# Wide Complex Tachycardias—The Differential Diagnosis Remains Wide and Complex

# a report by John M Miller, MD and Anil V Yadav, MD

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In the nearly 30 years since Wellens et al. categorized most of the currently used electrocardiographic (ECG) criteria for distinguishing supraventricular tachycardia (SVT) with aberrant interventricular conduction from ventricular tachycardia (VT), there have been relatively infrequent and largely incremental enhancements to the art and science of making this distinction. In most published series, a correct diagnosis can be made using these criteria in up to 90% of cases. Despite this, few clinical situations evoke more anxiety in physicians and nurses than a patient with wide complex tachycardia (WCT). Often, residents and even staff physicians are seen poring over the WCT ECG and muttering under their breath, "I should know this!" Why does this situation continue to present such a clinical challenge? And what are the consequences of making an incorrect diagnosis?

WCT has several potential causes, including VT; SVT with one of the following: aberrant interventricular conduction (SVT-A), atrioventricular (AV) conduction over an accessory pathway (Wolff-Parkinson-White), QRS widening due to drug effect/electrolyte abnormalities, or an abnormal bizarre baseline QRS (cardiomyopathy); or ventricular pacing. Although the proportion of cases falling into each category varies slightly depending on patient population, VT comprises about 67% of WCT in most series, with SVT-A accounting for another 25%. Thus, the most important distinction to make is between VT and SVT-A.

#### **Current Methods**

ECG differentiation of WCTs can be divided into two major areas: configurational (morphology of QRS) and relational (AV relationship during WCT). Configurational distinctions are based on QRS patterns that resemble aberrant



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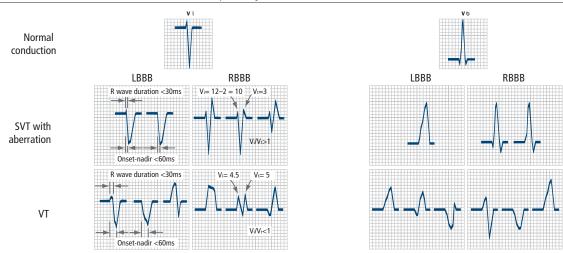
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conduction, and are thus more consistent with SVT-A, or do not resemble aberration patterns and are thus likely to be VT. Some of the more commonly used configurational criteria that are uncommon in SVT-A and thus strongly suggest VT are prolonged QRS duration (>140ms for right bundle branch block [RBBB] pattern QRS, >160ms for left bundle branch block [LBBB]); leftwards frontal plane axis (especially if between -90 and 180°); a fully concordant precordial R wave pattern (fully positive or fully negative); and specific patterns in leads  $V_1$  and  $V_6$  that are or are not compatible with aberrant conduction (see *Figure 1*). Particular combinations of bundle branch block and axis are distinctive, such as LBBB with rightwards inferior axis (+90–180°) that is almost never seen in SVT-A, and RBBB with normal axis (0–+90°) that is almost never seen in VT. Another pattern that is rare in SVT-A is the absence of any RS complex among the precordial leads; Brugada et al. used this as the basis of an algorithm that they found to have 99% sensitivity and 97% specificity for diagnosing VT.²

Capitalizing on the fact that in SVT-A the electrical vectors move rapidly at the beginning of the QRS and more slowly at the end (activating the 'locked' ventricle), whereas in VT slow conduction is the rule throughout the QRS complex, Vereckei and colleagues recently reported a new criterion that quantifies these differences.3 They calculated the ratio of the voltage amplitude change in the first versus last 40ms of a bi- or multiphasic QRS and found a ratio >1 suggested SVT-A and, if  $\le 1$ , VT was diagnosed. They also found that an initial R wave in lead aVR strongly suggested VT. This, as well as most of the published algorithms in the literature dealing with QRS configuration distinctions, reports predictive accuracies from 85 to 95%. Unfortunately, the configurational criteria are most helpful when the patient's baseline QRS complex is not significantly widened. If the baseline QRS is very abnormal, the configurational criteria lose much of their predictive capacity<sup>4</sup> with the exception that if the QRS complexes of the WCT are identical in configuration to those during baseline ECG, chances are good that the WCT is SVT.5 In many cases, however, the baseline ECG is simply not available for comparison. Recently, some of the long-trusted ECG differentiating criteria have undergone re-evaluation in light of changes in the patient population.<sup>6</sup> In particular, patients with heart failure are living longer with improved medical therapies. Many of these patients have widened, very abnormal baseline QRS complexes and some have episodes of SVT conducted with bizarre patterns that suggest VT but are identical to their baseline QRS configuration. Perhaps because of this, one differentiating criterion that has ceased to be helpful is a negative concordant precordial pattern. In a recent series of WCTs, this pattern was found as frequently in SVT (10%) cases as in VT (12%) whereas previously it was seen almost exclusively in VT.

In the relational area, the AV relationship during WCT is conceptually straightforward and has long been a useful tool in diagnosing WCTs, since—

Figure 1: Representative QRS Configurations for V<sub>1</sub> and V<sub>6</sub> in SVT-A and VT



Normal, narrow complexes are shown at the top for comparison. Typical (but not all-inclusive) QRS configurations for LBBB and RBBB types of SVT and VT are shown. Several differentiation criteria are displayed in measurements ( $v_i$  and  $v_t$  are absolute value of voltage change in the initial ( $v_i$ ) and terminal ( $v_t$ ) 40ms of any single bi- or multiphasic QRS complex).

with only extremely rare exceptions—SVTs have at least as many P waves as QRS complexes (AV ratio ≥1). In contrast, since VTs do not require atrial participation, the AV ratio is ≤1 (that is, AV dissociation or retrograde 2:1 or Wenckebach pattern is present). The AV relationship is independent of complicated morphological criteria as well as the pattern of the patient's QRS complex during normal rhythm. While these features of the AV relationship are attractive, it is often difficult to discern clear atrial activity during WCT, or the presence of atrial fibrillation or flutter confounds the issue. In a recent series, a diagnostic AV pattern was observed in only 36% of VTs; thus, while it is very specific, it is not a sensitive criterion. Since the AV relationship is such a specific differentiator when positive, some stepwise algorithms for analyzing WCTs incorporate it as a first step.

With a wealth of relatively good differentiating criteria, why is making the correct diagnosis of the cause of WCT so problematic? One reason is that many of the algorithms and rules are difficult to remember, and any diagnostic rule is only as good as the ability of the user to apply it correctly. Another limitation is that 'real-world' application of some of the criteria, even when recalled correctly, rarely replicates the original authors' results. In one study, a group of emergency physicians and cardiologists applied the Brugada criteria to a set of WCTs and found an average sensitivity of 85% and specificity of 57% compared with Brugada's >97% for both indices.

### **Consequences of Misdiagnosis**

Although correctly diagnosing the cause of a WCT may be difficult, the consequences of making an incorrect diagnosis can be deadly, such as if a patient with VT is mistakenly categorized as SVT and treated with repeated doses of adenosine followed by verapamil or beta-blockers to try to terminate the arrhythmia, or is not further evaluated and treated to prevent VT recurrences or sudden death. How can this happen? It is often tempting to assume that a patient who is awake, alert, and minimally uncomfortable during a WCT episode cannot have VT, and must therefore have SVT.

Unfortunately, this is simply erroneous. A more rational presumption, if one cannot make a definitive diagnosis, is to assume the arrhythmia is VT. This is, after all, the statistically safer choice (two-thirds of WCTs being VT in most series) as well as making certain that the most serious diagnosis is treated. If the patient is mistakenly diagnosed as having VT when their arrhythmia is really SVT, further evaluation will almost always sort out the correct diagnosis.

#### The Future

Although the correct diagnosis for a WCT episode can usually be made using existing criteria and algorithms, these remain somewhat cumbersome and difficult to remember in the urgency of a clinical situation. Owing to this, automated analysis (updated algorithms built into ECG machines) may take on a more important role, since the computer will not 'forget' the criteria or misapply them. Whether further refinements in differentiating criteria will produce a simple and all-encompassing discriminator is doubtful since, despite proper application of all criteria, a small proportion of WCTs simply do not follow the rules and will be misclassified. Any new criteria that are introduced should be simpler and more easily applied than existing ones, rather than more complex and difficult to apply, in order to be widely accepted.

## Summary

Over the years, a variety of useful criteria have been developed and refined for differentiating the cause of WCTs. While these criteria and the algorithms incorporating them have high sensitivity and specificity when correctly applied, they are often complex and cumbersome to recall and apply. When trying to diagnose a WCT, the stakes are high since misdiagnosis of VT as SVT can have disastrous consequences. If one is at all unsure of the correct diagnosis of a WCT after applying differentiating criteria to the best of his or her ability, the safest course is to assume the WCT is VT and treat accordingly, both because of the statistical likelihood that the rhythm is VT, as well as its greater seriousness. In so doing, patients with VT will be correctly managed and those who actually have SVT will eventually be correctly diagnosed.

<sup>1.</sup> Wellens HJJ, et al., Am J Med, 1978;64:27-33.

Brugada P, et al., Circulation, 1991;83:1,649–59.

<sup>3.</sup> Vereckei A, et al., Eur Heart J, 2007;28: 589–600.

<sup>4.</sup> Alberca T, et al., Circulation, 1997;96:3,527-33.

<sup>5.</sup> Dongas J, et al., Am J Cardiol, 1985;55: 717–21.

<sup>6.</sup> Miller JM, et al., Cardiology Clinics, 2006, Philadelphia: W. B.

Saunders: 439-51.

<sup>7.</sup> Isenhour JL, et al., Acad Emerg Med, 2000;7:769-73.