Differential Diagnosis and Treatment of Wide Complex Tachycardia

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When the diagnosis of a WCT is uncertain, the patient should be treated as if the rhythm is VT.
Treatment of WCT
Definitions

• Wide Complex Tachycardia (WCT) - a rhythm with QRS duration $\geq 120$ ms and heart rate $> 100$ beats/min

• Ventricular tachycardia - WCT originating below the level of His bundle

• SVT - tachycardia dependent on participation of structures at or above the level of His bundle
General Approaches to WCT

- **Clinical Characteristics** of the patient
  - Absence of structural heart disease makes SVT more likely.
    - But idiopathic VT can be seen!
  - History of structural heart disease makes VT more likely

- Classic RBBB or LBBB morphology argues STRONGLY for SVT with aberrancy

- Features suggestive of VT:
  - **QRS Morphology** not consistent with classic BBB
  - VA dissociation
  - Capture and fusion complexes

- **Brugada Algorithms**
Brugada algorithm

Absence of an RS complex in all precordial leads?
- Yes → VT
- No

R to S interval > 100 msec in one precordial lead?
- Yes → VT
- No

AV dissociation?
- Yes → VT
- No

Morphology criteria for VT present both in precordial leads V1-2 and V6?
- Yes → VT
- No → SVT with aberrant conduction

RBBB
Morphology criteria
LBBB

Absence of RS complex in all precordial leads
R to S interval > 100ms in one precordial lead
AV dissociation
### ECG distinction of VT from SVT with aberrancy

<table>
<thead>
<tr>
<th></th>
<th>Favors VT</th>
<th>Favors SVT with aberrancy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Duration</strong></td>
<td>RBBB: QRS &gt; 0.14 sec</td>
<td>&lt; 0.14 sec</td>
</tr>
<tr>
<td></td>
<td>LBBB: QRS &gt; 0.16 sec</td>
<td>&lt; 0.16 sec</td>
</tr>
<tr>
<td><strong>Axis</strong></td>
<td>QRS axis -90° to ± 180°</td>
<td>Normal</td>
</tr>
</tbody>
</table>
SVT with aberrancy

- Conduction to the ventricles via the His-Purkinje system, but with an abnormality
  - Right Bundle Branch Block (RBBB)
  - Left Bundle Branch Block (LBBB)
  - Intraventricular Conduction Delay (IVCD)
- These can be
  - Pre-existing BBB (helpful clue)
  - SVT-associated
- Any SVT can be conducted with aberrancy:
  - Sinus Tachycardia
  - Atrial tachycardia
  - Atrial flutter
  - Atrioventricular nodal reentrant tachycardia
  - Junctional Tachycardia
  - Atrioventricular Reentrant Tachycardia
WPW syndrome
(Antegrade conduction via accessory)

- Any SVT with antegrade conduction down an accessory pathway (WPW syndrome) will produce a wide QRS.
  - Slow myocyte-to-myocyte conduction arising from the ventricular insertion of the pathway
  - QRS morphology during tachycardia will look a lot like VT!
SVT vs VT

Coumel’s law
Absence of RS complex in all precordial leads
Capture or Fusion beat
AV dissociation
## Idiopathic monomorphic VT

<table>
<thead>
<tr>
<th></th>
<th>Adenosine-sensitive</th>
<th>Verapamil-sensitive</th>
<th>Propranolol-sensitive</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mech</strong></td>
<td><em>(Triggered activity)</em></td>
<td><em>(Fascicular reentry)</em></td>
<td><em>(Automaticity)</em></td>
</tr>
<tr>
<td>1) Exercise-induced</td>
<td></td>
<td>Fascicular</td>
<td>1) Exercise-induced</td>
</tr>
<tr>
<td>2) Repetitive monomorphic</td>
<td></td>
<td></td>
<td>2) Incessant</td>
</tr>
<tr>
<td><strong>Induction</strong></td>
<td>PES c/s cathecholamine</td>
<td>PES c/s cathecholamine</td>
<td>Cathecholamine</td>
</tr>
<tr>
<td><strong>ECG</strong></td>
<td>LBBB with inferior axis</td>
<td>RBBB with superior axis</td>
<td>RBBB, LBBB, Polymorphic</td>
</tr>
<tr>
<td></td>
<td>RBBB with inferior axis</td>
<td>RBBB with rt inferior axis</td>
<td></td>
</tr>
<tr>
<td><strong>Origin</strong></td>
<td>RVOT/LVOT</td>
<td>Lt posterior fascicle</td>
<td>RV/LV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lt anterior fascicle</td>
<td></td>
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</tbody>
</table>
RVOT VT

Coronal View

• No evidence of underlying structural heart disease

• Patients with symptoms not readily treated with medications are candidates for ablation.

• An ECG showing PVCs or VT can suggest the likely region of origin of the arrhythmia to assist in mapping.

• Mapping based on earliest activation
### Anterior vs. Posterior
- Lead I+, aVR > aVL
- Lead I-, aVR < aVL

### Caudal vs. Cranial
- aVL+ (>2 cm from PA)
- QRS > 140ms
- Notched R in inferior leads

### FW vs. Septal
- QRS duration ≥ 140 msec
- R-wave notching in inferior leads
- Lead V3 R/S ratio ≤ 1

<table>
<thead>
<tr>
<th>Category</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free wall versus septal sites</td>
<td>0.74</td>
<td>0.93</td>
<td>0.88</td>
</tr>
<tr>
<td>R-wave notching in inferior leads</td>
<td>0.79</td>
<td>0.99</td>
<td>0.94</td>
</tr>
<tr>
<td>Lead V3 R/S ratio ≤ 1</td>
<td>1.00</td>
<td>0.74</td>
<td>0.73</td>
</tr>
<tr>
<td>Anterior (leftward) versus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterior (rightward) sites</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative or isoelectric QRS in lead I</td>
<td>0.96</td>
<td>0.67</td>
<td>0.77</td>
</tr>
<tr>
<td>Caudal (&gt;2 cm from PV) versus Cranial Sites</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isoelectric or positive QRS in lead avL</td>
<td>0.86</td>
<td>1.00</td>
<td>1.00</td>
</tr>
</tbody>
</table>

PPV = positive predictive value; PV = pulmonary valve; RVOT = right ventricular outflow tract.

Wilber B, JCE 2005, 16:S52.
Localization
(QRS: Septal vs Free wall)

• QRS duration ≥ 140 msec
• QRS notching in inferior leads
• Lead V3 R/S ratio ≤ 1

• Majority arise 1-2 cm below the PV
• 20-30 % are free wall

Dixit et al, JCE 2003;14:1
Joshi et al, JCE 2005;16suppl:S52
Localization
(Lead I: Anterior vs Posterior)
Monomorphic ventricular tachycardia with LBBB morphology and an inferior axis

RVOT vs Aortic Sinus Cusp origin

1) total QRS duration
2) R-wave duration in leads V1 and V2
3) **R-wave duration index**, calculated as a percentage by dividing the QRS complex duration by the longer R-wave duration in lead V1 or V2
4) R/S-wave amplitude ratio in leads V1 and V2, measured from the QRS complex peak or nadir to the isoelectric line, expressed as a percentage
5) **R/S-wave amplitude index**, calculated from the greater percentage of the R/S-wave amplitude ratio in lead V1 or V2.
Fascicular VT

**Idiopathic left ventricular tachycardia**
- structurally normal heart
- Right bundle branch block + Left axis deviation
- verapamil-sensitive
- good longterm prognosis

**Mechanism of ILVT**
- Triggered activity
- microreentry
- Purkinje reentry

Anatomic extent of the reentry circuit in ILVT has not been defined.
Epicardial VT

A. pseudodelta wave ≥ 34 ms
B. The intrinsicoid deflection time ≥ 85 ms
C. RS complex duration ≥ 121 ms

Pseudodelta Wave
from the earliest ventricular activation (from the stimulation artifact in paced patients) to the earliest fast deflection in any precordial lead

Intrinsicoid Deflection Time
from the earliest ventricular activation (from the stimulation artifact in paced patients) to the peak of the R wave in V2

Shortest RS Complex
From the earliest ventricular activation (from the stimulation artifact in paced patients) to the nadir of the first S wave in any precordial lead

Idiopathic Epicardial Left Ventricular Tachycardia Originating Remote From the Sinus of Valsalva

Electrophysiological Characteristics, Catheter Ablation, and Identification From the 12-Lead Electrocardiogram

David V. Daniels, MD; Yen-Yu Lu, MD; Joseph B. Morton, MD; Peter A. Santucci, MD; Joseph G. Akar, MD, PhD; Alex Green, MD; David J. Wilber, MD

Background—Despite the success of catheter ablation for treatment of idiopathic ventricular tachycardia (VT), occasional patients have been reported in whom VT could not be ablated from the right or left ventricular endocardium or from the aortic sinus of Valsalva (ASOV).

Methods and Results—In 12 of 138 patients (9%) with idiopathic VT referred for ablation, an epicardial left ventricular site of origin was identified >10 mm from the ASOV. Coronary venous mapping demonstrated epicardial preceding endocardial activation by >10 ms (41±7 versus 15±11 ms before QRS onset; \(P<0.001\)). VT induction was facilitated by catecholamines and terminated by adenosine. Ablation through the coronary veins or via percutaneous transpericardial catheterization was successful in 9 patients; 2 required direct surgical ablation as a result of anatomic constraints. No ECG pattern was specific for epicardial VT. However, slowed initial precordial QRS activation, as quantified by a novel metric, the maximum deflection index, was more useful. A delayed precordial maximum deflection index ≥0.55 identified epicardial VT remote from the ASOV with a sensitivity of 100% and a specificity of 98.7% relative to all other sites of origin \(P<0.001\).

Conclusions—Although clinically underrecognized, idiopathic VT may originate from the perivascular sites on the left ventricular epicardium. The mechanism is consistent with triggered activity. It is amenable to ablation by transvenous or transpericardial approaches, although technical challenges remain. Recognition of a prolonged precordial maximum deflection index and early use of transvenous epicardial mapping are critical to avoid protracted and unsuccessful ablation elsewhere in the ventricles. (Circulation. 2006;113:1659-1666.)
Precordial MDI >0.55 reliably identified EPI VT

MDI : the maximum deflection index
TMD: time to maximum deflection in precordial lead
Take-home massages

► When the diagnosis of a WCT is uncertain
The patient be treated as if the rhythm is VT

► Several strategies or algorithms based upon ECG features