Objectives

1. Identify ECG changes associated with myocardial ischemia, injury, and infarction.
2. Associate lead views with the correlating area of the heart.
3. Identify abnormal ECG findings associated with various pathologies.
4. Discuss the management and therapies for identified pathologies.
5. Review clinical practice guidelines for the acute myocardial infarction patient; including anti-platelet, beta blocker, and statin therapies.

Bipolar Limb Leads

Einthoven’s triangle

Lead I
- Measures electrical potential between right arm (-) and left arm (+).

Lead II
- Measures electrical potential between right arm (-) and left leg (+).

Lead III
- Measures electrical potential between left arm (-) and left leg (+).

RL
- Anywhere above the ankle and below the torso – right

RA
- Anywhere between the shoulder and elbow – right

LL
- Anywhere above the ankle and below the torso – left

LA
- Anywhere between the shoulder and the elbow – left
Unipolar Limb Leads

avR – right arm (+)
avL – left arm (+)
avF – left foot (+)

Right foot is a ground lead.

Precordial or Chest Leads

<table>
<thead>
<tr>
<th>Lead</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>V1</td>
<td>4th intercostal space to the right of the sternum</td>
</tr>
<tr>
<td>V2</td>
<td>4th intercostal space to the left of the sternum</td>
</tr>
<tr>
<td>V3</td>
<td>Midway between V2 and V4, in 5th intercostal space</td>
</tr>
<tr>
<td>V4</td>
<td>Midaxillary line, in 5th intercostal space</td>
</tr>
<tr>
<td>V5</td>
<td>Anterior axillary level, in 5th intercostal space</td>
</tr>
<tr>
<td>V6</td>
<td>Midaxillary line, in 5th intercostal space</td>
</tr>
</tbody>
</table>

Lead Placement Matters

• Up to 50% of cases have the V1 and V2 electrodes above the 4th intercostal location, which can mimic an anterior MI and cause T wave inversion.

• Up to 33% of cases have the precordial electrodes misplaced, which can alter the amplitude and lead to a misdiagnosis.
Right Sided ECG

May be useful in the diagnosis of a right ventricular infarct.
- 19-51% of inferior MIs

Interventions
- Fluids
- Positive inotrope infusion

Coronary Arteries of the Heart

Some people have an additional coronary artery off the left main called the ramus or intermediate artery.

Right Coronary Artery (RCA) | Circumflex (Cx) | Left Coronary Artery (LCA,LAD)
--- | --- | ---
SA node – 55% people | SA node – 45% people | Anterior 2/3rds of septum, bundle branches
AV node, bundle of His – 90% people | AV node – 10% people | Left ventricle – anterior, apex, posterior
Lateral and posterior left ventricle | Lateral and posterior left ventricle | Minor portion of right ventricle
Posterior left ventricle papillary muscles | Posterior left bundle branch | Left atrium
Posterior division left bundle branch
### Wall Leads Coronary Artery Reciprocal changes

<table>
<thead>
<tr>
<th>Wall</th>
<th>Leads</th>
<th>Coronary Artery</th>
<th>Reciprocal changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>V1, V2, V3, V4</td>
<td>LAD branch of LCA</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, aVF</td>
<td>RCA</td>
<td>II, aVF</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL, V5, V6</td>
<td>Circumflex branch of LCA</td>
<td>V1, V3</td>
</tr>
<tr>
<td>Posterior</td>
<td>V1, V2</td>
<td>(ST depression, tall R waves)</td>
<td>RCA, Circumflex</td>
</tr>
<tr>
<td>Apical</td>
<td>V3, V4, V5, V6</td>
<td>LAD, RCA</td>
<td></td>
</tr>
<tr>
<td>Anterolateral</td>
<td>I, aVL, V1, V2, V3, V4, V5, V6</td>
<td>LAD, Circumflex</td>
<td>II, III, aVF</td>
</tr>
<tr>
<td>Septal</td>
<td>V1, V2</td>
<td>LAD</td>
<td></td>
</tr>
</tbody>
</table>

### Steps to Interpreting the ECG

**Basic rhythm steps**
- Rhythm
- Rate
- P Waves
- PR Interval
- QRS
- QT Interval

**Additional 12 Lead steps**
- Wall of the heart
- 3 I’s of a MI
- Axis Deviation
- Bundle Branch Blocks
- What’s not normal
- Ugly vs. Dangerous
An electrocardiography pearl

ECG is nothing more than a voltmeter and a stopwatch.

- **Timing - horizontal**
  - Rate, PR interval, QRS interval, QT interval

- **Voltage - vertical**
  - Scars decrease the voltage.
  - Thick muscle increases the voltage.

### Information at the top of the 12 Lead

<table>
<thead>
<tr>
<th>Name</th>
<th>ID</th>
<th>Date and Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date of Birth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PR interval</td>
<td>230</td>
<td></td>
</tr>
<tr>
<td>QRS duration</td>
<td>130</td>
<td>ms</td>
</tr>
<tr>
<td>QT/QTC</td>
<td>394/413</td>
<td>ms</td>
</tr>
<tr>
<td>P-R-T axes</td>
<td>64</td>
<td>52</td>
</tr>
</tbody>
</table>

- ST elevation now present in Inferior leads
- ST now depressed in Anterolateral leads
- T wave inversion now evident in Anterolateral leads

### QT Interval Prolongation

Normal is considered less than half of the R-R (when the heart rate is ~70).

#### Conditions Predisposing for Long QT > Torsades
- Baseline long QTc
  - >450 ms, esp > 500 ms
- Female gender
- Electrolyte disorder
  - Especially low K+ and Mg++
- Bradycardia < 50
- Structural heart disease
- Significant renal or hepatic dysfunction

#### Common causes:
- Medications
- Electrolyte imbalance
  - Hypokalemia
    - ST flattening, depression, develop U waves
- Hypomagnesemia
- Hypocalcemia
  - Normal T wave after prolonged QT interval
- CNS catastrophes
  - Stroke, seizure, coma, intra-cerebral or brainstem bleeding
  - Can produce bizarre ST-T waves and some of the longest QT intervals
### Medications that prolong QT interval

<table>
<thead>
<tr>
<th>Generic name</th>
<th>Brand name</th>
</tr>
</thead>
<tbody>
<tr>
<td>sotalol</td>
<td>Betapace</td>
</tr>
<tr>
<td>quinidine</td>
<td>Quiniglute</td>
</tr>
<tr>
<td>amiodarone</td>
<td>Cardarone, Pacerone</td>
</tr>
<tr>
<td>procainamide</td>
<td>Procan, Pronestyl</td>
</tr>
<tr>
<td>disopyramide</td>
<td>Norpace</td>
</tr>
<tr>
<td>nicardipine</td>
<td>Cardene</td>
</tr>
<tr>
<td>ibutilide</td>
<td>Corvert</td>
</tr>
<tr>
<td>dofetilide</td>
<td>Tikosyn</td>
</tr>
<tr>
<td>trimethoprim-sulfa</td>
<td>Bactrim</td>
</tr>
<tr>
<td>clarithromycin</td>
<td>Biaxin</td>
</tr>
<tr>
<td>erythromycin</td>
<td>EES, Erythrocin</td>
</tr>
<tr>
<td>ciprofloxacin</td>
<td>Cipro</td>
</tr>
<tr>
<td>levofloxacin</td>
<td>Levaquin</td>
</tr>
<tr>
<td>azithromycin</td>
<td>Zithromax</td>
</tr>
<tr>
<td>ampicillin</td>
<td>Omnipen</td>
</tr>
<tr>
<td>fluconazole</td>
<td>Diflucan</td>
</tr>
<tr>
<td>ketoconazole</td>
<td>Nizoral</td>
</tr>
<tr>
<td>foscarnet</td>
<td>Foscavir</td>
</tr>
<tr>
<td>cocaine</td>
<td></td>
</tr>
<tr>
<td>methadone</td>
<td>Methadone, Dolophine</td>
</tr>
<tr>
<td>pseudoephedrine</td>
<td>Sudafed</td>
</tr>
<tr>
<td>tacrolimus</td>
<td>Prograf</td>
</tr>
</tbody>
</table>

### QTc by Bazett’s Formula

**Step 1**
- Find the square root of the R-R interval
- Measure the R-R interval (in squares x 0.04) then press the square root button on a calculator.

**Step 2**
- Measure the QT interval
- Change the QT interval from seconds to milliseconds (QT .44 secs = 440 ms)

**Step 3**
- Divide the QT interval in ms by the square root of the R-R interval to calculate the QTc.

**Example:**

**Step 1**
- R-R is 19 squares x 0.04 = 0.76
- Press the square root button
- The square root of 0.76 is 0.87

**Step 2**
- QT interval is .48 sec or 480 ms

**Step 3**
- \( \frac{480}{0.87} = 552 \) (551.7) ms

### 12 Lead Format

<table>
<thead>
<tr>
<th>I</th>
<th>AVR</th>
<th>V1</th>
<th>V4</th>
</tr>
</thead>
<tbody>
<tr>
<td>II</td>
<td>AVL</td>
<td>V2</td>
<td>V5</td>
</tr>
<tr>
<td>III</td>
<td>AVF</td>
<td>V3</td>
<td>V6</td>
</tr>
</tbody>
</table>
3 I's of a MI

Injury
• ST elevation on the affected side

Infarction
• Significant Q waves

Ischemia
• Inverted T waves

Injury ST Elevation

General guidelines:
> 1 mm in limb leads
> 2 mm in chest leads

Acute injury is occurring. Heart attack is happening now.

Causes of ST Elevation

Acute MI
Injury pattern
Left BBB
Angina with coronary artery spasm
Early repolarization
Left Ventricular hypertrophy
Hyperkalemia

Tako Tsubo cardiomyopathy
Intracranial bleeds or other pathologies like tumors
Acute cor pulmonale
Myocarditis
Pericarditis
Cholecystitis
Myocardial tumors
Acute pancreatitis
Hypothermia
**Infarction**  **Significant Q Waves**

May or not develop. If they do – Q waves develop over 4 to 24 hours and remain for life.

![Significant Q-wave](image)

Significant Q waves are 25–33% of the R wave. Q > 0.038 seconds (almost one small box, see white)

---

**Q-Waves**

- **Physiologic / Insignificant**
- **Pathologic / Significant**

---

**Ischemia**  **Inverted T waves**

![Inverted T-wave](image)

Supply and Demand problem.
Determining Axis

Direction of the mean electrical vector.
Average direction of current flow.

Axis

<table>
<thead>
<tr>
<th>Axis</th>
<th>Lead I</th>
<th>Lead II</th>
<th>Lead III</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal 0-90</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physiologic Left Axis 0-40</td>
<td></td>
<td></td>
<td></td>
<td>aVF negative</td>
</tr>
<tr>
<td>Pathological Left Axis -40 to -90</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right Axis 90-180</td>
<td></td>
<td></td>
<td></td>
<td>aVF positive</td>
</tr>
<tr>
<td>Extreme Right Axis No Man’s Land</td>
<td></td>
<td></td>
<td></td>
<td>aVF negative</td>
</tr>
</tbody>
</table>

- ±0 to -30 (-40)
- +90 to +120

- Leads I, II, & III all positive
- Physiologically normal

O to +90
0 to −90

Physiological
- Too short, Too tall
- Obese
- Older
- Pregnant

Pathological
- Left Ventricular Hypertrophy
- Hyperkalemia
- Qs in Inferior MI
- Anterior hemiblock
- Left BBB
- WPW w/ Right sided accessory pathway
- Emphysema

Lead I positive
Lead II positive or negative
Lead III negative

+90 to +180

Physiological
- Normal in children and thin adults

Pathological
- Right Ventricular Hypertrophy
- Anterior-Lateral MI
- Posterior hemiblock
- Right BBB
- COPD w/ Pulmonary HTN
- Pulmonary Emboli
- WPW w/ Left sided accessory pathway
- ASD, VSD

Lead I negative
Lead II positive or negative
Lead III negative

+180 to -90

No Man’s Land

Ventricular Tachycardia
Ventricular Pacing
Hyperkalemia

Leads I, II & III negative
Signs and Symptoms of Acute Coronary Syndrome

Classic or usual
- Chest discomfort described as pain, pressure, ache, squeezing, burning or fullness
- Discomfort or pain in one or both arms
- Shortness of breath with or before chest discomfort
- Diaphoresis - sweating
- Anxiety

Atypical or not usual
- Back, abdominal, neck or jaw pain
- Weakness or fatigue
- Indigestion
- Nausea or vomiting
- Dizziness or lightheadedness

Prodromal symptoms or pre-heart attack symptoms can occur one to six weeks before include:
- Chest pain
- Pain in one shoulder blade or upper back
- Indigestion
- Unusual fatigue
- Anxiety
- Sleep disturbances
- Shortness of breath, especially if no previous awareness of heart disease

Acute Coronary Syndrome

- ST Elevated Myocardial Infarction - STEMI
  - ST segment is elevated above the isoelectric baseline
  - Classic presentation with elevated cardiac biomarkers
  - New LBBB – not diagnostic, 2013 update
- Non ST Elevated Myocardial Infarction - NSTEMI
  - ST and T-wave changes with elevated cardiac biomarkers
    - Depressed ST, inverted T wave
  - Classical or atypical presentation
- Angina, Unstable angina

Types of MI

Type 1
- Spontaneous MI related to ischemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection
- Non ST Elevation MI or ST Elevation MI

Type 2
- MI secondary to ischemia due to either increased oxygen demand or decreased supply
  - Respiratory distress, renal failure, sepis, shock
  - Not ischemia from thrombosis of coronary artery
  - **Document elevated troponin

Type 3
- Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of MI
  - Acute or subacute (presumably new ST elevation or new LBBB)
  - Evidence of fresh thrombus in the coronary artery by angiography

Type 4
- MI associated with coronary angioplasty or stent thrombosis

Type 5
- MI associated with coronary artery bypass grafting (CABG) occlusion
Pathological Types

Transmural AMI
- Infarct extends through the whole thickness of the heart muscle, usually resulting in complete occlusion of the area’s blood supply.
  - Associated with atherosclerosis involving a major coronary artery.
  - ST elevation, and Q-waves

Subendocardial AMI
- Involves a small area in the subendocardial wall of the left ventricle, ventricular septum, or papillary muscles.
  - Susceptible to ischemia.
  - ST depression, T-wave changes

AMI Clinical Practice Guidelines (CPGs)

During hospitalization
- 12 Lead ECG within 10 minutes
- Reperfusion strategies
- Aspirin within 24 hours before* or after arrival
- Smoking (tobacco) cessation advice/counseling

At Discharge
- Aspirin
- Beta-Blocker
- Statin
- ACE-I or ARB therapy for EF (ejection fraction) ≤ 40% - HFrEF, reduced left ventricular systolic dysfunction

STEMI Reperfusion Strategy

Door-to-needle within 30 minutes
- Thrombolytic (fibrinolysis) therapy
  - TNKase (tenecteplase)
  - Activase (t-PA, alteplase)
  - Retavase (r-PA, reteplase)
  - Streptokinase (Streptase)

Door-to-Balloon (D2B) within 90 minutes
- Percutaneous Coronary Intervention (PCI)
  - PTCA – Percutaneous transluminal coronary angioplasty
  - Atherectomy
  - Coronary artery stents
Percutaneous Coronary Intervention - PCI

Left heart catheterization with

- Angioplasty
- Atherectomy
- Coronary stenting
  - Bare metal (BMS)
  - Drug eluting (DES)

Antiplatelets

Aspirin
- 162 to 325 mg initially
- 81 (75-100 mg) daily
## Duration of Dual Antiplatelet Therapy

New 2016 guidelines

- [http://circ.ahajournals.org/content/134/10/e123](http://circ.ahajournals.org/content/134/10/e123)

- Generally
  - ASA indefinitely
  - Dual platelet therapy for 12 months
    - Less if high risk of bleeding

## Antiplatelet Options

<table>
<thead>
<tr>
<th>Name</th>
<th>Classification</th>
<th>Dosing - Std Concentration</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Aspirin            | Anti-platelet, attaches to TXA2
                    | 162-325 mg loading, then 81 (75-100) mg PO per day | Do not take with PPI, especially Prilosec; Caution in patients < 75 years old, < 60 kg. Box warning not to give if history of stroke or TIA. |
| Plavix (clopidogrel)| Anti-platelet, attaches to ADP P2Y12 site | 300-600 mg PO loading, then 75 mg daily | Do not take with PPI, especially Prilosec |
| Effient (prasugrel)| Anti-platelet, attaches to ADP P2Y12 site | 60 mg PO loading, then 10 mg daily | Caution in patients > 75 years old, < 60 kg. Box warning not to give if history of stroke or TIA. |
| Brilinta (ticagrelor) | Anti-platelet, attaches to ADP P2Y12 site | 180 mg PO loading, then 90 mg PO loading, then 75 mg daily | Aspirin to be limited to 75-100 mg/day |
| Integrin (eptifibatide)| Anti-platelet, attaches to GP IIb IIIa | 2 mcg/kg/min infusion 12 to 24 hours after PCI | Decrease to 1 mcg/kg/min for renal impairment |
| ReoPro (abciximab)  | Anti-platelet, attaches to GP IIb IIIa | 0.25 mg/kg bolus, then 10 mcg/min infusion x 18-24 hours or stop 1 hour after PCI | No renal dosing. 4 hour effect with half-life of 30 minutes |
Beta Blockers

- Reduce catecholamine levels
- Decrease myocardial ischemia and limit infarct size
- Reduce myocardial workload and oxygen demand
- Reduce heart rate and blood pressure
- Reduce supraventricular and malignant ventricular arrhythmias

Common Beta Blockers

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Daily Dose</th>
<th>Maximum Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carvedilol (Coreg)</td>
<td>3.125 mg BID</td>
<td>50 mg BID</td>
</tr>
<tr>
<td>Carvedilol extended release (Coreg CR)</td>
<td>10 mg daily</td>
<td>80 mg daily</td>
</tr>
<tr>
<td>Metoprolol succinate extended release (Toprol XL, generic)</td>
<td>12.5-25 mg daily</td>
<td>200 mg daily</td>
</tr>
<tr>
<td>Bisoprolol (Zebeta)</td>
<td>1.25 mg daily</td>
<td>10 mg daily</td>
</tr>
<tr>
<td>Atenolol (Tenormin)</td>
<td>50 mg daily</td>
<td>100 mg (200) daily</td>
</tr>
<tr>
<td>Metoprolol tartrate (Lopressor)</td>
<td>50 mg BID</td>
<td>450 mg divided in 2-3 doses</td>
</tr>
</tbody>
</table>

No Beta Blockers with Cocaine

If cocaine induced MI, no beta blocker
- BB may exacerbate the vasospasm induced by cocaine due to “unopposed” alpha effect
- Inhibits vasodilation

Beta Blockers

- Metoprolol – Lopressor, Toprol XL
- Carvedilol – Coreg
- Bisoprolol - Zebeta
- Atenolol – Tenormin
- Sotalol – Betapace
- Betaxolol – Kerlone
- Propranolol – Inderol
- Esmolol – Brevibloc (IV)
- Labetalol – Normodyne (IV)
HMG-CoA Reductase

Cholesterol is synthesized in the smooth endoplasmic reticulum by a series of chemical reactions. The first way to block cholesterol synthesis is to interrupt the conversion of HMG CoA to mevalonate.

HMG-CoA Reductase Inhibitors or Statins

2013 guideline update

- Lifestyle modification
  - Diet, exercise, lose weight
- Assess ASCVD risk
- Four Benefit Groups
  - Individuals with clinical ASCVD
  - Individuals with primary elevations of LDL-C >190 mg/dL
  - Individuals age 40-75 with diabetes and LDL-C of 70-189 mg/dL, without clinical ASCVD
  - Individuals without clinical ASCVD or diabetes who are age 40-75 with LDL-C of 70-189 mg/dL, and estimated 10 year ASCVD risk of ≥7.5%

Atorvastatin – Lipitor
Rosuvastatin – Crestor
Simvastatin – Zocor
Pravastatin – Pravachol
Lovastatin – Mevacor

Guidelines level to high or moderate intensity dosing.

Adverse effects – muscle aching, increase in liver enzymes

http://content.onlinejacc.org/article.aspx?articleid=1879710

High-Intensity Daily dose lowers LDL-C, on average, by approximately ≥50%
- Atorvastatin (40) – 80 mg
- Rosuvastatin 20 – 40 mg

Moderate-Intensity Daily dose lowers LDL-C, on average, by approximately 30% to <50%
- Atorvastatin 10 (20) mg
- Rosuvastatin (5) 10 mg
- Simvastatin 20-40 mg
- Pravastatin 40 (80) mg
- Lovastatin 40 mg

If going to check LDL
- Do within 24 hours
- Otherwise will be low and not reflective of usual level
PCSK9 Inhibitors

By blocking PCSK9’s ability to work, more receptors are available to get rid of LDL cholesterol from the blood and, as a result, lower LDL cholesterol levels.

Alirocumab (Praluent)
- 75 mg or 150 mg SQ every 2 weeks

Evolocumab (Repatha)
- 140 mg every 2 weeks or 420 mg once monthly
- 420 mg dose - Single use body infusor over 9 minutes or 3 injections within 30 minutes

These are additions to statin therapy. The benefit is to keep statin doses low.

Renin-Angiotensin-Aldosterone System

Angiotensin II is a very powerful vasoconstrictor.

ACE-I & ARBs

**ACE-I**
- Lisinopril – Prinivil, Zestril
- Captopril – Capoten
- Ramipril – Altace
- Enalapril – Vasotec
- Fosinopril – Monopril

Adverse effect – cough, angioedema, hyperkalemia
Watch renal function.

**ARB**
- Losartan – Cozaar
- Valsartan – Diovan
- Candesartan – Atacand

Tend not to have as many adverse effects. Cough not often seen.
ACE-I and ARBs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Daily Dose</th>
<th>Maximum Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>6.25 mg TID</td>
<td>50 mg TID</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 mg BID</td>
<td>10-20 BID</td>
</tr>
<tr>
<td>Fosinopril</td>
<td>5-10 mg daily</td>
<td>40 mg daily</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>2.5-5 mg daily</td>
<td>20-40 mg daily</td>
</tr>
<tr>
<td>Ramipril</td>
<td>1.25-2.5 mg daily</td>
<td>10 mg daily</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Daily Dose</th>
<th>Maximum Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Losartan</td>
<td>25-50 mg daily</td>
<td>50-150 mg daily</td>
</tr>
<tr>
<td>Valsartan</td>
<td>20-40 mg BID</td>
<td>160 mg BID</td>
</tr>
<tr>
<td>Candesartan</td>
<td>4-8 mg daily</td>
<td>32 mg daily</td>
</tr>
</tbody>
</table>

September 2017 AHA / ACA New Performance Measures for MI

1. Immediate angiography for resuscitated out-of-hospital cardiac arrest in STEMI patients
2. Noninvasive stress testing before discharge in conservatively treated patients
3. Early cardiac troponin measurement, within 6 hours of arrival
   - Baseline, 2 hours, 6 hours
4. Participation in a regional or national acute-MI registry
   - TJC Certifications need GWTG (Get With The Guidelines) registry

September 2017 AHA / ACA New Quality Measures for MI

1. Risk-score stratification for NSTEMI patients
2. Early invasive strategy, within 24 hours, in high-risk NSTEMI patients
3. Therapeutic hypothermia for comatose STEMI patients with out-of-hospital cardiac arrest
4. Aldosterone antagonist at discharge
5. Inappropriate in-hospital use of NSAIDS
6. Inappropriate prescription of prasugrel at discharge in patients with a history of prior stroke or TIA
7. Inappropriate prescription of high-dose aspirin with ticagrelor at discharge
Brugada Criteria for Ventricular Tach

<table>
<thead>
<tr>
<th>VT vs. SVT</th>
<th>Absence of an RS complex in all preordial leads</th>
<th>The longest R is S interval &gt;180 ms in any preordial lead</th>
<th>AV dissociation</th>
<th>Classical criteria for VT present both in lead V1-2 and V6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>VT diagnosed</td>
</tr>
<tr>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>SVT diagnosed</td>
</tr>
</tbody>
</table>

1. Is there extreme right axis?
2. Is there an RS complex?
3. Is the QRS wide?
4. Is there AV Dissociation?
5. Is there concordance in V1-V6?

Right versus Left BBB

Lopsided activation – one ventricle then the other.
Not all “rabbit ears” are BBBs.

RBBB

Look at V1 lead
- QRS is > 0.12 seconds
  - An incomplete BBB measures < 0.12 sec.

Right BBB is blocked:
- Electrical impulse is going Left > Right
- Right ventricle conducts later than left ventricle

Physiological
- Athletes
  - Increased muscle mass

Pathological
- CAD
  - More common with anterior MI
- Pulmonary HTN
- Inflammatory disease
- Lesions of the septum
- New RBBB after bypass surgery is a + periop MI
**LBBB**

Look at V1 lead
- QRS is > 0.12 seconds
  - An incomplete BBB measures < 0.12 sec.
Left BBB is blocked
- Electrical impulse is going Right > Left
- Left ventricle conducts later than right ventricle

Left bundle of His has 3 fascicles
- Anterior (superior)
- Posterior (inferior)
- Midseptum

**Pathological**
- CAD
  - More common with inferior MI
- Hypertension
- Dilated cardiomyopathy
- Calcified aortic valve, stenosis
- Aortic root dilation and aortic regurgitation
- Degenerative heart disease

---

**Left Atrial Hypertrophy**

**Causes**
- Hypertension
- Valvular Heart Disease
  - Mitral stenosis
  - Mitral regurgitation
  - Aortic stenosis
- Heart Failure
- Ventricular Septal Defect
- Cardiac myoma

**What will see**
- Broad or notched P-waves
- Prolonged P wave
  - V1 broad trough
  - II, II, & V4-V6 notched

---

**Right Atrial Hypertrophy**

**Causes**
- Lung disease
  - COPD
- Pulmonary Embolus
- Pulmonary Hypertension
- Right ventricular failure
- Tricuspid regurgitation or stenosis
- Atrial Septal Defects

**What will see**
- Tall, peaked P-waves
  - II, III, aVF
  - > 2.5 mm tall in the inferior leads
**Right Ventricular Hypertrophy**

**Causes**
- Increased right ventricular mass
  - Pulmonary stenosis or regurgitation
  - Primary pulmonary hypertension
  - Pulmonary embolus
  - Diastolic overload
  - Atrial septal defect
  - Congenital heart disease

**What will see**
- Right axis deviation
  - Tall “R” waves in right precordial leads
  - V1 most sensitive
  - Deep “S” waves in left precordial leads
  - V6

---

**Left Ventricular Hypertrophy**

**Causes**
- Increased LV muscle mass
  - Hypertension
  - Cardiomegaly
  - Cardiomyopathy
  - Aortic stenosis and regurgitation
  - Mitral regurgitation

**What will see**
- Left axis deviation
  - Measure
  - V1 or V2 Deepest “S” wave
  - PLUS
  - V5 or V6 Tallest “R” wave
  - #mm add up > 35 mm

---

**Aortic Stenosis**

**Left Ventricular strain pattern**
- Left Ventricular Hypertrophy
- Left atrial enlargement
- Left axis deviation
- Conduction defects
  - LBBB, RBBB, AV blocks

**Testing sequence**
- History and physical, Lab
- Chest x-ray and 12 Lead ECG
- Echocardiography and Doppler
- Cardiac catheterization*
  - 50% with critical AS have CAD

**ECG not diagnostic, may see**
- ST depression and T-wave inversion in anterior and lateral leads
- LV hypertrophy
  - Absence does not preclude AS
- Sub-endocardial ischemia
Hypertrophic Cardiomyopathy

Previously known as
- Hypertrophic obstructive cardiomyopathy – HCOM
- Idiopathic hypertrophic subaortic stenosis – IHSS

Number one cause of sudden cardiac death in young athletes (1-2%).
Inheritance is primarily autosomal dominant.

ECG changes
- Left ventricular hypertrophy pattern
  - Tall R waves
  - Large precordial voltages
  - Left atrial enlargement

12 Lead ECG as a Screening Test

United States does not require
  - Italy and Israel do

Issues
  - Placement of leads
  - 30% false positives

Ethical issues
  - Consent for screening
  - Who receives results
  - Who makes the determination of risk with participation in activities

Read more, including recommended 14 element screening at
http://circ.ahajournals.org/content/130/15/1303
Treatment and Management

Medical
• No highly strenuous activity
• Control blood pressure
  o Beta blockers
  o Calcium channel blockers
• Amiodarone
• Norpace (disopyramide)
• Cautious with diuretics
• Avoid inotropes, nitrates, sympathomimetic amines

Surgical
• Surgical septal myectomy
• Alcohol septal ablation
• Heart transplant

Tall R waves in V1-2

Not normal
• Posterior MI
• Right bundle branch block
• Right ventricular hypertrophy
• Hypertrophic cardiomyopathy

Wolff-Parkinson White

Sinus impulses bypass the AV node via an accessory pathway (AP) conduction.
• Uncommon - ~2 per 1,000 in the general population
• Can be right-sided, left-sided, anterior, or posterior – and sometimes more than a single AP.
• A very fast atrial fibrillation (250-300) – think WPW.
Accessory Pathway

Three key signs:
- Delta wave which may be positive or negative
- QRS widening
- Short PR interval

Treatment and Management

Acutely
- Adenosine
- Consult cardiology

Long Term
- Catheter ablation
- Flecaïnide (Tambocor)
- Sotalol (Betapace)
Pulmonary Embolus

Look at the sum of all in context with the clinical history.
• ECG is not diagnostic.
• Can strongly suggest before the V/Q or CT scan.

Old – S_I-Q_IIi-T_III “classic” finding is neither sensitive nor specific.

Suspect PE?

New onset dyspnea, pleuretic
Typically tachycardic
• Most common, seen in 44% of cases
  ➢ RBBB
    • Complete or incomplete
  ➢ Right Ventricular strain pattern
    • T wave inversion in V1, V2, V3, also V4
    • T Wave Inversion II, III, aVF
  ➢ Right axis deviation
    • Extreme right axis may occur between 0 and -90, giving appearance of left axis (pseudo left axis)
  ➢ Dominant R wave in V1
    • Manifestation of acute right ventricular dilation
  ➢ RA enlargement
    • Peaked P waves in lead II
  ➢ Wide S in Lead I, subtle S in V6
  ➢ ST elevation in aVR

Causes of PVCs

Bradycardia
• PVCs trying to help out

Hypoxia
• Evil of all evil

Electrolyte imbalance
• Potassium, magnesium, calcium

Medications
• Infusions we’ve started
• Medications not restarted

Stimulants
• Legal or illegal
References


References

