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 the 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>Location</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</td>
</tr>
<tr>
<td>V4</td>
<td>5th intercostal space at the midclavicular line</td>
</tr>
<tr>
<td>V5</td>
<td>Anterior axillary line at the same level as V4</td>
</tr>
<tr>
<td>V6</td>
<td>Midaxillary line at the same level as V4 and V5</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

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

### Coronary Arteries of the Heart

<table>
<thead>
<tr>
<th>Right Coronary Artery (RCA)</th>
<th>Circumflex (Cx)</th>
<th>Left Coronary Artery (LCA, LAD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SA node – 55% people</td>
<td>SA node – 45% people</td>
<td>Anterior 2/3rds of septum, bundle branches</td>
</tr>
<tr>
<td>AV node, bundle of His – 90% people</td>
<td>AV node – 10% people</td>
<td>Left ventricle – anterior, apex, posterior</td>
</tr>
<tr>
<td>Right atrium</td>
<td>Lateral and posterior left ventricle</td>
<td>Minor portion of right ventricle</td>
</tr>
<tr>
<td>Inferior left ventricle</td>
<td>Posterior left ventricle</td>
<td></td>
</tr>
<tr>
<td>Wall</td>
<td>Leads</td>
<td>Coronary Artery</td>
</tr>
<tr>
<td>------------</td>
<td>-----------</td>
<td>-----------------------</td>
</tr>
<tr>
<td>Anterior</td>
<td>V1, V2, V3, V4</td>
<td>LAD branch of LCA</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, aVF</td>
<td>RCA</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL, V5, V6</td>
<td>Circumflex branch of LCA</td>
</tr>
<tr>
<td>Posterior</td>
<td>V1, V2 (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>
</tr>
<tr>
<td>Anterolateral</td>
<td>I, aVL, V1, V2, V3, V4, V5, V6</td>
<td>LAD, Circumflex</td>
</tr>
<tr>
<td>Septal</td>
<td>V1, V2</td>
<td>LAD</td>
</tr>
</tbody>
</table>

### Steps to Interpreting the ECG

<table>
<thead>
<tr>
<th>Basic rhythm steps</th>
<th>Additional 12 Lead steps</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Wall of the heart</td>
</tr>
<tr>
<td>Rate</td>
<td>3 I's of a MI</td>
</tr>
<tr>
<td>P Waves</td>
<td>Axis Deviation</td>
</tr>
<tr>
<td>PR Interval</td>
<td>Bundle Branch Blocks</td>
</tr>
<tr>
<td>QRS</td>
<td>What's not normal</td>
</tr>
<tr>
<td>QT Interval</td>
<td>Ugly vs. Dangerous</td>
</tr>
</tbody>
</table>
Review of electrocardiography

ECG is nothing more than a voltmeter and a stopwatch.

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

Information at the top of the 12 Lead

<table>
<thead>
<tr>
<th>Last name, First name</th>
<th>ID #</th>
<th>Date and Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date of Birth</td>
<td>Gender</td>
<td>Race</td>
</tr>
<tr>
<td>66</td>
<td>66</td>
<td>200 ms</td>
</tr>
</tbody>
</table>

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 QT
  - >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
  - Like hypokalemia
  - 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>*methadone</td>
<td>Methadone, Dolophine</td>
</tr>
<tr>
<td>albuterol</td>
<td>Ventolin, Proventil</td>
</tr>
<tr>
<td>levalbuterol</td>
<td>Xopenex</td>
</tr>
<tr>
<td>salmeterol</td>
<td>Serevent</td>
</tr>
<tr>
<td>*haloperidol</td>
<td>Haldol</td>
</tr>
<tr>
<td>mesoridazine</td>
<td>Serentil</td>
</tr>
<tr>
<td>*risperidone</td>
<td>Risperdal</td>
</tr>
<tr>
<td>*chlorpromazine</td>
<td>Thorazine</td>
</tr>
<tr>
<td>*chlorpromazine</td>
<td>Thorazine</td>
</tr>
<tr>
<td>tacrolimus</td>
<td>Prograf</td>
</tr>
<tr>
<td>pseudoephedrine</td>
<td>Sudafed</td>
</tr>
<tr>
<td>phenylpropanolamine</td>
<td>Dexatrim, Acutrim</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 seconds 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
- 480 ÷ 0.87 = QTc of 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
- Acute corpulmonale
- 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 waves are 25–33% of the R wave.  
Q > 0.038 seconds

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

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</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>aVF positive</td>
</tr>
<tr>
<td>Physiologic Left Axis</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>aVF negative</td>
</tr>
<tr>
<td>Pathological Left Axis</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>Anterior Hemiblock</td>
</tr>
<tr>
<td>Right Axis 90-180</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>aVF positive</td>
</tr>
<tr>
<td>Extreme Right Axis No Man's Land</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>![Diagram]</td>
<td>aVF negative Ventricular in origin</td>
</tr>
</tbody>
</table>

### Normal

Some physiological normal found:
- 0 to -30 (-40)
- +90 to +120

### Left Axis

- 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 access pathway
  - Emphysema

**O to +90**

**0 to –90**
Physiological
- Normal in children and thin adults

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

---

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 equivalent since 2013 updated position*

- **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.
  - Coronary artery spasm, coronary embolism, anemia, arrhythmias, hypertension, or hypotension
  - Respiratory distress, renal failure, sepsis

Type 3
- Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of MI
  - Accompanied by 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

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

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.
- Subclassified into anterior, posterior, inferior, lateral, or septal.
- 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
- 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 left ventricular systolic dysfunction, EF (ejection fraction) < 40%

STEMI Reperfusion Strategy

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

Door-to-Balloon (D2B) within 90 minutes
- Angioplasty
  - PTCA – Percutaneous Transluminal Coronary Angioplasty
- Coronary artery stents
- Atherectomy Percutaneous Coronary Intervention

Percutaneous Coronary Intervention - PCI

- Atherectomy
- Angioplasty
- Coronary stenting
  - Bare metal (BMS)
  - Drug eluting (DES)
  - Bioresorbable (BVS)
Antiplatelet Therapy

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>
<tbody>
<tr>
<td>Aspirin</td>
<td>Anti-platelet, attaches to TXA2 site</td>
<td>162-325 mg loading, then 81 (75-100) mg PO per day</td>
<td>ASA to be limited to 75-100 mg/day. Do not take with PPI, especially Prilosec.</td>
</tr>
<tr>
<td>Plavix (clopidogrel)</td>
<td>Anti-platelet, attaches to ADP P2Y12 site</td>
<td>300-600 mg PO loading, then 75 mg daily</td>
<td>Do not take with PPI, especially Prilosec.</td>
</tr>
<tr>
<td>Effient (prasugrel)</td>
<td>Anti-platelet, attaches to ADP P2Y12 site</td>
<td>60 mg PO loading, then 10 mg daily</td>
<td>Caution in patients &gt; 75 years old, &lt; 60 kg. Box warning not to give if history of stroke or TIA.</td>
</tr>
<tr>
<td>Brilinta (ticagrelor)</td>
<td>Anti-platelet, attaches to GP IIb IIIa</td>
<td>180 mg PO loading, then 90 mg PO twice a day</td>
<td>ASA to be limited to 75-100 mg/day.</td>
</tr>
<tr>
<td>Integrilin (eptifibatide)</td>
<td>Anti-platelet, attaches to GP IIb IIIa</td>
<td>2 mcg/kg/min infusion 12 to 24 hours after PCI, decrease to 1 mcg/kg/min for renal impairment</td>
<td>Reversible in 2.5-4 hours. Don’t get patients OOB until 2-2.5 hours after infusion is shut off.</td>
</tr>
<tr>
<td>ReoPro (abciximab)</td>
<td>Anti-platelet, attaches to GP IIb IIIa</td>
<td>0.25 mg/kg bolus, then 10 mcg/min infusion x 18-24 hours or stop 1 hour after PCI</td>
<td>No renal dosing. 4 hour effect with half-life of 20 minutes</td>
</tr>
</tbody>
</table>
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

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

Common Beta Blockers

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>
</tbody>
</table>

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%

**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

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**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

**Aliruicumab (Praluent)**

- 75 mg or 150 mg SQ every 2 weeks

**Evolucomab (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.
Renin-Angiotensin-Aldosterone System

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 really 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 daily</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>
Patient Safety Indicators

AMI Quality Measures

<table>
<thead>
<tr>
<th>Quality Measure</th>
<th>Definition</th>
<th>Weight in Composite</th>
<th>Collection</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-Day Mortality</td>
<td>30-day, all cause, risk-standardized mortality rate following a hospitalization for AMI</td>
<td>50%</td>
<td>Claim-based per CIR (NQF #0013)</td>
</tr>
<tr>
<td>AMI Excess Days</td>
<td>Excess days in acute care, including emergency department, observation, and inpatient readmission days following a hospitalization for AMI</td>
<td>20%</td>
<td>Claim-based per CIR</td>
</tr>
<tr>
<td>HCAHPS Survey</td>
<td>Patient experience composite measure (risk in that rating measure not specific to AMI). Patient experience is evidence of a patient's level of comfort, discharge satisfaction, information, discharge instructions, and quietness.</td>
<td>20%</td>
<td>Patient Survey (NQF #190)</td>
</tr>
<tr>
<td>Hybrid AMI Mortality</td>
<td>30-day, risk-standardized AMI mortality rate, using a combination of claim data and vital data submitted by hospitals</td>
<td>10%</td>
<td>Voluntary submission (NQF #447D)</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
4. Participation in a regional or national acute-MI 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

**VT vs. SVT**

<table>
<thead>
<tr>
<th>Presence of an RS complex in all precordial leads</th>
<th>The longest R to S interval ≥100 ms in any precordial lead</th>
<th>AV dissociation</th>
<th>Classical criteria for VT present in both V1-2 and V6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>VT diagnosed</td>
<td>SVT diagnosed</td>
<td></td>
<td></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**

**Right bundle branch block characteristics**

- V1: rSR'
- V6: qRs

**Left bundle branch block characteristics**

- V1: rS
- V6: R

**RBBB - Right Bundle Branch Block**

QRS 162 ms
**RBBB**

<table>
<thead>
<tr>
<th>Look at V1 lead</th>
</tr>
</thead>
<tbody>
<tr>
<td>• QRS is ≥ 0.12 seconds</td>
</tr>
<tr>
<td>o An incomplete BBB measures &lt; 0.12 sec.</td>
</tr>
</tbody>
</table>

**Physiological**

- Athletes
  - Increased muscle mass

**Pathological**

- CAD
- Pulmonary HTN
- Inflammatory disease
- Lesions of the septum
- New RBBB after bypass surgery is a + periop MI

**Right BBB is blocked**

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

**LBBB – Left Bundle Branch Block**

| QRS 144 ms |

**Physiological**

- Athletes
  - Increased muscle mass

**Pathological**

- CAD
- Hypertension
- Dilated cardiomyopathy
- Calcified aortic valve, stenosis
- Aortic root dilation and aortic regurgitation
- Degenerative heart disease

**LBBB**

<table>
<thead>
<tr>
<th>Look at V1 lead</th>
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<tr>
<td>• QRS is ≥ 0.12 seconds</td>
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</table>

**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
Left Atrial Hypertrophy

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

Causes

Broad or notched P–waves

Prolonged P wave
  - V1 broad trough
  - I, II, & V4-V6 notched

What will see

Right Atrial Hypertrophy

www.pedcard.rush.edu
Right Atrial Hypertrophy

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

Tall, peaked P-waves
- II, III, aVF
- ≥ 2.5 mm tall in the inferior leads

Right Ventricular Hypertrophy

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

Right axis deviation
- Tall “R” waves in right precardial leads
  - V1 most sensitive
- Deep “S” waves in left precardial 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

Previous 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
- Deep, narrow “dagger-like” Q waves in lateral and inferior leads
- Giant T-wave inversion in apical HCM
- Left atrial enlargement
- Atrial fibrillation and SVTs are common

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
  - Beta blockers
  - Calcium channel blockers
- Amiodarone
- Norpace (disopyramide)
- Cautious with diuretics
- Avoid inotropes, nitrates, sympathomimetic amines

Surgical
- Surgical septal myectomy
- Alcohol septal ablation
- Heart transplant

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
WPW

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
Flecainide (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_T - 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

References


References


Disclaimer: The overview is not all inclusive and I recommend reviewing the ACC/AHA guidelines.