole
- Fluorouracil (systemic)
- Flupentixol[¶]
- Gabobenate dimeglumine
- Gemifloxacin[¶]
- Gilteritinib
- Halofantrine
- Haloperidol (oral)
- Imipramine[¥]
- Inotuzumab ozogamacin
- Isoflurane
- Meglumine antimoniate
- Midostaurin
- Moxifloxacin
- Nilotinib
- Olanzapine
- Ondansetron (IV > oral)
- Osimertinib
- Oxytocin
- Pazopanib
- Pentamidine
- Pilsicainide[◇]
- Pimozide
- Piperaquine
- Probucol[◇]
- Propafenone
- Propofol
- Sevoflurane
- Sparfloxacin[¶]
- Sunitinib
- Tegafur[¶]
- Terbutaline
- Thioridazine
- Toremifene
- Vemurafenib
- Voriconazole

Low risk[‡]

- Albuterol
- Alfuzosin
- Amisulpride (IV)[§]
- Amitriptyline[¥]
- Anagrelide
- Apomorphine
- Arformoterol
- Artemether-lumefantrine
- Asenapine
- Atomoxetine
- Benperidol
- Bilastine[¶]
- Bosutinib
- Bromperidol
- Buprenorphine[†]
- Buserelin
- Ciprofloxacin (Systemic)
- Cocaine (Topical)
- Degarelix
- Desipramine[¥]
- Fingolimod
- Fluoxetine
- Fluphenazine
- Fluvoxamine
- Formoterol
- Foscarnet
- Fostemsavir
- Gadofosveset
- Gepirone
- Glasdegib
- Goserelin
- Granisetron
- Hydroxychloroquine (rare reports)
- Hydroxyzine
- Iloperidone
- Indacaterol
- Itraconazole
- Ketoconazole (systemic)
- Lacidipine
- Lapatinib
- Macimorelin
- Maprotiline
- Mefloquine
- Mequitazine
- Metoclopramide (rare reports)
- Metronidazole (systemic)
- Mifepristone
- Mirtazapine
- Mizolastine
- Nelfinavir
- Norfloxacin
- Nortriptyline[¥]
- Ofloxacin (systemic)
- Olodaterol
- Osilodrostat
- Oxaliplatin
- Ozanimod[ΔΔ]
- Pacritinib
- Paliperidone
- Panobinostat
- Promazine
- Radotinib
- Ranolazine (due to bradycardia)
- Relugolix
- Rilpivirine
- Romidepsin
- Roxithromycin
- Salmeterol
- Sertraline
- Siponimod
- Solifenacin
- Sorafenib
- Sulpiride
- Tacrolimus (systemic)
- Tamoxifen
- Telavancin
- Teneligliptin
- Tetrabenazine
- Trazodone
- Triclabendazole

▪ Deutetrabenazine ▪ Dexmedetomidine** ▪ Dolasetron ▪ Donepezil ▪ Efavirenz ▪ Eliglustat ▪ Eribulin ▪ Etrasimod ▪ Ezogabine	▪ Lefamulin ▪ Leuprolide ▪ Leuprolide-norethindrone ▪ Levalbuterol ▪ Levomepromazine (methotrimeprazine) ▪ Levomethadone ▪ Lithium ▪ Loperamide¶¶ in overdose ▪ Lopinavir	▪ Paroxetine ▪ Pasireotide ▪ Pefloxacin ▪ Periciazine¶ ▪ Pimavanserin ▪ Pipamperone ▪ Pitolisant ▪ Ponesimod ▪ Primaquine	▪ Triptorelin ▪ Tropisetron¶ ▪ Vardenafil ▪ Vilanterol ▪ Vinflunine ▪ Voclosporin ▪ Vorinostat ▪ Zuclopenthixol

This is not a complete list of all corrected QT interval (QTc)-prolonging drugs and does not include drugs with either a minor degree or isolated association(s) with QTc prolongation that appear to be safe in most patients but may need to be avoided in patients with congenital long QT syndrome depending upon clinical circumstances. A more complete list of such drugs is available at the CredibleMeds website. For clinical use and precautions related to medications and drug interactions, refer to the UpToDate topic review of acquired long QT syndrome discussion of medications and the drug interactions program.

AV: atrioventricular; IV: intravenous; QTc: rate-corrected QT interval on the electrocardiogram.

* Classifications provided by Lexicomp according to US Food & Drug Administration guidance: Clinical Evaluation of QT/QTc Interval Prolongation and Proarrhythic Potential for Non-Antiarrhythmic Drugs – Questions and Answers; Guidance for Industry US Food and Drug Administration, June 2017 (revision 2) available at: https://www.fda.gov/downloads/Drugs/GuidanceComplianceRegulatoryInformation/Guidances/UCM073161.pdf as updated August 8, 2023 (https://www.fda.gov/media/170814/download) with additional data from CredibleMeds QT drugs list[1,2]. The use of other classification criteria may lead to some agents being classified differently by other sources.

¶ Not available in the United States.

Δ In contrast with other class III antiarrhythmic drugs, amiodarone is rarely associated with torsades de pointes; refer to accompanying text within UpToDate topic reviews of acquired long QT syndrome.

◇ Withdrawn from market in most countries due to adverse cardiovascular effects.

§ IV amisulpride antiemetic use is associated with less QTc prolongation than the higher doses administered orally as an antipsychotic.

¥ Some other cyclic antidepressants (ie, amoxapine, protriptyline, trimipramine) may also prolong the QT interval, but data are insufficient to identify level of risk with confidence; refer to UpToDate content on cyclic antidepressant pharmacology, administration, and side effects.

‡ The "low risk" category includes drugs with limited evidence of clinically significant QTc prolongation or TdP risk; many of these drugs have label warnings regarding possible QTc effects or recommendations to avoid use or increase ECG monitoring when combined with other QTc prolonging drugs.

† Rarely associated with significant QTc prolongation at usual doses for treatment of opioid use disorder, making buprenorphine a suitable alternative for patients with methadone-associated QTc prolongation. Refer to UpToDate clinical topic reviews.

** The United States FDA labeling for the sublingual preparation of dexmedetomidine warns against use in patients at elevated risk for QTc prolongation. Both intravenous (ie, sedative) and sublingual formulations of dexmedetomidine have a low risk of QTc prolongation and have **not** been implicated in TdP.

¶¶ Over-the-counter; available without a prescription.

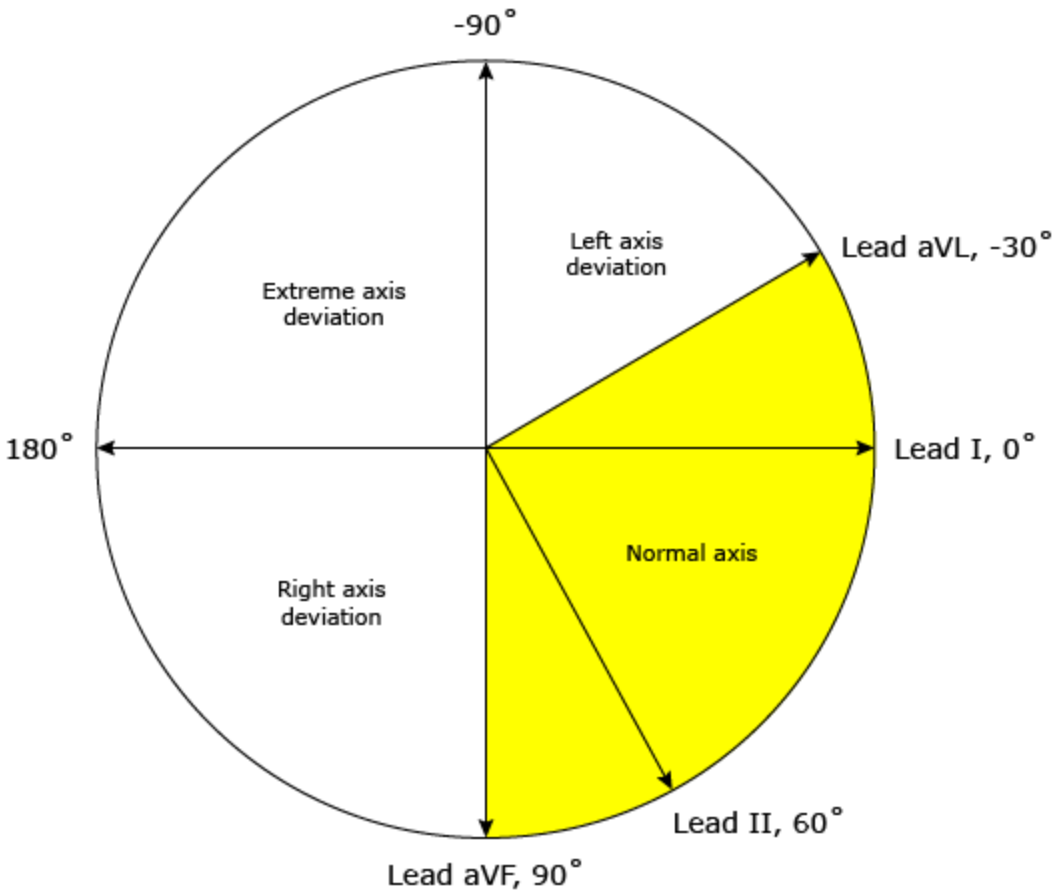
ΔΔ Not associated with significant QTc prolongation in healthy persons. Refer to UpToDate clinical topic for potential adverse cardiovascular (CV) effects in patients with CV disease.

Data from:

1. *Lexicomp Online. Copyright ©1978-2024 Lexicomp, Inc. All Rights Reserved.*
2. *CredibleMeds QT drugs list website sponsored by Science Foundation of the University of Arizona. Available at http://crediblemeds.org/.*

Graphic 57431 Version 151.0

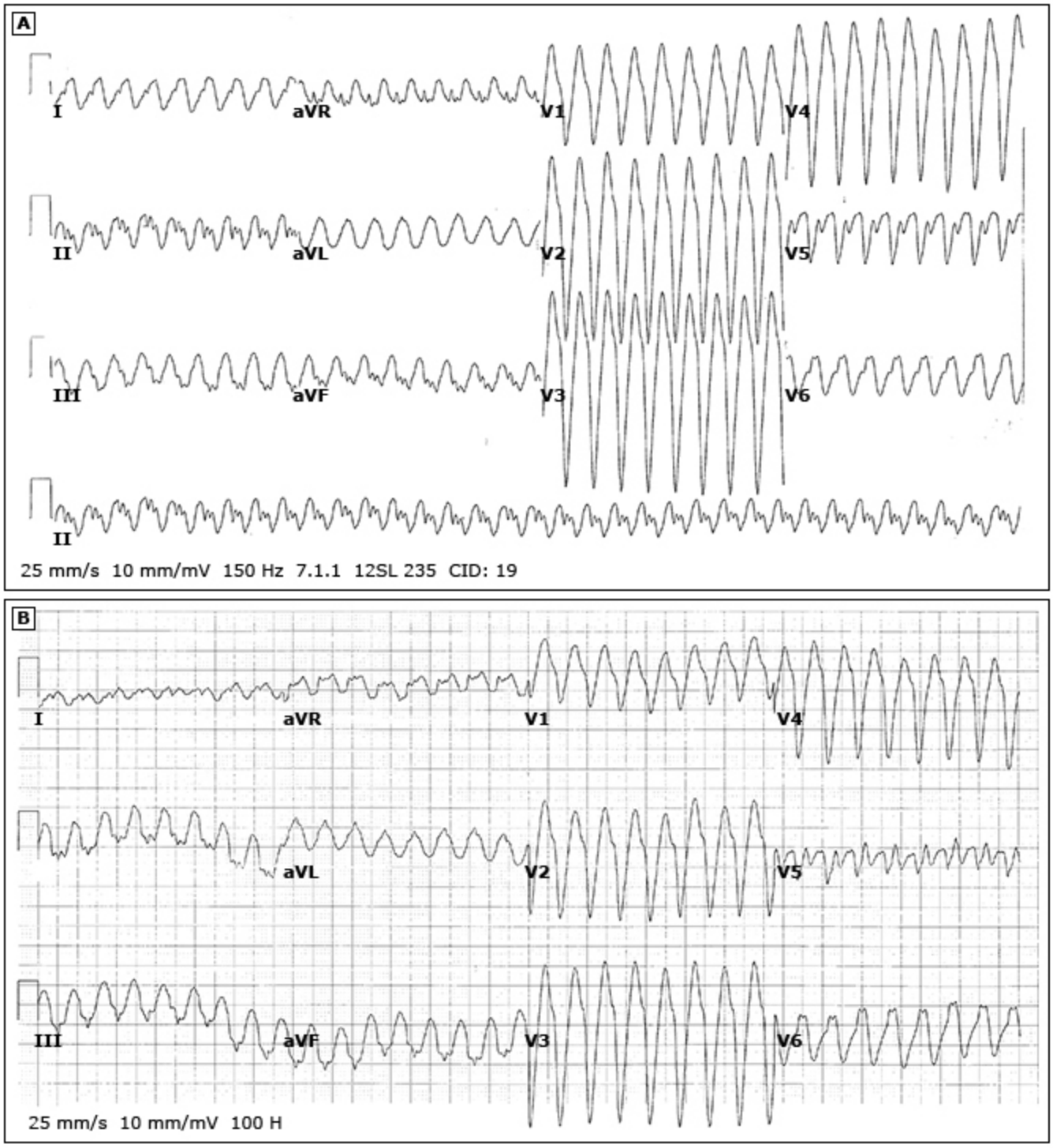
Calculation of frontal plane axis



If the QRS complex is positive in leads I and II, it falls between -30 and 90° and is normal, as indicated by the yellow area. If the QRS complex is negative in I and positive in aVF, there is right axis deviation. If the QRS complex is positive in I and negative in II, there is left axis deviation. If the QRS complex is negative in I and aVF, there is extreme axis deviation.

Graphic 85682 Version 1.0

Ventricular tachycardia

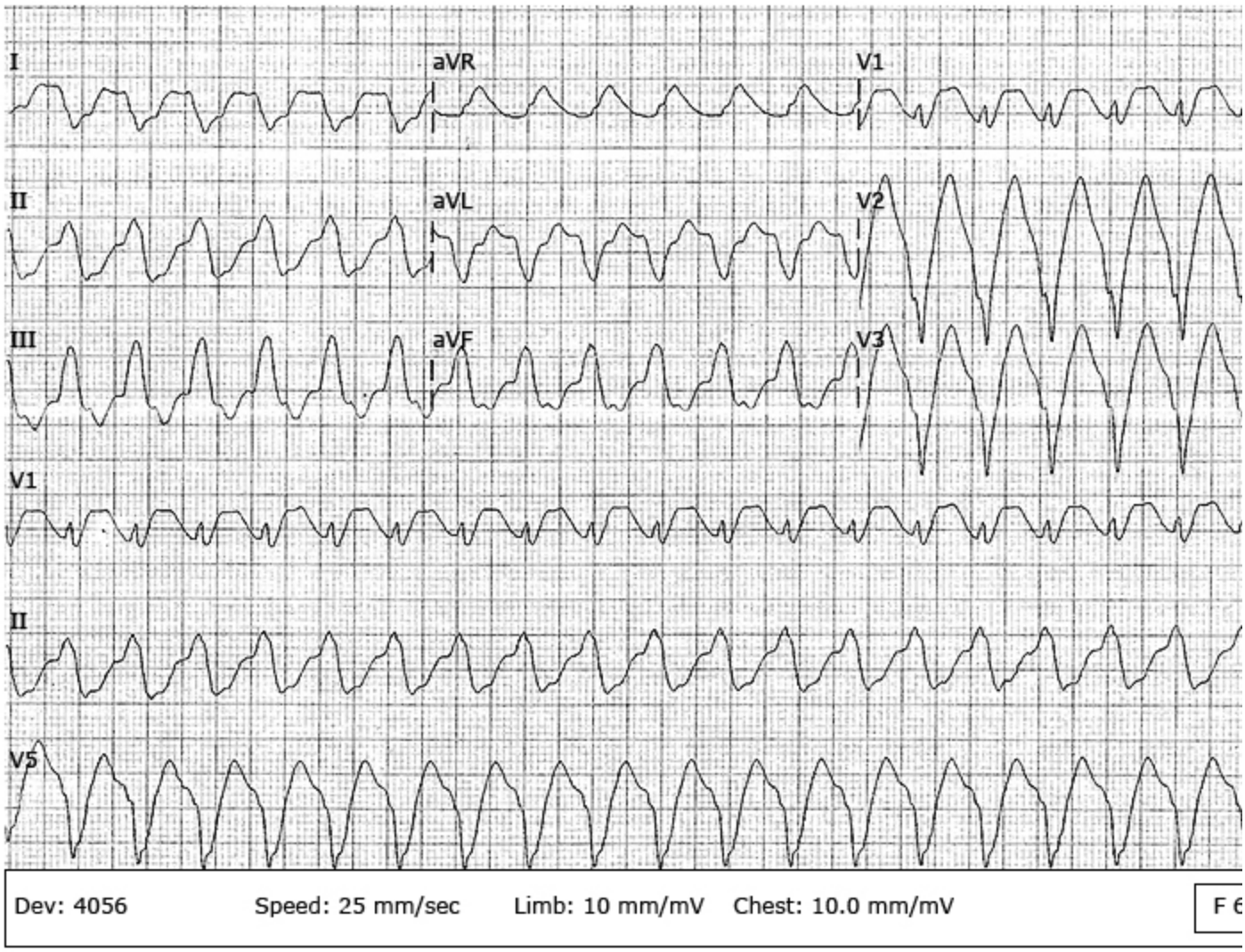


An example of concordance/absence of rS complex and initial R wave in AVR.

Courtesy of Leonard Ganz, MD.

Graphic 129901 Version 1.0

Wide complex tachycardia with wide concordance

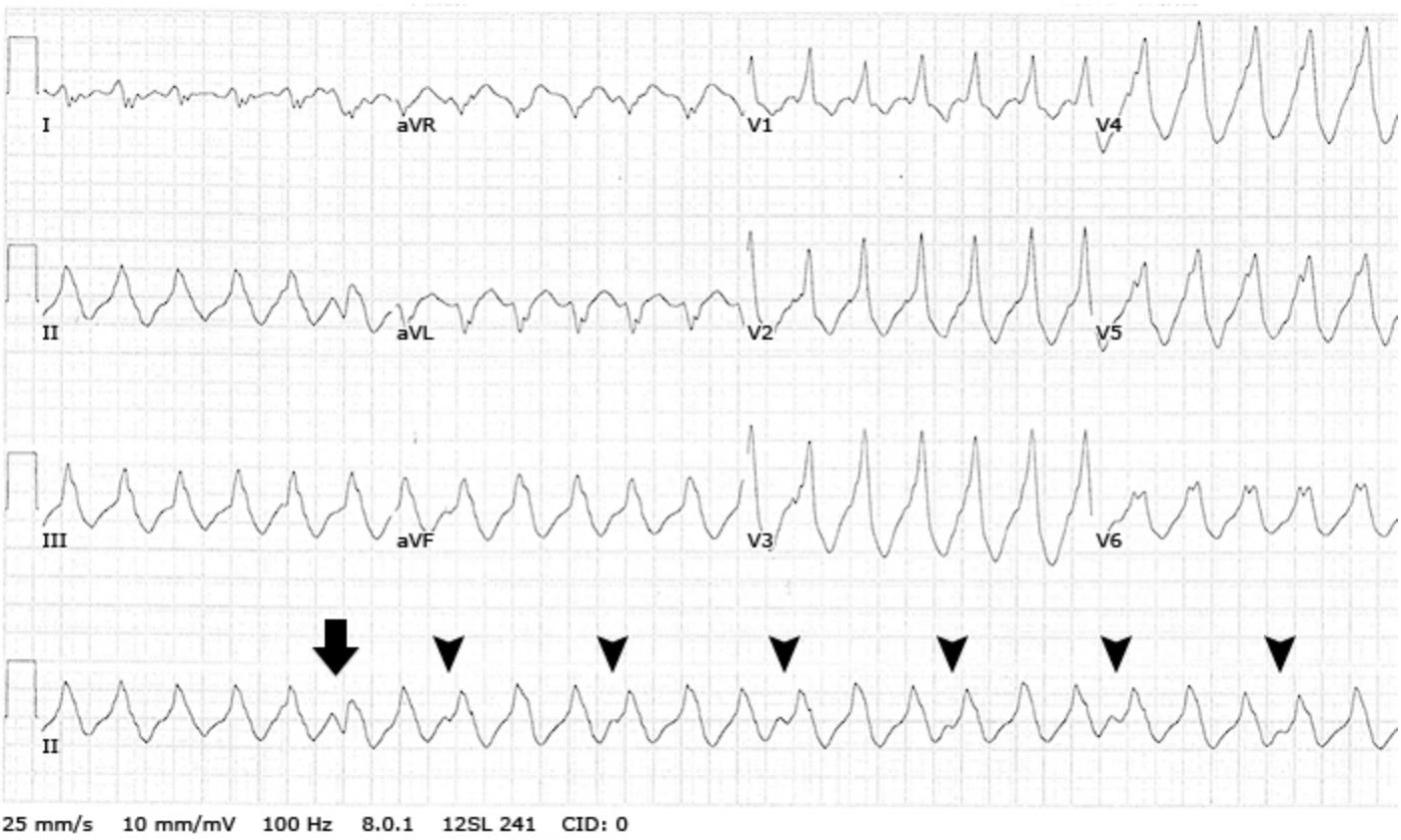


Extreme tachycardia with wide complex.

Courtesy of Leonard Ganz, MD.

Graphic 129926 Version 2.0

Ventricular tachycardia with positive concordance



Monophasic R waves are noted in all the precordial leads; this is positive concordance. Dissociated P waves are evident in the lead II rhythm strip and in lead aVR. Thick arrow shows artifact. Arrowheads show P waves, marching out at approximately 54 beats per minute. P waves are less evident on the left side of the lead II rhythm strip.

Courtesy of Leonard Ganz, MD.

Graphic 129857 Version 1.0

Atrioventricular dissociation

Independent activation of the atria and ventricles results in no fixed relationship between the P waves (arrows) and the QRS complexes; the PR intervals are variable in a random fashion.

Graphic 52123 Version 2.0

Normal rhythm strip

Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Fusion beats

The rhythm strip in a patient with sustained ventricular tachycardia shows a fusion beat and a capture beat. The fusion beat occurs when a supraventricular impulse (following the first P wave) causes ventricular activation, which fuses with the complex originating in the ventricle, producing a hybrid complex. The complex following the second P wave has the appearance of a normal QRS complex and is known as a capture beat.

Graphic 72600 Version 4.0

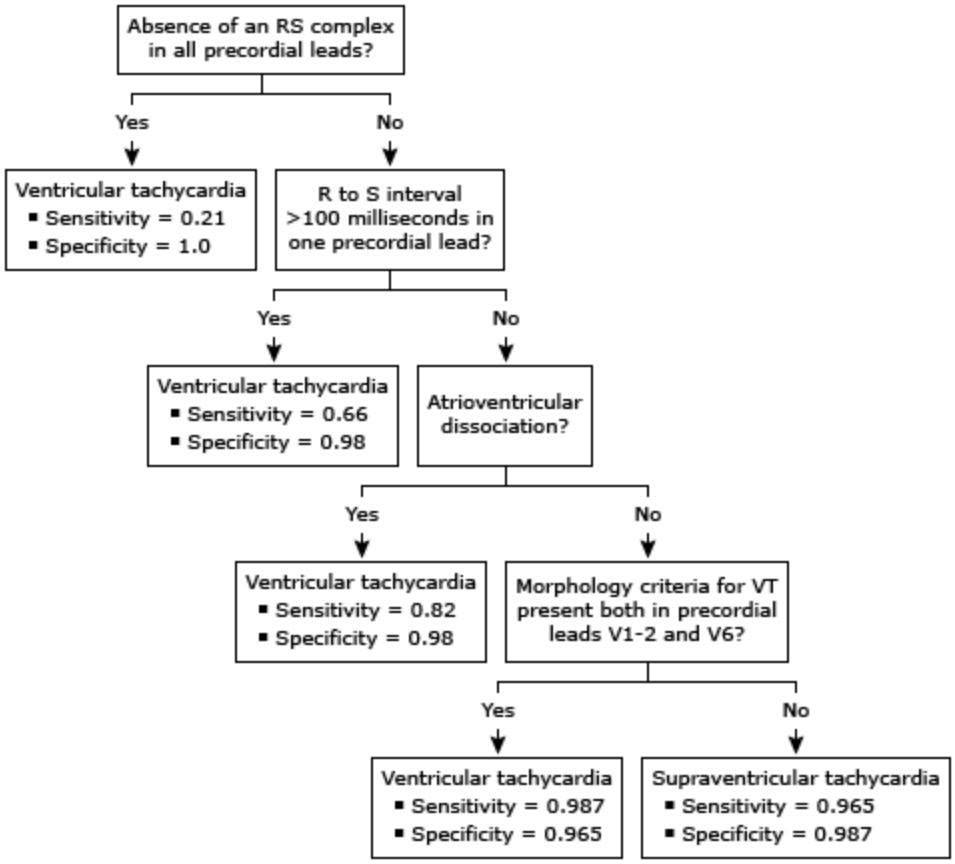
Normal rhythm strip

Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

Brugada algorithm for the diagnosis of ventricular tachycardia or supraventricular tachycardia in a patient with wide QRS complex tachycardia



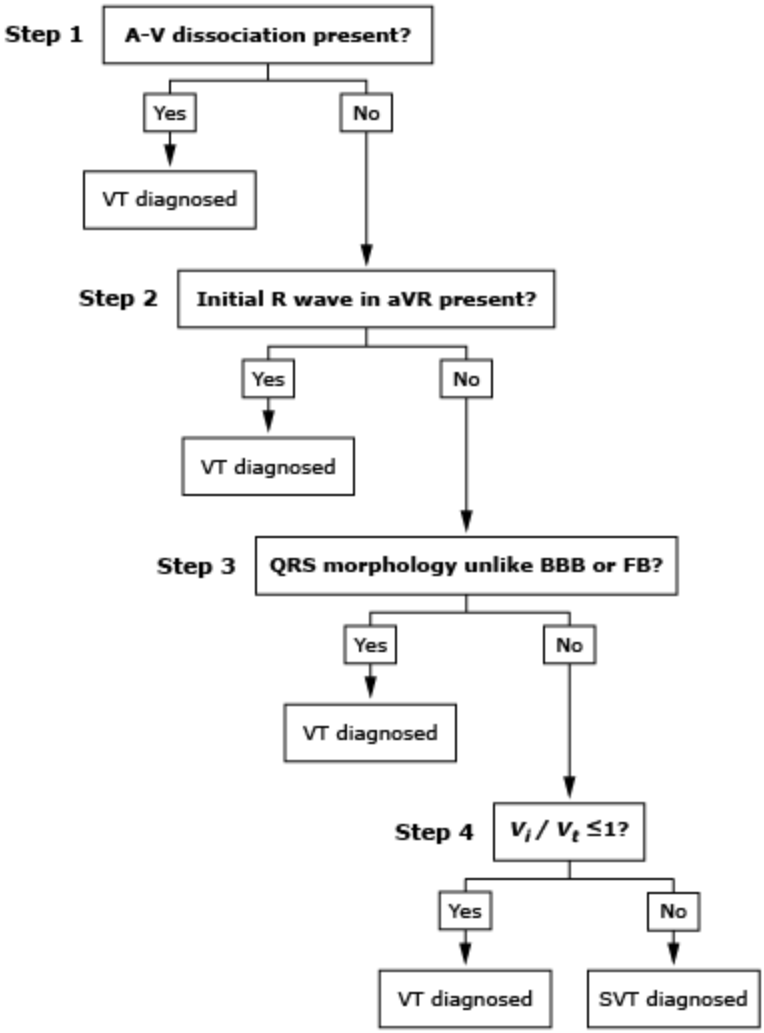
Brugada algorithm for distinguishing VT from SVT.

VT: ventricular tachycardia; SVT: supraventricular tachycardia.

Adapted from: 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.

Graphic 73159 Version 4.0

Vereckei algorithm for the diagnosis of ventricular tachycardia (VT) or supraventricular tachycardia (SVT) in a patient with wide QRS complex tachycardia

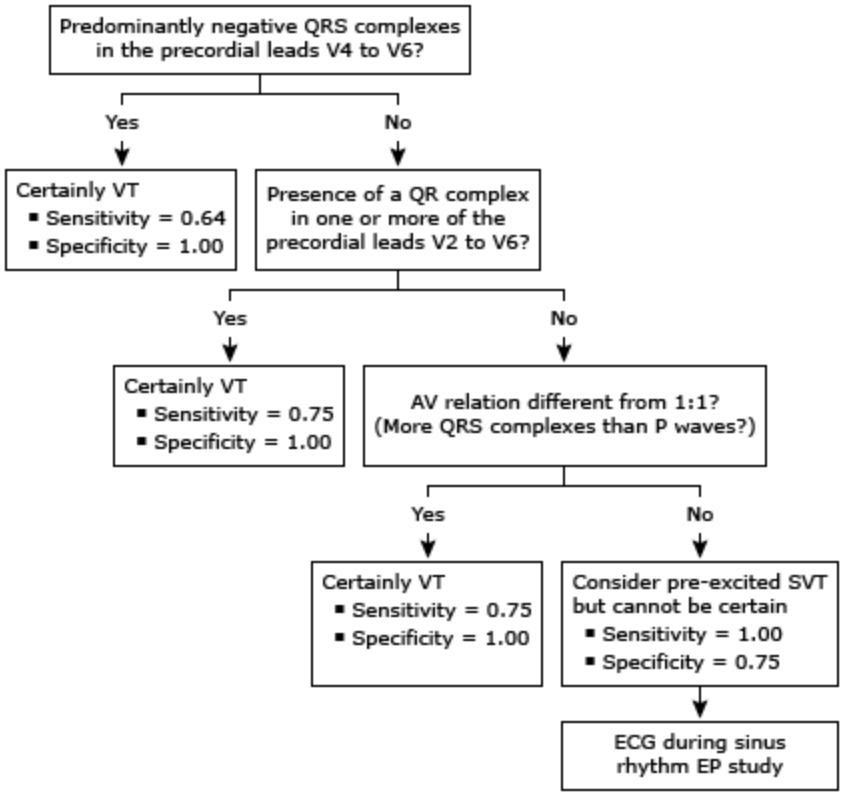


Vereckei algorithm for the diagnosis of wide complex tachycardias. In step 4, V_i represents the magnitude of voltage change in the initial 40 milliseconds of the QRS complex, while V_t represents the magnitude of voltage change in the terminal 40 milliseconds of the QRS complex. The initial V_i and terminal V_t voltages should be measured from the same biphasic or multiphasic QRS complex.

A-V: atrio-ventricular; BBB: bundle branch block; FB: fascicular block; SVT: supraventricular tachycardia; VT: ventricular tachycardia.

Graphic 79624 Version 6.0

Algorithm for the diagnosis of wide QRS tachycardia in the setting of ventricular pre-excitation

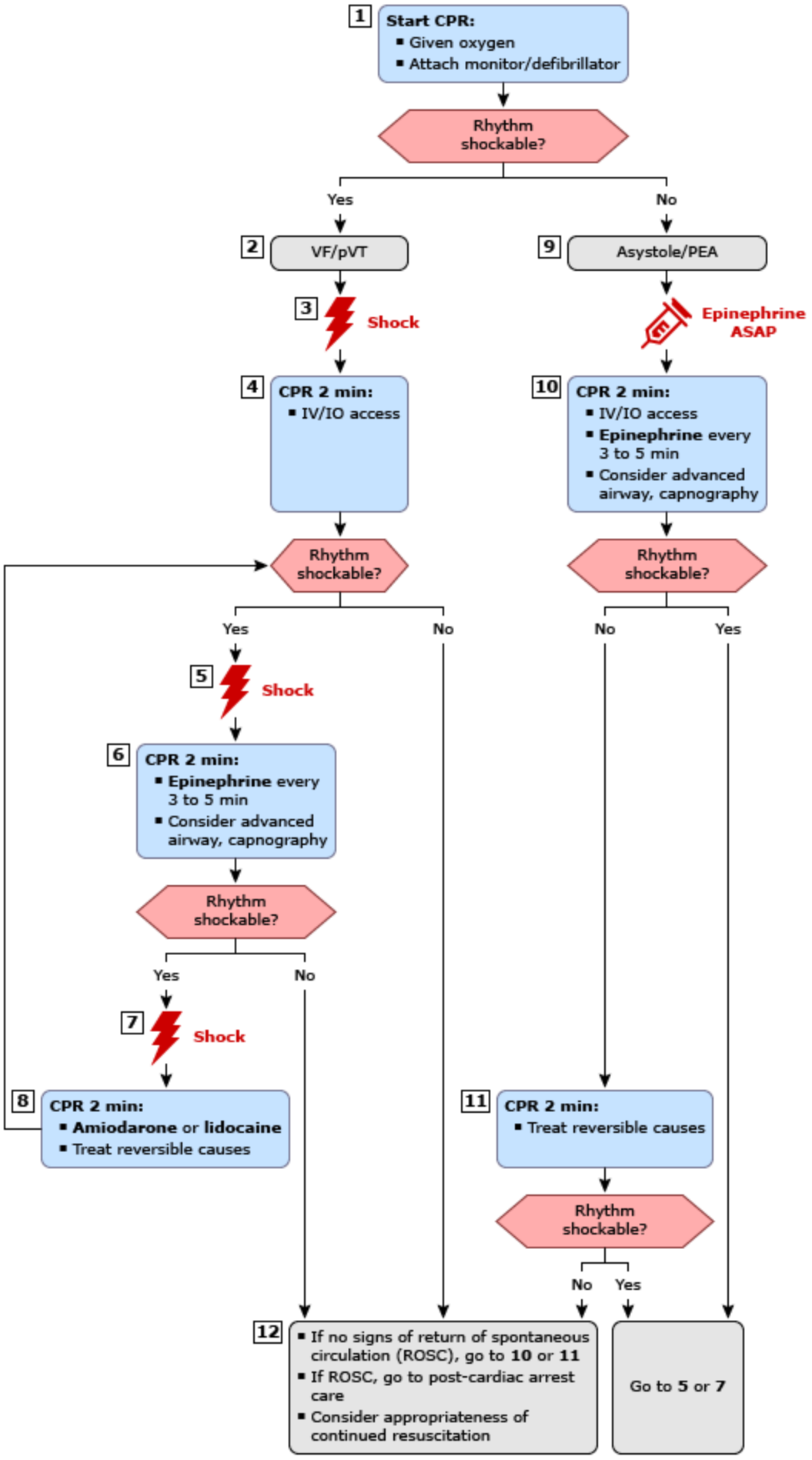


Algorithm for distinguishing VT from SVT in the setting of pre-excitation syndrome.

VT: ventricular tachycardia; AV: atrioventricular; SVT: supraventricular tachycardia; ECG: electrocardiography; EP: electrophysiologic.

Graphic 59336 Version 8.0

Adult cardiac arrest algorithm



CPR quali

- Push hard (at least 2 inch
fast (100 to 120/min) and
chest recoil.
- Minimize interruptions in
- Avoid excessive ventilatio
- Change compressor every
sooner if fatigued.
- If no advanced airway, 30
ventilation ratio.
- Quantitative waveform ca
  - If $PETCO_2$ is low or decr
CPR quality.

Shock energy for d

- **Biphasic:** Manufacturer r
(eg, initial dose of 120 to
unknown, use maximum
and subsequent doses sh
and higher doses may be
- **Monophasic:** 360 J.

Drug thera

- **Epinephrine IV/IO dos**
1 mg every 3 to 5 minute
- **Amiodarone IV/IO dos**
First dose: 300 mg bolus.
Second dose: 150 mg.
or
- **Lidocaine IV/IO dose:**
First dose: 1 to 1.5 mg/kg
Second dose: 0.5 to 0.75

Advanced ai

- Endotracheal intubation o
advanced airway.
- Waveform capnography o
confirm and monitor ET t
- Once advanced airway in
breath every 6 seconds (1
with continuous chest com

Return of spontaneous c

- Pulse and blood pressure.
- Abrupt sustained increase
(typically ≥40 mmHg).
- Spontaneous arterial pres
intra-arterial monitoring.

Reversible c

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Graphic 129983 Version 10.0