Bedside Cardiac Monitoring for All Practice Settings:
Implementing Evidence at the Point of Care

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Thought for the Day

“Professional nursing practice can only advance as much as individual nurses are aware that a knowledge gap exists in their practice, feel empowered to access further learning, and integrate evidence based competencies into their professional practice to provide safe, effective, efficient, patient centered, equitable care.”

www.tigersummit.com
History of Cardiac Monitoring

• 1962 1\textsuperscript{st} monitoring begins in Sydney Australia by Dr. Desmond Julian who noted: “All wards admitting patients with acute myocardial infarction should have a system capable of sounding an alarm at the onset of an important rhythm change and recording the rhythm automatically on an ECG....the provision of the appropriate apparatus would not be prohibitively expensive if these patients were admitted to special intensive care units. Such units should be staffed by suitably experienced people throughout the 24 hours.”

History of Cardiac Monitoring

• Later that year the 1\textsuperscript{st} CCU in the US opened in Kansas City by Dr. Hugh Day
• Early cardiac monitoring focused on observing heart rates and monitoring for life threatening arrhythmias
• Monitoring equipment has changed greatly over the past 40+ years
Despite advances in technology, the need for human on-site interpretation of the ECG is as important as it was 40 years ago for the following reasons:

- With the high sensitivity capabilities of the current monitors numerous alarms must be evaluated to prevent over treatment
- More aggressive approaches to treatment of myocardial ischemia improves outcomes requiring ongoing surveillance
- Complex device technology requires expert analysis of ECG monitoring data
- Many more drugs have been that prolong QT intervals – failure to identify patients at risk increases the incidence of sudden cardiac death
- Only humans, not monitors, can determine the goals of monitoring for individual patients
Short supply of skilled healthcare professionals with expertise in electrocardiography and cardiac monitoring for the following reasons:

- Multipurpose ICUs have replaced pure CCUs
- Shortage of critical care nurses results in under trained nurses working in the ICU
- Medical students, residents, and cardiology trainees are often inadequately trained in ECG interpretation and learn even less about cardiac monitoring leads, technology, and interpretation

Staff Qualifications

- Dedicated “monitor watcher”
  - Pros and cons
  - Investing in advanced monitoring technology may be more cost effective than dedicated “watchers”
- Combination of monitors from several units at a remote site by a dedicated “watcher”
  - Not recommended UNLESS expertise of the remote monitor watcher is superior and training cannot be provided to nurses on each monitored unit.
- Pagers that signal the nurse with monitor alarms that displays arrhythmia is helpful
Staff Qualifications

- Monitoring needs are different for each unit requiring different training for each unit
- Staff proficiencies should be determined based on unit population and purpose of monitoring
- Formal orientation to include both didactic and hands on practice with return demonstration
- Accurate electrode placement is an essential part of the orientation process

Understanding Specific ECG Abnormalities

- Normal Rhythms
- Intraventricular conduction defects
  - Bundle branch blocks
  - Aberrant conduction
- Tachyarrhythmias
  - Supraventricular
    - AV reentrant
    - AV nodal reentrant
    - A fib / flutter
    - Multifocal atrial tachycardia
    - Atrial tachycardia
    - Junctional ectopic tachycardia
  - Ventricular
    - Accelerated V arrhythmias
    - Nonsustained / sustained polymorphic VT
    - Prolonged QT interval associated VT
    - VF
- Bradyarrhythmias
- Premature complexes
  - Supraventricular
  - Ventricular
- Pacemaker Electrocardiology
  - Failure to capture, pace, or sense
  - Failure to capture both ventricles in biventricular pacing
- ECG abnormalities in acute myocardial infarction
  - ST segment elevation or depression
  - T Wave inversion
- Muscle or other artifacts
Understand General Electrophysiology Concepts

- Automaticity
- Excitation
- Conduction
- Sinus node physiology
- AV node physiology
- Wide and narrow QRS complexes
- Observation with arrhythmias
  - Sustained vs nonsustained
  - Monomorphic vs polymorphic
  - Stable vs nonstable
  - Symptomatic vs asymptomatic
  - Association with heart disease vs no heart disease
- Syncope
- Hemodynamic effects of arrhythmias
  - Influence of rate
  - Influence of heart disease
  - Influence of A-V synchrony
  - Influence of LV synchrony
- Function of Implantable devices
- Acute myocardial ischemia
  - STEMI
  - ST recovery of successful reperfusion
  - Reperfusion arrhythmias
  - NonSTEMI
  - Transient ischemia
- Effects of common antiarrhythmic drugs, rate control vs rhythm control

Specific Monitoring Skills

- Operation of monitoring system
- Recognition of limitations of computerized algorithms
- Proper skin prep
- Accurate lead placement
- Setting heart rate, ST alarm parameters
- Measurement of HR
- Measurement of intervals (with calipers)
- Recognition of atrial activity
- Evaluating pauses
- Diagnosis of specific rhythms
- Recording from postoperative epicardial wires
- Ability to intervene (unit protocols for responding, reporting, and documenting) in patients with:
  - Bradycardia
  - Tachycardia
  - Syncope
  - Cardiorespiratory arrest
  - Implantable devices
  - Temporary pacemakers
  - Transcutaneous pacemakers
Electrical Conduction Pathway

- SA Node
- AV Node
- Bundle of His
- AV Junction
- Right and Left Bundle Branches
- Anterior and Posterior Fascicles
- Purkinje Fibers

WAVES and COMPLEXES

- **P wave**: atrial depolarization
- **QRS**: ventricular depolarization
- **T wave**: ventricular repolarization
- **PR interval**: AV conduction time
- **QRS width**: intraventricular conduction time
- **ST Segment**: entire ventricular depolarization
- **QT interval**: used to reflect ventricular repolarization time
QRS Complex

• Not every QRS complex contains a Q wave, R wave and S wave!!
• Q – always negative (below baseline)
• R – first positive above the baseline
• R’ – second positive above the baseline
• S – negative deflection following R wave or second component to entirely – complex
• S’ – second negative deflection

Let’s Practice
Let’s Practice

ECG Paper – Horizontal Axis

Normal speed 25 mm/ sec
• Smallest box 1mm x 1mm
• 1 small box 0.04 sec
• 1 large box 0.20 sec
• 5 large boxes 1.0 sec
Measuring Rate on Irregular Rhythms

- **Irregular rhythms**
  - Count number of R-R intervals in a 6 second strip and multiply by 10

\[ 6 \times 10 = 60 \]

Measuring Rate on Regular Rhythms

- **Regular rhythms**
  - Count number of large boxes between R waves and divide into 300:

\[
\begin{align*}
1 &= 300 \\
2 &= 150 \\
3 &= 100 \\
4 &= 75 \\
5 &= 60 \\
6 &= 50 \\
7 &= 43 \\
8 &= 37 \\
9 &= 33
\end{align*}
\]

\[ 300 \div 4 = 75 \]
Calculating Rate

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<th>Number of Fifths between Consecutive QRS Complexes</th>
<th>Rate Is:</th>
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<tr>
<td>1</td>
<td>300</td>
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<tr>
<td>2</td>
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Understanding the Bedside Monitor Starts With Understanding the 12 Lead ECG
## Anatomy of a 12 Lead ECG

**The Basis of Bedside Cardiac Monitoring**

### Limb Leads

- I, II, III
- ♥ Standard Limb Leads
- ♥ Two electrodes used
- aVR, aVL, aVF
- ♥ Augmented Lim Leads
- ♥ One electrode used

### Precordial Leads

- V1, V2, V3, V4, V5, V6
- ♥ Also called Chest leads
Frontal vs. Horizontal Planes

- **Frontal plane:**
  - Leads I, II, III,
  - aVF, aVL, aVF
- **Horizontal plane:**
  - V leads

Dual and Single Electrode Leads

**Dual Electrode Leads**

- One positive electrode
- One negative electrode
- Records difference in electrical potential between selected electrodes

- Leads I, II, and III

**Single Electrode Leads**

- One positive electrode
- One negative reference point
  - Zero electrical potential
  - Center of heart

- Leads aVR, aVL, aVF
- V1-V6

**Note:** All leads are bipolar.
Importance of the Positive Electrode
Reason 1

• Consider the positive electrode the “the camera” (exploring electrode)

Electrode Placement
Limb Leads

Bedside Monitoring

12 Lead ECG
The Ground

• Note: Nothing travels toward the right leg as a positive electrode.

• The right leg is the ground used to absorb any excess electrical activity.

Standard Limb Leads
Leads I, II, III

Each Standard Limb Lead uses 2 surface electrodes per lead
Standard Limb Leads (Dual Electrode Leads)
Monitoring Lead Placement: Leads I, II, III

Augmented Limb Leads
Leads aVR, aVL, aVF

Each Augmented Limb Lead uses one surface electrode per lead
Augmented Limb Leads (Single Electrode Leads)
Monitoring Lead Placement: aVR, aVL, aVF

- Nothing Specific to the LV
- High Lateral Wall of LV
- Inferior Wall of LV

6 Limb Leads
Chest (Precordial) Leads
(Single Electrode Leads)
Electrode Placement
Chest (Precordial) Leads

- Lead V₁
  - 4ᵗʰ ICS, RSB
- Lead V₂
  - 4ᵗʰ ICS, LSB
- Lead V₃
  - Midway Between V₂ & V₄
- Lead V₄
  - L midclavicular line, 5ᵗʰ ICS
- Lead V₅
  - L anterior axillary line, same level as V₄
- Lead V₆
  - L midaxillary line, same level as V₄

A Closer Look at Chest Leads
The Point of View of the Positive Electrode

- V₁ – Septum
- V₂ – Septum
A Closer Look at Chest Leads
The Point of View of the Positive Electrode

• V3 – Anterior
• V4 – Anterior

A Closer Look at Chest Leads
The Point of View of the Positive Electrode

V5 – Low Lateral       V6 – Low Lateral
6 Precordial (Chest) Leads

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<th>aVR</th>
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<th>V4</th>
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<tbody>
<tr>
<td>+ Left Arm</td>
<td>+ Right Arm</td>
<td>+ 4&lt;sup&gt;th&lt;/sup&gt; ICS, RSB</td>
<td>+ L MCL, 5&lt;sup&gt;th&lt;/sup&gt; ICS</td>
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<td>Septal Wall</td>
<td>Anterior Wall</td>
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<th>aVL</th>
<th>V2</th>
<th>V5</th>
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</thead>
<tbody>
<tr>
<td>+ Left Leg</td>
<td>+ Left Arm</td>
<td>+ 4&lt;sup&gt;th&lt;/sup&gt; ICS, LSB</td>
<td>+ L anterior axillary, same level as V&lt;sub&gt;4&lt;/sub&gt;</td>
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<tr>
<td></td>
<td></td>
<td>Septal Wall</td>
<td>Low Lateral Wall</td>
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<th>aVF</th>
<th>V3</th>
<th>V6</th>
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<td>+ Left Leg</td>
<td>+ Left Leg</td>
<td>+ Midway Between V&lt;sub&gt;2&lt;/sub&gt; &amp; V&lt;sub&gt;4&lt;/sub&gt;</td>
<td>+ L midaxillary line, same level as V&lt;sub&gt;4&lt;/sub&gt;</td>
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<td>Anterior Wall</td>
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The Importance of the Positive Electrode

Reason 2

Normal Ventricular Depolarization

1. Septum depolarizes from left to right
2. Both ventricles depolarize from endocardium to epicardium
3. Basal portions of ventricles depolarize last
4. Mean direction of depolarization is downward, leftward, and posterior

How Leads Record

- Positive electrode is the recording electrode or “camera lens”
- Negative electrode or reference point tells camera which way to shoot
- If positive electrode sees depolarization approaching it, it records an upright complex
- If positive electrode sees depolarization heading away from it, it records a negative complex.

If depolarization is proceeding perpendicular to a lead, no deflection is recorded.
A Closer Look at Lead I

• Lead 1 Normals
  – P waves: Upright and gently rounded
  – QRS Complex: Upright
  – T Waves: Upright and smaller than QRS

A Closer Look at Lead II

• Lead II normals
  – P wave: upright and gently rounded
  – QRS: upright
  – T wave: upright and smaller than QRS
A Closer Look at Lead III

• Lead III normals
  – P wave: upright and gently rounded
  – QRS Complex: Upright
  – T wave: Upright and smaller than QRS

A Closer Look at aVR

• aVR Normals
  – P wave: inverted
  – QRS: inverted (rSr’ or rS)
  – T wave: inverted
A Closer Look at aVL

• aVL Normals
  – P waves: Upright or inverted
  – QRS: Upright or inverted
  – T wave: Upright or inverted (but no down sloping of ST)

A Closer Look at aVF

• aVF Normals
  – P waves: Upright and gently rounded
  – QRS: Upright
  – T wave: Upright and smaller than QRS
A Closer Look at V1

- Normal V1
  - P wave: inverted, upright or biphasic
  - QRS: inverted with rS pattern
  - T waves: inverted or upright

A Closer Look at V2

- V2 Normals
  - P waves: upright
  - QRS: inverted; rS pattern
  - T waves: upright, inverted
A Closer Look at V3

- V3 Normals
  - P wave: upright
  - QRS: equiphasic; RS pattern
  - T waves: Upright

A Closer Look at V4

- V4 Normals
  - P Wave: Upright
  - QRS: Upright; qRs
  - T wave: Upright
A Closer Look at V5

• V5 Normals
  – P wave: Upright
  – QRS: upright; qRs pattern
  – T wave: Upright

A Closer Look at V6

• V6 Normals
  – P wave: upright
  – QRS: upright; qRs pattern
  – T wave: upright
Normal V1-6: R Wave Progression

- The R wave becomes taller and the S wave becomes smaller as the electrode is moved from right to left.
- This pattern is called R wave progression.

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<tr>
<td>+ Left Arm</td>
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<td>+ 4th ICS, RSB</td>
<td>+ L MCL, 5th ICS</td>
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<tr>
<td>High Lateral Wall</td>
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<td>+ Left Leg</td>
<td>+ Left Arm</td>
<td>+ 4th ICS, LSB</td>
<td>+ L anterior axillary, same level as V₄ Low Lateral Wall</td>
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<td>+ Left Leg</td>
<td>+ Left Leg</td>
<td>+ Midway Between V₂ &amp; V₄</td>
<td>+ L midaxillary line, same level as V₄ Low Lateral Wall</td>
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<td>Inferior Wall</td>
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5/23/2012
Utilizing the Bedside Monitor to Provide 12 Lead ECG Information

Many Options
- 3 lead
- 5 lead
- 6 lead
- Hard Wired
- Telemetry
- Derived ECG

Comparing Bedside Monitoring to the 12 Lead ECG

- Remember View of Positive Electrode (Camera)
- Importance of Lead Placement
- Identify Correct Lead on Rhythm Strip
Rating System for Recommendations

**Class I Recommendations**
- Cardiac monitoring indicated in most, if not all, patients in this group.

**Class II Recommendations**
- Cardiac monitoring may be of benefit in some patients but is not considered essential for all patients.

**Class III Recommendations**
- Cardiac monitoring is not indicated because a patient’s risk of a serious event is so low that monitoring has no therapeutic benefit.
Three Reasons for Bedside Cardiac Monitoring

- Arrhythmia Detection
- Ischemia Monitoring
- QT Interval Monitoring

WELCOME TO ARRHYTHMIA MONITORING

The default reason for cardiac monitoring.
Class I Arrhythmia Monitoring Recommendations

- Patients resuscitated from cardiac arrest
- Patients in early phase of Acute Coronary Syndromes (including “Rule Outs”)
- Patients with unstable coronary syndromes and newly diagnosed high-risk coronary lesions
- Adults and children who have undergone cardiac surgery
- Patients after nonurgent percutaneous coronary interventions with complications
- Patients after ICD implant or pacer lead placement if pacer dependent
- Patients with temporary pacemaker or transcutaneous pacing pads
- Patients with AV block

Class I Arrhythmia Monitoring Recommendations

- Patients with Arrhythmias complicating Wolff-Parkinson-White Syndrome with rapid anterograde conduction over an accessory pathway
- Patients with Long-QT Syndrome and associated ventricular arrhythmias
- Patients with intraaortic balloon pump
- Patients with acute heart failure/ pulmonary edema
- Patients with indications for intensive care
- Patient under going diagnostic / therapeutic procedures requiring conscious sedation or anesthesia
- Patients with any other hemodynamically unstable arrhythmias
- Diagnosis of arrhythmias in pediatric patients
# Arrhythmia Monitoring

### Candidates
- Primary purpose for all patients on cardiac monitor

### Purpose
- Detection of and prompt intervention for **life threatening** arrhythmias

### Leads of Choice
- V1
- V6 (or MCL6)

### Diagram

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<td>VT vs Aberrancy</td>
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<tr>
<td>+ Left Leg</td>
<td>+ Left Arm</td>
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<td>+ L anterior axillary, same level as $V_4$</td>
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Understanding Arrhythmias
Physiologically

What does P wave represent?
Can we see sinus node?
Normal PR interval represent (prolonged?)?
What does skinny QRS represent?
Wide QRS?
What pacemaker options exist?
What should be the end result of each sinus impulse?

Understanding the Origin of Arrhythmias

- **Disorder of impulse initiation**
  - Abnormal automaticity
    - Enhanced
    - Abnormal
  - Triggered mechanism: disturbance in recovery or repolarization (less common)
    - Early or delayed after depolarizations

- **Disorder of impulse conduction**
  - Reentrant Circuit *(Most common)*
Difference Between Reentry and Abnormal Impulse Initiation

Ectopic focus in paroxysmal atrial fib (near pulmonary veins)

Ectopic focus in VT from ischemia or electrolyte abnormalities

Examples of Reentrant Tachycardias: Atrial
Examples of Reentrant Tachycardias: SVTs

Examples of Reentrant Tachycardias: Ventricular

> 90% of monomorphic VT is associated with scar!
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 for defibrillation?)
- Check the blood pressure (need for cardioversion?)
- Check the ECG (determine the rhythm)

Ectopy Versus Aberrancy

- **Ectopy:** Ventricular Tachycardia
- **Aberrancy:** SVT conducted aberrantly (with a bundle branch block)
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**

Positive Concordance: Cannot rule out antidromic tachycardia in WPW

**AV Dissociation**

Only seen in 30% VTs

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

- Ventricular tachycardia may also have retrograde P waves (retrograde P waves do not confirm VT)
Sinus Capture Beat: Another Way to Prove AV Dissociation

Axis

- **Axis**
  - Extreme axis is strong indicator of ventricular ectopy
  - Right axis deviation confirms ventricular ectopy with LBBB pattern
  - 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
- If hands are different, always place the left hand over the right.

“Handy” Method of Axis Calculation developed by J. Cooper, PhD., American College of CV Nursing
Axis Practice

Axis Practice
Axis Practice

Axis Practice
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<td><strong>AXIS</strong></td>
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<td>+ 4th ICS, LSB Septal Wall</td>
<td>+ L anterior axillary, same level as V4 Low Lateral Wall</td>
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<td><strong>AXIS</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Morphology (Shape)**

**Ventricular Ectopy compared to Aberrancy (BBB)**

**Morphology Challenges:**
- BBB Reentrant VT
- Idiopathic RVOT
- Antidromic tachycardia
Bedside Cardiac Monitoring

- 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 !!**

Standard Lead Placement

6 Lead System

Standard 6 Lead Placement
Modified 5 Lead System

Standard 6 Lead Placement
5 Lead Placement (No C6)

Modified 5 lead Placement-
MCL6

• Standard 3 Lead Electrode Placement

• 3 Lead Placement for Modified Chest Lead 1 (MCL1)
  Lead 3 (MCL6)
Normal Depolarization

- QRS complex is 0.06-.10 sec

Bundle Branch Block

- QRS complex is 0.12 sec or greater
- Incomplete BBB measures from 0.10 to 0.11
Right Bundle Branch Block

- $V_1 = rsR'$
- Triphasic complex rsR' pattern - positive
- Or an M shaped R wave with right peak taller
- Or a qR pattern

- $V_6 = qRS$
- Triphasic complex
- qRs with wide S waves
- Positive

QRS = .12 sec or more
Left Bundle Branch Block

- **V1**
  - Wide QS or rS complex - negative
  - Slick downstroke
  - Nadir <0.06 sec

- **V6**
  - Wide R wave with no initial septal q wave - positive
Comparison of Morphology in Lead V1

- RBBB
- VT from Left Ventricle
- VT from Right Ventricle
- LBBB

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

VT from Left Ventricle
- VT with RBBB pattern or LVT

VT from Right Ventricle
- VT with LBBB pattern or RVT

Lead V1 (VT Patterns)
- VT with RBBB pattern or LVT
- VT with LBBB pattern or RVT
Comparison of Morphology in Lead V1

RBBB
VT from Left Ventricle

LBBB
VT from Right Ventricle

Bundle Branch Block Morphology in Lead V6

RBBB

LBBB
Ventricular Ectopy Morphology in Lead V6

Left Ventricular Tachycardia
- $\sqrt{QS}$
- $R/S < 1$
- QS complex
- r wave followed by S wave with R:S ratio < 1

Right Ventricular Tachycardia
- Any Q Wave
- QS wave

Methodology for Differentiation Using ECG / Bedside Monitoring

Step 1: Axis (if 12 lead)
Step 2: V1 (morphology)
Step 3: V6 (morphology)
Step 4: Back to V1 (confirm morphology)

Note: Bedside monitoring starts with step 2
Practice EKGs

RBBB Above are two examples of RBBB morphology in lead V1. The first strip shows the classic rsR' pattern. The second strip shows a qR pattern. Patients who have infarcted their septum lose the first r wave and will typically demonstrate a qR pattern.
It is important to recognize RBBB and LBBB morphology in lead V1 (and document) when patients are in SR. This skill allows you to better differentiate between VT and SVT with BBB (aberrancy) when the patient is in a wide complex tachycardia.

In the examples above the patient is in an atrial flutter. In the first strip the patient is conducting with a normal QRS width. The second strip the patient is now in a 2:1 atrial flutter with an increased ventricular rate resulting in the right bundle becoming refractory. Therefore the patient conducts with a RBBB.
These strips are all from the same patient. None of these episodes of non sustained VT were documented. Nor, was there any documentation of provider notification. These arrhythmias were discovered while preparing for discharge. EP was consulted at that time which resulted in a delay in treatment and subsequent discharge.

Case Study
Clinical Pearls for Ventricular Arrhythmias

- V-fib seldom is seldom preceded by warning arrhythmias
  - Prophylactic lidocaine not indicated
- R on T PVCs are typically only important first 24 hours of myocardial infarction
- Bigeminy may need treated if cardiac output effected
- Ventricular ectopy (as infrequent as 15% burden) can result in heart failure

Clinical Pearls for Ventricular Arrhythmias

- Potential reversible causes
  - Hypokalemia: K < 3.2 mEq/L (cause or result)
  - Magnesium < 1.5 mEq/dL
  - Ischemia
  - Use of inotropic agents
Using Evidence Based Monitoring Makes You A STAR!

Three Reasons for Bedside Cardiac Monitoring

Arrhythmia Detection

Ischemia Monitoring

QT Interval Monitoring
Ischemia (ST) Monitoring

- **Purpose**
  - To monitor changes in ST segments (compared to baseline) in select leads

- **Leads of Choice**
  - Based on area of known or potential ischemia
  - Anterior wall / Left anterior descending coronary
    - Lead V3
  - Inferior wall / Right Coronary artery
    - Lead III
  - Lateral wall / Circumflex coronary artery
    - Lead V6

---

<table>
<thead>
<tr>
<th>Lead 1</th>
<th>aVR</th>
<th>V1</th>
<th>V4</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ Left Arm</td>
<td>+ Right Arm</td>
<td>+ 4&lt;sup&gt;th&lt;/sup&gt; ICS, RSB</td>
<td>+ L MCL, 5&lt;sup&gt;th&lt;/sup&gt; ICS</td>
</tr>
<tr>
<td>High Lateral Wall</td>
<td></td>
<td>Septal Wall</td>
<td>Anterior Wall</td>
</tr>
<tr>
<td></td>
<td></td>
<td>VT vs Aberrancy</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lead 2</th>
<th>aVL</th>
<th>V2</th>
<th>V5</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ Left Leg</td>
<td>+ Left Arm</td>
<td>+ 4&lt;sup&gt;th&lt;/sup&gt; ICS, LSB</td>
<td>+ L anterior axillary, same level as V&lt;sub&gt;4&lt;/sub&gt;</td>
</tr>
<tr>
<td>Inferior Wall</td>
<td>High Lateral Wall</td>
<td>Septal Wall</td>
<td>Low Lateral Wall</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lead 3</th>
<th>aVF</th>
<th>V3</th>
<th>V6</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ Left Leg</td>
<td>+ Left Leg</td>
<td>+ Midway Between V&lt;sub&gt;2&lt;/sub&gt; &amp; V&lt;sub&gt;4&lt;/sub&gt;</td>
<td>+ L midaxillary line, same level as V&lt;sub&gt;4&lt;/sub&gt;</td>
</tr>
<tr>
<td>Inferior Wall</td>
<td>Inferior Wall</td>
<td>Anterior Wall</td>
<td>Low Lateral Wall</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ST - LAD</td>
<td>VT vs Aberrancy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Low Lateral Wall</td>
</tr>
</tbody>
</table>

| | | ST - RCA | |
| | | | |

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5/23/2012
Class I
ST Segment Monitoring Recommendations

- Patients in early phase of Acute Coronary Syndromes (including “Rule Out”)
- Patients who present to ED with chest pain or anginal equivalent symptoms
- Patients who have had nonurgent percutaneous coronary interventions with suboptimal results
- Patients with possible variant angina resulting from coronary vasospasm

Class II
ST Segment Monitoring Recommendations

- Patients postacute MI
- Patients after nonurgent uncomplicated percutaneous coronary intervention
- Patients at high risk for ischemia after cardiac or noncardiac surgery
- Pediatric patients at risk of ischemia or infarction resulting from congenital or acquired conditions
Class III

ST Segment Monitoring Recommendations

- Patients with left bundle branch block
- Patients with ventricular paced rhythms
- Patients with other confounding arrhythmias that obscure the ST Segment
- Patients who are agitated

Methods To Improve ST Segment Monitoring

- Identification of body position changes
- Careful skin preparation
- Consistent lead placement
- Tailoring alarm parameters to patients baseline ST level
- Understand goals of monitoring in the individual patient
- Analyze ECG print out rather than just graphic trends
<table>
<thead>
<tr>
<th>Body Position</th>
<th>Careful Prep</th>
<th>Lead Placement</th>
</tr>
</thead>
<tbody>
<tr>
<td>• STs may fluctuate with body position changes</td>
<td>• ECG noise impedes accurate diagnosis</td>
<td>• Mark electrode placement</td>
</tr>
<tr>
<td>• May cause false alarms</td>
<td>• Skin prep essential to good tracing</td>
<td>• Waveform changes may occur with as little as 1cm change in location</td>
</tr>
<tr>
<td>• ST should be evaluated with patient in the supine position</td>
<td>• Clipping to remove hair</td>
<td>• Assess change in ST for true change or change lead location</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tailor Alarms</th>
<th>Understand Goals of Monitoring</th>
<th>Analyze ECG Printout</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Alarms must be set to reflect each individual patient’s baseline</td>
<td>• Monitor for silent ischemia</td>
<td>• Graphic trends are capable on most monitors with ST segment monitoring</td>
</tr>
<tr>
<td>• 1mm above and below for precordial leads</td>
<td>• Monitor for recurrent ischemia “Bad Alarm”</td>
<td>• Convenient for quick identification of ischemia</td>
</tr>
<tr>
<td>• 0.5 mm for limb leads</td>
<td>• Monitor for ST recovery after intervention with fibrinolytic or PCI</td>
<td>• Should never replace evaluation of rhythm strips</td>
</tr>
<tr>
<td>• 2mm reasonable in the more stable patient (helps eliminate false alarms)</td>
<td>• “Good Alarm”</td>
<td>• When in doubt always verify with a 12 lead ECG</td>
</tr>
</tbody>
</table>

- ECG noise impedes accurate diagnosis
- Skin prep essential to good tracing
- Clipping to remove hair
- Remove skin oils with abrasion (dry 4x4)
- Keep electrodes in original package – start to dry 20 minutes after opening
- Mark electrode placement
- Waveform changes may occur with as little as 1cm change in location
- Assess change in ST for true change or change lead location
- Monitor for silent ischemia
- Monitor for recurrent ischemia “Bad Alarm”
- Monitor for ST recovery after intervention with fibrinolytic or PCI “Good Alarm”
- Graphic trends are capable on most monitors with ST segment monitoring
- Convenient for quick identification of ischemia
- Should never replace evaluation of rhythm strips
- When in doubt always verify with a 12 lead ECG
ST Segment

- In limb leads the ST segment is normally isoelectric but may be slightly elevated or depressed by less than 1mm.
- In precordial leads ST segment elevation is normally not more than 1 to 2 mm (small elevation normal in many people).

Clinical Application:
1) Do not accept any ST elevation in limb leads
2) Do not accept any ST depression in chest leads

The “J” Point

- Point where the QRS complex and the ST segment meet.

Clinical Application: There can be ST segment elevation with no J point elevation.
ST Depression from Atrial Repolarization

- ST segments are measured 60 to 80 msec from J point.

T Waves

- Represents ventricular repolarization
- Slightly asymmetrical
- Usually oriented in the same direction as the previous QRS
- Not normally > than 5mm (limb leads) to 10 mm (precordial) high

Clinical Application:
Do not accept any T wave that is too big in any lead.
ECG Assessment Priorities

1) Assess for ST segment elevation first
   – ST elevation and need for reperfusion

2) Assess for T wave inversion next
   – Non STEMI or
   – Unstable angina (ischemia)

3) Assess for ST segment depression third
   – Ischemia

Patterns of ST Elevation Injury

- Hyperacute T Wave
  – As early as 2 minutes after occlusion

- J Point Elevation
Patterns of ST Elevation Injury

- Subtle ST Elevation
  Forming Broad T Wave

Subtle Inferior Elevation
Most Obvious J Point Elevation

Hyper Acute T Wave with J Point Depression
Post Hyper Acute T Waves

T Waves Too Big??????
Answer: YES
(same patient 2 hours later)

T Wave Inversion: Key Points

- T wave should be positive in lead I and II
- **Normal inversion is rare in V2 – V6**
- Inversion in lead III, aVL and aVF may be normal
- Inversion in V1 is common - always compare to previous ECG

- **T Wave Inversion Associated With Ischemia/Infarction**
  - Deep T wave Inversion
  - Disproportionate T wave Inversion (in relation to QRS voltage)
  - New or changing T wave Inversion
  - QTc usually increased
2 Types of T Wave Inversion: NSTEMI or Ischemia

- Terminal T wave inversion
- Symmetrical T wave inversion

More on T Wave Inversion

- T wave inversion is a “warning” (for ischemia or injury) unless.............

-The T wave inversion is after a STEMI
  - After a STEMI T wave inversion is expected
  - Terminal T wave inversion is a sign of reperfusion after a STEMI
  - Symmetrical T wave inversion will develop after terminal T inversion
ECG Changes After STEMI

Non Reperfused
- T wave enlargement
- ST elevation
- Q wave formation or loss of R wave amplitude
- ST stabilization
- **T wave inversion** (within 48 - 72 hours) before ST resolution
- ST resolution
- **T waves stays inverted** for period of time (takes weeks to months)
- Possible disappearance of Q waves

Reperfused
- Earlier ST normalization and stabilization
- T wave inversion may accelerate
  - Terminal T wave inversion initially
  - T waves deepen symmetrically over time
- Q wave development is less pronounced or even absent

ST Evolution: Pseudo Normalization

[Diagram showing ECG changes]
T WAVE INVERSION

Both Bad and Good

Practice ECG 1 of 2
ST Depression

- Horizontal ST Depression
- Down Sloping of ST Segment

ST Segment Changes

- Primary Changes (Most Important)
  - What is the problem? (injury or ischemia)
  - Where is the problem? (what wall of ventricle)

- Reciprocal Changes
  - Reciprocal changes may be present in STEMI
    - ST segment depression in leads reciprocal to those with ST elevation
    - Reciprocal changes can help confirm primary
    - May Potentially Be Confused as the Primary Problem

Clinical application: Before calling ST segment depression ischemia – double check the reciprocal leads for missed ST segment elevation
<table>
<thead>
<tr>
<th>Lead 1</th>
<th>Lead 2</th>
<th>Lead 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Arm</td>
<td>Right Arm</td>
<td>Left Leg</td>
</tr>
<tr>
<td>High Lateral Wall</td>
<td>High Lateral Wall</td>
<td>In Inferior Wall</td>
</tr>
</tbody>
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<tr>
<th>aVR</th>
<th>V1</th>
<th>V2</th>
<th>V3</th>
<th>V4</th>
<th>V5</th>
<th>V6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Arm</td>
<td>4th ICS, RSB</td>
<td>4th ICS, LSB</td>
<td>Midway Between V2 &amp; V4</td>
<td>L MCL, 5th ICS</td>
<td>L anterior axillary, same level as V4</td>
<td>L midaxillary line, same level as V4</td>
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<td>Septal Wall</td>
<td>Septal Wall</td>
<td>Anterior Wall</td>
<td>Anterior Wall</td>
<td>Low Lateral Wall</td>
<td>Low Lateral Wall</td>
</tr>
</tbody>
</table>

Value of Lead 3 in Reciprocal Changes
Value of Lead V3 in Reciprocal Changes

Using the “V” Telemetry Lead to Assess the Right Ventricle and Posterior Wall of the Left Ventricle

These are vulnerable areas of myocardium: Not directly assessed on 12 Lead!
Associated Changes in Inferior MIs

• ST depression in V1-V3
  – *Think posterior injury*

• Isolated ST elevation in VI
  (But don’t rely on V1)
  – *Think RV involvement*
Right Sided and Posterior Quick Look with V1 Lead on Bedside Monitor

- **Right Sided Lead**
  - Place electrode in V4R Position
  - 5th ICS RMCL
  - Attach V monitoring lead (Brown Lead) to electrode
  - Assure monitor lead selector is on V
  - If ST elevation noted → RV Infarct
  - Run strip and clearly mark “V4 Right Lead”

- **Posterior Lead**
  - Place electrode in V8 position
  - Under tip of left scapula same level as V6
  - Attach V monitoring lead (Brown Lead) to electrode
  - Assure monitor lead selector is on V
  - If ST elevation noted → Posterior Infarct
  - Run strip and clearly mark “V8 Posterior Lead”
ECG showing ST segment elevation in the inferior Leads (II, III, and aVF) with reciprocal depression in Leads I and aVL. There is also depression in V2 and V3 most likely representing reciprocal changes from ST elevation in the posterior leads. This is an ideal patient for a 16 lead ECG to assess for injury to the right ventricle and posterior wall of the left ventricle.

Same Patient as Previous 12 Lead:
Do to hypotension the point of care nurse used the V lead from bedside monitoring to record a V4R lead. This recording confirms RV injury and this knowledge was used to guide treatment.
ST Segment Monitoring

A SUCCESS Story!!

The next 2 slides show the following:
1. Admission ECG for a patient with an anteroseptal / lateral wall STEMI.
2. ECG post intervention for same patient.
   1. Note: The T waves have not yet inverted post intervention. Ideally T waves will begin to invert after an intervention showing evidence of reperfusion.

REMEMBER: T wave must invert within 48-72 hours after a STEMI (the sooner the better). Failure of T waves to invert after a STEMI is indicative of post infarction regional pericarditis and the patient is at higher risk for myocardial rupture.
The strip below assessing ST segments in V3 was done 48 hours post STEMI (same patient as previous 2 ECGs). The failure of the T waves to invert is indicative of post infarction regional pericarditis with increased risk of myocardial rupture. The patient was hypotensive, which raises the concern for cardiac tamponade as the etiology of the hypotension. This assessment finding was communicated to the cardiologist.

The patient’s echocardiogram showed a large pericardial effusion and the patient subsequently underwent a surgical pericardial window.
ST Segment Monitoring

An Example to Improve Practice

- Please review the ECG on the next slide demonstrating ST segment elevation in leads II, III, aVF, and V4, V5, and V6.
- The patient was admitted to CCU from Woodlawn post CABG. The ECG on the next slide was approximately 5 weeks post CABG.
- The ECG changes on the next slide occurred with a hemoglobin < 8.0.
- The patient continued to have a hemoglobin level below normal for the remainder of the hospital stay. However, 5 days later it was charted that the patient met exclusion for ST segment monitoring.
- Important points:
  - Graft occlusion is a potential complication post CABG.
  - Anemia decreases myocardial oxygen supply and contribute to an acute coronary event.

Just 5 days after this ECG with ongoing anemia.
ST Segment Monitoring

An Example to Improve Practice

- This patient received ST segment monitoring on admission to the hospital due to admitting diagnosis of chest pain.
- Please read the results on the next slide of the patient’s stress test (first report) and cardiac catheterization results (second report).

**IMPRESSION:**

Within the limits of the study, suspect moderate to significant ischemia involving the anterior, anteroseptal, anterolateral, and lateral walls. Possible transient ischemic dilatation with stress, suggesting multivessel disease.

Significantly reduced LV function, LVEF of 27%.

**FINAL IMPLICATIONS:**

1. Significant disease is noted involving the second diagonal branch, ramus intermedius artery, circumflex proper and right coronary artery.
2. Significant gradient on pullback into the aorta with a gradient of 40 mmHg.
3. LV function appears to be in the 50% range, but this may be overestimated because there was significant ectopy during that time.
4. Dominant right system.
5. Recommend an Echo at this time to assess gradient on pullback. The patient does have what appears to be a large mass in the abdominal area. This could represent infection vs. malignancy. Will proceed at this time with that work up and then we can re-consider the cardiac issues later. She is currently pain-free and asymptomatic.
6. Patent stent in the proximal left circumflex, which is a 4.0x18 bare metal Driver stent from 2009.
ST Segment Monitoring

An Example to Improve Practice

• Although the patient needed further revascularization, it was unable to be performed because the patient required an urgent surgery, resulting in the inability to continue clopidogrel or prasugrel after the placement of an intracoronary stent.
• The decision was made to proceed with the high risk urgent surgery (due to inability to revascularize an ischemic patient) and later proceed with coronary intervention pending post op recovery and results of urgent surgery.

ST Segment Monitoring

An Example to Improve Practice

• The patient received ST segment monitoring preoperatively.
• The patient returned to CCU post operatively but did not receive post operative ST segment monitoring.

• Note: There is high risk for perioperative ischemia and infarction in high risk surgical patients and therefore ST segment monitoring should have been continued.
Three Reasons for Bedside Cardiac Monitoring

Arrhythmia Detection

Ischemia Monitoring

QT Interval Monitoring

- **Purpose**
  - To monitor for increase in QT interval to identify and intervene in patients at high risk for Torsades de Pointes

- **Leads of Choice**
  - **Lead where an accurate QT Interval can be measured**
  - Patient can be changed to another lead to run a strip to measure QT or 12 lead can be done if QT not easily measured in V1 or V6

- **Notes:**
  - QT interval needs to be adjusted for HR
  - V2 and V3 usually have the longest QT
  - Dynamic changes are most important
  - Abnormal findings are uncovered during abrupt changes in the R to R
The Electronics

Action Potential of Cardiac Cells

- Phase 0: Rapid depolarization – Sodium Influx
  (beginning of QRS complex)
- Phase 1: Brief, rapid initiation of repolarization
The Electronics

- **Phase 2:** Plateau phase
  - Calcium Influx (greater than potassium efflux) correlates with ST segment

- **Phase 3:** Sudden acceleration in the rate of repolarization - Potassium Efflux Correlates with T wave

- **Phase 4:** Resting membrane potential

**QT represents both depolarization and repolarization**
Class I
Slow conduction (widen QRS). Some prolongation of refractory period (prolong QT interval).

Class III
Marked prolongation of refractory period (prolong QT interval).

Lead II or V5
QRS
P
RR Interval
baseline
Tangent
QT
Bazett Formula

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

QT Dynamics

- Linear regression analysis

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>
</tbody>
</table>


QTc .50 sec (500 msec or more is dangerous and should be considered an ominous sign of impending Torsade’s de Pointes.)
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.


Class I
QT Interval Monitoring Recommendations

• Patients administered an antiarrhythmic drug known to cause Torsades de Pointes
• Patients who overdose from a potentially proarrhythmic agent
• Patients with new onset bradyarrhythmias
• Patients with severe hypokalemia or hypomagnesemia
Class II

QT Interval Monitoring Recommendations

• Patients who require treatment with antipsychotics or other drugs with possible risk of Torsades de Pointes
• Patients with acute neurologic events

Class III

• Healthy patients administered drugs that pose little risk for Torsades de Pointes

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
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](http://www.QTdrugs.org)
- [www.torsades.org](http://www.torsades.org)
- Class Ia and Class III antiarrhythmics
- Some antihistamines
- Some antibiotics
- Some antipsychotics
- Some antidepressants
- Some sedatives
- Some gastric motility agents
### 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

### Acquired Torsade's De Pointes

- **Warning Signs:**
  - QTc prolongation
    - Usually greater than 0.5 sec
  - T Wave aberration or T wave alternans
  - Prominent U waves
  - Couple of PVCs and couplets
  - Initiated by short long RR interval (Pause dependent)

- **Short bursts:** QRS peaks first appear to be up and then to be down (Can degenerate into V fib)
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.
QT Interval Monitoring

- 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..
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
  
  role of 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
Heart Blocks – AV Blocks

- Classification
  - 1\textsuperscript{st} Degree
  - 2\textsuperscript{nd} Degree
    - Type I (Wenckebach)
    - Type II
  - High Grade
  - Third Degree

2nd Degree AV Blocks

- One P Wave at a time fails to conduct to ventricle
- Type I (Wenckebach)
  - Conduction fails in AV node
    - There will be problem with PR interval
    - Narrow QRS complex means block is in AV node and not below
- Type II
  - Conduction fails below the AV node and usually involves both bundles
    - There will be no problem with the PR interval
    - QRS complex is usually wide (Can be narrow due to location of block (below node but not involving both bundles) – less common
Wenckebach (2nd Degree Type I)

- Sinus node fires regularly
- Disease in AV node
- Group beating is noted
- First P-R of group of often longer than normal with progressive lengthening of the P-R until a beat is not conducted
  - PR problem because of physiological location
- In absence of BBB QRS is normal
  - Normal QRS width because of physiological location
- Conduction ratios may be 2:1, 3:2, 4:3 etc.
- May develop 2:1 conduction if sinus rate increases
  - Verify the block is still type I
  - P-R longer than normal
  - Absence of prolonged QRS
- Treatment: Often none
  - Acutely with symptoms: Atropine or TTVP
  - Atropine will work because of physiological location

Wenckebach (2nd Degree Type I)

[Electrocardiogram images]

Treatment: Often none
- Acutely with symptoms: Atropine or TTVP
- Atropine will work because of physiological location
2\textsuperscript{nd} Degree AV Block Type II

- Similar to Type I however no progressive lengthening of P-R interval
  - Physiological problem does not involve AV node
- Disease within or below bundle of His
- P-R interval is fixed with normally conducted beats
- QRS: wide

- If 2:1 conduction look for:
  - Normal P-R interval with conducted beats
  - Wide QRS complex
- Treatment: Usually requires permanent pacing

The above rhythms occurred in a patient who had digoxin toxicity. Neither rhythm disturbance was reported at the time of occurrence other than the posting of the rhythm strips on the chart. The point of care nurse on the following shift notified the provider. The bottom strip represents second degree heart block type I. The patient’s digoxin had been discontinued based on a digoxin level. The patient’s beta blocker was discontinued after the reporting of the arrhythmia.
Heart Blocks - High Grade AV Block

- Two or more consecutive atrial impulses are blocked.
- P waves: Regular, but 2 or > fail to conduct to the ventricles
- QRS: Narrow in type I & wide in type II
- Ventricular Rate: Slow, often symptomatic
- Treatment: Atropine for Type I
  - Pacing for Type II - Usually

Third Degree AV Block – Complete

- No atrial impulses are conducted to the ventricles
- One form of AV dissociation
- Ventricular Rate: Maintained by ventricular escape (wide QRS) or by pacemaker coming from His bundle (narrow QRS – less common)
- Symptomatic if develops acutely
- May be well tolerated if develops overtime
- Treatment: Perm. Pacer
Potential Causes of Bradycardias in Critical Care

- Propofol
- Cardiac disorders and medications
- Vasovagal
- CNS injury
- Hypothyroid
- Hypothermia
- Multiple other

Rule of thumb: Pace if cause cannot be reversed.

AV Nodal Re-entrant Tachycardia (AVNRT)

- A PAC initiates atrial depolarizations which travel via the slow AV nodal pathway. (the fast pathway is refractory (blocked) due to previous SA node depolarization.)
- At the AV node exit depolarizations travel antegrade to depolarize the ventricles and retrograde up the fast pathway to depolarize the atria.
- This cycle repeats.
AV Nodal Re-entrant Tachycardia
AV Nodal Re-entrant Tachycardia
AV Nodal Reentrant Tachycardia (typical)

- Most common supraventricular tachycardia
- Least likely to be life threatening
- **Narrow QRS has no visible P waves**
  - Simultaneous depolarization
- Or, P waves are so close to QRS they look like part of it (pseudo R waves in V1 and pseudo R waves in inferior leads)

Importance of Rhythm Onset

![Diagram of ECG showing P' wave and V1 lead]

- V1 lead with P' wave and ECG tracing
Importance of Rhythm Onset

The patient above demonstrates the most common form of SVT: AV Nodal Reentrant Tachycardia (AVNRT). One of the keys to accurately interpreting a tachycardia is to look at the ONSET. AVNRT is typically triggered by a PAC that is conducted by a long PR interval. This is a patient who is several weeks post CABG. In strip 1 you see she has a run of atrial tachycardia and the last P wave in the run is conducted by a long PR interval. This triggers her narrow complex SVT. The patient was successfully converted with IV adenosine. Beta blockers were also used to suppress atrial activity.

COME to a Level 4 or Beyond the Core Curriculum class to learn more about AVNRT.
Sustained AV Nodal Reentrant Tachycardia

Treatment for AVNRT

- Vagal (teach patient)
  - Valsalva
  - Carotid massage
  - Facial cold water immersion
- Adenosine or non-dihydropyridine calcium channel blockers (stable)
  - Adenosine preferred
  - Contraindications
  - Benefits of long acting agents
- DC Cardioversion (unstable)
Special Considerations in Atrial Arrhythmias: Lewis Lead

The Lewis Lead

When P waves are not clearly seen in a rhythm strip (see lead 3 above), the Lewis lead can be very helpful in assessing for the presence of atrial activity.

As seen in the Lewis lead above this patient is clearly in an atrial flutter. The atrial flutter is not as obvious in the lead III rhythm strip.
Lewis Lead
Atrial Lead: Atrial Pacing Wire

General Principles for Atrial Arrhythmias

- Atrial Fibrillation
  - Rate control is first priority
  - Optimize rate control based on clinical assessment of perfusion
  - Hemodynamic instability
    - BP < 90 systolic or HR > 150 BPM
- Anticipate need for rhythm control with atrial flutter
- Critical care setting associated with increased catecholamine levels
  - Treat infection
  - Treat inflammation
  - Correct electrolytes
<table>
<thead>
<tr>
<th>Class</th>
<th>Specific Medications</th>
<th>Purpose of Medication</th>
<th>Major Cardiac Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I A</td>
<td>Disopyramide</td>
<td>Rhythm Control</td>
<td>Torsade de pointes, HF</td>
</tr>
<tr>
<td></td>
<td>Procainamide</td>
<td>Rhythm Control</td>
<td>Torsade de pointes</td>
</tr>
<tr>
<td></td>
<td>Quinidine</td>
<td>Rhythm Control</td>
<td>Torsade de pointes</td>
</tr>
<tr>
<td>Class I B</td>
<td>Not used in atrial fibrillation</td>
<td>Rhythm Control</td>
<td>Ventricular tachycardia, HF, Atrial Flutter</td>
</tr>
<tr>
<td>Class I C</td>
<td>Flecainide</td>
<td>Rhythm Control</td>
<td>Torsade de pointes (rare) * Organ toxicity</td>
</tr>
<tr>
<td></td>
<td>Propafenone</td>
<td>Rhythm Control</td>
<td>Torsade de pointes</td>
</tr>
<tr>
<td>Class II</td>
<td>Beta Blockers</td>
<td>Rate Control</td>
<td>Torsade de pointes, HF</td>
</tr>
<tr>
<td>Class III</td>
<td>Amiodarone</td>
<td>Rhythm / Rate Control</td>
<td>Torsade de pointes, HF, Beta blocker side effects</td>
</tr>
<tr>
<td></td>
<td>Dofetilide</td>
<td>Rhythm Control</td>
<td>* Organ toxicity</td>
</tr>
<tr>
<td></td>
<td>Ibutilide</td>
<td>Rhythm Control</td>
<td>Torsade de pointes</td>
</tr>
<tr>
<td></td>
<td>Sotalol (also contains beta blocker)</td>
<td>Rhythm Control (also controls rate)</td>
<td>Torsade de pointes, HF, Beta blocker side effects</td>
</tr>
</tbody>
</table>

**ADDITIONAL CLINICAL APPLICATIONS**

*Bedside Monitoring*
**Derived ECG**

**E:** Lower extreme of the sternum (Brown +)

**A:** Left mid-axillary line, same transverse line as E (Black +)

**S:** Sternal manubrium (Red -)

**I:** Right mid-axillary line, same transverse line as E (White -)

**G:** Fifth electrode is the ground and can be placed anywhere on the torso (Green no polarity)

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**Hyperkalemia**
Delta Wave of Pre-excitation Syndrome

- 60 to 70% of WPW shows evidence in SR

- Left sided accessory pathway: Positive delta wave in V1
- Right sided accessory pathway: Negative delta wave in V1
Arrhythmias of WPW (AVRT or CMT)

Manifest accessory pathway
- PR ≤ 120 ms
- Delta wave present
- QRS ≥ 120 ms
- Repolarization abnormal

Concealed accessory pathway
- PR normal
- Delta wave absent
- QRS normal
- Repolarization normal

Orthodromic SVT

Antidromic SVT

Atrial fibrillation
Critical Thinking Guideline for Cardiac Monitoring

**NEEDS OF THE PATIENT DRIVE THE DECISIONS**

- Ejection fraction < 30%
- Implantable cardioverter defibrillator
- Non ischemic cardiomyopathy
- Syncope as reason for admission
- Current frequent premature beats or short runs of tachycardias

**Patient Characteristics**

- Arrhythmia monitoring: Use V1 and V6 (or MCL6) as primary monitoring leads
### Patient Characteristics
- New administration of class I or class III antiarrhythmics
- Electrolyte abnormalities (hypokalemia, hypomagnesemia, hypocalcemia)
- QTc > 0.45 seconds
- Receiving Haldol or other high risk medications

### Monitoring Priorities
- Use arrhythmia monitoring leads as baseline monitoring leads.
- Measure QTc interval q 4 hours with rhythm interpretation in lead where QT interval can be clearly defined. Document and record lead used for measurement. Use consistent lead in the measuring of QTc.

### Patient Characteristics
- Stable acute coronary syndrome (ACS) or rule out ACS as reason for admission
- Admission symptoms suspicious for ischemia (shortness of breath, nausea, fatigue, etc)
- Admission with heart failure with history of recent revascularization

### Monitoring Priorities
- Ischemic monitoring: Use V3 and lead III are primary ischemia detection leads if area of ischemia or culprit vessel is unknown
- If known choose ischemia monitoring leads based on ECG footprint during active ischemia – document reason for use of chosen leads
- Perform ECG with posterior leads during symptomatic episodes with non-diagnostic standard 12 Lead
- Simultaneously monitor in V1 for arrhythmia detection for patients admitted to ICU or step down level of care
- Note: Whenever possible in ICU and step down level patients a 6 lead telemetry system should be used in order to monitor V1 for arrhythmia detection and the second V lead for ischemia monitoring.
Patient Characteristics

- High risk (hemodynamic or electrical instability) ACS

Monitoring Priorities

- Patients will typically be monitored with 5 lead hardwire due to other monitoring needs
- V1 must be used as primary monitoring lead in any unstable patient
- Secondary monitoring can be a limb lead or modified chest lead to aid in either arrhythmia interpretation or ischemia detection

Pulling it All Together
Final Case Study: ECG 1

Final Case Study: ECG #2
Final Case Study: ECG #3

Final Case Study: ECG #4
Final Case Study: ECG #5
Final Case Study: ECG #6

Moral of today’s class: Treat the patient and not the ECG.
A Final Thought:

We must not, in trying to think about how we can make a big difference, ignore the small daily differences we can make which, overtime, add up to big differences that we often cannot foresee.

-Marian Wright Edelman