The differential diagnosis of wide QRS complex tachycardia

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ABSTRACT

Wide complex tachycardia is defined as a cardiac rhythm with a rate greater than 100 beats/min (bpm) and a QRS complex duration greater than 0.10 to 0.12 seconds (s) in the adult patient; wide complex tachycardia (WCT) in children is defined according to age-related metrics. The differential diagnosis of the WCT includes ventricular tachycardia and supraventricular tachycardia with aberrant intraventricular conduction, including both relatively benign and life-threatening dysrhythmias. This review focuses on the differential diagnosis of WCT with a discussion of strategies useful in making the appropriate diagnosis, when possible.

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1. Introduction

A wide complex tachycardia is defined as a cardiac rhythm with a rate greater than 100 beats/min (bpm) and a QRS complex duration greater than 0.10 to 0.12 seconds (s) in the adult patient; wide complex tachycardia (WCT) in children is defined according to age-related metrics [1]. WCTs can result from either supraventricular or ventricular rhythm disturbances. Thus, the differential diagnosis of the WCT includes ventricular tachycardia (VT) and supraventricular tachycardia (SVT) with aberrant intraventricular conduction (SVT-AC); aberrant conduction results from pre-existing or rate-related bundle branch block (BBB), ventricular pre-excitation, or dysfunction of the intraventricular conduction system due to various factors (toxic, metabolic, cardiac injury, infectious, etc.). From the electrocardiographic perspective, the rhythm differential diagnosis is broad, including both relatively benign and life-threatening dysrhythmias; furthermore, the diagnosis of specific rhythms is often not possible, at least initially during the early phase of evaluation and management [2,3].

In this article, we discuss the differential diagnosis of WCT, with a focused discussion of the various dysrhythmias encountered in this challenging electrocardiographic presentation. In addition, the electrocardiographic features useful in the distinction of VT from SVT-AC and a review of the various diagnostic algorithms are presented.

2. Electrocardiographic differential diagnosis

The differential diagnosis of the WCT includes VT and SVT-AC. Earlier literature suggests that VT is the most common diagnosis in the WCT patient [4]. While this is likely true in certain clinical settings, such as the coronary care unit, catheterization laboratory, and electrophysiology laboratory, the opposite is found in other clinical settings, such as the medical intensive care unit and emergency department (Fig. 1) [2,4,5,6]. VT includes both monomorphic and polymorphic ventricular tachycardia. The diagnostic possibilities within the SVT-AC category are numerous, including supraventricular dysrhythmias with BBB (sinus tachycardia [ST], atrial fibrillation [AF] or flutter, atrioventricular nodal re-entrant tachycardia [AVNRT]), with ventricular pre-excitation (Wolff-Parkinson-White syndrome [WPW]-related atrial fibrillation and antidromic atrioventricular re-entrant tachycardia [AVRT]), and with dysfunctional intraventricular conduction system (toxic, metabolic, etc.).

3. Electrophysiology of ventricular impulse conduction

The QRS complex morphology and width is determined by the electrical depolarization of the ventricles. During normal conduction, electrical impulses pass from the atrium through the atrioventricular node (AVN) to the rapidly conducting His bundle. The His bundle crosses into the ventricular myocardium, separating into the right and left bundles which initially course in the superficial subendocardium; the left bundle divides into anterior and posterior bundles. These bundles progressively divide to form an intricate Purkinje network within the
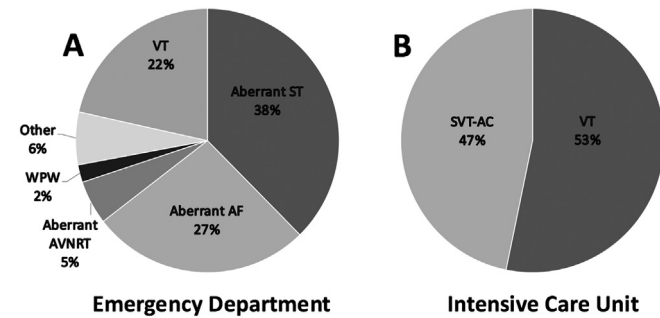
Fig. 1. Occurrence rates of specific dysrhythmias presenting as a wide complex tachycardia **A.** in the emergency department and **B.** in the intensive care unit [23]. Abbreviations: ventricular tachycardia (VT); sinus tachycardia (ST); atrial fibrillation (AF); atrioventricular nodal re-entrant tachycardia (AVNRT); Wolff-Parkinson-White Syndrome (WPW); and supraventricular tachycardia with aberrant ventricular conduction (SVT-AV).

ventricular trabeculae which covers most of the endocardial surface of the ventricles [7]. Ventricular myocytes are depolarized from the terminal Purkinje fibers with depolarization spreading less rapidly from the endocardium to the epicardium [8,9].

A change in QRS complex width, axis or morphology is caused by abnormal ventricular depolarization, resulting from prolonged His Purkinje conduction (as in SVT-AC) or by an abnormal focus of ventricular activation outside the His-Purkinje system (as in VT). In the patient with SVT-AC, impulse movement along the Purkinje fibers is slowed or blocked by degenerative fibrosis as seen in BBB, by medication and other toxins, or by metabolic change (e.g. hyperkalemia). In the patient with VT, a change in ventricular activation will produce the altered QRS complex; ventricular pacing, from either transcutaneous, transvenous, or implanted pacemaker also alter the ventricular activation. All such events, whether involving SVT-AC or VT, will change the appearance of the QRS complex.

4. Supraventricular tachycardia with aberrant ventricular conduction

A range of supraventricular dysrhythmias are encountered Multiple rhythms are considered in this broad category of SVT-AC (Fig. 2). These rhythms have several features in common, including a supraventricular focus, a rapid rate, and a widened QRS complex.

In aberrantly conducted **sinus tachycardia**, the QRS complex is most often widened due to a fixed or rate-related BBB (Fig. 2A); other causes of aberrantly conducted ST include certain poisoning events with sodium channel blocking agents, significant hyperkalemia, or a dysfunctional intraventricular conduction system (Fig. 2B). In addition to the rapid rate, regular rhythm, and widened QRS complex, ST with bundle branch block is electrocardiographically characterized as follows: positively oriented P waves in leads I, II, and III; direct, consistent P wave-QRS
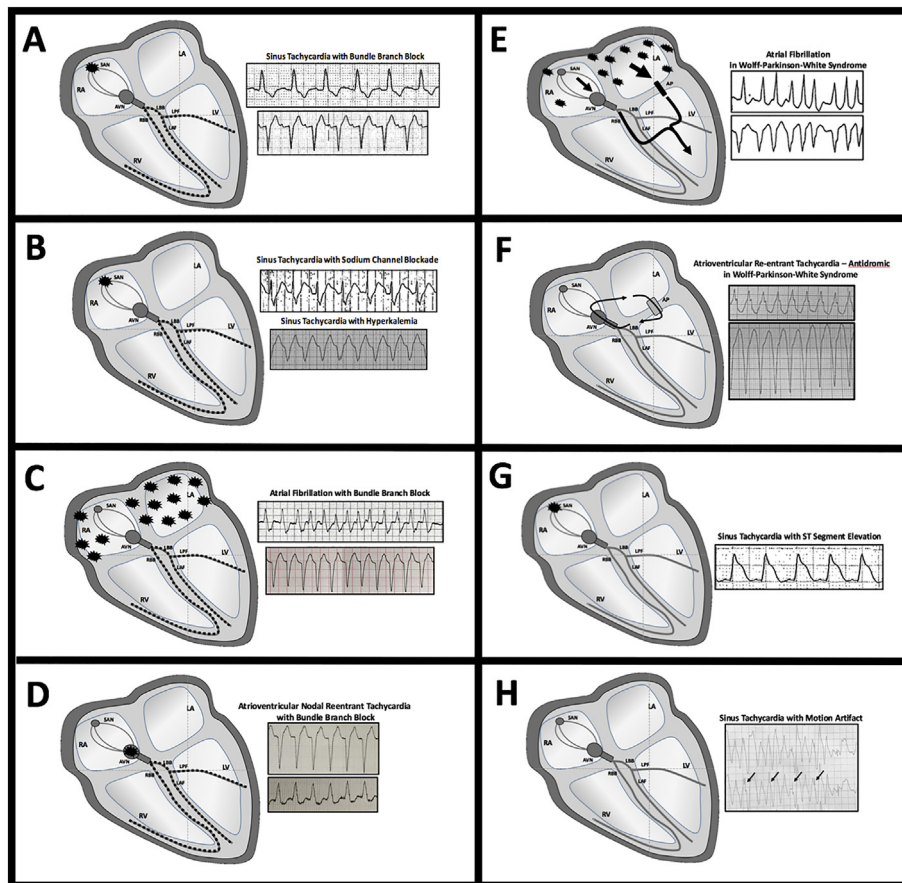

Fig. 2. Supraventricular ventricular tachycardia with aberrant intraventricular conduction. **A.** Sinus tachycardia with bundle branch block. The dotted line indicates bundle dysfunction due to bundle branch block. **B.** Sinus tachycardia with sodium channel blockade toxicity and hyperkalemia. The dotted line indicates bundle dysfunction due to toxic and metabolic issues. **C.** Atrial fibrillation with bundle branch block. The dotted line indicates bundle dysfunction due to bundle branch block. **D.** Atrioventricular nodal re-entrant tachycardia with bundle branch block. The dotted line indicates bundle dysfunction due to bundle branch block. **E.** Atrial fibrillation with Wolff-Parkinson-White syndrome. The arrows indicate the direction of the depolarization wave. **F.** Atrioventricular nodal re-entrant tachycardia – antidromic in the Wolff-Parkinson-White syndrome. The arrows indicate the direction of the depolarization wave. **G.** Sinus tachycardia with giant R wave (ST segment elevation) mimicking a widened QRS complex in a patient with STEMI. **H.** Electrocardiographic artifact mimicking wide complex tachycardia. The arrows indicate portions of QRS complexes occurring from patient's normal rhythm. Abbreviations: right atrium (RA); left atrium (LA); right ventricle (RV); left ventricle (LV); sinoatrial node (SAN); atrioventricular node (AVN); right bundle branch (RBB); left bundle branch (LBB); left anterior fascicle (LAF); and left posterior fascicle (LPF).

complex relationship for each electrical beat; and a broad rate range, from 100 to 160 bpm [10]. QRS complex duration is variable, ranging from 0.12 to 0.14; significantly more variation is encountered in situations involving hyperkalemia and poisonings.

Atrial fibrillation (Fig. 2C) is a common dysrhythmia, particularly in the elderly population with chronic heart disease. AF can present in an uncontrolled fashion with rapid ventricular response (RVR); the QRS complex can be widened as a result of either a fixed or rate-related BBB. AF with RVR and BBB is electrocardiographically characterized as follows: absence of distinct P waves; widened QRS complexes with consistent morphology in any single lead; and significant irregularity of the RR interval (a most significant clue to the diagnosis of AF with aberrant conduction).

Atrioventricular nodal re-entrant tachycardia (Fig. 2D), commonly known as paroxysmal supraventricular tachycardia, can present with a widened QRS complex due to a fixed or rate-related bundle branch block. In addition to the rapid rate, regular rhythm, and widened QRS complex, AVNRT is electrocardiographically characterized as follows: absence of normal P waves; retrograde P waves in approximately 70% of cases; QRS complex duration of approximately 0.12 to 0.14 s; and a very broad rate range, from 120 to 260 bpm, typically 170 to 180 bpm [11].

Ventricular pre-excitation syndromes, most frequently WPW, can present with various dysrhythmias, including AF and AVRT. **WPW-related AF** (Fig. 2E), the second most commonly encountered WPW dysrhythmia, is potentially malignant due to extremely rapid ventricular rates, exceeding 240 bpm at times; these rapid rates are largely a consequence of impulse conduction via the accessory pathway (AP). This dysrhythmia has an unusual, characteristic appearance with extremely rapid rate, significant irregularity, and beat-to-beat variation in the QRS complex morphology in any single lead. This variation in the QRS complex configuration results from varying contributions of the ultimate ventricular depolarization from two separate depolarization waves, reaching the ventricle via the AP and the AVN simultaneously. The resulting ventricular depolarization is a fusion of these two separate impulses; for any given beat, the contributions to ventricular depolarization from these two waves differ, producing the beat-to-beat variation in the QRS complex configuration.

Another of the classic **WPW** dysrhythmias, **antidromic AVRT** (Fig. 2F), presents with a widened QRS complex. This dysrhythmia is also malignant for the same reasons as WPW-related AF, the potential for extreme ventricular rates with associated hemodynamic compromise. In this dysrhythmia, the impulse moves anterograde from atria to ventricles by the AP, traverses the ventricular myocardium to the AVN, and returns to atrial tissues via retrograde conduction at the atrioventricular node. The conduction is described as "antidromic" with respect to the direction of impulse movement at the AVN. In addition to the rapid rate, regular rhythm, and widened QRS complex, antidromic AVRT is electrocardiographically characterized as follows: absence of P waves;

widened QRS complexes with unchanging morphology and duration ranging from 0.12 to 0.16 s; and extremely rapid rates, ranging from 180 to 260 bpm [11].

Other causes of SVT-AC include sinus tachycardia with a dysfunctional ventricular conduction system, for instance occurring after resuscitation from cardiac arrest, which will present initially with a WCT. Sinus tachycardia with ST segment elevation myocardial infarction (STEMI), particularly the giant R wave, can mimic a widened QRS complex, producing an apparent WCT (Fig. 2G). Lastly, electrocardiographic artifact can mimic WCT (Fig. 2H).

5. Ventricular tachycardia

VT is a malignant dysrhythmia, originating from any part of the intraventricular conduction system or ventricular myocardium [12]. The dysrhythmia manifests as a WCT consisting of at least three consecutive ventricular complexes with a rate greater than 120 bpm; VT rates range from 130 to 200 bpm, typically 170 to 190 bpm [12]. Slower forms of monomorphic VT, with rates from 130 to 160 bpm, can be encountered in patients with end-stage heart failure, resulting from severe forms of dilated cardiomyopathy, or in patients chronically maintained on amiodarone. Ventricular rhythms with rates of 100–120 bpm are referred to as accelerated idioventricular rhythm and should not be confused with VT as they are typically transient and self-terminating, frequently in the presence of reperfusion therapy for STEMI.

VT can present in either a monomorphic or polymorphic QRS complex configuration. Monomorphic VT (MVT) is characterized by a uniform QRS morphology and constant rate in any single lead (Fig. 3A), whereas polymorphic VT (PVT) demonstrates at least two or more QRS complex morphologies with varying rate (Fig. 3B). MVT is more common and may occasionally demonstrate evidence of atrioventricular dissociation (AVD), fusion complexes, and capture beats. The QRS complex axis tends to remain constant in MVT. In contrast, PVT often demonstrates a shifting axis; this shifting axis is particularly true for a variant of PVT known as torsade de pointes (TdP) in which the axis appears to shift back and forth, "twisting about the point" (Fig. 3B). TdP should be distinguished from the general term PVT; the Tdp form of polymorphic VT is diagnosed with the characteristic pattern and prolongation of the QTc interval [12]. Clinically, monomorphic VT results from a structurally abnormal heart, such as produced by scarring and/or fibriosis, often in the setting of remote myocardial infarction or cardiomyopathy; re-entry (2 distinct conduction pathways with a conduction block in one pathway and a region of slow conduction in the other) is the most common mechanism encountered in monomorphic VT. Conversely, polymorphic VT frequently occurs as a consequence of abnormal repolarization, frequently seen in patients with acute coronary ischemia, electrolyte abnormalities, medication toxicity, and congenital anomalies; with polymorphic VT, either triggered activity (early or late
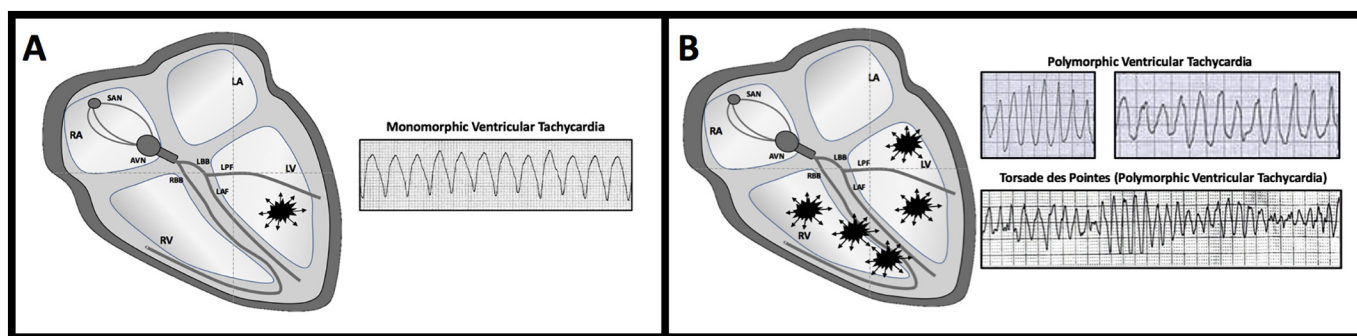

Fig. 3. Ventricular tachycardia. **A.** Monomorphic ventricular tachycardia. **B.** Polymorphic ventricular tachycardia and torsade des pointes. Abbreviations: right atrium (RA); left atrium (LA); right ventricle (RV); left ventricle (LV); sinoatrial node (SAN); atrioventricular node (AVN); right bundle branch (RBB); left bundle branch (LBB); left anterior fascicle (LAF); and left posterior fascicle (LPF).

after-depolarizations) or abnormal automaticity (accelerated abnormal impulse generation) is responsible for dysrhythmia formation.

6. Differentiation of supraventricular tachycardia with aberrant conduction from ventricular tachycardia

A history of prior heart disease is found commonly in patients with WCT and is suggestive of VT; prior myocardial infarction (MI), heart failure and recent angina pectoris have positive predictive values for the diagnosis of VT of 98%, 100%, and 100%, respectively [13]. Older age is also associated with a higher chance of VT [13]. Of course, older age and prior cardiac history do not always equal VT. Examination findings are of limited value in making this distinction; furthermore, clinical stability (or lack thereof) should not be used as a discriminator between VT and SVT-AC [14]. These clinical findings must be considered in conjunction with the electrocardiogram.

Several ECG features can suggest VT. Atrioventricular dissociation is considered a hallmark for VT (Fig. 4A). In cases of VT without retrograde conduction to the atria, sinus rhythm continues and is independent of ventricular activity, producing P waves that are dissociated from the QRS complexes, resulting in AVD. There are several important caveats with respect to AVD and VT. Firstly, AVD occurs in approximately 50% of VT presentations; thus, its electrocardiographic absence does not rule out VT. Secondly, AVD can also rarely occur with AVNRT, a form of SVT-AC; in other words, while strongly suggestive, AVD is not completely specific for VT. AVD is best detected in leads where the P wave is most prominent, usually the limb leads.

Capture and fusion beats, another manifestation of AVD, strongly suggests the diagnosis of VT (Fig. 4B). Intermittent AV conduction down the His-Purkinje system can result in these beats. In the patient with VT, an independent atrial impulse can occasionally cause ventricular depolarization via the normal conducting system. Such a supraventricular impulse, if conducted and able to trigger a depolarization within the ventricle, will result in a QRS complex. If the resulting QRS complex occurs earlier than expected and is narrow, the complex is called a capture beat. Fusion beats occur when a sinus beat conducts to the ventricles via the AVN and joins, or fuses, with a ventricular beat originating from the abnormal ventricular focus. These two electrical impulses combine, resulting in a QRS complex of intermediate width and differing morphology compared with the other beats of MVT.

The ECG during sinus rhythm (before or after the episode of WCT) can also provide important information. Findings suggestive of VT include the presence of Q waves indicative of prior MI, QT interval prolongation, or Brugada syndrome while other features potentially indicate SVT-AC, such as WPW and QRS complex abnormalities related to medications or hyperkalemia. Pre-existing BBB can be helpful as an indicator of SVT-AC yet the reader is cautioned to recall that bundle branch block is marker of chronic heart disease and therefore increased chance of VT [15]. If PR interval prolongation is noted during sinus rhythm, it is unlikely that SVT-AC will conduct in a rapid 1:1 pattern, sufficient to cause a wide complex tachycardia.

Several ECG algorithms have been developed to differentiate SVT-AC from VT with varying diagnostic accuracies [15,16,17,18,19,20,21,22]. Early ECG criteria focused on several features of the QRS complex, including QRS complex concordance (Fig. 4C & D) in the precordial leads (all QRS complexes demonstrating similar polarity, suggestive of VT); negative concordance is particularly suggestive of VT. In addition, RBBB morphologic changes in lead V1, the so-called "rabbit ear" configuration, are used to describe the double peak (RR′ wave) of the QRS complex; a larger amplitude initial peak (Rr′ wave) is suggestive of VT whereas a taller secondary peak (rR′ wave) is seen with SVT-AC [15, 16,17,18,19,20,21,22].

In 1978, Wellens and colleagues used His bundle recordings to determine the origin of the WCT and developed the Wellens' "classical criteria" [18]. This algorithm uses the presence of ventriculo-atrial dissociation (VAD) and QRS complex features to distinguish VT from
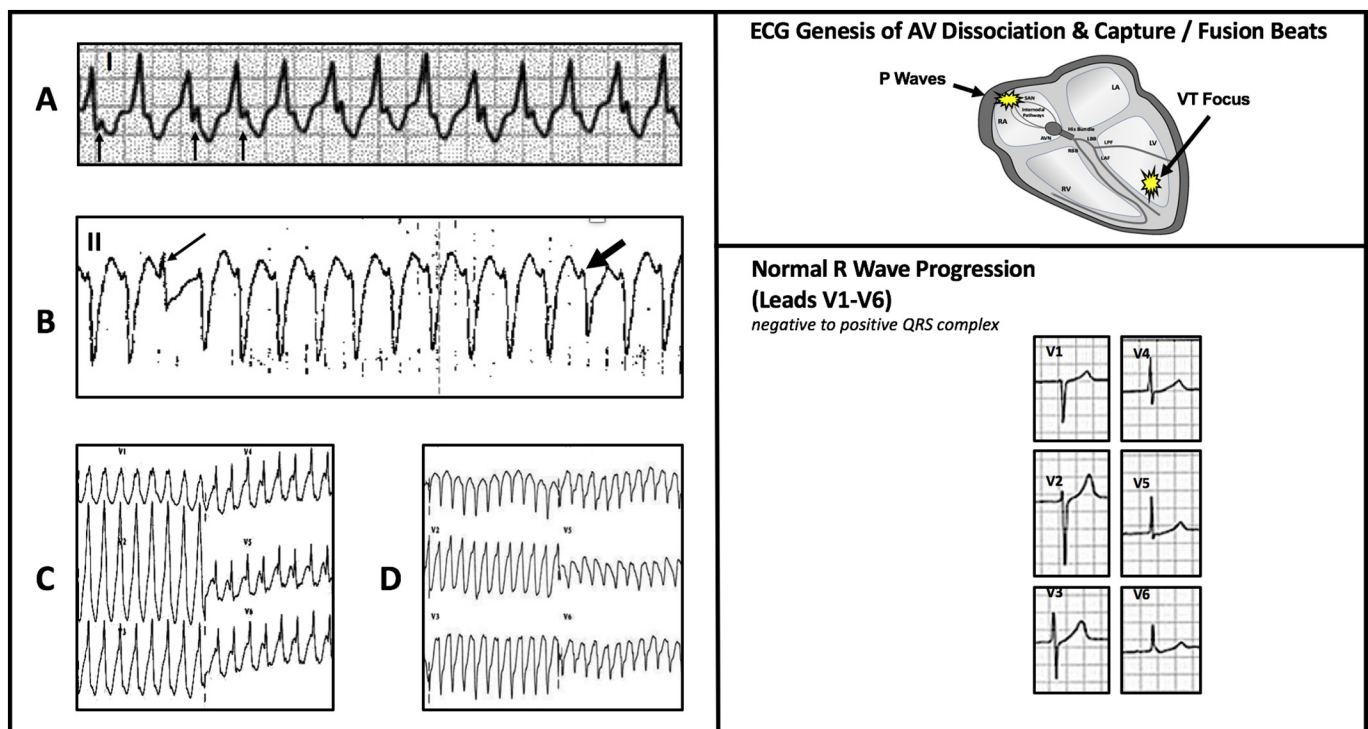

Fig. 4. Electrocardiographic findings suggestive of ventricular tachycardia. **A.** Atrioventricular dissociation. Note P waves (arrows) occurring within the wide QRS complexes. **B.** Capture (small arrow) and fusion (large arrow) beats. **C.** Positive QRS complex concordance in leads V1 to V6. **D.** Negative QRS complex concordance in leads V1 to V6. The cardiac silhouette in the upper right of this figure illustrates the concept of atrioventricular dissociation and genesis of capture and fusion beats in ventricular tachycardia. In the lower right of this figure is a depiction of normal R wave progression in the precordial leads from V1 to V6; compare this R wave progression to that encountered in VT with loss of this R wave progression, with resulting either entirely negative or positive QRS complexes from leads V1 to V6.

SVT-AC when typical RBBB or LBBB configurations are encountered. VT is more likely in RBBB configuration with a QRS complex duration of greater than 0.14 s; additional QRS complex criteria suggestive of VT include QR, R or RSr′ wave configurations in lead V1; and an RS ratio greater than 1 or QS wave in lead V6. VT is more likely in LBBB configuration with a QRS complex duration greater than 0.16 s; additional QRS complex criteria suggestive of VT include: in lead V1, an initial R wave greater than 0.03 s, slurring or notching of the downstroke of the S wave or QRS complex onset to nadir of S wave greater than 0.07 s; and in lead V6, any Q wave [18] .Kindwall (1988) uses criteria similar to Wellens [18] in leads V1 and V6 for distinguishing VT from SVT-AC [19].

The Brugada algorithm (1991) [15] uses the absence of RS wave or the R to nadir of S wave greater than 0.10 s in any precordial lead, together with Wellens' classical criteria, to suggest VT. Vereckei (2008), focusing solely on QRS complex criteria in lead aVR, notes the following criteria are suggestive of VT: initial R wave, initial R or q wave greater than 0.04 s, notching of the negative limb of the QRS complex, and lower voltage change during the initial 0.04 s compared to the terminal 0.04 s of the QRS complex [20,21].

Despite the use of detailed ECG criteria, WCT remains misdiagnosed, particularly with dysrhythmias demonstrating extremely rapid ventricular rates. Incorporating clinical and ECG data into a single algorithm can improve diagnostic accuracy. Becker and Crijns [22] have suggested the combination of 3 clinical criteria to increase the positive predictive value supporting a VT diagnosis, as compared to traditional ECG interpretation or single algorithms, including the following: VT diagnosed via any of the ECG algorithms, AVD diagnosed on ECG or echocardiography, and past history of MI, cardiomyopathy, congenital heart disease, or cardiac surgery [17].

A recent review [2] applied several of these diagnostic algorithms to undifferentiated WCT, noting that the more contemporary decision rules performed equally to the classic Wellens and Brugada criteria with only moderate diagnostic accuracy [2,15,18]. Additionally, significant variation in test characteristics existed across these rules [2]. Many of these algorithms have excluded patients using antiarrhythmic drugs, demonstrating pre-existing bundle branch block, or experiencing other dysrhythmias (e.g. fascicular VT or WPW-related dysrhythmias). All these factors reduce the specificity of the various ECG algorithms to correctly diagnose VT [19]. Furthermore, the rather cumbersome nature of the various ECG features, coupled with less-than-impressive test characteristics and rather poor inter-observer reliability, have limited their use outside of the cardiology community.

In the final analysis, if the diagnosis remains uncertain and typical BBB morphology is absent, VT should be the default diagnosis – the clinician is cautioned to avoid a default diagnosis of SVT-AC. A prudent approach suggests that, when uncertain, it is preferable to assume VT rather than SVT-AC.

7. Summary with management implications

Establishing the rhythm diagnosis in the WCT patient is a challenge; the specific rhythm diagnosis is frequently not possible in the early stage of evaluation and management. Furthermore, common medical opinion incorrectly holds that WCT with intact perfusion is supraventricular in origin; continuing along with this common misconception, unstable WCT is always VT [14]. In fact, both of these statements are incorrect; these two misconceptions can contribute to incorrect management strategies and consequently adverse patient outcome, including death. The presenting hemodynamic status is not predictive of the rhythm origin or the diagnosis [14]. Irrespective of the clinical stability, a prudent approach suggests that, when uncertain, it is preferable to assume VT rather than SVT-AC.

Many of these dysrhythmias present in the setting of complex medical events, not infrequently with significant instability; early resuscitative therapies often must be empiric in nature, initiated before the final diagnosis is established. Management can involve therapies focusing on the underlying event, responsible for the dysrhythmia and its clinical impact while, in other instances, treatments can be solely aimed at rhythm control and / or conversion. Several rhythm diagnoses in this WCT differential consideration require specific management, addressing the underlying issue. For instance, sinus tachycardia, rhythms related to toxic or metabolic issues, and wide complex tachycardias with dysfunctional intraventricular conduction do not usually benefit from direct rhythm therapy; rather, treatments aimed at the underlying, causative events are most appropriate. Atrial fibrillation and atrial flutter present in truly complex scenarios, at times with significant underlying illness; in other instances, these two atrial dysrhythmias can present solely as abnormal rhythms without active, underlying process – thus, a consideration of the clinical presentation will suggest the most appropriate treatment strategy. Lastly, AVNRT, AVRT, WPW-related atrial fibrillation, and ventricular tachycardia usually are managed with therapy aimed primarily at rhythm conversion; of course, underlying medical issues should also be addressed, should they exist.

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