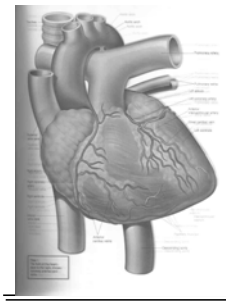
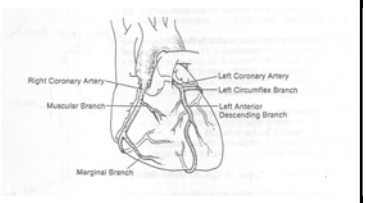
Ischemia and ST changes



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

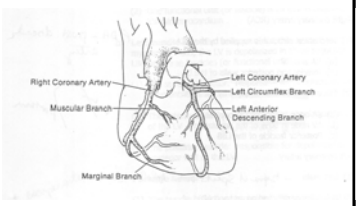
Right Coronary Artery

- **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**



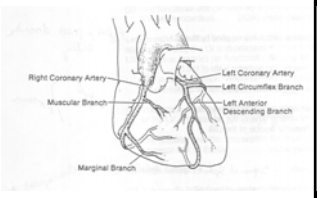
Left Anterior Descending Artery (LAD)

- **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

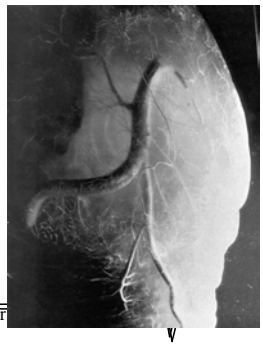
- Occurrence
  - Occurs in large or small arteries
  - Usually occurs near an artery damaged by plaque
- Factors that precipitate vasospasm
  - cold exposure
  - anxiety, fear, hostility
  - exercise, hyperventilation
- Factors that prevent vasospasm
  - ~~nitroglycerine, calcium blockers~~
  - endothelial 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 susceptibilty to vasospasm in arteries with much less occlusion
- Use caution in the interpretation of angiography results

Coronary Collaterals

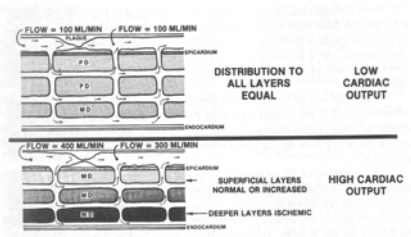
- Primary stimulus is hypoxia
- Occurs in humans in vessels with > 75% occlusion
- Occurs rapidly, *min in dogs*
- Gradual onset of occlusion, more collaterals, better outcome
- Use of exercise in rehab
  - to promote collateral development?
  - to increase CA size (Clarence Demarr Mr. Marathon)



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 platlets.
- Platlets 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

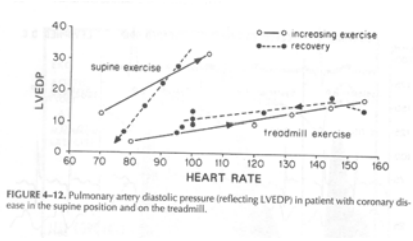


FIGURE 4–12. Pulmonary artery diastolic pressure (reflecting LVEDP) in patient with coronary disease in the supine position and on the treadmill.

Supine position, CBV increases by 200-300 ml
Increases LVEDP
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

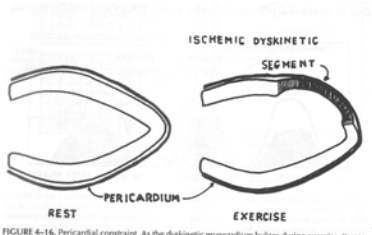


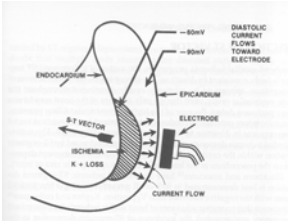
FIGURE 4–16. Pericardial constraint. As the dyskinetic myocardium bulges during exercise, it uses up the maximum pericardial distensibility so that the normal diastolic expansion cannot occur, thus resulting in a restrictive process that causes an increase in LVEDP.

Pain and Ischemia

- Cause of pain in ischemia is unknown
  - metabolites? bradykinins, prostoglandins?
- 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 26% had pain

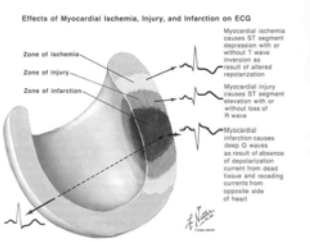
Mechanism of ST depression

- 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



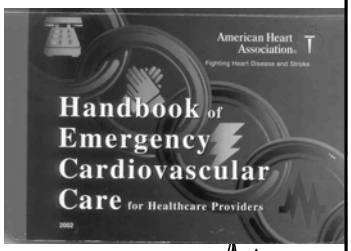
ST Elevation

- 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

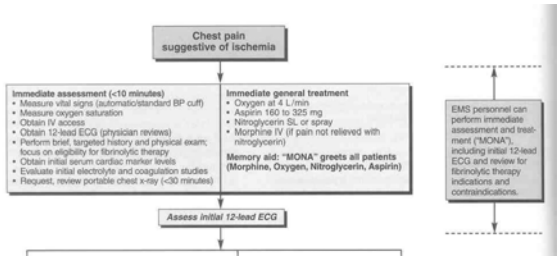


Acute Coronary Syndromes Treatment

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



AHA Chest Pain Algorithm pg 29

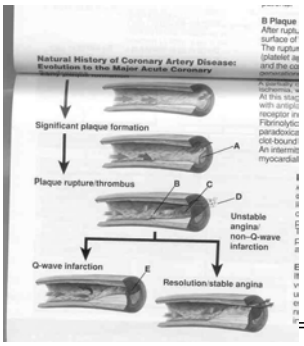


MONA greets all patients

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

History of CAD pg 28



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

Chest Pain Alogrithm, cont.

Assess 12 lead EKG

ST depression/ T inversion

ST elevation new LBBB

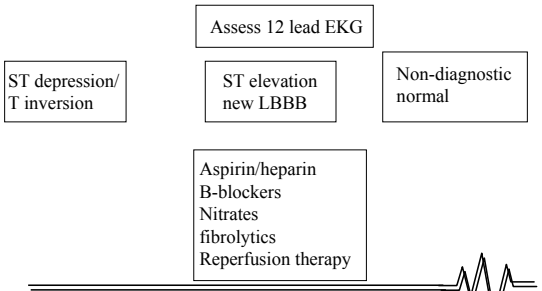
Non-diagnostic normal

Aspirin/Heparin
Antiplatlet therapy:
Glycoprotein IIb/IIIa inhibitors
B-blockers
nitrates

ST depression treatment

- A partially occluded artery causes ischemia
- Caused by thrombin-rich platlets
- Antiplatlet agents (aspirin and GP llb/llla inhibitors are most effective)
- fibrinolytic agents may paradoxically accelerate occlusion
- B-blockers to decrease contraction
- Nitrates to vasodilate and increase blood flow

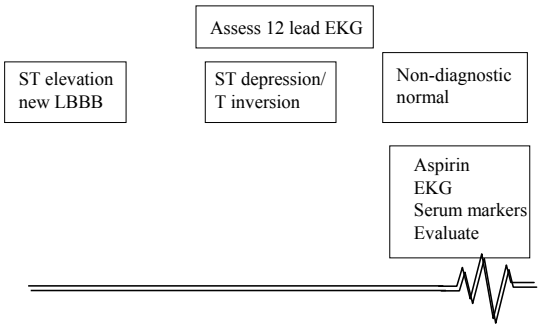
Chest Pain Alogrithm, cont.



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

Chest Pain Alogrithm, cont.



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

Ischemia vs. Myocardial Infarction

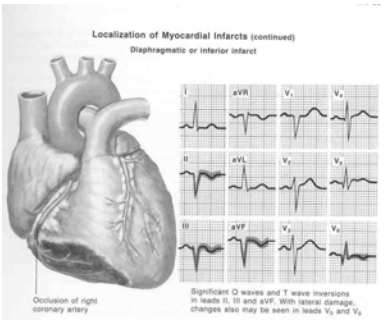
- Ischemia
  - hypoxic tissue
  - due to inadequate bf/oxygen requirement
  - ST depression
- MI
  - occluded artery(s)
  - tissue necrosis
  - elevated ST segment
  - may or may not have Q wave changes

Q waves and MI

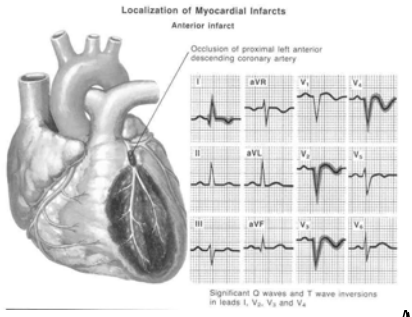
- Small Q waves (septal depol) are usual in leads I, aVL, V5 and V6 *(the lateral leads)*
- Q Criteria for MI
  - duration $\geq$ 0.04 sec or
  - amplitude $\geq$ 1/4 of the R wave in the same lead
- Present when damage involves the entire thickness of the myocardial wall

Localization of MI and Ischemia

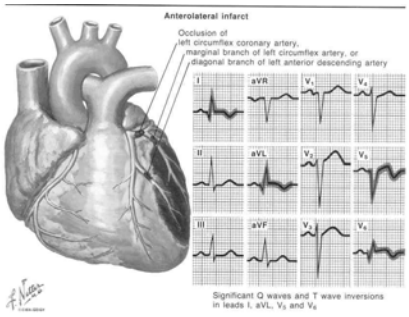
- EKG leads can be used to determine which area (sometimes even vessels) of the heart are affected



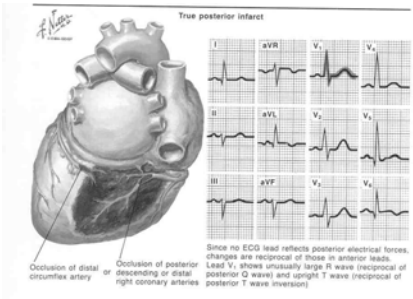
Inferior leads: II, III, aVF



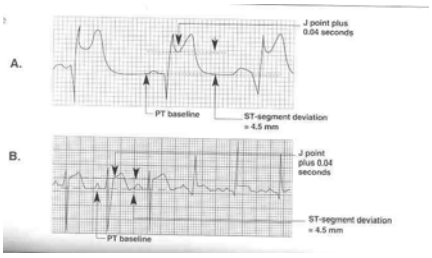
Anterior leads: V3, V4



Lateral leads: I, aVL, V5, V6



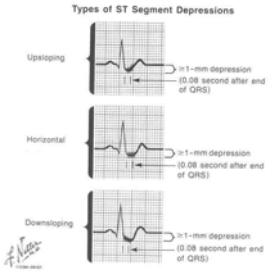
How to measure ST changes



0.08 seconds for ACSM

Types of ST depressions

- Upsloping
  - least specific
  - 30-40% false positive
  - females
- Horizonal
  - ~10% false positives
- Downsloping
  - most sensitive
  - 5-10% false positive in middle aged males
  - < 5% with chest pain



ST prognosis

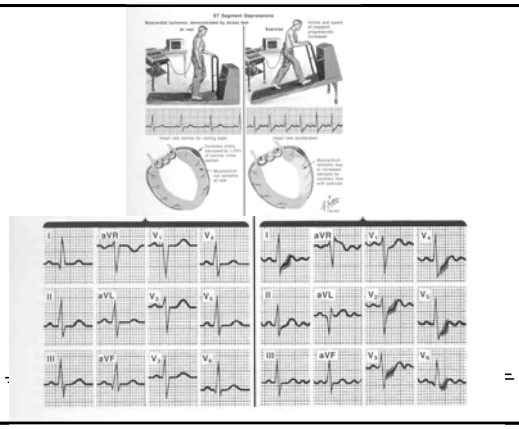
- The greater the mm of depression or elevation, the greater the amount of tissue affected
- The greater the number of leads with the change, the greater the amount of tissue affected
- The earlier in the stress test that the changes occur, the more severe the condition
- Simultaneous occurrence of other 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?