Approach to the Differentiation of Wide QRS Complex Tachycardias

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The differentiation of wide QRS complex tachycardias presents a challenging diagnostic dilemma to many physicians despite multiple published algorithms and approaches.1 The differential diagnosis includes supraventricular tachycardia conducting over accessory pathways, supraventricular tachycardia with aberrant conduction, antidromic atrio-ventricular reentrant tachycardia, supraventricular tachycardia with QRS complex widening secondary to medication or electrolyte abnormalities, ventricular tachycardia (VT) or electrocardiographic artifacts. The correct diagnosis is essential since it has significant prognostic and treatment implications. In this article we will discuss the factors which support the diagnosis of VT as well as some algorithms useful in the evaluation of regular, wide QRS complex tachycardias.

Appearance
It is a somewhat common misconception that patients with ventricular tachycardias are almost always hemodynamically unstable.2 The patient’s blood pressure cannot be used as a reliable sign for the differentiation of the origin of an arrhythmia. In a small study by Garratt et al. clinically detectable variation of the first heart sound and examination of the jugular venous pressure were noted to be useful for the diagnosis of a ventricular origin of the arrhythmia.3

Substrate
The assessment of a patient’s history may support the increased probability of an arrhythmia originating in the ventricle. A wide QRS complex tachycardia in a patient older than 35 years is more likely to be VT.4 A known history of coronary artery disease, previous myocardial infarction or cardiomyopathy makes VT a probable diagnosis. A history of ischemic heart disease or congestive heart failure is 90 % predictive of a ventricular origin of an arrhythmia.4 Patients with hypertrophic obstructive cardiomyopathy are prone to have VT.5 A known history of arrhythmogenic right ventricular dysplasia or cathecolaminergic polymorphic VT should also point towards a ventricular origin of the tachycardia.

Tetralogy of Fallot is a common cyanotic congenital lesion.6 Patients with both unrepaired and repaired conditions are at risk of having VT.7,8 Patients with a history of Duchenne muscular dystrophy, Becker muscular dystrophy, myotonic dystrophy, Friedreich’s ataxia, and Emery–Dreifuss muscular dystrophy are at increased risk of developing cardiomyopathies.9 Thus a diagnosis of VT should be considered in these patients presenting with wide complex tachycardias. A history of both short and long QT syndromes makes a ventricular origin of the tachycardia likely as well.10–12 However, patients with a short QT syndrome and the Brugada syndrome are more likely to present with ventricular fibrillation rather than VT. Infiltrative diseases of the heart such as cardiac amyloidosis or sarcoidosis may also predispose patients to ventricular arrhythmias.13,14 Interestingly enough, VT is also common in patients with Chagas’ disease.15

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Medications should be carefully reviewed. Vaughan Williams Class I and Class III antiarrhythmic medications, multiple medications that prolong the QT, and digoxin at toxic levels may cause VT.

The Electrocardiogram

A careful review of the electrocardiogram (ECG) may provide clues to the origin of a wide QRS complex tachycardia. The presence of atrioventricular dissociation strongly favors the diagnosis of VT. However, it may also be observed in atrioventricular junctional tachycardia in the absence of retrograde conduction. Even though capture and fusion beats are not frequently observed, their presence suggests VT.

Comparison with the baseline ECG is an important part of the process. A change in the QRS complex morphology or axis by more than 40°, as well as a QRS axis of -90° to -180° suggests a ventricular origin of the arrhythmia. An entirely positive QRS complex in lead augmented vector right (aVR) also supports the diagnosis of VT. When the sinus rhythm with wide QRS becomes narrow with a tachycardia, this indicates VT. The morphology of a tachycardia similar to that of premature ventricular contractions seen on prior ECGs increases the probability of a ventricular origin of the arrhythmia.

One approach to the interpretation of wide QRS complex tachycardias is to divide them into right bundle branch block morphology (QRS complex being predominantly positive in lead V1) and left bundle branch block morphology (QRS complex being predominantly negative in lead V1). Wide complex tachycardias with right bundle branch block morphologies are more likely to be of ventricular origin if the following criteria are met:

- QRS complex duration of more than 140 ms;
- the presence of positive concordance in the precordial leads;
- a superior axis of the QRS complex;
- the presence of a qR, R or RS complex or an RSR’ complex where R is taller than R’ and S passes through the baseline in V1; and
- an R to S ratio of more than one in V6.

Left bundle branch block morphology tachycardias are more likely to be VT if they have the following features:

- QRS complex duration of more than 160 ms;
- the presence of negative concordance in the precordial leads;
- the presence of an rS complex in V1; and
- mostly negative QS complex in V6.

In addition to these criteria, the presence of an R wave of more than 30 ms duration, notching of the downstroke of the S wave, or duration from the onset of the QRS to the nadir of the S wave in leads V1 or V2 of greater than 60 ms and any Q wave in lead V6 favors the ventricular origin of an arrhythmia. A protocol for the differentiation of a regular, wide QRS complex tachycardia was published by Brugada et al. It consisted of four diagnostic criteria:
the absence of an RS complex in all precordial leads;  
an R to S wave interval of more than 100 ms in any of the precordial lead;  
the presence of atrio-ventricular dissociation; and  
the presence of morphologic criteria for VT in leads V1–2 and V6.

The presence of any of these criteria supports the diagnosis of VT. Morphologic criteria for right bundle branch block for lead V1 are: the presence of monophasic R wave, QR or RS morphology; for lead V6: Larger S wave than R wave, or the presence of QS or QR complexes. For left bundle branch block morphology the criteria include: for V1–2: an R wave of more than 30 ms duration, notching of the downstroke of the S wave, or duration from the onset of the QRS to the nadir of S wave of more than 70 ms; for lead V6: the presence of a Q or RS complex. For the final assessment at least one criterion for both V1–2 and V6 have to be present to diagnose VT. Although this is an excellent protocol, with a sensitivity of 88–92 % and specificity of 44–73 % for VT, it requires remembering multiple morphologic criteria.25,26 The majority of the protocols use supraventricular tachycardia as a default diagnosis of wide QRS complex tachycardia. Only the presence of specific ECG criteria is used to diagnose the arrhythmia as VT. Unlike previous protocols, VT was used as a default diagnosis by Griffith et al.27 Only the presence of typical bundle branch criteria assigned the arrhythmia’s origin to be supraventricular. A Bayesian diagnostic algorithm, with assignment of different likelihood ratios of different ECG criteria from historically published protocols used by Lau et al., was found to have very good diagnostic accuracy.28 However, this protocol did not incorporate certain important features, such as atrioventricular dissociation, as they could not be ascertained in all cases. Interestingly enough, no statistically significant difference in sensitivity and specificity was found between the Brugada, Griffith and Bayesian algorithm approaches.25

In 2007, Vereckei et al. proposed an algorithm for the differentiation of monomorphic wide QRS complex tachycardias.26 It consisted of four steps. If a patient meets a criteria at any step then the diagnosis of VT is made, otherwise one proceeds to the next step. The four criteria are:

- the presence of atrio-ventricular dissociation;
- the presence of an initial R wave in lead aVR;
- a QRS morphology that is different from bundle branch block or fascicular block; and
- the algebraic sum of the voltage of the first 40 ms divided by the last 40 ms is less than or equal to one.

Table 1: Summary of the Brugada and Vereckei Protocols

<table>
<thead>
<tr>
<th>Step</th>
<th>Brugada et al.26</th>
<th>Vereckei et al. (#1)26</th>
<th>Vereckei et al. (#2, based solely on lead aVR)29</th>
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<tbody>
<tr>
<td>I</td>
<td>The absence of an RS complex in all precordial leads</td>
<td>The presence of atrio-ventricular dissociation</td>
<td>The presence of an initial R wave</td>
</tr>
<tr>
<td>II</td>
<td>An R to S wave interval of more than 100 ms in any precordial lead</td>
<td>The presence of an initial R wave in aVR</td>
<td>The presence of an initial q or r wave of &gt;40 ms duration</td>
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<tr>
<td>III</td>
<td>The presence of atrio-ventricular dissociation</td>
<td>A QRS morphology that is different from bundle branch block or fascicular block</td>
<td>The presence of a notch on the descending limb of a negative onset and predominantly negative QRS complex</td>
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<td>IV</td>
<td>The presence of morphologic criteria for VT in leads V1–2 and V6 (as described earlier in this text)</td>
<td>The ratio of the sum of voltage changes of the initial over the final 40 ms of the QRS complex being less than or equal to one</td>
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aVR = augmented vector right; VT = ventricular tachycardia.

Table 2: Factors Increasing the Likelihood of Ventricular Tachycardia when Reviewing Wide QRS Complex Tachycardias

1. Appropriate ‘substrate’
2. Atrio-ventricular dissociation, capture and fusion beats
3. QRS axis of -90° to -180°
4. Change in QRS axis by more than 40°
5. Narrowing or QRS complex with tachycardia
6. Positive R wave in lead augmented vector right

This algorithm has a better sensitivity and specificity than the Brugada criteria being 95.7 and 95.7 %, respectively.26 More recently, a new protocol using only lead aVR to differentiate wide QRS complex tachycardias was introduced by Vereckei et al.29 It consists of four steps:

- the presence of an initial R wave;
- the presence of an initial q or r wave of > 40 ms duration;
- the presence of a notch on the descending limb of a negative onset and predominantly negative QRS complex; and
- the ratio of the sum of voltage changes of the initial over the final 40 ms of the QRS complex being less than or equal to one.

Similar to the previous algorithm, only one of the four criteria needs to be present. The sensitivity and specificity of this protocol are 96.5 and 95.7 %, respectively, which is similar to the previous algorithm published by this group.25 To reinforce the material we would like to offer
two ECGs for review (see Figures 1 and 2). Table 1 summarizes the Brugada and Vereckei protocols. All three algorithms should be considered when reviewing the sample electrocardiograms.

Conclusion
The differentiation of wide QRS complex tachycardias remains a diagnostic challenge. Making the correct diagnosis has important therapeutic and prognostic implications. There are multiple approaches and protocols, each having its own pros and cons. No protocol is 100 % accurate. In this article we try to summarize approaches which we consider optimal for the evaluation of patients with wide QRS complex tachycardias. From our perspective, the last protocol by Verekei et al. is one of the easiest to use while having a good sensitivity and specificity. Thus we recommend the following approach: evaluating the ‘substrate’ for the arrhythmia, then evaluating the ECG for fusion beats, capture beats and atrioventricular dissociation. Careful attention should subsequently be paid to the potential change in the width and axis of the QRS complex when comparing it to the QRS complex of the baseline ECG. See Table 2 for the initial approach to the differentiation of wide QRS complex tachycardias preferred by our group. We recommend using a protocol that one is most familiar and comfortable with and supplementing it with the steps from other protocols to improve the accuracy of the diagnosis. However, when in doubt, treat the arrhythmia as if it was VT, as approximately 80 % of wide QRS complex tachycardias are of ventricular origin.20,31

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