

Acute Coronary Syndrome

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CCRN



Angina & MI Interventions

• **All interventions relate to oxygen supply & demand**

- There is a decreased O₂ supply in hypoxemia, such as anemia
- An increased O₂ demand in tachycardia, increased preload, increased afterload
- The goal is to increase O₂ supply (increase coronary blood flow) & decrease O₂ demand (decrease ht rate, decrease preload & afterload)



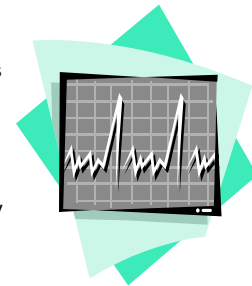
Cardiovascular Assessment

- Subjective Data: Personal/Fam HX, CP, Dyspnea, weight changes etc.
- Physical Assessment: Skin, extremities, BP, JVP, Lungs, Precordium
- Objective Data: Labs: CPK/ troponin
- Hypo/ Hyperkalemia
- Hypocalcemia
- Serum NA



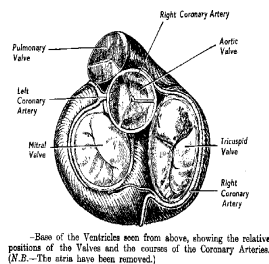
EKG: "Einthoven's Triangle"

- **I, II, III** measure differences in activity between the limb leads
- **AVR, AVL, AVF** measure activity between the heart & the limbs
- **V1-V6** measure activity of heart on horizontal plane



Testing: Non invasive

- **Holter monitor**
- **Echocardiogram: TTE and TEE**
- **Exercise stress test**
- **Nuclear cardiography:**
Thallium Muga
(multiple gated acquisition scan)



Cardiac Catheterization: Coronary Angiogram

- **Most definitive: most invasive**
- **Purpose:** To define clinically suspected lesion, assess pathophysiology of cardiac Dz, information R/T LV function
- CO= SVxHR
- **Right Ht Cath:** venous access ; brachial or femoral: measures pressures: RA-RV-PA-PCWP
- **Left Ht cath:** arterial access; checks patency of coronary arteries



Cardiac Cath : Followup Care

- **Bedrest 4-24 hours (Dependent if PCI was done post cath)**
- Supine
- Insertion site nonflexed: check pulse, color skin temp with each VS check
- Different closure devices: ex. Angioseal
- Check ACT prior to sheath pull; usually can pull sheath when ~ 150
- **Complications: Bleeding/ hematoma, R/P Bleed**

CAD Treatments

- Medications
- PTCA
- PTCA/ Stents (vessel must be considered @ about 80% blocked)
- Rotoblade
- Radiation therapy
- CABG

Other Measurements

- Pulmonary artery catheter: PAP: left ventricular function, norm:25/9 mean=15
- PCWP= approx. left atrial pressure & LVEDP
- CVP: right atrial pressure
- CO=4-8L/min

- Electrophysiology Studies (EPS): To evaluate dysrhythmias
- CPK/ MB/ Index & Troponin

Prinzmental Angina

- CP @ rest or sleep
- Coronary artery spasm
- May or may not have coronary atherosclerosis
- Rx: calcium channel blockers

Myocardial Infarction

- Life Threatening: formation of dead tissue
- Coronary artery occlusion: may be spasm
- Types: Anterior, Lateral, Inferior, Posterior
- All three layers: Transmural
- Through one or two layers: Nontransmural (nonQ wave MI; non STEMI)
- Phys. Exam: pale, anxious, clammy, diaphoretic, SOB, nausea
- Tests: Enzymes, CBC, BMP, CXR

Myocardial Infarction

- Anterior: I, AVL, V1- V4
- Lateral: I, AVL, V5- V6
- Inferior: II, III, AVF
- Posterior: V1, V2 (Tall R wave with ST depression)

- All 3 layers: Transmural infarct
- Not all 3 layers: Non Transmural infarct

Infarction Zones

- **Ischemia:** ST depression, T wave inversion, Both changes above are seen in both leads
- **Injury:** (1)*subendocardial:* still reversible, ST down 1 mm, transient, returns to baseline when pain subsides
(2)*subepicardial:* ST/T elevated, usually with Prinzmetal angina; often precedes an MI
- **MI:** Q wave, ST elevated in affected leads, ST depressed in leads 180 degrees from infarcted areas; T wave changes for weeks or rest of life
- **Subendocardial MI:** (no Q waves)

MI: Complications

- **Dysrhythmias:** Ventricular
- **CHF/ Pulmonary Edema**
- **Cardiogenic Shock**
- **Papillary muscle dysfunction:** follows impaired blood flow to coronary arteries
- **Papillary muscle rupture:** 1st week post MI; floppy & inefficient valves

MI: Complications

- **Dressler's Syndrome:** pericarditis post MI; fever, cp, dyspnea; EKG changes. CP intensifies with thoracic movement & deep inspiration
- **Ventricular aneurysm:** Occurs in weeks to years later
- **Ventricular wall rupture:** death from cardiac tamponade

MI Complications

- **Cardiac cripple syndrome:** patient resists resuming ADL's due to anxiety, fear of death, & disability
- **Thromboemboli:** thrombus formation in the left ventricle; leads to systemic circulation, pulmonary emboli can arise from deep leg veins. Early ambulation Important!!
- **ICU Psychosis:** confused, personality changes, fear of death, sleep deprivation, death. Adverse effects from drugs, hypotension, with impaired cerebral blood flow; isolation from family

MI Complications

- Hiccups (Inferior wall MI)
- N&V: Dysrhythmias
- Abdominal distention: constipation
- Curling's stress ulcer: GI prophylaxis

Cardiac Rehabilitation

- **Phase I:** CCU & medical Unit: Disease management, pt. & family education, activity progression, low level exercise
- **Phase II:** Discharge to Home & Community: Begin supervised exercise program or walk program @ home. Education, vocational counseling, multiple evaluations & testing

Cardiac Rehabilitation

- **Phase III:** Maintenance: Emphasis on resuming normal lifestyle, continued evaluation & assessment, individual & prescriptive exercise program
- **Goals:** *risk stratification, limitation of potential adverse psychological consequences of CVD*
- Modification of lifestyle, alleviation of symptoms, reduction of morbidity & mortality, improvement of function, decreasing probability of future clinical S&S of CAD

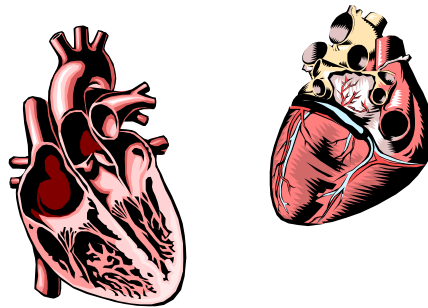
Dietary Issues

- **400 mg: Na = 1 Gm salt**
- **4 Gm Na diet: “no added salt”**
- **2 Gm Na diet: “all salt eliminated from cooking”**
- Salt is 40% sodium
- All foods contain some sodium
- **Salt substitutes contain potassium**
- **Teach patients to read labels**
- Beware of processed foods

Pericarditis

- Acute
- Chronic
- Refer to handout
- Differentiate between pericardial effusion and pericardial tamponade

Pericarditis



Pericarditis

- During initial healing period
- Develops from pericardial irritation due to cellular debris & exudates in infarct area
- May last up to 3 months
- **S & S:** Atypical, long lasting chest pain, low grade fever, chills, diaphoresis, dyspnea, pericardial friction rub, persistent ST segment elevation, dysrhythmias & atrial fibrillation

Pericarditis

- **Diagnosis:** CXR, Echocardiogram (may show pericardial effusion), CK/ MB elevated, elevated Sed rate, & elevated WBC
- **Pain:** sharp, stabbing, localizes to the left or right, shoulder, arm, elbow or neck. Aggravated by inspiration, coughing swallowing, coughing. **Relieved by sitting up & leaning forward**

Pericarditis

- **Friction Rub:** Grating, scratchy, scraping sound
- **Dyspnea** is from compression of bronchi by distended pericardium
- **Treatment:** Bedrest, NSAIDS, ASA, Motrin, sometimes steroids, antibiotics if bacterial
- Pericardectomy: surgical removal of pericardium for recurrent disabling pericarditis; may do pericardial window

Chronic Pericarditis

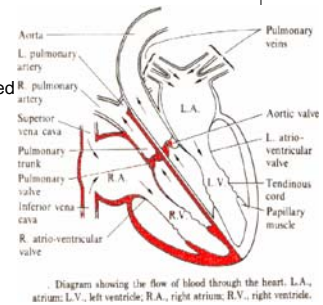
- **Chronic pericardial effusion occurs after pericardial inflammation**
- Constrictive pericardium leads to the pericardium being scarred & rigid
- **Effusive Pericarditis:** constrictive pericarditis; thickened visceral pericardium leading to cardiac constriction
- **Adhesive Pericarditis:** Involves entire pericardium & adjoining mediastinal structures

Complications of Acute & Chronic Pericarditis

- **Pericardial Effusion:** excess pericardial fluid accumulates. ***If it builds up slowly, the pericardium stretches & accomodates up to 2 Liters***.....However, Rapid accumulation bars pericardial stretch; as little as 80- 200 ml can cause cardiac compression

Complications

- **Cardiac Compression:** Muffled heart sounds, paradoxical pulse, decreased CO, decreased BP, increased CVP, tachycardia & distended neck veins
- **Diagnosis:** **Echocardiogram, CXR, EKG**



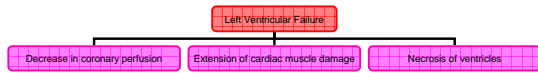
Cardiac Tamponade

- Rapid accumulation of fluid; decreased CO from pressure, decreased ventricular filling therefore, the pericardium compresses the ventricles
- **BECK'S TRIAD: decreased BP, increased CVP, & muffled heart sounds**
- Distended neck veins, tachycardia, dyspnea, **pulsus paradoxus**
- **Treatment: Pericardiocentesis: Pt in ICU with drain**

Cardiogenic Shock

- **Definition:** Impaired ability of the heart to function as a pump. Decrease in systemic blood flow. Resultant in inadequate tissue perfusion. Often associated with Anterior Wall MI. Results fro severe left vantricular failure
- **Causes:** MI (lose 40-45% of myocardium), acute dysrhtymia, severe CHF, cardiac tamponade, cardiomyopathy, spontaneous damage to heart valves

Pathophysiology



- The cardiac output is severely affected....inadequate tissue oxygenation & severe hypoperfusion of the tissues...which will lead to **metabolic acidosis**

Cardiogenic Shock

- **Marked Depressed Cardiac Output....hypotension....inadequate tissue perfusion**

Clinical Presentation:

- **Increased CVP**
- **Decreased CO**
- **Decreased CI**
- **Hypotension (sys<80)**
- **PCWP (> 18 mm Hg)= pulmonary congestion & peripheral edema**
- **Increased HR**
- **Cold, clammy skin, oliguria (< 20 ml/HR)**

Cardiogenic Shock: Management

- Vasopressors: Dopamine & Dobutamine
- Alpha adrenergic stimulators: Norepinephrine
- Afterload increases & deleteriously affects cardiac muscle
- Vasodilators: Nipride & NTG IV
- IABP
- Other measures to improve cardiac function: improve O₂, decrease workload, improve LV function, decrease pt. stress
- Please refer to handout