Cardiac Arrhythmias: Wide Complex Tachycardias

Seton Medical Center
Raising the Bar for Excellence in Cardiac Care

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Classification of Ventricular Arrhythmia by Electrocardiography

- Nonsustained ventricular tachycardia (VT)
  - ♥ Monomorphic
  - ♥ Polymorphic
- Sustained VT
  - ♥ Monomorphic
  - ♥ Polymorphic
- Bundle-branch re-entrant tachycardia
- Bidirectional VT
- Torsades de pointes
- Ventricular flutter
- Ventricular fibrillation

ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death
Ventricular Flutter
Spontaneous conversion to NSR (12-lead ECG)
VF with Defibrillation (12-lead ECG)
Common Cause of Monomorphic VT
Bundle Branch Reentrant Ventricular Tachycardia

- Malignant
- Treated with ablation
Acute Management of Ventricular Arrhythmias

- Wide complex tachycardia presumed to be VT if diagnosis is unclear
- DC cardioversion with sedation if hemodynamically unstable
- Don’t assume VT cannot be well tolerated!
- The rate, size of the heart and presence of additional complications are often more important than the source of the tachycardia

✓ Check the patient (need to defib?)
✓ Check the blood pressure (need to cardiovert?)
✓ Check the ECG (determine the rhythm)
Criteria for Differentiating Ectopy from Aberrancy

- Patient history / assessment
- QRS Width
- Concordance
- AV Dissociation
- Axis
- Morphology

**Note:**
VT is much more common than supraventricular tachycardia with bundle branch aberration. In wide QRS tachycardias VT is the right answer up to 80% of the time. A wide complex tachycardia is always considered ventricular in origin if the diagnosis is uncertain.
Patient History

- Acute ischemia / injury (Abnormal automaticity)
- Post myocardial infarction / ischemic cardiomyopathy (Reentrant circuit within myocardium)
- Non ischemic dilated cardiomyopathy (Bundle branch reentrant VT)

QRS Width

- The wider the QRS – VT is favored – However:
- SVT with LBBB will have a wider QRS than SVT with RBBB
- Other causes of SVT with wider than expected QRS: antidromic tachycardia and patients on Class I antiarrhythmics or amiodarone
- Not all VT is significantly wide
  - VT originating from septum more narrow than VT from free wall
  - If QRS more narrow than sinus rhythm = VT
Negative Concordance
AV Dissociation

- Independent atrial and ventricular activity (AV dissociation) is diagnostic for ventricular ectopy

**Only seen in 30% VTs**

- Ventricular tachycardia may also have retrograde P waves (retrograde P waves do not confirm VT)
AV Dissociation: Fusion or Capture Beats
Axis: Mean Wave of Depolarization

• **Extreme axis is strong indicator of ectopy**

• Right axis deviation confirms ectopy with LBBB pattern (right ventricular tachycardia)

• Ventricular tachycardia rarely occurs with normal axis
Let Your Hands Determine Axis

• Use Lead I and aVF
• Left hand represents QRS in Lead I
• Right hand represents QRS in aVF
• Fingertips will point in the same direction as the QRS complex
Normal Axis:
+0 to +90 Degrees

• Lead I: Upright QRS

• aVF: Upright QRS

• It’s always “normal” to be on the up and up
Right Axis Deviation:
+90 to +180 Degrees

• Lead I: Downward QRS

• aVF: Upward QRS

• Your right hand is up so this is “right” axis deviation
Left Axis Deviation:

0 to –90 degrees

• Lead I: Upright QRS

• aVF: Downward QRS

• Your left hand is up so this is “left” axis deviation
Extreme (Right Superior) Axis: -90 to –180 Degrees

- Lead I: Downward QRS
- aVF: Downward QRS
- Fingertips are both facing downward therefore the axis is "down and out" (opposite of normal)
Axis Practice
Axis Practice
Note: Left axis can be a normal shift leftward or a true left axis deviation.

Clinical Pearl:
The key is to look at lead II. If the QRS is upright in lead II leftward axis is within normal limits. If the QRS in lead II is not upright then left axis deviation is present.
Axis Practice

EXTREME – RIGHT SUPERIOR
Morphology (Shape)

Ventricular Ectopy compared to Aberrancy (BBB)

Morphology Challenges:
- BBB Reentrant VT
- Idiopathic RVOT
- Antidromic tachycardia
V1 and V6 are gold standard monitoring leads for ectopy versus aberrancy.

Bundle branch block patterns and ventricle ectopy can be differentiated by using the morphology of these leads.

DON’T rely on Lead II!!
Practice Alert
Dysrhythmia Monitoring

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Issued April 2008
Comparing Bedside Monitoring to the 12 Lead ECG

- Remember View of Positive Electrode (Camera)
- Importance of Lead Placement
- Identify Correct Lead on Rhythm Strip
Standard Lead Placement
5 or 6 Lead System

Standard 6 Lead Placement
Standard 5 Lead Placement
Modified 5 Lead System

Standard 5 Lead Placement
V1

Modified 5 Lead Placement
V1 & MCL6

V and
Standard 3 Lead Electrode Placement

Modified Chest Lead (MCL1) 3 Lead Placement

Lead I becomes MCL1
Lead II becomes MCL6

LA, left arm; LL, left leg; RA, right arm.

Figure 3  Electrode placement for a 3-lead system
Morphology Waves

- Not every QRS complex contains a Q wave, R wave and S wave!!
- Normal QRS width 0.05 to 0.10 sec
- Q – always negative (below baseline)
  - Normal Q not wider than 0.03 sec
- R – first positive above the baseline
- R’ – second positive above the baseline
- S – negative deflection following R wave or second component to entirely – complex

Remember: The alphabet says Q then R then S
Right Bundle Branch Block
Lead V1
Left Bundle Branch
Lead V1

- Nadir is the distance from the onset of the QRS complex to the lowest point of the S Wave
- Measure from the beginning of the QRS complex to the bottom valley or peak of the QRS
- The nadir in V1 should be < 0.06 sec for LBBB (slick down stroke)
Comparison of Morphology in Lead V1

RBBB

VT from Left Ventricle

LBBB

VT from Right Ventricle

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Physiological Critical Thinking Questions?

• In a right BBB – which ventricle depolarizes first?
• In a left BBB – which ventricle depolarizes first?

• If VT starts in the left ventricle – which ventricle depolarizes first?
• If VT starts in the right ventricle – which ventricle depolarizes first?
Comparison of Morphology in Lead V1

RBBB

VT from Left Ventricle

Left ventricle first

LBBB

VT from Right Ventricle

Right ventricle first

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Left Ventricular Ectopy

Lead V1

• Right Bundle Branch shaped
  – R wave with an early left peak (Rr’)
  – R wave with a single peak
  – q wave followed by R wave

Can also be shape of RBBB
Right Ventricular Ectopy
Lead V1

• **LBBB shaped**
  - Primarily negative wide rS complex
    - > 0.06 sec
  - r wave broader than 0.03 sec
  - Slurring on the down stroke

Note: LBBB shaped VT can come from RV or septum.
VT from RV includes: Idiopathic VT, BB Reentrant VT, Arrhythmogenic right ventricular dysplasia, VT from Brugada Syndrome
Lead V1 (VT Patterns)

VT with RBBB pattern or LVT

VT with LBBB pattern or RVT
# Comparison of Morphology in Lead V1

<table>
<thead>
<tr>
<th>RBBB</th>
<th>LBBB</th>
<th>VT from Left Ventricle</th>
<th>VT from Right Ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image1" alt="RBBB" /></td>
<td><img src="image2" alt="LBBB" /></td>
<td><img src="image3" alt="VT from Left Ventricle" /></td>
<td><img src="image4" alt="VT from Right Ventricle" /></td>
</tr>
</tbody>
</table>

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Bundle Branch Block Morphology in Lead V6

RBBB

LBBB

No Q
Ventricular Ectopy Morphology in Lead V6

**Left Ventricular VT**
- QS complex
- r wave followed by S wave with R:S ratio < 1

**Right Ventricular VT**
- Any Q Wave
- QS wave

Morphology in V6 for LVT
Morphology in V6 for RVT
Methodology for Differentiation Using ECG / Bedside Monitoring

Nice to Knows: AV dissociation, Negative concordance V1-V6, V6 changed from upright to negative, axis changed to right superior

YES

VT

NO

V1 positive QRS?

SVT with RBBB or VT

Evaluate QRS Morphology

V1 negative QRS?

SVT with LBBB or VT

SVT RBBB

SVT LBBB

VT

VT

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Practice ECG 1
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 2

AV Dissociation or Negative Concordance

Extreme Axis or V6 Negative
Practice ECG 3
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 4
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 5

AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 6

AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 7

AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative

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Practice ECG 8

AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 9

AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 10

AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 11

AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Practice ECG 12
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Vital Signs Stable

12 lead ECG Interpretation:
- Atrial Fibrillation
- RBBB with Left Anterior Hemiblock
Practice ECG 14
AV Dissociation or Negative Concordance
Extreme Axis or V6 Negative

Vital Signs Stable
12 lead ECG Interpretation: Non Specific Intraventricular Conduction Delay??
Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Linking to the Bedside Monitor Practice ECG 2

Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Linking to the Bedside Monitor Practice ECG 3

Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Linking to the Bedside Monitor Practice ECG 5

Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Linking to the Bedside Monitor Practice ECG 7

Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Dissociation or Negative Concordance
Extreme Axis or V6 Negative
Case Study

SVT RBBB

SVT LBBB

LVT

RVT

ORR RM3760 8/7/05 14:54:38 HR 136 SINUS TACHY PVC Ø NBP 112/45 (65) 25 mm/sec
Case Study
Case Study
Morphology Challenges
QT (CONGENITAL AND ACQUIRED)
QT Interval

• Measured from beginning of QRS complex to the end of the T wave

• Reflects both ventricular depolarization (QRS) and ventricular repolarization (T wave)

• Used most specifically to reflect ventricular repolarization
Technical Issues for Consideration

- Role of QRS width in QT interval
- Manual versus computer generated measurement
- U waves
- End of T wave in biphasic T wave
- Which lead for measurement
- RR interval in irregular rhythms
- Calculated measurement via 12 Lead ECG
- Bedside monitor calculation via e-calipers
- Continuous QT interval monitoring software
  - Qtip Study
Which Lead for Measurement

• QT usually measures longest V2 or V3
• T wave is often clearest V5 or V6

• For repetitive QT interval monitoring in an inpatient setting:
  – Pick a lead with a well defined T wave
  – Use the same lead consistently
U Waves and Biphasic T waves.
Heart Rate Adjustment

- QT interval needs to be adjusted for HR
- QT does not adjust to HR on a beat to beat basis

- **Dynamic changes are most important**
- Abnormal findings are uncovered during abrupt changes in the R to R

- **Irregular heart rhythms (i.e. atrial fibrillation) remain a clinical challenge**
Assessing for Risk of Torsades de Pointes in Atrial Fibrillation

• Print a long rhythm strip to assess over the course of the strip if the interval from the R wave to the peak of the following T wave is more than 50% of the proceeding RR interval.

• If so this is considered too long a QT interval and the risk for Torsades de Pointes is increased.

Measurements are using seconds.

**Bazett Formula**

- Formula not reliable at slow rates (under estimates); over estimates QT interval at fast HRs

**QT Dynamics**

- Linear regression analysis
Practicing the Bazett Formula

• HR 70
  - QT = .43 sec
  - R to R = .84 sec
  - \( \frac{.43}{.9165} = QTc .469 \)

• HR 38
  - QT = .80
  - R to R = 1.56 sec
  - \( \frac{.80}{1.28} = QTc .641 \)
## Expected QTc Intervals

<table>
<thead>
<tr>
<th></th>
<th>1 to 15 Years</th>
<th>Adult Males</th>
<th>Adult Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt; .44 seconds</td>
<td>&lt; .43 seconds</td>
<td>&lt; .45 seconds</td>
</tr>
<tr>
<td>Borderline</td>
<td>.44 to .46 seconds</td>
<td>.43 to .45 seconds</td>
<td>.45 to .47 seconds</td>
</tr>
<tr>
<td>Prolonged</td>
<td>&gt; .46 seconds</td>
<td>&gt; .45 seconds</td>
<td>&gt; .47 seconds</td>
</tr>
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</table>


**QTc .50 sec (500 msec or more is dangerous and should be considered an ominous sign of impending Torsade's de Pointes.**
Dangers of Abnormal Repolarization

• Places of unequal repolarization can set up for reentrant tachyarrhythmias

• There can be the development of early after depolarizations
What are Early After Depolarizations?

• Right after repolarization (or during) there is a transient sub threshold depolarization
  – Can occur during Phase II or III of the cardiac action potential
  – If an early after depolarization reaches threshold a second upstroke occurs and a triggered beat follows
  – The triggered beat may have its own after depolarization that reaches threshold – thus causing another triggered beat

• Thought to be etiology of Torsade's de Pointes
  – Acquired
  – Congenital
More on Early After Depolarizations

• Precipitating Factors
  – Hypokalemia
  – Hypomagnesemia
  – Heightened sympathetic tone
  – Slow heart rate
  – Prolonged repolarization (QT interval)
Early R-wave

T-wave

TdP initiated by R-on-T phenomenon caused by LQTS (QT=500ms).
Cardiac Ion Channel Abnormalities

- Long QT Syndrome (LQTS)
- Brugada disease
- Idiopathic short QT
  - < 300 to 340 msec

- Diagnosed by family history and ECG

- Note: Patients with heart failure can develop channelopathies
LQTS

• QTc > 450 ms
• Genetic defect in either potassium (LQT1 or LQT2) or sodium (LQT3) channels
  – Delayed repolarization (1 and 2)
  – LQT1 and LQT2 = 95%
    • Beta blockers
  – LQT3 = 5%
    • Beta blockers may be harmful
• Autosomal dominant trait
• 1 in 2500
• QT prolongation important risk factor for SCD
  • QTc < 440 ms  / < 5%
  • QTc 460 to 500 ms  / 20%
  • QTc > 500 ms  / 50%
Each Type of Congenital QT Looks Differently in Terms of T Wave Morphology

- Interestingly – some acquired Torsade's may be preceded by T wave morphology looking like congenital LQTS

- Long QT 1: wide, broad-based T waves
- Long QT 2: low amplitude, often notched T waves
- Long QT 3: long ST segment and tall, peaked T waves
Brugada Syndrome

• Disorder of cardiac sodium channel
• Autosomal dominant
  – Most common in Asia
  – Typical patient young male age 30 to 50 who is otherwise healthy.
• ST elevation in anterior precordial leads
  – Cove
  – Saddleback
• ECG can be dynamic
• In children temperature spike may uncover
  – Treat aggressively
• Syncope
  • 2 year risk of SCD approximately 30%
• ICD recommended
Quinidine and Isoproterenol two most common drugs used to prevent electrical storms.
Torsade's De Pointes

• Recognition of this life-threatening arrhythmia is important because it is not treated like other VTs

• Two groups: Acquired and congenital

• Acquired
  • Drugs prolonging repolarization
    – Most often as a result of blocking the potassium channel
  • Electrolyte abnormalities
    – Low potassium
    – Low magnesium
  • Severe bradycardias / pauses
More on Drugs that Prolong Repolarization (blocking of potassium channel efflux)

• www.QTdrugs.org
• www.torsades.org

• Class Ia and Class III antiarrhythmics
• Antihistamines
• Antibiotics
• Antipsychotics
• Antidepressants
• Sedatives
• Gastric motility agents
• Anticancer agents
• Opiate agonists

✓ Risk
✓ Possible Risk
✓ Conditional
Other Risk Factors for Torsade's de Pointes

- Rapid (IV) administration of QT prolonging agent
- Renal or hepatic dysfunction
- Female gender (particularly for drug induced)
- Advanced age
- Anorexia
- Heart disease
- Poly pharmacy
Class I........................
Slow conduction (widen QRS).
Some prolongation of refractory period (prolong QT interval).

Class III
Marked prolongation of refractory period (prolong QT interval).
Warning signs for Torsades de Pointes

• Increase QTc from predrug baseline of 60 ms,
• Marked QTc interval prolongation .500 ms
• T-U wave distortion that becomes more exaggerated in the beat after a pause
• Visible (macroscopic) T-wave alternans
• New-onset ventricular ectopy, couplets
• Nonsustained polymorphic ventricular tachycardia initiated in the beat after a pause.
Torsade's de Pointes

• Class I
  – Discontinue offending drugs
    • Note: Class IA drug induced TdP usually appears soon after the initial administration of the drug
  – Correct electrolytes
    • Magnesium
    • Potassium
  – Increase HR
    • Isoproterenol
      – 2 mcg/min then titrate to HR of 100 beats per minute
    • Temporary pacing at rate of 100 to 110
    • Permanent pacing if bradycardia or CHB cannot be resolved.

• Defibrillation if sustained
  – However, continue to assess for and treat cause
More on Magnesium in Torsade's de Pointes

- 2 Gm IV bolus over 1-2 minutes
  - Followed in 15 minutes by another bolus if necessary
  - May start continuous infusion at rate of 3-20 mg/min

- Benefit occurs without shortening of QT interval and in presence of normal Magnesium level
Case Example
Case Example
Case Example
QT Interval Monitoring Case Example

- Patient admitted for syncope after having motor vehicle crash while driving.
- Long standing history of paroxysmal atrial fibrillation – on dofetilide (Tykosin) for several years.
- Recent chemotherapy for breast CA resulting in a reduction of EF.
- Recent increase in carvedilol and lisinopril per general cardiology to improve EF.

- Next slide is admission ECG. Note the QTc interval.
- ABNORMAL ECG -

Unconfirmed Diagnosis
1. Strip 1: QTc consistent with admission ECG.
2. Strip 2: Marked QTc prolongation when patient asleep.
3. Initial run of ventricular tachycardia initiated by PVC firing at end of T wave,
Same patient with sustained Torsades de Pointes. Treated effectively with 2 grams IV Magnesium (magnesium level was normal at baseline). Magnesium is the drug of choice to stabilize the cardiac membrane. Dofetilide (Tikosyn) was also discontinued.

Note: Although the patient had been on dofetilide (Tikosyn) for several years, the recent change in ejection fraction and increase in beta blocker therapy increased her risk for Torsades de Pointes.
Polymorphic VT with normal QT:

- Seen frequently in ischemic conditions
  - Think revascularization
  - Think beta blockers
Special Considerations: Polymorphic VT (normal QT)

• DC cardioversion with sedation when unstable
• IV beta-blockers if ischemia suspected
  • Improve mortality
• IV amiodarone in absence of abnormal repolarization
  – Amiodarone better than placebo
  – Magnesium not better than placebo
• Urgent angiography to exclude ischemia
• Lidocaine may be reasonable if ischemia suspected
• Check electrolytes
• Consider any other potential reversible cause
Congratulations!!!