Ischemia and ST changes
- Coronary Arteries
- Mechanisms of ischemia
- Treatment
- Ischemia and MI
- EKG changes

Right Coronary Artery
- RCA Supplies
  - RA and RV
  - Inf and post. walls of the LV
  - SA node in 55% of people
  - AV node in 90% of people
  - Posterior fascicle of the LBB

Left Anterior Descending Artery (LAD)
- LAD Supplies
  - anterior wall of LV
  - LA and IVS
  - Apex of the heart
  - RBB
  - anterior fascicle of the LBB

Circumflex Artery
- Supplies
  - Lateral wall of LV
  - inferior and posterior wall of LV (10% of population)
  - septal perforator of LBB
  - SA (45% of population)
  - AV node (10% of population)

Law of Supply & Demand
- Oxygen delivery
  - luminal diameter
  - driving pressure - resistance to flow
  - hemoglobin content
  - blood viscosity
- Oxygen requirement
  - heart rate
  - wall tension
  - contractile state

Vasospasm
- Occurrence
  - Occurs in large or small arteries
  - Usually occurs near an artery damaged by plaque
- Factors that precipitate vasospasm
  - cold exposure
  - anxiety, fear, hostility
  - exercise, hyperventilation
- Factors that prevent vasospasm
  - nitroglycerine, calcium blockers
  - endothelial factors
Occlusions > 70% cause ischemia?

- Frequently taught that perfusion is not limited until a plaque occludes 70-80% of the lumen
- Untrue at high velocities of flow
- Plaque may increase susceptibility to vasospasm in arteries with much less occlusion
- Use caution in the interpretation of angiography results

Coronary Collaterals

- Primary stimulus is hypoxia
- Occurs in humans in vessels with > 75% occlusion
- Occurs rapidly, min in dogs
- Gradual onset of occlusion, more collaterals, better outcome
- Use of exercise in rehab
  - to promote collateral development?
  - to increase CA size (Clarence Demarr, Mr. Marathon)

Trigger Mechanisms for Ischemia

- Passive collapse of a vessel near a stenotic region
- Spasm, related to sympathetic tone
- Plaque rupture produces an ulcerated region that attracts platelets.
- Platelets attracted to plaque cause production of a powerful vasoconstrictor (thromboxane A2)
- Protective mechanisms = prostacyclin and nitric oxide are made by the endothelium and are vasodilators and plaque inhibitors.

Why the endothelium becomes ischemic first

- > blood flow, < bf to endothelium

Vasodilatory Reserve

- VR = ability to increase coronary flow
  - usually 8-fold ability in humans
  - decreases in arteries with occlusion
  - Syndrome X = persons with LV hypertrophy with normal coronary arteries except, they have a reduced vasodilatory reserve (endothelin mechanism?)
    - nitric oxide
    - adenosine (↑ coronary bf during hypoxia)

Effect of Posture on Angina

- Supine position, CBV increases by 200-300 ml
- Increases LV EF
- Greater endocardial ischemia
**ACSM Post-Exercise Guidelines (pg 106, ACSM guidelines)**

- Normal stress testing
  - cool-down for 3-5 minutes at low workload, recording EKG and BP
- Clinical stress testing
  - Record 10 sec of EKG in the upright posture, then the patient should be supine during the post-exercise period for EKG
  - more sensitive method to detect ST changes

**Protective Action of decreased contractility**

- Ischemic region soon loses contractility
- Reduction in wall motion and sometimes even a paradoxical bulge appears in the ischemic region even before ATP is depleted (met trigger, ↓ pH?)
- Decreased contraction promotes increased blood flow to this region—reduces injury?
- Wall motion changes are used to assess for ischemia (echocardiography)

**Pericardial Hypothesis of ischemia**

**Pain and Ischemia**

- Cause of pain in ischemia is unknown
  - metabolites? bradykinins, prostaglandins?
- Subendocardial ischemia with ST depression often occurs without pain
- Absence of pain is of no value in predicting CAD
- Silent myocardial infarction
- Silent ischemia
  - in 2703 patients with a positive stress test, only 66% had pain

**Mechanism of ST depression**

- K+ is lost from the ischemic tissue
- positive ion loss produces a current vector toward the endocardium, opposite the mean QRS vector
- appears as ST depression on the EKG

**ST Elevation**

- Occurs with myocardial injury
  - Ellstad, occurs with a transmural injury
  - Occurs when the tissue is damaged, before it becomes necrotic and has no electrical activity
**Acute Coronary Syndromes Treatment**

- AHA Handbook
- pages 28-52
- ischemia algorithm
- treatment rationale
- EKG interpretation
- drug effects

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**MONA**

- Oxygen
  - may reduce ischemic injury
- Nitrates
  - dilates coronary arteries
- Morphine
  - take for pain if nitroglycerin does not help
- Aspirin
  - inhibits thromboxane
    - dissolves fibrin in the clot and prevents platelet aggregation

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**AHA Chest Pain Algorithm pg 29**

MONA greets all patients

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**History of CAD pg 28**

A. Unstable plaque
B. Plaque rupture
  - platelets aggregate
  - thrombin clot
C. Angina
  - anti-platelet agents
  - GP IIb/IIIa, aspirin
D. Microemboli
  - cardiac markers
E. Occlusive thrombus
  - MI with Q waves
  - Fibrinolytics
  - Percut. Coron. Interv (PCI)

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**Chest Pain Algorithm, cont.**

Assess 12 lead EKG

<table>
<thead>
<tr>
<th>ST depression</th>
<th>T inversion</th>
<th>Non-diagnostic normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST elevation</td>
<td>new LBBB</td>
<td></td>
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</tbody>
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Aspirin/Heparin
- Antiplatelet therapy
- Glycoprotein IIb/IIIa inhibitors
- B-blockers
- nitrates

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**ST depression treatment**

- A partially occluded artery causes ischemia
- Caused by thrombin-rich platelets
- Antiplatelet agents (aspirin and GP IIb/IIIa inhibitors are most effective)
- Fibrinolytic agents may paradoxically accelerate occlusion
- B-blockers to decrease contraction
- Nitrates to vasodilate and increase blood flow
**Chest Pain Algorithm, cont.**

- Assess 12 lead EKG
- ST depression/T inversion
- ST elevation/new LBBB
- Non-diagnostic normal

**ST elevation treatment**

- May indicate complete occlusion
- Clot must be dissolved asap to minimize cardiac damage
- Prompt fibrinolytics to dissolve the clot (pg 62)
- Percutaneous coronary intervention to open the artery
- B-blockers to decrease contraction
- Nitrates to vasodilate and increase bf

**Ischemia vs. Myocardial Infarction**

- Ischemia
  - hypoxic tissue
  - due to inadequate bf/oxygen requirement
  - ST depression
- MI
  - occluded artery(s)
  - tissue necrosis
  - elevated ST segment
  - may or may not have Q wave changes

**Non-Diagnostic EKG**

- Monitor EKG for elevation or depression
- Monitor cardiac markers for MI
  - CK-MB isoforms (early markers of necrosis)
  - troponin
- Consider imaging
- Look for other causes of chest pain

**Q waves and MI**

- Small Q waves (septal depol) are usual in leads I, aVL, V5 and V6 (the lateral leads)
- Q Criteria for MI
  - duration ≥ 0.04 sec or
  - amplitude ≥ 1/4 of the R wave in the same lead
- Present when damage involves the entire thickness of the myocardial wall
Localization of MI and Ischemia

- EKG leads can be used to determine which area (sometimes even vessels) of the heart are affected

Inferior leads: II, III, aVF

Anterior leads: V3, V4

Lateral leads: I, aVL, V5, V6

How to measure ST changes

0.08 seconds for ACSM
Types of ST depressions

• Upsloping
  – least specific
  – 30-40% false positive
  – females
• Horizontal
  – ~10% false positives
• Down sloping
  – most sensitive
  – 5-10% false positive in middle-aged males
  – < 5% with chest pain

ST prognosis

• The greater the mm of depression or elevation, the greater the amount of tissue affected
• The greater the number of leads with the change, the greater the amount of tissue affected
• The earlier in the stress test that the changes occur, the more severe the condition
• Simultaneous occurrence of other indicators (pain, T waves, Q waves) increases probability of a true positive result

Other Causes of ST depression

• Ventricular hypertrophy
  – LV, leads I, aVL, V4-V6 (lateral leads)
  – RV, leads V1, V2
• RBBB
  – V1, V2
• LBBB
  – I, aVL, V5, V6
• Drugs, esp. digitalis

Review

• A 55 year old man complaining of chest pain
• Resting EKG and blood pressure are normal
• Bruce treadmill stress test
  – Stage 3
  – 3.3 mph, 14% grade
  – Subject complains of chest pain
  – the following EKG changes are seen

Review cont.

• What do the EKG changes tell you?
  – Mild ischemia, severe ischemia with damage, or MI?
  – right or left side of the heart is affected?
  – Upsloping, horizontal, or downsloping change?
  – What do you think about the prognosis? Accurate?
• What do you do next?
  – Continue the exercise test?
  – Cool down procedures?
Review, cont.

- If the pain continues and gets worse, what treatments should this bring to mind?
  - Immediate treatments for all patients with chest pain?
  - Long-term treatment based on EKG?