History of Cardiac Monitoring

• 1962 1st 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.”

Despite advances in technology, the need for human over site in the interpretation of the ECG is as important as it was 40 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

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
    - Prolonged QT interval associated VT
  - VF
- Bradyarrhythmias
- Premature complexes
  - Supraventricular
  - Ventricular
- Pacemaker Electrocardiography
  - 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
- AV node physiology
- AV 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
- Syncpe
- Hemodynamic effects of arrhythmias
  - Influence of rate
  - Influence of heart disease
  - Influence of A-V synchrony
  - Influence of UV 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
- 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

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
Electrical Conduction Pathway

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

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

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

Measuring Rate on Regular Rhythms

- Regular rhythms
  - Count number of large boxes between R waves and divide into 300:
    - 1 = 300
    - 2 = 150
    - 3 = 100
    - 4 = 50
    - 5 = 30
    - 6 = 3

\[ \frac{300 + 4}{6} = 75 \]
Calculating Rate

<table>
<thead>
<tr>
<th>Number of Beats</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>300</td>
</tr>
<tr>
<td>2</td>
<td>150</td>
</tr>
<tr>
<td>3</td>
<td>100</td>
</tr>
<tr>
<td>4</td>
<td>75</td>
</tr>
<tr>
<td>5</td>
<td>60</td>
</tr>
<tr>
<td>6</td>
<td>50</td>
</tr>
<tr>
<td>7</td>
<td>43</td>
</tr>
<tr>
<td>8</td>
<td>37</td>
</tr>
<tr>
<td>9</td>
<td>33</td>
</tr>
<tr>
<td>10</td>
<td>30</td>
</tr>
</tbody>
</table>

Utilizing the Bedside Monitor to Provide 12 Lead ECG Information

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

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
  - Lead 1 (MCL1)
  - Lead 3 (MCL6)

• 3 Lead Placement for Modified Chest

Importance of the Positive Electrode

• Consider the positive electrode the “the camera” (exploring electrode)
Comparing Bedside Monitoring to the 12 Lead ECG

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

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)

2004

AHA Scientific Statement

Practice Standards for Electrocardiographic Monitoring in Hospital Settings
An American Heart Association Scientific Statement From the Council on Cardiovascular Nursing, Clinical Cardiology, and Cardiovascular Disease in the Young
Endorsed by the International Society for Computerized Electrocardiology and the American Association of Critical-Care Nurses
Barbara J. Drew, RN, PhD; Charles Robert M. Cobif, MD; Marijeen Fork, RN, PhD; Elizabeth S. Kasztner, MD; Mitchell W. Knopfler, MD; Mitchell M. Laks, MD; Irene W. Macfarlane, DS; FRCP; Claire Somerby, RN, PhD; Steven Swyn, MD; George F. Van Hare, MD
Circulation. 2004;110:2721-2746
doi: 10.1161/01.CIR.0000145144.56673.59
http://circ.ahajournals.org/cgi/content/full/110/17/2721

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

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)

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:
  - VT with LBBB will have a wider QRS than VT with RBBB
  - Other causes of VT 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

Axis Practice

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 !!

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 with RBBB pattern or LVT
VT with LBBB pattern or RVT

VT with RBBB pattern or LVT
VT with LBBB pattern or RVT

VT with RBBB pattern or LVT
VT with LBBB pattern or RVT

VT with RBBB pattern or LVT
VT with LBBB pattern or RVT
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.

Practice EKGs

- 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 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.
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

Potential reversible causes
- Hypokalemia: K < 3.2 mEq/L (cause or result)
- Magnesium < 1.5 mEq/dL
- Ischemia
- Use of inotropic agents

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

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>
<tr>
<td>Keep electrodes in original package—start to dry 20 minutes after opening</td>
<td>Remove skin oils with abrasion (dry 4x4)</td>
<td></td>
</tr>
</tbody>
</table>

Body Position

- STs may fluctuate with body position changes
- May cause false alarms
- ST should be evaluated with patient in the supine position

Careful Prep

- 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

Lead Placement

- 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
## Tailor Alarms
- Alarms must be set to reflect each individual patient's baseline.
- 1mm above and below for precordial leads.
- 0.5 mm for limb leads.
- 2mm reasonable in the more stable patient (helps eliminate false alarms).

## Understand Goals of Monitoring
- 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.

## Analyze ECG Printout
- 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).

## ST Segment
- 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.

## 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.

## T Waves Too Big?????
- Clinical Application:
  Do not accept any T wave that is too big in any lead.
Answer: YES  
(same patient 2 hours later)

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

Hyper Acute T Wave with J Point Depression

Post Hyper Acute T Waves

Patterns of ST Elevation Injury
- Subtle ST Elevation  
  Forming Broad T Wave
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

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

2 Types of T Wave Inversion: NSTEMI or Ischemia

- 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

ECG Changes After STEMI

<table>
<thead>
<tr>
<th>Non Reperfused</th>
<th>Reperfused</th>
</tr>
</thead>
<tbody>
<tr>
<td>T wave enlargement</td>
<td>Earlier ST normalization and stabilization</td>
</tr>
<tr>
<td>ST elevation</td>
<td>T wave inversion may accelerate</td>
</tr>
<tr>
<td>Q wave formation or loss of R wave amplitude</td>
<td>— Terminal T wave inversion initially</td>
</tr>
<tr>
<td>ST stabilization</td>
<td>— T waves deepen symmetrically over time</td>
</tr>
<tr>
<td>T wave inversion (within 48 - 72 hours) before ST resolution</td>
<td>• Q wave development is less pronounced or even absent</td>
</tr>
<tr>
<td>ST resolution</td>
<td></td>
</tr>
</tbody>
</table>
Right Sided and Posterior Quick Look with V1 Lead on Bedside Monitor

- **Right Sided Lead**
  - Place electrode in V4R Position
  - 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”

---

**ST Evolution:**

Pseudo Normalization

- Reocclusion
- Terminal T-wave inversion
- Pseudo-normalization of T-wave
- Increased ST elevation

---

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.

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.

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).
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.

An Example to Improve Practice

- The patient received ST segment monitoring preoperatively.
- The patient returned to CCU postoperatively but did not receive postoperative 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

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

• 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: Na⁺ Channel Blockers
Class II: Calcium Channel Blockers
Class III: K⁺ Channel Blockers
Class IV: Calcium Channel Blockers

Measurements are using seconds.
### Expected QTc Intervals

<table>
<thead>
<tr>
<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>
</tr>
<tr>
<td>Borderline</td>
<td>.44 to .46 seconds</td>
<td>.43 to .45 seconds</td>
</tr>
<tr>
<td>Prolonged</td>
<td>&gt; .46 seconds</td>
<td>&gt; .45 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 know 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

**QT Interval Monitoring Recommendations**

- 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
- 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
  - Couplets 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 carvediol 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.

Polymorphic VT with normal QT:
- Seen frequently in ischemic conditions
- 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

Special Considerations: Polymorphic VT (normal QT)
- 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.
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 atrial flutter. The atrial flutter is not as obvious in the lead III rhythm strip.

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

Antiarrhythmics in Atrial Fibrillation

<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</td>
<td>Disopyramide, Procainamide, Quinidine</td>
<td>Rhythm Control</td>
<td>Torsade de pointes, HF</td>
</tr>
<tr>
<td>Class I B</td>
<td>Not used in atrial fibrillation</td>
<td>Rhythm Control</td>
<td>Torsade de pointes, HF</td>
</tr>
<tr>
<td>Class I C</td>
<td>Flecainide, Propafenone</td>
<td>Rhythm Control</td>
<td>Ventricular tachycardia, HF, Atrial Flutter</td>
</tr>
<tr>
<td>Class II</td>
<td>Beta Blockers</td>
<td>Rate Control</td>
<td>Torsade de pointes (rare) *Organ toxicity</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>Class IV</td>
<td>Calcium Channel Blockers</td>
<td>Rate Control</td>
<td></td>
</tr>
</tbody>
</table>

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

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

Monitoring Priorities

- 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 Halldol 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**

- Ischemia monitoring: use V3 and lead II as primary ischemia detection leads if area of ischemia or culprit vessel is unknown.
- If known 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 V2 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 V6 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 hardware 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.

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**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