Ischemic Heart Disease

- Is most commonly due to atherosclerosis in coronary arteries
- Ischemia occurs when blood supply to tissue is deficient
  - Causes increased lactic acid from anaerobic metabolism
- Often accompanied by angina pectoris (chest pain)

CHEST PAIN

ST Segment

- Normal ST Segment is flat (isoelectric)
  - Same level with subsequent PR segment
- Elevation or depression of ST segment by 1 mm or more, measured at J point is ABNORMAL
- “J” (Junction) point is the point between QRS and ST segment
What’s a J point and where is it?

- J point – point to mark end of QRS and beginning of ST segment
  - Evaluate ST elevation 0.04 seconds after J point
  - Based on relationship to the baseline
  - Used in assessing ST elevation

Ischemic Heart Disease

- Detectable by changes in S-T segment of ECG
- Myocardial infarction (MI) is a heart attack
  - Diagnosed by high levels of creatine phosphate (CPK) & lactate dehydrogenase (LDH)

Measuring for ST Elevation

- Find the J point
- Is the ST segment >1mm above the isoelectric line in 2 or more contiguous leads?

Characteristic changes in AMI

- ST segment elevation over area of damage
- ST depression in leads opposite infarction
- Pathological Q waves
- Reduced R waves
- Inverted T waves

Myocardial Insult

- Ischemia
  - lack of oxygenation
  - ST depression or T wave inversion
  - permanent damage avoidable
- Injury
  - prolonged ischemia
  - ST elevation
  - permanent damage avoidable
- Infarct
  - death of myocardial tissue; damage permanent; may have Q wave
**Variable Shapes Of ST Segment Elevations in AMI**

ST elevation
- Occurs in the early stages
- Occurs in the leads facing the infarction
- Slight ST elevation may be normal in V1 or V2

Deep Q wave
- Only diagnostic change of myocardial infarction
- At least 0.04 seconds in duration
- Depth of more than 25% of ensuing R wave

T wave changes
- Late change
- Occurs as ST elevation is returning to normal
- Apparent in many leads

Bundle branch block
- Anterior wall MI
- Left bundle branch block
Sequence of changes in evolving AMI

A - pre-infarct (normal)
B - Tall T wave (first few minutes of infarct)
C - Tall T wave and ST elevation (injury)
D - Elevated ST (injury), inverted T wave (ischemia), Q wave (tissue death)
E - Inverted T wave (ischemia), Q wave (tissue death)
F - Q wave (permanent marking)

Evolution of AMI

Diagnostic criteria for AMI

• Q wave duration of more than 0.04 seconds
• Q wave depth of more than 25% of ensuing r wave
• ST elevation in leads facing infarct (or depression in opposite leads)
• Deep T wave inversion overlying and adjacent to infarct
• Cardiac arrhythmias

Location of infarct combinations
Complications of Lateral Wall MI

- Monitor for lethal heart blocks
  - Second degree type II – classical
  - Third degree heart block – complete
- Treat with TCP
  - Consider sedation for patient comfort
  - Monitor for capture
  - Monitor for improvement by measuring level of consciousness and blood pressure

Complications of Inferior Wall MI

- May see Mobitz type I – Wenckebach
  - Due to parasympathetic stimulation & not injury to conduction system
- Hypotension
  - Right ventricle may lose some pumping ability
  - Venous return exceeds output, blood accumulates in right ventricle
  - Less blood being pumped to lungs to left ventricle and out to body
  - Develop hypotension, JVD, with clear lung sounds
- Treated with additional fluid administered cautiously
- EMS to contact Medical Control prior to NTG administration
Complications of Septal Wall MI V1 & V2

- Monitor for lethal heart blocks
  - Second degree type II – classical
  - Third degree heart block – complete
    - Treat with TCP
- Rare to have a septal wall MI alone
  - Often associated with anterior and/or lateral wall involvement

Complications of Anterior Wall MI V3 & V4

- Occlusion of left main coronary artery – the "widow maker"
  - Cardiogenic shock and death without prompt reperfusion
- Second degree AV block type II
  - Often symptomatic
  - Often progress to 3rd degree heart block
  - Prepare to initiate TCP
- Third degree heart block – complete
  - Rhythm usually unstable
  - Rate usually less than 40 beats per minute
  - Prepare to initiate TCP

ST T changes - MI anteroseptal/// axis???

- ST T in V1-V5/ aVL
- Axis
ST DEPRESSION

ST in V1-V4

ISCHEMIC T WAVES
LAHB RBBB
Patient Presenting with Coronary Chest Pain – AMI Until Proven Otherwise

- Oxygen
  - May limit ischemic injury
  - New trends/guidelines coming out in 2011 SOP’s
- Aspirin - 324 mg chewed (PO)
  - Blocks platelet aggregation (clumping) to keep clot from getting bigger
  - Chewing breaks medication down faster & allows for quicker absorption
  - Hold if patient allergic

Acute Coronary Syndrome Medications cont.

- Nitroglycerin - 0.4 mg SL every 5 minutes
  - Dilates coronary vessels to relieve vasospams
  - Increases collateral blood flow
  - Dilates veins to reduce preload to reduce workload of heart
    - Watch for hypotension
    - If inferior wall MI (II, III, aVF), contact Medical Control prior to administration
    - If pain persists, move to Morphine
    - Check for recent male enhancement drug use (ie: viagra, cialis, levitra)
    - Side effect could be lethal hypotension
Acute Coronary Syndrome Region X EMS Medications cont.

• Morphine - 2 mg slow IVP
  – Decreases pain & apprehension
  – Mild venodilator & arterial dilator
  – Reduces preload and afterload
  – Given if pain level not changed after nitroglycerin
  – Give 2mg slow IVP repeated every 2 minutes as needed
  – Max total dose 10 mg

T wave

• The normal T wave is asymmetrical, the first half having a more gradual slope than the second half
• The T wave should generally be at least 1/8 but less than 2/3 of the amplitude of the corresponding R wave
• T wave amplitude rarely exceeds 10 mm
• Abnormal T waves are symmetrical, tall, peaked, biphasic or inverted.

T wave

• As a rule, the T wave follows the direction of the main QRS deflection. Thus when the main QRS deflection is positive (upright), the T wave is normally positive.
• Other rules
  – The normal T wave is always negative in lead aVR, but positive in lead II.
  – Left-sided chest leads such as V4 to V6 normally always show a positive T wave.

Hyperkalemia: peaked T waves

See a normal EKG.

Hypokalemia: prominent U waves

See a normal EKG.
**QT interval**

- QT interval decreases when heart rate increases
- A general guide to the upper limit of QT interval. For HR = 70 bpm, QT<0.40 sec.
  - For every 10 bpm increase above 70 subtract 0.02 sec.
  - For every 10 bpm decrease below 70 add 0.02 sec.
- As a general guide the QT interval should be 0.35-0.45 sec, and should not be more than half of the interval between adjacent R waves (R-R interval).

**Long QT Syndrome**

- Electrocardiogram from 5 year old girl with long QT syndrome

**Prolonged QTc**

- During sleep
- Hypocalcemia
- Ac myocarditis
- AMI
- Drugs like quinidine, procainamide, tricyclic antidepressants
- Hypothermia
- HOCM

**Shortened QT**

- Advanced AV block or high degree AV block
- Jervell-Lange-Neilson syndrome
- Romano-ward syndrome

- Digitalis effect
- Hypercalcemia
- Hyperthermia
- Vagal stimulation
**QT Interval**

- The QT interval increases slightly with age and tends to be longer in women than in men.
- Bazett's correction is used to calculate the QT interval corrected for heart rate (QTc): \( \text{QTc} = \frac{\text{QT}}{\sqrt{R-R \text{ in seconds}}} \)

**U wave**

- Normal U waves are small, round, symmetrical and positive in lead II, with amplitude < 2 mm (amplitude is usually < 1/3 T wave amplitude in same lead)
- U wave direction is the same as T wave direction in that lead
- More prominent at slow heart rates and usually best seen in the right precordial leads.
- Origin of the U wave is thought to be related to afterdepolarizations which interrupt or follow repolarization

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**Final Impression**

* Does the ECG correlate with the clinical scenario? *