Differentiating Wide Complex Tachycardia

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- Definitions
- Causes
- Electrocardiographic features
- Diagnostic Criteria
- **Wide Complex Tachycardias (WCT)** - QRS duration > 120 ms and heart rate > 100 beats/min.

- **Ventricular Tachycardia (VT)** - 3 or more consecutive ventricular beats with a rate of 100 beats/min or more.

- **Nonsustained VT (NSVT)** – tachycardia < 30 seconds duration.
- Sustained VT: Last > 30 seconds
- Causes significant hemodynamic symptoms
- Requires therapeutic intervention for termination
- Monomorphic VT – uniform and stable QRS appearance in any given lead
- Polymorphic VT – continuously varying QRS morphology and/or axis in a single lead during an episode
- **Supraventricular tachycardia (SVT)** – any tachycardia using the normal AV conduction system for ventricular excitation, w/ the tachycardia originating in the atria or the AV node and requiring the AV node for its maintenance.

- **Aberrant conduction (aberrancy)** – conduction delay or block in the His-Purkinje system during antegrade conduction of impulses over the normal AV-conduction system resulting in a wide, abnormal QRS.
Pre-excitation syndrome – AV conduction can occur via the normal conduction system and via an accessory AV pathway

The two pathways create the substrate for a reentrant circuit, facilitating AV reentrant tachycardia (AVRT)

- Orthodromic AVRT – antegrade conduction over the AV node and retrograde conduction via the accessory pathway; QRS is narrow unless there is aberrant conduction
- Antidromic AVRT – antegrade conduction over the accessory pathway and retrograde conduction over the AV node or a second accessory pathway; QRS is wide
- Pre-excited tachycardias – ventricular activation occurs predominately or exclusively via an accessory pathway
- VT - most common cause of WCT (80% of cases in unselected populations and more than 95% in pts w/ structural heart disease

- Can originate anywhere below the AV node: His bundle, bundle branches, fascicles, Purkinje fibers, and ventricular myocardial tissue

- Fixed conduction block – when there is a baseline block during sinus rhythm due to pathological lesions in the conduction system

- Seen as baseline left bundle branch block (LBBB), right bundle branch block (RBBB), or a nonspecific intraventricular conduction delay (IVCD)
- Orthodromic AVRT w/ aberrancy
- Pre-excitation tachycardias
- Antidromic AVRT
- Relatively uncommon, occurring in about 6% of cases
- Difficult to differentiate from VT because ventricular activation begins outside the normal intraventricular conduction system in both
- Atrial fibrillation with an accessory pathway
- Tachycardia's w/ two accessory pathways
- Pacemakers and cardiac resynchronization therapy
Electrocardiograms

- **Rate:** limited use in distinguishing VT from SVT as there is too much overlap

- Consider Atrial flutter when **HR** is ~150 beats/min

- **Regularity:** VT is generally regular, though there can be slight variation in the RR intervals

- Slight irregularity at the onset ("warm--up phenomenon")- favors VT

- Grossly irregular WCT likely represents:
  1) AF w/ aberrant conduction,
  2) AF w/ conduction over an accessory pathway,
  3) polymorphic VT

- Uniformity of the RR intervals' favors SVT
- **Axis:** mean QRS axis in the normal range favors SVTw/aberration

- Right superior axis of -90 to ± 180° strongly suggests VT (sometimes called “northwest” or extreme right axis)

- Should compare the axis during SR,, an axis shift during WCT of > 40° favors VT

- In RBBB-like WCT, axis to the left of -30° suggest VT

- In LBBB-like WCT, axis to the right of +90° suggests VT
- QRS duration: generally, a wider QRS favors VT
- In RBBB--like WCT,, duration > 140 msec suggests VT
- In LBBB--like WCT,, duration > 160 msec suggest VT
- QRS duration > 160 msec is a strong predictor of VT regardless of bundle--branch block morphology
- Except in cases of SVT w/ an AV accessory pathway and the presence of drugs capable of slowing intraventricular conduction
  - Such as class 1A or class 1C agents or amiodarone
- QRS duration < 140 msec does not exclude VT
- VT originating from the septum or w/in the His-Purkinje system
Concordance: present when QRS complexes in all 6 precordial leads (V1-V6) are monophasic w/ the same polarity (90% specificity for VT)

Positive concordance – All entirely positive w/ totally, monophasic R waves

Most often due to VT but can also occur in rare cases of antidromic AVRT w/ a left posterior accessory pathway

Negative concordance -- All entirely negative w/ deep monophasic QS complexes

Concordance is not present if any of the 6 leads has a biphasic QRS (qR or RS complexes)

Absence of concordance is not diagnostically helpful
- **AV dissociation:** atrial activity that is independent of ventricular activity occurs in 20-50% off VT and almost never in SVT.

- When the atrial rate is slower than the ventricular rate, this strongly suggest VT.

- Atrial rate faster than the ventricular rate suggests a SVT, such as atrial flutter or AT w/ 2:1 AV conduction.

- There is a consistent relationship between the P waves and the QRS complexes.
- Presence of this largely establishes the diagnosis of VT but its absence is not helpful.
- May sometimes not be evident on ECG.
- Some cases of VT, the ventricular impulses conduct retrograde through the AV node and capture the atrium, preventing dissociation.
- **Echo beat** – VT impulse conducted retrogradely through the AV node to produce atrial capture that, on to the ventricles, produces a narrow QRS complex

- **Fusion beat** – when one impulse originating from the ventricle and a second supraventricular impulse simultaneously activate the ventricular myocardium

- Morphology is intermediate between that of a sinus beat and a purely ventricular complex

- **Capture beat** – normal conduction momentarily “captured” control of ventricular activation from the VT focus
QRS Morphology:

- most approaches involve classifying the WCT as having RBBB—like pattern or LBBB—like pattern
- RBBB-like pattern: QRS polarity is positive in leads V1 and V2
- LBBB-like pattern: QRS polarity is negative in leads V1 and V2
- **RBBB pattern associations:**
  - **Lead V1:** monophasic R or biphasic qR complex favors VT
  - Triphasic RSR” or RsR” complex (‘’rabbit--ear’’ sign) favors SVT,, except if the left peak of the RsR” complex is taller than the right peak
  - **Lead V6:** rS complex (R wave smaller than S wave) favors VT
  - Rs (R wave larger than S wave) complex favors SVT
LBBB pattern associations:

- Lead V1 or V2: broad initial R wave of >30 msec duration favors VT
- Slurred or notched downstroke of the S wave favors VT
- Duration from the onset of the QRS complex to the nadir of the QS or S wave of M 60 msec favors VT
- Absence of an initial R wave or a small initial R wave of < 30 msec favors SVT
- A swift, smooth downstroke of the S wave in w/ a duration of < 60 msec favors SVT
- Lead V6: any Q or QS wave favors VT
- Absence of a Q wave favors SVT
Brugada Criteria

- Stepwise approach in which four criteria for VT are sequentially evaluated and if any are satisfied, the diagnosis of VT is made;

  if none are fulfilled then diagnosis off SVT is made by exclusion

  1) Lead V1-V6 are inspected for an RS complex, if none then concordance is present

  2) If an RS complex is present, the longest interval in any lead between the onset of the R wave and the nadir of the S wave (RS interval) is measured VT if the RS interval is > 100 msec
3) If the RS interval is < 100 msec, then evaluate for the presence of AV dissociation to diagnose VT.

4) If the RS interval is < 100 msec and AV dissociation is not evident, then evaluate the QRS morphology for V1-positive and V1-negative WCT.

If either the V1-V2 or the V6 criteria are not consistent w/ VT, then SVT is assumed.
Brugada algorithm

1. Absence of an RS complex in all precordial leads?
   - yes → VT diagnosed
   - no →

2. The longest R to S interval >100 ms in any precordial lead?
   - yes → VT diagnosed
   - no →

3. A-V dissociation?
   - yes → VT diagnosed
   - no →

4. Morphology criteria for VT present both in leads $V_{1,2}$ and $V_{6}$?
   - yes → VT diagnosed
   - no → SVT diagnosed
## Brugada Algorithm: Steps

<table>
<thead>
<tr>
<th>Step</th>
<th>Cumulative Sensitivity for VT</th>
<th>Cumulative Specificity for VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Absence of an RS complex in all precordial leads.</td>
<td>21%</td>
<td>100%</td>
</tr>
<tr>
<td>2. Precordial RS interval &gt;100 ms</td>
<td>66%</td>
<td>98%</td>
</tr>
<tr>
<td>3. VA dissociation</td>
<td>82%</td>
<td>98%</td>
</tr>
<tr>
<td>4. Morphological criteria for VT</td>
<td>99%</td>
<td>97%</td>
</tr>
</tbody>
</table>

Brugada P. *Circulation* 1991;83:1649-1659
New aVR Algorithm

- Hypothesized reasons for using aVR:

- During SVT w/ BBB, the initial septal activation and the latter main ventricular activation waterfront move away from lead aVR, creating a negative QRS complex in lead aVR

- Exception to this generalization is occurs in inferior myocardial infarction where there is the loss of the initial inferiorly directed forces creating an initial r wave (rS complex) during NSR or SVT

- Because an initial dominant R wave in aVR is incompatible w/ SVT, its presence suggest VT, typically originating from the inferior or apical region
Useful for detecting VT originating from sites other than the inferior or apical wall, but would not show an initial R wave in aVR.

Would rather show a slow, initial upward vector of variable size pointing toward aVR even if the main vector of the VT points downward and creates a predominately negative QRS in lead aVR.

Exceptions would be VT originating from the most basal sites of the interventricular septum or free wall.
- Stepwise approach similar to the Brugada criteria
- used to analyze monophonic WCT:
  1) Evaluate for the presence of an initial R wave
  2) Evaluate for the presence of an initial r or q wave with width M 40 msec
  3) Evaluate for notching on the descending limb of a negative onset, predominately negative QRS complex
New aVR algorithm

In lead aVR:

Step 1. Presence of an initial R wave?
- No
- Yes → VT diagnosed

Step 2. Presence of an initial r or q wave >40 ms?
- No
- Yes → VT diagnosed

Step 3. Presence of a notch on the descending limb of a negative onset and predominantly negative QRS?
- No
- Yes → VT diagnosed

Step 4. $v_i/v_t \leq 1$?
- No
- Yes → VT diagnosed

SVT diagnosed
New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia

András Verecke, MD,* Gábor Duray, MD,† Gábor Szénási, PhD,‡ Gregory T. Altemose, MD, FHRs,§ John M. Miller, MD, FHRs§
We analyzed the RWPT at lead II, defined as the duration of the QRS from the initiation of depolarization until the first change of the polarity, independent of whether the QRS deflection was positive or negative.

A R-wave peak time $\geq 50$ ms at DII is a simple and highly sensitive criterion that discriminates VT from SVT in patients with wide QRS complex tachycardias.

Comparison of five electrocardiographic methods for differentiation of wide QRS-complex tachycardias

Marek Jastrzebski¹*, Piotr Kukla², Danuta Czarnecka³, and Kalina Kawecka-Jaszcz³

Europace Advance Access published February 14, 2012
ECG criteria for epicardial VT

<table>
<thead>
<tr>
<th>Criteria</th>
<th>ENDO</th>
<th>EPI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>QRS duration (ave ms ± std dev)</td>
<td>192 ± 42</td>
<td>220 ± 45</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Pseudo-delta wave (ave ms ± std dev)</td>
<td>33 ± 21</td>
<td>56 ± 28</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Intrinsicsal deflection time (ave ± std dev)</td>
<td>78 ± 42</td>
<td>114 ± 40</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Shortest RS complex (ave ms ± std dev)</td>
<td>124 ± 44</td>
<td>157 ± 47</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Maximum deflection index (ave % ± std dev)</td>
<td>39 ± 13</td>
<td>51 ± 11</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Presence of a Q wave in lead I (%)</td>
<td>4</td>
<td>91</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Absence of Q waves in inferior leads (%)</td>
<td>42</td>
<td>99</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

PdW ≥ 34 ms
SN 73% & SP 63%

IDT ≥ 85 ms
SN 83% & SP 70%

SRS ≥ 121 ms
SN 74% & SP 57%

MDI ≥ 55
SN 30% & SP 89%

q in lead I
SN 91% & SP 96%

No q in inferior leads
SN 99% & SP 58%
1. ECG suggests epicardial VT exit site
   - A: Yes
   - B: No

2. Prior unsuccessful endocardial ablation?
   - C: No
   - D: Yes

3. Define scar location with CE imaging:
   - E: Sub-epicardial or mid-myocardial scar
     - F: Yes
     - G: No

4. Consider likelihood of epicardial circuit for underlying substrate
   - H: Low

Perform Endocardial Mapping and Ablation

- ECG Criteria (ref 40)
  1) Pseudo-delta >34 ms
  2) IDT (V2) >85 ms
  3) Shortest RS complex >121 ms
  4) ORS duration >211 ms

- ECG Criteria for NICM (ref 44)
  1) Absence of inferior Q wave
  2) Pseudodelta ≥75 ms
  3) MDI >0.59
  4) Presence of Q wave in lead I

- Probability of Epicardial Focus (ref 7)
  Normal: 6%
  ICM: 16%
  NICM: 35%
  ARVC: 41%
  Other CM: 18%

Consider Obtaining Epicardial Access for Mapping & Ablation

Circulation. 2012;126:1752-1769
### Table 5. Summary Of ECG Criteria For Regular WCT—The Griffith(I) Algorithm

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

### Table 4. Summary of Griffith ECG Morphology/Axis Criteria

<table>
<thead>
<tr>
<th>Classic RBBB Pattern</th>
<th>Classic LBBB Pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lead V1:</strong> rSR’</td>
<td><strong>Lead V1 &amp; V2:</strong> QS</td>
</tr>
<tr>
<td>- R’ &gt; r</td>
<td></td>
</tr>
<tr>
<td>- S cuts baseline</td>
<td>• Time to S nadir &lt; 70 ms</td>
</tr>
<tr>
<td><strong>Lead V6:</strong> RS</td>
<td><strong>Lead V6:</strong> R</td>
</tr>
<tr>
<td>- R &gt; S</td>
<td>• NO ‘Q’ allowed</td>
</tr>
<tr>
<td>- small initial ‘q’ allowed (&lt; 2 mm depth, &lt; 40 sec width)</td>
<td>• Either ‘RR’ or monophasic R</td>
</tr>
</tbody>
</table>

**Regular WCT**
First, evaluate QRS criteria listed in Step 1. If QRS criteria are met AND axis criteria (in Step 3) are not met AND no AV dissociation is present, then the diagnosis is SVT with AVC. All other WCTs are VT.

**Irregular WCT**
First, exclude polymorphic VT. Then, evaluate Step 1 for all other WCTs. If QRS criteria in Step 1 are met, then the diagnosis is AF with AVC. Otherwise, the diagnosis is AF with preexcitation.
Case 1

- 65 yo
- CAD (anterior MI), LVEF: 35%, NYHA I
- bb, ACE, spironolactone, amiodarone
- incessant wide QRS tachycardia
Case 1
Case 1

1. VT left ventricular origin (apex)

2. SVT with aberration

3. VT right ventricular origin (apex)

4. Antidromic SVT via an AP
mid-diastolic potentials
Case 2

- 60 yo man
- prior inferior MI
- LVEF: 45%
Case 2

1. VT left ventricular origin

2. SVT with aberration

3. VT right ventricular origin

4. Antidromic SVT via an AP
Case 2, activation mapping
Case 3

- 17 yo female
- ECG during SR: unremarkable
- Transthoracic echocardiography: normal LV and RV
Case 3
Case 3

1. RVOT VT

2. SVT with aberration

3. LVOT VT

4. Idiopathic left fascicular VT
Case 4

- 35 yo man
- Syncope during exercise
- ECG during SR: negative T waves V1–3
- Transthoracic echocardiography: RV dilation
Case 4
Case 4

1. SVT with aberration

2. RVOT VT due to ARVC

3. Epicardial VT due to ARVC

4. Mitral annulus VT
Case 5

- 27 yo man
- ECG during SR: unremarkable
- Holter monitoring: sustained tachycardia
- Verapamil terminates the tachycardia
Case 5

1. Mitral annulus VT
2. SVT with aberration
3. LVOT VT
4. Idiopathic left fascicular VT
Case 5

presystolic potential
Case 5

aVF
II
aVL
III
V1
V2
V3
V4
V5
V6
Map D
Map P
HBEP
HBED
Case 6

• 67 yo

• no structural heart disease

• Transthoracic echocardiography and coronary angiography ruled out structural heart disease

• MRI was normal

• referred to our hospital for EPS/ablation
Case 6
Case 6

1. VT left ventricular origin (apex)

2. SVT with aberration

3. VT right ventricular origin (apex)

4. Antidromic SVT via an AP
Case 6

pace mapping
11–12 of 12 leads
Case 6

propagation map

voltage map
Case 7

- **41 yo**
- **no structural heart disease**
- Transthoracic echocardiography and exercise test ruled out structural heart disease.
- Referred to our hospital for EPS/ablation
Case 7
Case 7

1. VT left ventricular origin

2. Idiopathic left fascicular VT

3. VT right ventricular origin (apex)

4. Antidromic SVT via an AP (Mahaim fiber)
ΣΑΣ ΕΥΧΑΡΙΣΤΩ