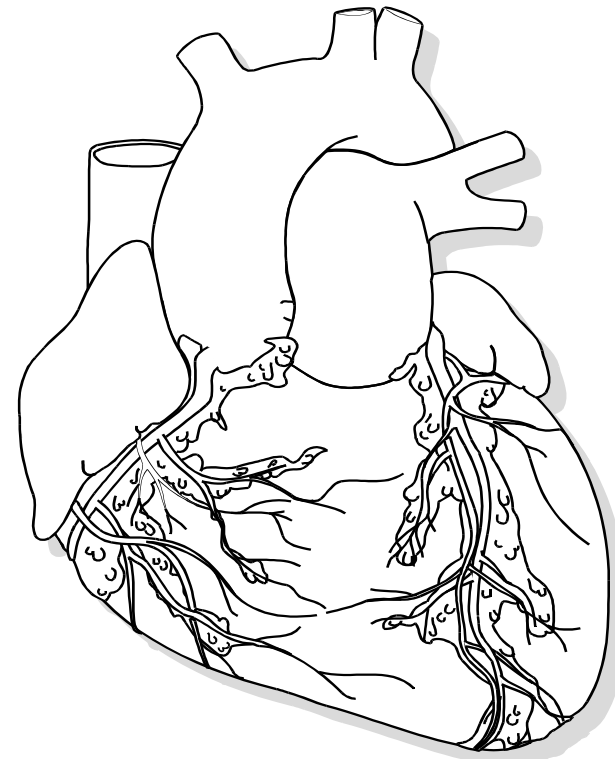


# **CARDIOVASCULAR EMERGENCIES *REVIEW-I***

**CORE CONTENT SYNTHESIS-BASED ON  
ABEM, SAEM, ACEP & ACLS OUTLINES**

## **● CLINICAL PEARLS**

- **David Riley, MD**
- **Director of Ultrasound Training & Resident Didactics**
- **Department of Emergency Medicine**
- **St. Luke's Roosevelt Hospital Center**
- **Columbia University College of Physicians & Surgeons**



# PATHOPHYSIOLOGY I

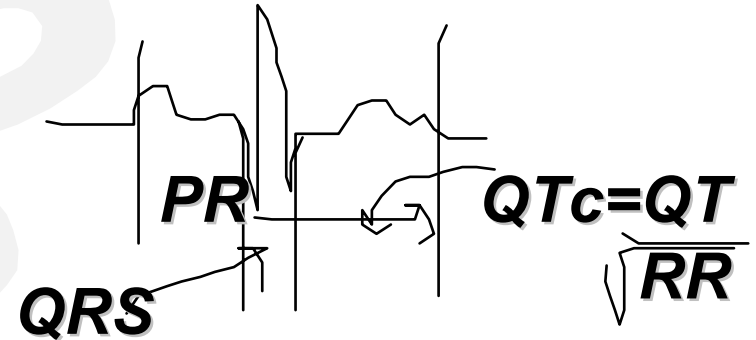
ECG EVALUATION: rate, rhythm, intervals, segments, axis, waves

I up + aVf up = nl axis

I up + aVf down = LAD

I down + aVf up = RAD

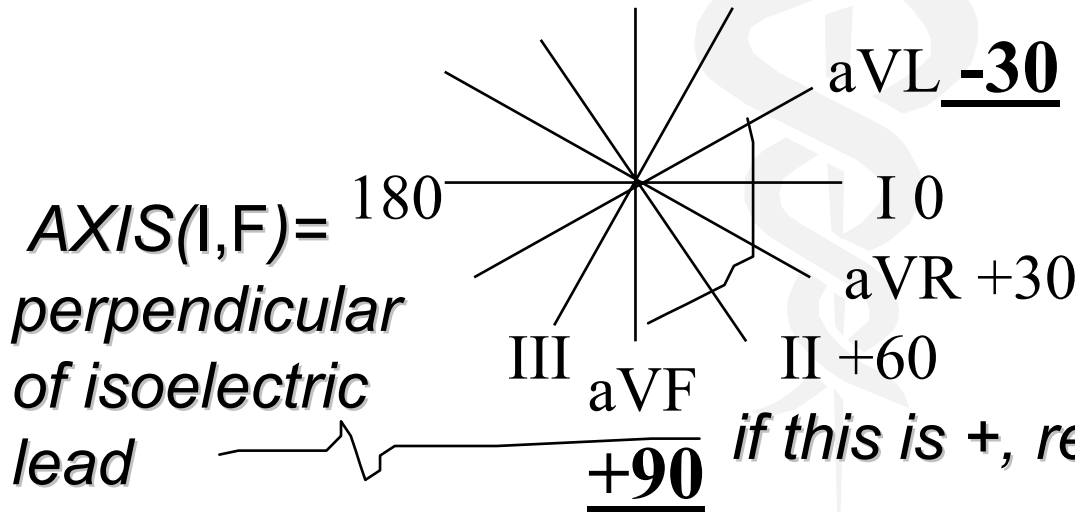
low voltage: air(copd), fat, water, cardiomyopathy



**-30 to +90: normal axis**

**-30 to -90: LAD**

**+90 to +180: RAD**



# Electrical Axis

- Northwest axis=no man's land (-) I & F
  - -90 to -180
  - Causes:
    - EMPHYSEMA, HYPERKALEMIA, LEAD TRANSPOSITION, CARDIAC PACING, VT
- Causes of right axis deviation
  - nl in peds, RVH, COPD, LPIB, PE, ASD, VSD
- Causes of left axis deviation
  - LASB, Q waves of inf-AMI, emphysema, hyperkalemia, WPW-right accessory, tric. atres.

# PATHOPHYSIOLOGY II

- **DO<sub>2</sub>=O<sub>2</sub> DELIVERY=O<sub>2</sub> CONTENT \* CARDIAC INDEX**, nl 550-600cc/min/m<sup>2</sup>
- O<sub>2</sub> CONTENT=(Hbg/dl\*%sat\*1.36cc/g) +(PO<sub>2</sub>\*.003cc of O<sub>2</sub>/mmHg/dl)
- **CARDIAC INDEX=CI=CARDIAC OUTPUT/BSA**, nl:2.5-4L/min/m<sup>2</sup>
- BODY SURFACE AREA(ht-cm+wt-kg - 60/100)=BSA
- OXYGEN CONSUMPTION=VO<sub>2</sub>=120cc/min/m<sup>2</sup>
- **SvO<sub>2</sub>=mixed venous oxygen saturation: 70-75% normal**
- O<sub>2</sub>er=oxygen extraction ratio=VO<sub>2</sub>/DO<sub>2</sub>(\*100); 20-30% normal
- CARDIAC OUTPUT=HR\***SV(75cc-nl)**(preload,afterload,contractility). nl 4-6 L/min
- **BP=CO \* SVR**(800-1200dynes/sec/cm<sup>2</sup>), **Mean Arterial Pressure=MAP=D + S-D/3**
- SVR=systemic vas. resistance=MAP-CVP\*80/CO= normal 800-1200 dyne/sec/cm<sup>2</sup>
- PVR=pulmonary vas. resistance=PAP(10-20mmHg) - PCWP \*80/CI, NL 150-250
- R ATRIAL PRESSURE=RAP=**CVP, NL 2-5 mm Hg**
- PULMONARY CAP WEDGE PRESSURE=**PCWP=LVEDP, NL 6-12mm Hg**
- **CORONARY PERFUSION PRESSURE = AORTIC PRESSURE - RAP**

# Pathophysiology III

- Shock Index =  $HR/SBP$  0.5-0.7 is normal, **>1 is BAD**
- ACLS Goals maximize  $CPP=AoP-RAP$
- Mixed Venous O<sub>2</sub> saturation: best index for continued bleeding in trauma(base deficit also), ie:  $SvO_2 = RA(VBG)$  if )2 saturation <60% this is a blood transfusion indication

# CARDIOVASCULAR EFFECTS OF AGING

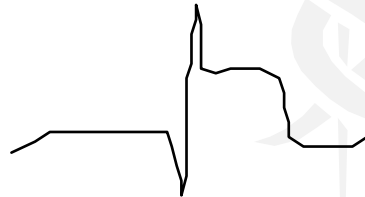
- \*1. Diminished early (passive) left ventricular filling (DIASTOLIC DYSFUNCTION) with increased dependence on the atrial kick to fill the ventricle (thus more prone to HYPOTENSION, w/ decreased LV stroke output, in a.fib. or SVT)
- 2. Operating further up the Frank-Starling curve - LESS RESERVE
- 3. Decreased inotropic and chronotropic responses to catecholamines

# Normal R wave Progression

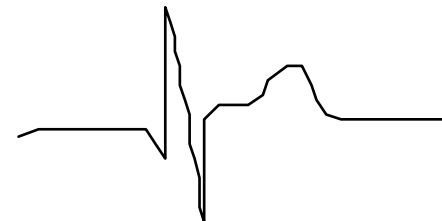


# ST SEGMENT ELEVATIONS

- 1. Injury Associated with Myocardial Infarction
- 2. Prinzmetal variant angina( espec. At night-chest pain)
- 3. Ventricular aneurysm(big source of mural thrombi - emboli)
- 4. Pericarditis: PR dep.& PR elevaiton-aVr: ST/T >0.25
- 5. Early repolarization-normal variant: ST/T <0.25 (V3-V6 primarily)
  - notched J point; young men typically
- 6. LBBB: T waves normally discordant: Espec. In V1-2
- 7. Paced ventricular rhythm
- 8. Hypothermia (causes AV block)
- 9. Hyperkalemia (causes AV block): in Anteroseptal Leads V1-4
- 10. Drug Induced, ie TCA's=Rx. low BP w/NE
- 11. Brugada's Syndrome = RBBB + V1-3 ST elevations



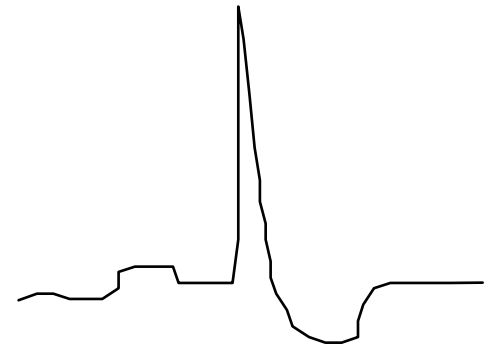
*convex up AMI*



*concave up BER*

# ST SEGMENT DEPRESSION

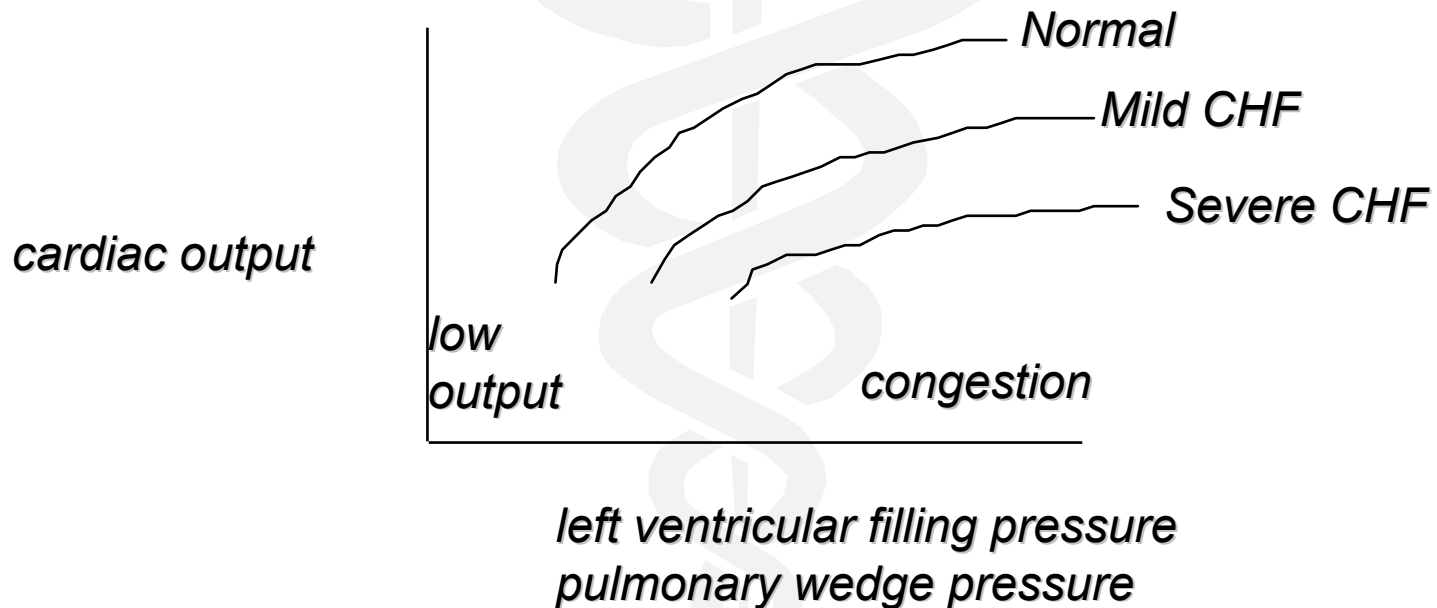
- 1. Absolute ischemia, due to coronary artery narrowing
- 2. Relative ischemia, due to increased demands of the myocardium; hypertrophy, anemia, hypoxia
- 3. Subendocardial(non-Q wave) myocardial infarction
- 4. Digitalis effect
- 5. V1-V2 possible POSTERIOR AMI:
  - UPRIGHT T-WAVES V1-V2
- 6. Hypokalemia



# ACQUIRED DISEASES OF THE MYOCARDIUM

- 1. CARDIAC FAILURE
- 2. CARDIOMYOPATHY
- 3. ISCHEMIC HEART DISEASE
- 4. ENDOCARDITIS
- 5. VALVULAR HEART DISEASE
- 6. MYOCARDITIS

# 1. CARDIAC FAILURE



*Definition: Heart cannot meet the demands of the circulation in all conditions, OR it can meet the demands with GENERALIZED INCREASED PRESSURES*

# Congestive Heart Failure

- **NEW YORK HEART ASSOCIATION FUNCTIONAL CLASSIFICATION:**
  - CLASS I: SX ONLY W/>NL ACTIVITY
  - CLASS II: SX DURING NL ACTIVITY
  - CLASS III: SX W/MINIMAL ACTIVITY
  - CLASS IV: SX AT REST
- CAUSES OF **CHF EXACERBATIONS**: NON-COMPLIANT W/MEDS; ISCHEMIA-AMI:Killip Classification(I-IV same as boxes), INFECTION, ANEMIA, ARRHYTHMIA, STRESS, >Na, PE, RHEUM-OTHER VALVE PROBLEM, THYROID DISEASE, PREGNANCY(HIGH CO STATE), A-V FISTULA, ETOH
- **Exercise intolerance is key symptom**

# Congestive Heart Failure

- THE HOT, COLD, OLD  
AND SICK  
HEART.....WALK  
THEM SLOWLY UP THE  
STARLING CURVE

# A. RIGHT OR LEFT VENTRICULAR FAILURE?

- HALLMARK OF HEART FAILURE IS **EXERCISE INTOLERANCE: (DOE>PND>Orthopnea)**
- **LHF: ORTHOPNEA & PAROXYSMAL NOCTURNAL DYSPNEA; RALES; S3(IF PRESENT:HIGHLY PREDICTIVE OF DECREASED EF)(LATE DIASTOLIC FILLING WAVE), S3 BEST HEARD WITH PT ON L SIDE, USE BELL OF STETHESCOPE AT APEX OF HEART with patient on L side, PULSUS ALTERNANS (severe CHF)**
- **RHF: EDEMA OF FEET AND ANKLES PRIMARILY; JVD; HEPATOMEGALY, HEPATOJUGULAR REFLUX; JUGULAR VENOUS DISTENTION, COPD**

# DYSPNEA: CARDIAC OR PULMONARY ORIGIN?

- B-Naturetic Peptide (**BNP**): NEJM, Maisel, 2002, 347: 161-68
- >100 pg/ml: 83% Diagnostic accuracy for CHF
- <50 pg/ml: 96% Negative predictive value for CHF
- USE OF PEAK FLOW METER MAY BE HELPFUL **YET/BNP = #1**
- **PEFR > 150 SUGGESTIVE OF CONGESTIVE HEART FAILURE: Dyspnea 1st then cough: PND**
- **PEFR < 150 SUGGESTIVE OF CHRONIC LUNG DISEASE: Cough 1st then dyspnea: PND**

# **B.ISOLATED RIGHT HEART FAILURE**

- 1. **PULMONARY EMBOLISM (S1, Q3, T3)**
- 2. TRICUSPID STENOSIS
- 3. TRICUSPID REGURGITATION
- 4. RIGHT ATRIAL TUMOR
- 5. CARDIAC TAMPONADE
- 6. CONSTRICTIVE PERICARDITIS
- **7. PULMONARY ARTERIAL  
HYPERTENTION:COPD; Primary:dyspnea,  
fatigability & women-raynaud's**
- 8. RIGHT VENTRICULAR INFARCTION
- 9. INTRINSIC LUNG DISEASE
- 10. Ebstein's Anomaly( tricuspid stenosis & right ventricular hypoplasia: atrialization of the RV)

# C. LEFT OR BIVENTRICULAR FAILURE

- 1. HYPERTENSIVE HEART DISEASE (75%)
- 2. AORTIC INSUFFICIENCY
- 3. MITRAL STENOSIS
- 4. MITRAL REGURGITATION
- 5. MOST CARDIOMYOPATHIES (ETOH-DILATED-SYST-<EF)
- 6. ACUTE MYOCARDIAL INFARCTION (CORONARY DIS.)
- 7. RESTRICTIVE CARDIOMYOPATHY=DIASTOLIC FAILURE W/NL EF
- 8. MYXOMA
- 9. AORTIC STENOSIS
- 10. MYOCARDITIS: VIRAL/LYME/HIV
- 11. SUPRAVENTRICULAR ARRHYTHMIAS
- 12. CARDIAC SHUNTS
- 13. HIGH CARDIAC OUTPUT STATES (ANEMIA (sickle cell disease), BERIBERI, SYSTEMIC A-V FISTULAE, PAGET'S DISEASE, CARCINOID TUMOR (ileal-tricuspid regurgitation, bronchial-mitral regurgitation), THYROTOXICOSIS (remember apathetic))

# ECG EVIDENCE OF LEFT VENTRICULAR HYPERTROPHY

- **LVH: VOLTAGE CRITERIA**  
**SENSITIVE(SNOOT) YET NOT**  
**SPECIFIC(SPIN)**
  - **S IN V1 + R IN V5 OR V6 >35mm**
  - **S IN V1 OR R IN V5-V6 >25**
  - **R IN aVL >11**
  - **strain pattern: flipped T waves in aVL, V4-V6**
  - **LEFT AXIS DEVIATION (>-30)**
- **D.Dx: severe HTN, AS, IHSS**

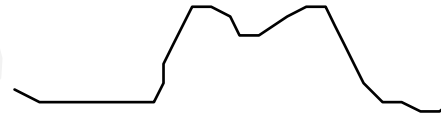
# ECG EVIDENCE OF RIGHT VENTRICULAR HYPERTROPHY

- RIGHT AXIS DEVIATION (+90)
- R/S RATIO  $> 1$  IN V1-V2: POOR R-WAVE PROGRESSION
- R/S RATIO  $< 1$  IN V6
- RIGHT VENTRICULAR LEADS V1-V3 FLIPPED T WAVES: LIKE IN CHILDREN

# ECG EVIDENCE OF LEFT ATRIAL HYPERTROPHY

- **P-MITRALE**: BEST SEEN IN I,II, aVL & V4-V6:(ANT-LATERAL LEADS)

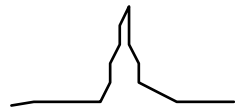
- NOTCHED P WAVE:



- V1 INVERTED P WAVE, >1mm & >1mm WIDE
- THINK OF MITRAL STENOSIS:  
RHEUMATIC HEART DISEASE

# ECG EVIDENCE OF RIGHT ATRIAL HYPERTROPHY

- **P PULMONALE**: PEAKED (>3mm) P WAVES IN II, III & aVF (**INFERIOR**):



- V1 UPRIGHT P WAVES
- **SEEN IN COR PULMONALE-COPD**
- **Room air: 55 pO<sub>2</sub>/88 O<sub>2</sub> sat club or 56/89 + p-pulmonale : medicare will pay for home oxygen**

# RADIOLOGY OF CHF or “Dropsy” ON CXR

- 1. STAGE 1: PULMONARY VASCULAR REDISTRIBUTION TO THE UPPER LUNG FIELDS, CEPHALIZATION (PAWP 15-20)
- 2. STAGE 2: INTERSTITIAL , EDEMA **KERLY B LINES**( interlobar edema-best seen in **R lower lung** ) (PAWP 20-25): Forrester-Diamond Boxes 2 & 4: Goal is box 1:CI>2.2 & PCWP<18
- 3. STAGE 3: ALVEALAR EDEMA; FRANK PULMONARY EDEMA (PAWP >25)

# Pulmonary Edema + Small Heart on CXR

- ARDS: NON-CARDIAC PULMONARY EDEMA (ASA, SEPSIS, PROPOXYPHENE (Darvon) = wide QRS & seizures, PANCREATITIS)
- MITRAL STENOSIS
- MASSIVE AMI
- CONSTRICTIVE PERICARDITIS

# CONGESTIVE HEART FAILURE & PULMONARY EDEMA:RX-UNLOAD ME

- 1. SIT THE PATIENT UP
- 2. OXYGEN, high FLOW, mask-NRB
- 3. LASIX 1MG/KG IV or Bumex, Zaroxilyn
- 4. Nitroglycerine start at 5-10 ug/min if sbp>100
- 5. Nitroprusside start at 0.3- 0.5 ug/kg/min
- 6. Dobutamine 3-15 ug/kg/min IV (SBP > 100)
- 7. Dopamine 6-20ug/kg/min IV is preferred if shock is present (SBP< 90)
- 8. Norepinephrine 0.5-30 ug/min IV (SBP < 70)
- 9. Positive pressure ventilation; CPAP, BIPAP
- 10. check digitalis level
- 11. Morphine 4mg IV
- 12. Ace inhibitors, Amrinone, ALBUTEROL IF BRONCHOSPASM
- 13. Dialysis-ESRD (induce diarrhea w/sorbitol-kaexylate)
- 14. Consider CPAP & BIPAP before Intubation

# How to Mix Drips?

- Nitroglycerine/Nipride: 50mg in 250cc of D5W: 3 microdrops/min = 10 micrograms/min (Titrate BP for SPB >110 with 3 microdrop increases every 5 minutes)
- Dopamine/Dobutamine: TRICK to get 10mcg/kg/min: Add 150mg in 250cc D5W & the patients weight in KG = number of microdrops/minute

# CHF-P.Edema & ESRD

- Ultimate Rx = Dialysis
- If dialysis is not immediately available:  
250-500cc phlebotomy; sorbitol 25-50cc  
of oral; if anuric BIG LASIX IV, ie 200-  
300 mg can help along with the  
standard nitrates & inotropes

## **2. CARDIOMYOPATHIES**

- **DISEASES WHICH DIRECTLY DAMAGE THE MYOCARDIUM, STRUCTURALLY AND FUNCTIONALLY: low voltage=low # myocytes**
- **1. CONGESTIVE DILATED (DECREASED EF)**
- **2. RESTRICTIVE (nl or decreased EF)**
- **3. HYPERTROPHIC (nl or inc EF)**

# CONGESTIVE

## CARDIOMYOPATHY

- **SYSTOLIC PUMP FAILURE (DECREASED EJECTION FRACTION) = S3 at PMI(Not due to (HTN, CAD:top two causes),valve)**
- BIVENTRICULAR CHF & EMBOLI
- SYMPTOMS OF CHF WITH EMBOLI( flank pain and hematuria, neurologic deficit, pulseless cyanotic extremity)
- CAUSES:cocaine, idiopathic, coronary disease,HTN, neuromuscular disease, toxins; **ALCOHOL**, heavy metals, adriamycin; infections; coxsackieB, bacterial(diphtheria); infiltrative; amyloidosis, hemochromatosis; postpartum; metabolic; glycogen storage disease, thiamine deficiency(beriberi), **HIV**, hypothyroidism, hyperthyroidism; collagen vascular disease
- TREATMENT: **#1- ACEI'S**, DIURETICS, DIGITALIS, VASODILATORS, & ANTICOAGULANTS IF IN AFIB

# RESTRICTIVE CARDIOMYOPATHY-Poor

Prognosis: Pts have increased filling pressures

- **● DIASTOLIC PUMP FAILURE (NI. or decreased EJECTION FRACTION), increased filling pressures**
- **CAUSES:** Majority idiopathic( septum asymmetric ), hemachromatosis, amyloidosis, sarcoidosis, scleroderma: all have symmetrical wall thickening
- **Treatment: #1:Diuretics; CCB's, Beta-Blockers.**  
digoxin(should not be used with amyloidosis (USUALLY HAS HIGH GRADE AV BLOCK: may need pacer), vasodilators
- **2 Reversible Causes:** chelation therapy with deferoxamine may help hemachromatosis, steroids for sarcoidosis, scleroderma

# HTN Diastolic Dysfunction & APE

- Acute Pulmonary Edema: in SEVERE long standing Hypertension patients often due to diastolic dysfunction
- Left ventricular wall remains thick & stiff (CAN'T RELAX), and the left ventricle fills partially with fluid back up into the lungs
- Treatment: Aggressive afterload reduction with SL & IV NITRATES & 25mg SL captopril

# HYPERTROPHIC

## CARDIOMYOPATHY (IHSS)

- LEFT VENTRICULAR HYPERTROPHY WITHOUT VENTRICULAR DILATATION: Diastolic Dysfunction (dec. compliance)
- CLINICALLY: DYSPNEA ON EXERTION, SYNCOPE, CHEST PAIN, PALPITATIONS, SUDDEN DEATH, EXAM; S<sub>4</sub> (due to decreased compliance of the ventricles), SYSTOLIC EJECTION MURMUR AT APEX, DO A VALSALVA OR STANDING TEST (decrease preload) IN A YOUNG PERSON WITH EXERTIONAL CHEST PAIN (SQUATING OR HAND GRASP WILL DECREASE THE MURMUR BY INCREASING PRELOAD), valsalva: increases the murmur
- TREATMENT: BETA-BLOCKERS AS THEY PROLONG DIASTOLIC FILLING TIMES TO DECREASE OUTFLOW OBSTRUCTION
- AGENTS WHICH INCREASE CONTRACTILITY OR REDUCE VENTRICULAR VOLUME SHOULD BE AVOIDED AS THEY CAN INCREASE OUTFLOW OBSTRUCTION

# IHSS & Aortic Stenosis

- **IHSS & AS ARE BOTH AFTERLOAD DEPENDENT**
- **DECREASING AFTERLOAD IN PATIENTS WITH IHSS & AS CAN DRAMATICALLY REDUCE BLOOD PRESSURE**
- **IHSS: Normal:CO, EF, end systolic & diastolic volumes, increased LV filling pressure, decreased diastolic Relaxation**
- **Warning: Nitrates w/ AS, IHSS, RV-AMI, volume depletion & pulmonary stenosis (ie non-repaired Tetralogy): ALL CAN DECREASE BP - SHOCK**

# 3. ISCHEMIC HEART DISEASE

## 18 LEAD ECG

I      aVR      V1      V4      V7      VR3

II      aVL      V2      V5      V8      VR4

III      aVF      V3      V6      V9      VR5

**ANTERIOR LEADS: V1 - V4**

**LATERAL LEADS: I & AVL; V5-V6**

**INFERIOR LEADS: II, III & AVF**

**SEPTAL LEADS: V1-V2**

**RVLEADS: KEY LEAD**

**V4R      POSTERIOR LEADS: V7 -  
V9**

**V1-V3 ST seg depression in MI**

**LAD**

*diagonal or circumflex a.*

**RCA or circumflex a.**

*septal perf. br. LAD*

**RV branch RCA**

*post. descending a.*

# Right Ventricle AMI

- 20% of Inferior wall AMI are RV-AMI: right ventricular branch of the right coronary artery
- Dx: V4R, V5R: right sided leads - obtain if any Inferior ST, Q or T wave changes
- Be very careful with nitrates, as volume loading, ie 1-2L is often required

# Acute Coronary Syndromes In The ED

- “SOMEONE COULD WIN THE NOBEL PRIZE IN MEDICINE AND SAVE THE COUNTRY BILLIONS OF DOLLARS BY FINDING A WAY TO DIAGNOSE ACUTE MYOCARDIAL INFARCTION WITH 100% CERTAINTY”

*DAVID RILEY, M.D.*



# Myocardial Ischemia: Timeline

- 1st Diastolic dysfunction
- 2nd Systolic dysfunction
- 3rd ECG changes
- 4th Symptoms of cardiac ischemia

# ACS & Infectious Agents

- Possible links: CMV, chlamydia pneumoniae, helicobacter pylori, HSV
- Koch's 4-postulates have yet to be fulfilled:
  - 1. Pathogen must be present in nearly all cases of disease
  - 2. Pathogen must be isolated from the diseased host & grown in culture
  - 3. Disease must be reproduced when the culture is inoculated into the healthy host
  - 4. Organism must be recovered from the experimentally infected host

# CHEST PAIN-HISTORY AND RISK FACTORS MATTER MOST

*HEBERDEN'S 1789: ANGINA=CHOKING*

- CLASSIC SYMPTOMS: SUBSTERNAL CHEST PRESSURE, SOB, DIAPHORESIS, NAUSEA, PALPITATIONS, RADIATION TO MEDIAL ASPECT OF ONE OR BOTH UPPER EXTREMITIES, NECK, OR JAW, SYNCOPÉ
- RISK FACTORS: HYPERCHOLESTEROLEMIA,
- TOBACCO, HTN, DIABETES, FAMILY HISTORY-M55,W65  
HYPERURICEMIA, OBESITY, PRIOR HISTORY, POST-MENOPAUSAL WOMEN(Peripheral.VAS.DIS.), SEDENTARY, TYPE A, LVH, COCAINE USE, HYPERINSULINEMIA, HYPOTHYROIDISM, STEROID USE, MALE SEX, ADVANCED AGE, Ear lobe crease, arcus senilis < 50y/o
- ANGINA DECUBITUS: angina w/lying flat(pericarditis & GERD,pneumothorax): inc. preload, inc. MVO<sub>2</sub>-demand on the heart and chest pain

***ADMIT THE HISTORY***

# Chest Pain w/ Lying Down , Valsalva & Hand Grip

- A pearl to remember is that even myocardial ischemia can worsen with recumbency (angina decubiti) due to an increase in venous return and a temporary greater workload.
- Valsalva: Warning: causes a 45% reduction in coronary blood flow velocity=lethal dysrhythmia
- Hand Grip: increases MVO<sub>2</sub>; like an exercise test

# CHEST PAIN: WHAT CAN HELP YOU CLINICALLY

- PHYSICAL EXAM? NO!
- NORMAL ECG? NO!
- NORMAL ENZYMES? NO!
- NO CHEST PAIN? NO!

atypical presentations, ie “silent MI “  
(approximately 20% of MI’s are silent!!),  
especially in diabetics, **elderly,**  
**alcoholics**

# Non-Invasive Tests in Patients with Chest Pain

- META-ANALYSIS: NEJM; 2001; 344;1840-44

<u>Test</u>	<u>Sensitivity</u>	<u>Specificity</u>
-------------	--------------------	--------------------

● exercise ECG	68	77
----------------	----	----

● STRESS		
----------	--	--

● ECHO	<u>76</u>	88
--------	-----------	----

# ACS & Serum Markers

- CK-MB peak @ 12-18 hrs post occlusion
- Troponin I & T: peak @ 6 hrs then stay elevated for days
- Myoglobin: peak @ 1-2 hrs but back to baseline at 6-10 hrs (window only)
- Myoglobin/Carbonic anhydrase III ratio has higher sensitivities than myoglobin alone
- Troponin T/I may rise with unstable angina : **33% of the time- Elevated**

# Troponin I

- SLRHC: If LYTIC Given: only CK-MB
- ACS & NO LYTIC: ONLY Troponin I & Do Not Draw CK-MB
- TROPONIN I:
  - Normal: 0-0.4 ng/ml
  - **POSITIVE: >0.4 ng/ml**

# False Positive Troponin I



- HIV
- RENAL FAILURE
- SEPSIS
- MYOCARDITIS
- DILATED CARDIOMYOPATHY
- CNS PATHOLOGY
- SEVERE COPD

# UNSTABLE ANGINA

- **POORLY DEFINED:** crescendo angina( more severe or frequent) superimposed on chronic stable angina; angina at rest or with minimal activity, NEW ONSET ANGINA=UNSTABLE
- **25-50% OF PATIENTS WITH UNSTABLE ANGINA HAVE A PARTIALLY OCCLUDING INTRACORONARY THROMBUS WHICH MAY PROGRESS TO AMI**
- **TREATMENT:**serial cardiac enzymes, CCU-telemetry ,oxygen, anti-anginals(beta-blockers or calcium channel blockers), NO TPA/PTCA ASPIRIN( 51-72<sup>0</sup>% mortality reduction), intravenous nitrates, IbIIIa Rc inhibitors
- **heparin IV (Weight Based Protocol) Goal PTT 46-70, CHECK PTT 6 HOURS AFTER BOLUS OR CHANGES. ESPECIALLY FOR INTERMEDIATE (NEW T-WAVE INVERSION) OR HIGH RISK(ST 1 mm CHANGES)**

# Does Heparin Help?

- In Unstable Angina: prevents a partial clot from progressing to complete occlusion
- In Acute Myocardial Infarction:  
**decreases the complications** of embolic problems, ie LV aneurysm and CHF emboli

# IV Heparin

- ACLS 2000: New Protocol based on Key Study in NEJM by Judith Hochman, MD at SLRHC
- <70 kg: 60U/kg IV bolus & 12U/kg/hr drip
- >70 kg: 4000U Max bolus & 900U/hr drip
- Goal PTT: **46-70**: only check PTT 6 hours after the bolus!!!

# IV Heparin

- Range Dosing 60-70 U/kg Bolus
- 12-15 U/kg/hr Infusion
  
- Women require less Heparin:  
60 / 12
- *Diabetics require more Heparin:*  
*70 / 15*
- Elderly require less Heparin:  
60 / 12

# UNSTABLE ANGINA & LOW-MOLECULAR WEIGHT HEPARIN

- IN PATIENTS WITH UNSTABLE ANGINA, OR NON Q-WAVE AMI
- LOW-MOLECULAR WEIGHT HEPARIN (ENOXAPARIN) *BOTH IMPROVES IMPORTANT CLINICAL OUTCOMES AND SAVES MONEY* RELATIVE TO THERAPY WITH STANDARD UNFRACTIONATED HEPARIN

# **UNSTABLE ANGINA: High Risk = ST seg Depression**

- SLRHC Study: All Get IIbIIIa Inhibitors + either LMWH or Unfractionated Heparin
- **If Patient Going to Cath Lab they MUST get IIbIIIa Inhibitor: 30% Reduction in Mortality with ST segment elevation AMI**

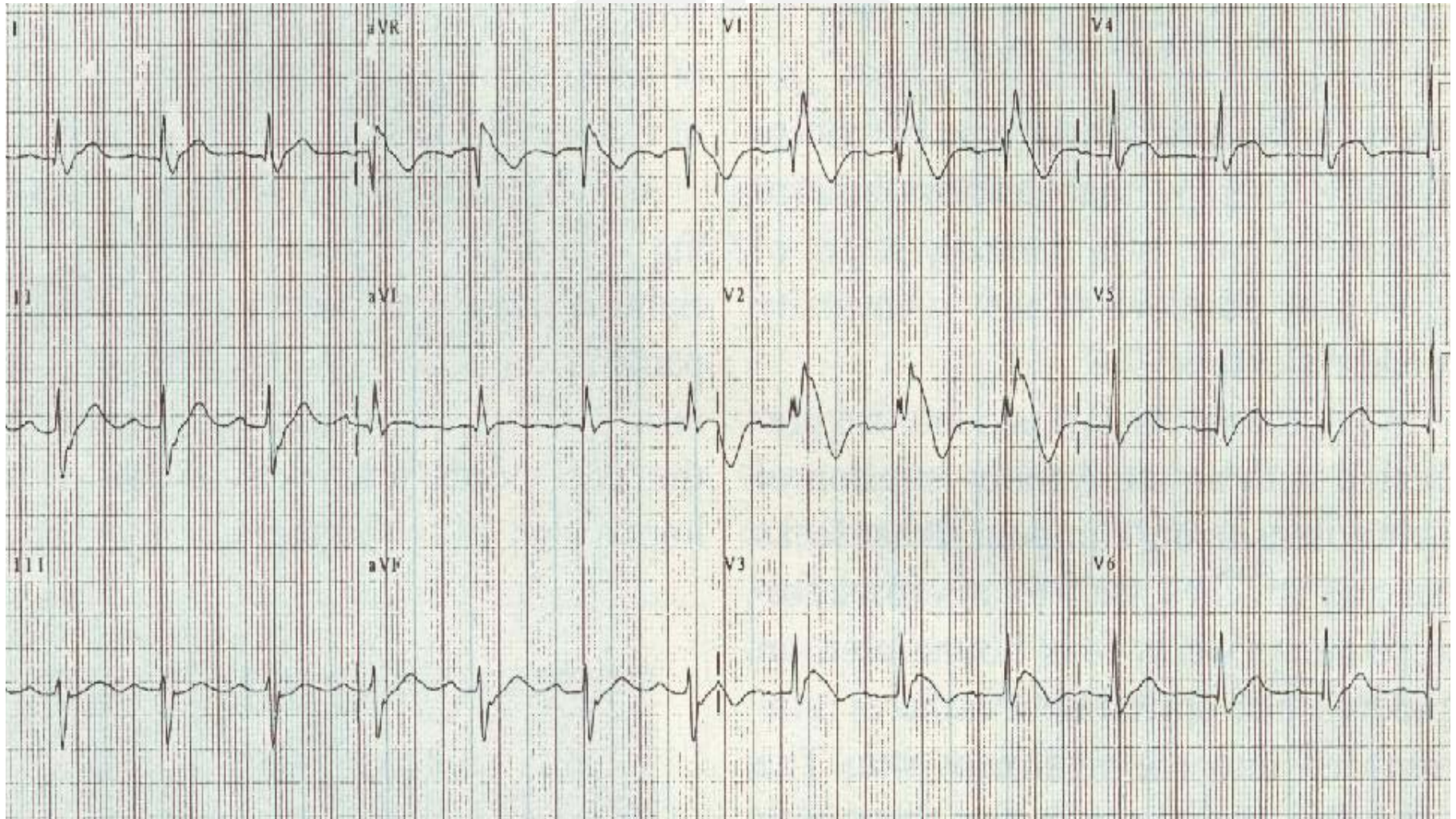
# UNSTABLE ANGINA AND INDICATIONS FOR THE CATH.

- PATIENTS WITH UNSTABLE ANGINA who carry high risk markers need angiography
- Patients with chronic stable angina who begin to fail medical therapy
- Those patients with low-risk clinical profiles and who respond to medical therapy may NOT NEED CATH & ANGIO, a noninvasive test such as a stress sestamibi-technesium, or thallium scan(cold spot:need Na/K ATPase) may suffice
- Pyrophosphate scan: hot spot=necrosis
- Patient with new systolic murmur after AMI

# Wellens' Syndrome

- Recognition of this ECG pattern associated with a critical stenosis of the proximal left anterior descending coronary artery can prevent a devastating anterior AMI
- ECG: biphasic T-waves in V2 & V3
- Rx: need CATH, and not stress test=can precipitate death

# What is the Diagnosis?



# Brugada's Syndrome

- First described at the U. of Barcelona in 1992
- Autosomal dominant with variable expression
- Potentially fatal arrhythmias
  - ventricular fibrillation
  - torsades de pointes
- Hx of syncope or aborted sudden death
- Usually become symptomatic in childhood or young adulthood

# Brugada's Syndrome

- Unique ECG findings
  - right bundle branch block
  - ST elevation in leads V1-V3
  - DDX: acute MI with RBBB
- Provocative tests in the CCU
  - iv procainamide elicits the ST elevation
- Treatment
  - automatic implantable defibrillator

# Brugada's Syndrome

Mortality due to sudden death

		# pts
AICD	0%	35
BB or CCB	26%	15
No treatment	31%	13

# B. AMI: Stop the Wavefront of Ischemia

- RECIPROCAL CHANGES ON ECG -DOWN SLOPING ST SEGMENTS IN LEADS OPPOSITE TO ST SEGMENT ELEVATION IN PATIENTS WITH AMI
- INFERIOR AMI: RECIPROCAL CHANGES IN LEADS V1-V4 (MOST RELIABLE-INF)
- ANTERIOR AMI: RECIPROCAL CHANGES IN LEADS II, III, & aVF
- POSTERIOR AMI: RECIPROCAL CHANGES IN LEADS V1, V2, V3 SEGMENT DEPRESSION + UPRIGHT T WAVES
- Reciprocal changes increase the positive predictive value for the electrocardiographic diagnosis of AMI to over 90%

# AMI AND Q-WAVES

- ABNORMAL Q WAVES ARE A COMMON FINDING EARLY IN THE COURSE OF ACUTE MYOCARDIAL INFARCTION
- THE ABSENSE OF Q WAVES DOES NOT EXCLUDE AN INFARCTION OF ANY AGE

# CHEST PAIN & LBBB

## GUSTO-I DATA

- IS THERE ST-SEG ELEVATION  $\geq 1$ mm THAT IS CONCORDANT W/QRS: odds ratio=25, LR+=9.54(3.1-17.3), Score=5
- IS THERE ST SEG DEPRESSION  $\geq 1$ mm IN LEADS V1-V3: odds ratio=6, LR+=6.58(2.6-16.1) Score=3
- IS THERE ST SEG ELEVATION  $\geq 5$ mm THAT IS DISCORDANT W/QRS: odds ratio=4.3, LR+=3.63(2-6.8), Score=2
- Accurate Dx: 90% specificity= Score 3 & >
- NOT: Prospectively validated

# **AMI & NEURAL NETWORKS**

- **MAY HAVE A FUTURE ROLE IN DX.**
- **THE NEURAL NETWORK TRAINING PROCESS CONSISTS OF THE RETROSPECTIVE SELECTION OF A LARGE NUMBER OF PATIENTS WHO ARE KNOWN OR NOT KNOWN TO HAVE HAD AN AMI. THE CASES ARE USED TO DERIVE A SET OF TRAINING PATTERNS FOR THE COMPUTER NEURAL NETWORK**

# THE PRIMARY PREVENTION OF MYACARDIAL INFARCTION: ED MD PUBLIC EDUCATIONAL ROLE

- Cessation of smoking
- Lowering the cholesterol level
- Treatment of hypertension
- Maintain a physically active life
- Avoidance of obesity
- Maintain nl. Glucose tolerance: 8hrs fasting & BS>126 = diabetes
- Postmenopausal estrogen therapy
- Mild-mod alcohol consumption
- Prophylactic low-dose aspirin

# AMI : TREATMENT

- 1. MONITOR, O<sub>2</sub>, ECG ASAP; ED MD'S GOOD AT DX AMI, IV
- **2. IF INFERIOR LEADS INVOLVED DO A RIGHT SIDED STAT ECG & CHECK V4R**
- 3. RV AMI: CAREFUL WITH NITRO & PUSH FLUIDS, STILL USE THROMBOLYSIS
- 4. Nitroglycerine 0.4mg q5' times 3 & IV start at 10ug/min if SBP>100
- 5. morphine 3mg = 25mg demerol(better in INF-AMI bec. Vagolytic)
- 6. ASPIRIN: 160-325 MG PREVENT THE PLUG
- 7. THROMBOLYTICS VS ANGIOPLASTY (PCTA) VS CABG
- 8. Beta Blockade, IV atenolol or metoprolol 5mg IV q 5 min times 3 barring any absolute contraindications; bradycardia(<60), hypotension, COPD & asthma, 2nd and 3rd deg AV B
- 9. IV HEPARIN TO PREVENT REOCCLUSION
- **10. IF ST DEPRESSION IN V1-V3  
W/UPRIGHT T'S: CHECK V7-V9:PWAMI**

# AMI : TREATMENT

- **AVOID HYPOTENSION WHEN GIVING THROMBOLYTICS**
- GUSTO III: rPA Study: Quicker Bolus
- Gusto IV: IIbIIIa (Rheopro) + 1/2 Dose rPA= Dissolve White & Red Clot with Fewer Bleeding Complications

# Acute Coronary Syndromes

- **Beta Blockers, ie metoprolol 5mg IV every 5 minutes times three (barring any contraindications) markedly reduce the morbidity and mortality of acute coronary syndromes, ie AMI, UA**
- **Goal HR = 60**

# Metoprolol Contraindications

- 1: PR interval  $>240\text{ms}$
- 2: Asthma history
- 3: CHF or Rales on auscultation
- 4: SBP  $< 95$
- 5: Heart Rate  $< 50$

# Angina: Cardiology Consults

- Sx refractory to aggressive medical Rx & ongoing chest pain
- Dynamic ECG changes
- Pulmonary edema ( PCWP >18 )
- **Cardiogenic shock: Consider Surgeon**
- New or worsening MR murmur or known or suspected critical aortic stenosis

# THROMBOLYTIC THERAPY

- “DEAD MEAT DON’T BEAT”
- GOLDEN 30 MINUTES
- INCLUSION CRITERIA(miss many MI’s); chest pain c/w AMI >20-30min, not relieved by nitroglycerine & ST seg elevations 1/2-1mm in limb leads, 2mm-precordial leads, OLD AGE OK TO LYSE
- WORRY ABOUT ASCENDING AORTIC ROOT DISSECTION: INF AMI-ECG
- Absolute Contraindications: active bleeding, puncture of a non-compressible vessel, possible aortic dissection, active intracranial malignancy, recent major surgery (<1week), acute pericarditis, allergy, SBP>180, DBP>110
- Relative Contraindications: major trauma-surgery 6-8wks, recent stroke, prolonged CPR>10minutes, hx of PUD, diabetic retinopathy, severe HTN, severe liver disease, pregnancy, cancer, hx bleeding diathesis, SBP>160, DBP>100

# THROMBOLYTIC REGIMENS

- STREPTOKINASE: 1.5 MILLION UNITS OVER 1 HOUR IV \$
- APSAC(anisoylated plasminogen-strepto activator complex): 30 UNITS IV OVER 2-5 MINUTES \$\$\$\$\$
- TISSUE PLASMINOGEN ACTIVATOR: 15 MG BOLUS IV, .75MG/KG OVER 30 MIN UP TO 50 MG, THEN .5MG/KG UP TO 35 MG OVER THE NEXT 1 HR (+HEPARIN) \$\$\$\$
- RETEPLASE (rPA): 10.8 units IV over 2', repeat in 30' \$\$\$\$
- TNKase: 0.5mg/kg Single 5 second bolus: maximum of 50mg; 70kg man = 35mg \$\$\$\$\$

# THROMBOLYTIC MULTICENTER TRIALS

	<u>GISSI-2</u>	<u>ISIS-3</u>	<u>GUSTO-1</u>
<i>YEAR</i>	1990	1992	1993
<i># PATIENTS</i>	12,490	39,913	41,021
<i>Mortality t-PA</i>	9.0%	10.3%	<b>6.3%</b>
<i>STK</i>	8.6%	10.6%	<b>7.3%</b>
<i>Hemorrhage stroke</i>			
<i>t-PA</i>	0.3%	0.66%	<b>0.7%</b>
<i>STK</i>	0.25%	0.24%	<b>0.5%</b>
<i>Heparin</i>	SQ	SQ	<i>IV with t-PA</i>

# GUSTSO STUDY: SOME FACTS & CRITICISMS

- ABSOLUTE REDUCTION IN MORTALITY OF 1%, YET 14% SURVIVAL BENEFIT with t-PA, also, t-PA will reduce CHF, cardiogenic shock, malignant ventricular arrhythmia, and provides a better ejection fraction
- **WARNING: STREPTOKINASE + IV HEPARIN**, caused more hemorrhagic stroke than STK without heparin & adding IV heparin to STK did not improve efficacy
- Elderly Patients: >75y/o did as well with streptokinase and were more prone to stroke, which occurred more frequently with t-PA
- TPA COST 10 TIMES MORE THAN STK

# **RECOMMENDATIONS BASED ON GUSTO**

- **FRONT LOADED TPA & IV HEPARIN:  
BEST FOR YOUNGER PATIENTS  
WITH ANTERIOR OR LARGE  
INFERIOR WALL MI W/I 4-6HRS OF  
PAIN ONSET**
- **STREPTOKINASE : ELDERLY:>70  
PATIENTS, THOSE WITH SMALL  
INFERIOR MI OR IF THERAPY IS  
APPLIED >6HRS (TO AVOID THE  
SMALL TPA INCR. STROKE RISK)**

# Adjunctive Therapies Used to Prevent REOCCLUSION

- 1. ANTIPLATELET THERAPY WITH ASPIRIN IS ESSENTIAL, Ticlid(TTP worry), or Plavix if ASA intolerance
- 2. IV HEPARIN ( accelerates the formation of antithrombin III complexes which inhibit thrombin) IS USEFUL ESPECIALLY WITH TPA
- IIBIIIA receptor platelet aggregation inhibitors have a definite role

# THROMBOLYSIS COMPLICATIONS

- ALLERGIC RXNS ESPECIALLY WITH **STREPTOKINASE: HYPOTENSION: RX W/IVF & DO NOT STOP THE THROMBOLYTIC!**
- BLEEDING: MOST AT SITE OF AN INVASIVE PROCEDURE
- INTRACRANIAL BLEEDS <1%
- HOW TO REVERSE THROMBOLYSIS: **stop all meds;** reverse heparin with **protamine 1mg/100U heparin** not to exceed 50mg in 10 min; cryoprecipitate and fresh frozen plasma, espec. if the fibrinogen level is <100mg/dl; platelets can be given for prolong. bleeding t.; **e-amino caproic acid**, that prevents the binding of t-PA and plasmin to fibrin

# Thrombolytic Reversal Drugs

- Protamine 1mg/100U heparin:  
WARNING: protamine given alone is a blood thinner
- e-amino caproic acid: blocks plasminogen to plasmin(lyses fibrin), thus more fibrin is present; especially good for oral bleeding in VWD, and factor 8 &9 deficiencies

# POST MI MORBIDITY PREDICTORS: OPEN CORONARY ARTERY GIVES BEST PROGNOSIS

- RV AMI IN INF AMI: IS AN INDEPENDENT PREDICTOR OR POOR PROGNOSIS (UNLESS ANGIOPLASTY IS DONE: THEN GOOD PROGNOSIS), COMPLICATIONS, IN-HOSPITAL MORTALITY, ALWAYS CHECK V4R W/INF
- HOLTER MONITORING: LOW HEART RATE VARIABILITY ASSOCIATED WITH INCREASED RISK OF SUDDEN CARDIAC DEATH
- SIGNAL AVERAGED ELECTROCARDIOGRAPHY: ( LATE POTENTIALS (TERMINAL 40ms) AT THE END OF THE QRS); 70-90% OF PATIENTS WITH LATE POTENTIALS HAVE HAD SUSTAINED AND INDUCIBLE VENTRICULAR TACHYCARDIA AFTER AMI
- SUCCESSFUL THROMBOLYSIS MINIMIZES BOTH LOW HEART RATE VARIABILITY, AND POSITIVE SIGNAL AVERAGED ELECTROCARDIOGRAPHY LATE POTENTIALS

# INDICATIONS FOR ANGIOPLASTY - PTCA

- CONTRAINDICATIONS TO THROMBOLYSIS
- FAILED LYTIC THERAPY
- HIGH RISK PATIENTS: MASSIVE ANTERIOR AMI,  
PERSISTENT SINUS TACHYCARDIA,  
CARDIOGENIC SHOCK
- NO ROLE FOR PTCA IN PATIENTS WHO ARE SUCCESSFUL WITH THROMBOLYSIS
- PTCA IF DONE W/I 60 MIN. APPEARS TO HAVE BETTER PROGNOSIS THAN LYTICS

# C. CARDIOGENIC SHOCK

- Impairment of >25% myocardium - CHF
- Impairment of >40% myocardium(ie with AMI) - cardiogenic shock (CI<2.2, PCWP>18, SVR>2000, MAP<80)
- Other causes: RV AMI; rupture of necrotic free wall(instant PEA), septal or papillary muscle(30' hypotension)(inf wall, acute mitral regurgitation-only definitive **tx is surgical**, only if intraortic balloon pumping and vigorous vasodilator and inotropic therapy is available); pericardial tamponade
- Best Treatment is PREVENTION with reperfusion
- Rx: initial fluid challenge, may need dobutamine vs dopamine vs IABP(CONTRAINED. IN AI, Balloon is up in Diastole to allow better diastolic coronary & ventricle filling & perfusion) & surgery

# SHOCK: Bedside Diagnosis

- 1st look at SKIN:  
warm, flushed = distributive shock,  
mottled = low vol., obstructive, cardiogenic
- 2nd look at NECK VEINS: if flat = low vol.,  
if distended = obstructive, cardiogenic
- 3rd listen to LUNG SOUNDS: if  
decreased on one side = tension pneumo, if  
rales = cardiogenic shock, if  
clear = obstructive shock (PE, RV-AMI, air  
embolus)

# Shock: When to TUBE?

- Hypoxia + Hypotension = Tube
- AMI + Hypotension = Tube
- PE + Hypotension = Tube
- Delta MS + Hypotension = Tube

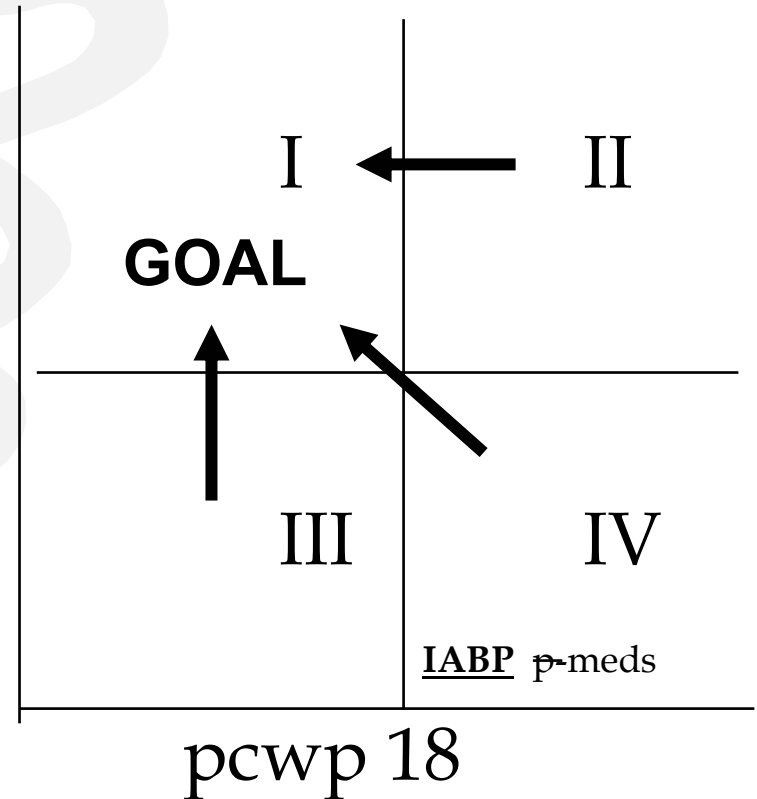
# FORRESTER, DIAMOND, CHATTERJEE: AMI AND HEART FAILURE - CARDIAC INDEX & PCWP

**I:** no pulmonary congestion  
no hypoperfusion. **GOAL RX**

**II:** pulm. congestion  
Rx: lasix, ACEI's  
**CI 2.2**

**III:** hypoperfusion, RV-AMI, JVD  
Rx: normal saline, dobutamine

**IV:** pulm. congest. & hypoperfusion  
Rx: dobutamine 3mcg/kg/min & dopamine 6mcg/kg/min, lasix,  
ACEI, IABP & Cardiac Surgery



# Forrester Diamond Boxes at the Bedside

- Distributive shock=box I
- Obstructive shock=box III
- Hypovolemic shock=box III
- Cardiogenic shock=box III & IV
- CHF + Pulmonary Edema=box II
- Cardiac Index  $<2.2$ : If pulse pressure is  $<25\%$  of SBP (91% accuracy)
- PCWP  $>18$ : bibasilar rales & CHF on CXR,  $\pm$  abdominojugular reflux is 95% specific

# Air Embolism & Obstructive Shock: Box III

- Need 5 cc/kg = 350cc of air in the circulation to cause shock
- Causes: central lines, especially subclavian & IJ; penetrating and severe blunt trauma; blast injuries (explosion, lightning); drink H<sub>2</sub>O<sub>2</sub>-OD
- Rx: HBO, Left lat. Decubitus position so air is localized in RV & w/central catheter, potentially aspirated

# Forrester Diamond Boxes & Mortality

- **Box I = 2%**
- **Box II = 10%**
- **Box III = 22%**
- **Box IV = 55%**

# AMI: OTHER COMPLICATIONS

- **DYSRHYTHMIAS: BRADYCARDIA - INFERIOR AMI (AVOID LIDOCAINE, USE ATROPINE .5MG IV, Demerol is vagolytic & is better than MS ); PVCs, OR POST VF THEN USE LIDOCAINE 1.5MG/KG IV BOLUS & 2-4MG/MIN**
- **CONDUCTION DISTURBANCES:AV BLOCKS; 1st deg AV & Mobitz I 2nd deg AV block usually INF AMI and respond to atropine. However, Mobitz II 2nd deg AV block or 3rd deg AV block are assoc. with ANT. wall AMI, are more severe and need pacemaker therapy; new bifascicular blocks with AMI need pacing therapy also**
- **Ventricular Aneurysm: noncontractile outpouching of the left ventricle - may rupture. ULTRASOUND MAY DX.**

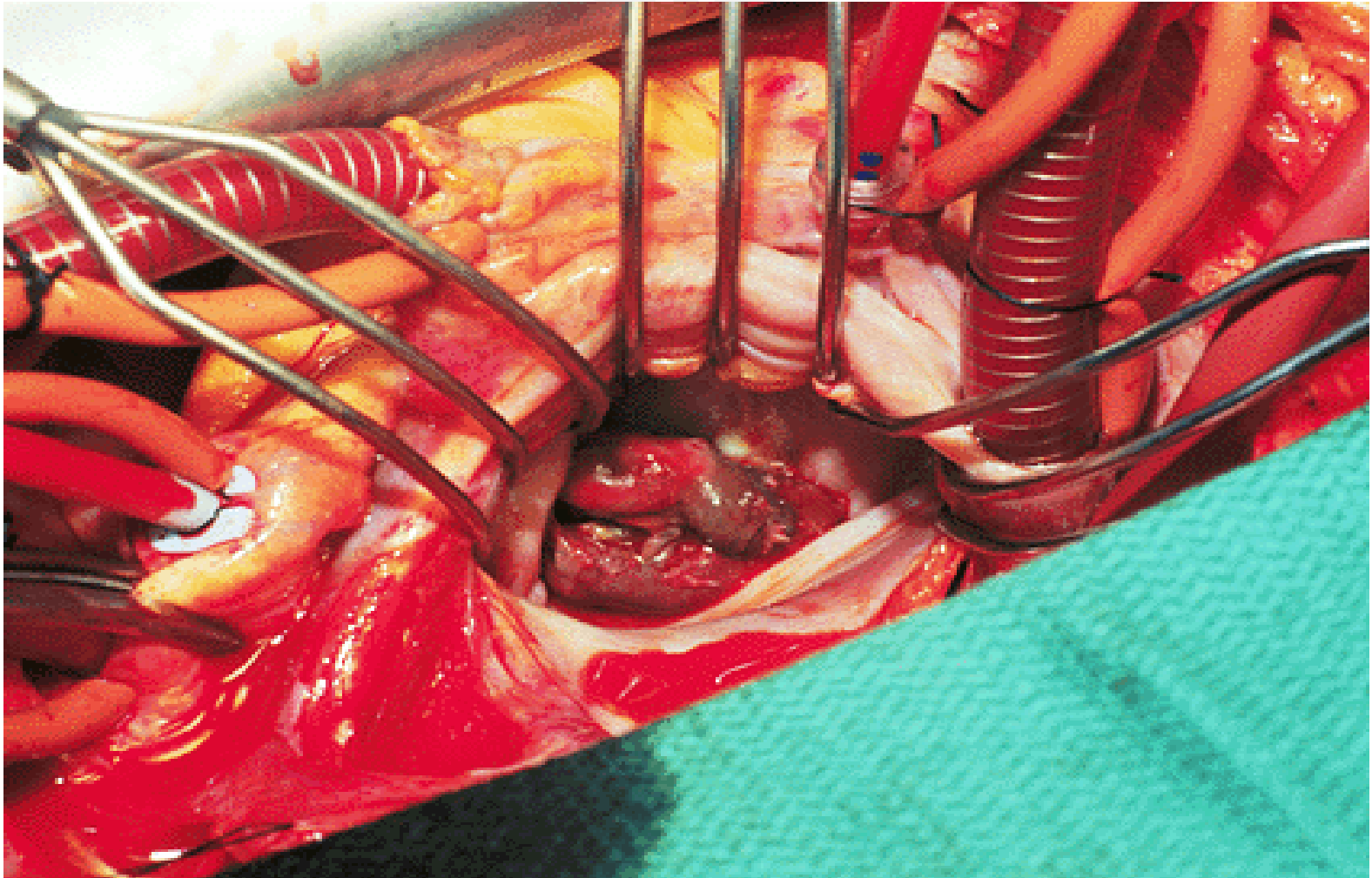
# POST AMI PERICARDITIS

- **1. EARLY PERICARDITIS 24-72 HRS AFTER AMI (NECROSIS-MECH.): Usually with TRANSMURAL, Q-wave AMI**
- **2. LATE PERICARDITIS (DRESSLER SYNDROME-autoimmune) 1-12 WEEKS POST AMI: CHARACTERIZED BY FEVER, PERSISTANT AND RECURRING PERICARDITIS; RX WITH NSAIDS AND CORTICOSTEROIDS**
- **DX: Sed Rate Usually Elevated**

# 4. INFECTIVE ENDOCARDITIS (IE)

- **RISK FACTORS:** IVDA, mitral valve prolapse, rheumatic heart disease, congenital heart disease ( patent ductus arteriosus, VSD, bicuspid aortic valve, coarctation of the aorta ), **ALL CONGENITAL HEART DISEASE**
- **MITRAL**>>**AORTIC**>**TRICUSPID**>**PULMONIC**
- **MYCOTIC ANEURYSM (infected piece of vegetation seeds a distant vessel producing aneurysm): OSLER 1855**
- **ORGANISMS:** non-IVDA-strep. Viridans & other HACEK oral flora; IVDA-staph. aureus; prosthetic valves- strep. Epidermidis; fungal; **Cancer & marantic endocarditis**
- **DIAGNOSIS:** blood cultures, fever, risk factors, **\*ECHOCARDIOGRAPHY(espec. transesophageal)**
- **Indications for Surgery:** acute valvular dysfunction, myocardial invasion, antibiotic resistant org. and persistent sepsis, intractable CHF, nonfatal emboli

# **Infective Endocarditis + Shock = Call Cardiac Surgeon**



# INFECTIVE

## ENDOCARDITIS

- Intravenous Drug Users: worry about **staph. aureus**, gram negative ie pseudomonas & serratia & fungi; the **tricuspid valve** (with septic emboli and recurring pneumonias)
- Empirical Treatment: native valves - **vancomycin & gentamycin in IVDA; in non IVDA - nafcillin & gent.; in prosthetic valve IE - ampicillin, vanco, & gentamycin**
- Complications of IE: heart failure(espec if aortic v: **CALL CARDIAC SURGEON-STAT.**), embolization, neurologic deficits, mycotic aneurysm, renal failure

# INFECTIVE ENDOCARDITIS

- STROKE + ENDOCARDITIS =  
ABSOLUTE TPA  
CONTRAININDICATION
- DUE TO THE POSSIBILITY OF A  
CEREBRAL MYCOTIC ANEURYSM
- Tricuspid vegetation through a PFO  
(cryptogenic stroke)

# INFECTIVE ENDOCARDITIS PREVENTION

- PRIMARY DENTAL AGENT IS NOW AMOXICILLIN AND NOT PENICILLIN 1990  
AHA CHANGE: 2 DOSES
- AMOXICILLIN 3.0 GM PO 1 HOUR BEFORE THE PROCEDURE AND 1.5 GM 6 HOURS AFTER THE INITIAL DOSE ( ERYTHROMYCIN OR CLINDAMYCIN IF ALLERGIC TO PCN)
- Add clinda/flagyl if anaerobe worry, and gent/tobra if gram negative worry

# 5. VALVULAR HEART DISEASE: PE

to Dx, ECHO=extent

- A. STENOSIS: MORE CRITICAL DISEASE
- B. REGURGITATION: MORE BENIGN NATURAL HISTORY

**MITRAL STENOSIS=**

**POST-STREP RHEUM**

**FEVER(95% aortic also): Dx-**

**12y/o, Sx-31y/o, 38y/o die**

**DX: HX(new AF in a young**

**person) & DIAST MURMUR, ECHO.**

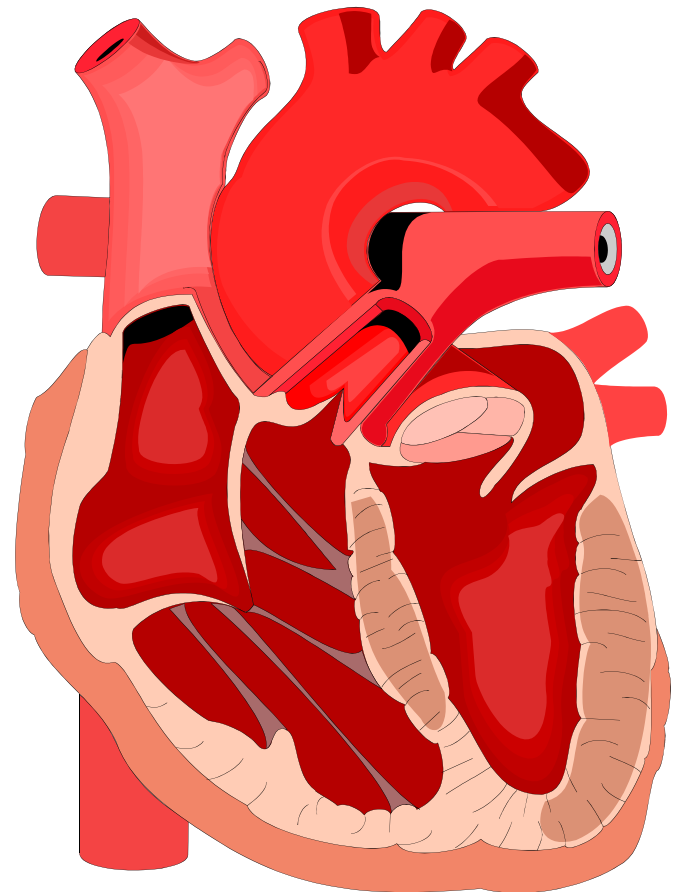
**RX: BEST PREVENTION**

**IF NOT PREVENTED, THE**

**ONLY OPTIONS ARE**

**SURGICAL; IE BALLOON**

**VALVULOPLASTY**



# MITRAL STENOSIS

- ACUTE PULMONARY EDEMA AND PREGNANCY (inc. CO)  
THINK MITRAL STENOSIS:  
latent rheumatic disease; P-mitrale (ant-lat leads)

# A. AORTIC REGURGITATION

- **AR: CAUSES; #1 A-AR ENDOCARDITIS, dissection, penetrating trauma ,#1 C-AR LONG STANDING HTN, INFLAMMATION(RHEUM, SYPHILIS), CONNECTIVE TISSUE ( MARFAN'S, EHLERS-DANLOS), CONGENITAL, TORN CUSP(TRAUMA, DISSECTING ANEURYSM, INF. ENDOCARDITIS)**
- **AR: DIAGNOSIS; DIASTOLIC MURMUR: Best Heard leaning forward on inspiration, WIDE PULSE pressure(chronic: QUINCKE'S pulses-visible pulsations in the nail beds), AUSTIN FLINT MURMUR (FUNCTIONAL MS MURMUR DUE TO REGURGITANT JET HITTING MITRAL VALVE)**
- **AR: TREATMENT; MAJOR ISSUE IS THE TIMING OF SURGERY, AFTERLOAD REDUCTION THERAPY WITH ACE INHIBITORS MAY BUY TIME, To inc. Forward flow**
- **NO BALLOON PUMP: IE BOX IV=SURGERY ONLY**

# AORTIC STENOSIS (AS)

- COMMON; WE ALL DEVELOP AS WITH ADVANCED AGING, RHEUMATIC CAUSE ALSO, AND CONGENITAL-BICUSPID V. CAUSES(#1<60)
- EXAM: CAROTID PARVUS(WEAK) TARDUS(SLOW):Neck Radiation
- CLASSIC TRIAD OF SX: DYSPNEA, SYNCOPE, AND ANGINA(Due to  $MVo_2 > \text{supply}$ ); Decr. BP
- DX: LVH, FORCEFUL APICAL PULSE, CHF, SYSTOLIC MURMUR ,ENLARGED LEFT ATRIUM; BEST TEST IS DOPPLER ECHOCARDIOGRAPHY
- RX: PATIENTS WITH MILD AS (valve area  $>1.0\text{cm}^2$ ) HAVE AN EXCELLENT PROGNOSIS, THOSE WITH SYMPTOMS, OR ASYMPTOMATIC WITH A valve area  $<0.8\text{cm}^2$ , NEED AORTIC VALVE REPLACEMENT
- After load Dependent Lesion: can't tol. Decreased SBP

# Split Second Heart Sound

- **Increased splitting of S2:** RBBB, Atrial septal defect
- **Paradoxical splitting of S2:**  
pulmonic valve closing before aortic:  
LBBB, Aortic stenosis w/ slowly moving aortic valves

# B. MITRAL REGURGITATION

- MR: CAUSES, RHUEM, SLE, INF.  
ENDOCARDITIS & MITRAL VALVE PROLAPSE,  
ANY LV DILATATION(CHF)
- MR: DX, LOUD HOLOSYSTOLIC APEX  
MURMUR , BIG V-WAVE ON ATRIAL PRESSURE  
CURVE
- MR: RX, MOST PRESENT ILL AND NEED  
SURGERY, YET BENIGN NATURAL HISTORY

# MITRAL V. PROLAPSE

- MVP: MOST COMMON VALVULAR DISEASE  
5% OF POP, AUTOSOMAL DOMINANT, RISKS  
(ENDOCARDITIS, MR, SUDDEN DEATH, SVT'S,  
EMBOLI) , **OVER DIAGNOSED WITH ECHO**,  
PROLONGED QTc, Inferior: Flipped T's, II, III &  
aVF
- MVP: SX, MOST ASSYMPTOMATIC, CHEST  
PAIN WITH EXERTION, **MID-SYSTOLIC  
CLICK(chordae tendinae checks its  
prolapse into the atrium)**, palpitat.
- MVP: RX, no Sx=no Rx & close f/u, BETA  
BLOCKERS if palpitations, antibiotic prophylaxis  
for dental or urinary tr. Procedure, NTG DOES  
NOT RELIEVE CHEST PAIN

## 6. MYOCARDITIS: Admit All

- CAUSES: bacterial (diphtheria, lyme, strep, **mycoplasma**, meningococcus), viral (coxsackie B, echo, influenza, **HIV, hepatitis**), protozoal (**Chaga's (trypanosoma cruzi: peripheral blood smear) disease, toxoplasmosis**), radiation, chemicals, adriamycin; **ELEVATED ESR**
- SX: CP, **new onset CHF**, TACHYCARDIA
- TREATMENT: SUPPORTIVE, ANTIBIOTICS, STEROIDS FOR VIRAL, REST; IF CHF IS PRESENT, USE CARE AS THE INFLAMED MYOCARDIUM IS SENSITIVE TO DIGOXIN, **NO NSAIDS**

# DISEASES OF THE PERICARDIUM

- 1. PERICARDITIS
- 2. PERICARDIAL EFFUSION & TAMPONADE

# 1. PERICARDITIS: Admit All

- CAUSES: trauma, uremia, AMI(acute and delayed - Dressler's), meds(hydralazine, procainamide), infections(bacterial, fungal, TB), rheum, radiation, tumors
- CLINICAL: SHARP PRECORDIAL CP MADE WORSE WITH LAYING DOWN, AND RELIEVED BY SITTING UP; PERICARDIAL SYS FRICTION RUB(TRIPHASIC:2D,1S).
- DX: ECHOCARDIOGRAPHY #1; ECG ST ELEVATE, PR INTERVAL DEPRESSION, aVr-elevation, ST/T>0.25
- PR seg depression = atrial current of injury
- RX: THE UNDERLYING DISORDER, NSAIDS, CORTICOSTEROIDS; SOME PATIENTS MAY NEED HOSPITALIZATION TO OBSERVE FOR THE DEVELOPMENT OF TAMPONADE OR CONSTRICTIVE COMPLICATIONS(pericardial knock, Kussmaul's sign=JVD w/inspiration, big y-descent=passive atrial emptying, Rx.=surgery)

# PERICARDITIS ECG CHANGES

- STAGE I: DIFFUSE ST ELEVATION (NONANATOMIC DIST.), PR SEG DEPRESSION INFERIORLY
- STAGE II: ST NORMALIZES, T WAVE DECREASED BUT NOT INVERTED
- STAGE III: T WAVE INVERSION
- STAGE IV: ECG NORMALIZED
- NOTE: SHOULD NEVER SEE ST ELEVATION AND FLIPPED T'S AT SAME TIME

## 2. PERICARDIAL EFFUSION / TAMPONADE

- CLASSIC CLINICAL FINDINGS: BECK'S TRIAD- HYPOTENSION, JVD, DISTANT HEART TONES, NARROW PULSE PRESSURE, TACHYCARDIA, TACHYPNEA, **PULSUS PARADOXUS**, CXR-CARDIOMEGALLY, LOW QRS VOLTAGE & ELECTRICAL ALTERNANS ON ECG;  
**ECHOCARDIOGRAM IS BEST**: LOOK FOR DIASTOLIC COLLAPSE OF THE **RIGHT ATRIUM FIRST**, THEN RIGHT VENTRICLE
- DIFF DX OF HYPOTENSION AND JVD: TENSION PNEUMOTHORAX, PE, SVC OBSTRUCTION-SYNDROME
- TREATMENT: O2 5-10L/MIN; 2 LARGE BORE IV-NS AND **AGGRESSIVE VOLUME RESUSITATION**; CARDIAC MONITOR; PERICARDIOCENTESIS, OR PERICARDIAL WINDOW, **PENETRATING TRAUMA: THORACOTOMY**
- Atrial pressure tracing: big X-descent= equalization of all 4 chambers in diastole w/**active** atrial emptying; Y-descent =passive atrial emptying

# Ultrasound & Tamponade

- IVC Plethora: dilated IVC without inspiratory collapse on subcostal ultrasound: suggests tamponade
- Diastolic collapse of the right ventricle = impending hypotensive tamponade

# PULSUS PARADOXUS

- DEFINED: MORE THAN 10mmHG DROP IN SYSTOLIC BP DURING INSPIRATION
- SEEN IN: TAMPONADE, COPD, BRONCHIAL ASTHMA, CONSTRICTIVE PERICARDITIS, RV-AMI
- MECHANISM: DECREASED LV STROKE OUTPUT: SYSTEMIC VEINS (UNLIKE THE PULMONARY VEINS) ARE EXTRATHORACIC, THUS THERE IS INCREASED RV FILLING DURING INSPIRATION, SHIFTING THE INTERVENTRICULAR SEPTUM TOWARDS THE LV RESULTING IN IMPAIRED LV FILLING, AND DECREASED LV STROKE OUTPUT.

# The Chubby Blue 7day old Boy

- Hx: 9lb 2oz boy, 7days old, discharged from hospital on day 2 w/no problems during pregnancy/labor/delivery. Child was feeding every 3 hrs on d/c
- Mother brings chubby boy to the ED stating that he can't feed for more than 2-3 minutes when he turns blue, is irritable, starts crying and is more blue, is mildly SOB, and is sweating. No N/V/D, or fevers.

# The Chubby Blue 7day old Boy

- PE: Irritated blue appearing chubby boy, HR 125, RR 45, pulse ox 91% RA, Tr-99
- HEENT: blue tongue, no lid edema
- Lungs: clear
- Cardiac: RRR, S1 & loud S2 + systolic ejection murmur heard maximally in the pulmonic area radiating to the back
- Pulses & BP are equal in all 4 extremities

# The Chubby Blue 7day old Boy

- PE: Abdomen: mild hepatomegaly, non-tender, stool heme neg
- Neuro: moving all 4's well, irritable
- Lab: Hct 50, pO<sub>2</sub> 65, pCO<sub>2</sub> 35
- lytes=nl, CO<sub>2</sub>-20
- CXR: decreased pulmonary markings
- ECG: RVH

# The Chubby Blue 7day old Boy

- Differential Dx.?
- Additional w / u?
- Treatment in the ED?

# Peds Cyanosis & Tachypnea

- Hyperoxia test: 100% for 10 min O<sub>2</sub> if pO<sub>2</sub> increases >20torr most likely a pulmonary cause; if <20 most likely cardiac
- Tachypnea:
  - pulmonary(O<sub>2</sub> improves): structural, infection
  - metabolic(normal oxygenation room air): inborn errors of metabolism, dehydration, sepsis
  - cardiac(oxygen does not improve): acquired, congenital

# PINK BABIES

- LEFT TO RIGHT SHUNT
- ATRIAL SEPTAL DEFECT
- VENTRICULAR SEPTAL DEFECT
- PATENT DUCTUS ARTERIOSUS (PGE1 used to keep open & indomethacin used to close)
- CAN RESULT IN VOLUME OVERLOAD W/RV HYPERTROPHY (PULM HTN) AND SHUNT REVERSAL: EISENMENGER'S SYNDROME
- RX: DEPENDS CLINICAL SUSPICION: lasix 1mg/kg IV for preload reduction, digoxin/ dopamine/ dobutamine, & nifedipine: consult PEDIATRIC CARDIOLOGIST

# GRAY BABIES: LV OUTFLOW OBSTRUCTION

- LV: HYPOPLASTIC LEFT HEART SYNDROME
- LV: COARCTATION OF THE AORTA (CHECK BP IN ALL FOUR EXTREMITIES)
- RV: pulmonic stenosis
- FIRST WEEK OF LIFE SYNDROMES
- CAN DETERIORATE INTO CYANOSIS, ACIDOSIS AND SHOCK
- **DEFINITE CANDIDATE FOR PGE1 INFUSION (REMEMBER ELECTIVE INTUBATION)**
- CALL THE PEDIATRIC CARDIOLOGIST

# BLUE BABIES: R TO L

## SHUNT

- TRANSPOSITION OF THE GREAT VESSELS
- TRICUSPID ATRESIA (EBSTEIN'S ANOMALY)
- TOTAL ANOMALOUS PULMONARY VENOUS RETURN
- ABOVE 3: CYANOSIS W/I FIRST WEEK
- TRUNKUS ARTERIOSUS
- SEVERE PULMONARY STENOSIS W/INTACT SEPTUM OR VSD
- TETRALOGY OF FALLOT: VSD, PULM. STENOSIS, RIGHT VH, OVERRIDING AORTA
- CONSULT PEDIATRIC CARDIOLOGIST AS PATIENTS MAY NEED PGE1 INFUSION, AND RX FOR HYPER TET SPELL

# Tetralogy of Fallot

- Mixture of systemic venous blood into the systemic arterial supply: air & bacteria in blood is not filtered by the lung bed thus air filters should be used on IV lines
- Mixed blood must get back to the pulmonary system: yet anything that raises pulmonary resistance or lowers systemic resistance increases the R to L shunt & cyanosis

# **HYPER TET SPELLS: TOF**

- **Basic problem is increased r to l shunting w/decreased pulmonary perfusion: increased pulmonary vas. Resistance (resp. Illness), or decreased systemic vascular resistance (hypotension, fever, dehydration) can trigger this.**
- **The child hyperventilates which increases systemic venous return(negative thoracic pump) which returns more flow into a restricted rv outflow tract resulting in more r-l shunt worsening cyanosis and a vicious cycle that must be broken.**

# **HYPER TET SPELLS: TOF**

- **PLACE INFANT IN KNEE-CHEST(SQUAT POSITION): INC. PRELOAD = GREATER RV FILLING**
- **MORPHINE SULFATE 0.2mg/kg/dose TO DECREASE HYPERVENTILATION**
- **PROPRANOLOL 0.1 mg/kg IV TO ALLOW GREATER RV FILLING**
- **INCREASE AFTERLOAD: IVF, PHENYLEPHRINE, KETAMINE**
- **CALL THE PEDIATRIC CARDIOLOGIST**

# PROSTAGLANDIN E1 INFUSION TO MAINTAIN PDA PATENCY

- PEDIATRIC CYANOSIS/SHOCK IN 1ST 4 WEEKS OF LIFE: PGE1 Useful When:
  - PDA PROVIDES ALL OR PORTIONS OF THE PULMONARY BLOOD FLOW: PULMONARY ATRESIA
  - PDA PROVIDES ALL OR PORTIONS OF THE SYSTEMIC BLOOD FLOW: HYPOPLASTIC LEFT HEART, OR CRITICAL AORTIC COARCTATION
  - PDA SERVES AS A SITE OF MIXING FOR POORLY OXYGENATED SYSTEMIC BLOOD W/OXYGENATED BLOOD:TRANSPOSITION W/INTACT VENT. SEPTUM
- DOSE: 0.05-0.1 MCG/KG/MIN
- WARNING: PGE1 CAN CAUSE RESPIRATORY DEPRESSION: APNEA (ELECTIVE INTUBATION), HYPOTENSION, SEISURES, AND HYPERTHERMIA

# RECOGNITION OF CHF IN INFANTS

## ● CARDINAL SIGNS

- RIGHT SIDED FAILURE: HEPATOMEGALY, LID EDEMA(DDx:periorb cellulitis, nephrotic synd, dermatomyositis) (JVD AND EXTREMITY EDEMA RARE)
- LEFT SIDED FAILURE: TACHYPNEA, DYSPNEA, SWEATING ON FEEDING(stress test for babies),  
RALES
- BOTH: CARDIOMEGALY, FAILURE TO THRIVE, TACHYCARDIA

# PEDIATRIC SHOCK: EARLY

- COMPENSATED = EARLY: Autoregulation is working
  - INCREASED HEART RATE
  - NORMAL BLOOD PRESSURE
  - PALE SKIN W/PROLONGED CAPILLARY REFILL >2-3 SECONDS
  - PINK & WARM SKIN(SEPTIC SHOCK)
  - URINE OUTPUT LOW (NL 1-2cc/kg/hr)
  - RESPIRATORY ALKALOSIS
  - RESTLESS BEHAVIOR
- 25% OF BLOOD VOL. LOST: NORMAL BLOOD VOLUME OF CHILDREN= 80-90 cc/kg

# PEDIATRIC SHOCK: LATE

- DECOMPENSATED SHOCK: lactate production, and autoregulation can't keep tissues perfused
  - HR INCREASED MARKEDLY
  - BP NL TO SLIGHTLY LOW ( $SPB=70+2*AGE(yrs)$ )-  
low 5%ile: 90=50%ile)
  - PULSE PRESSURE DECREASED (DIASTOLIC RISING=HYPOVOLEMIA w/compens. sympathetic tone)
  - SKIN MOTTLED W/PROLONGED CAPILLARY REFILL
  - METABOLIC ACIDOSIS/ MINIMAL URINE
  - PERSISTANT IRRITABILITY/LETHARGY
  - SIGNS OF INCREASED CAPILLARY LEAK
- Very Difficult to get out of this state

# PEDIATRIC ASYSTOLE AND PULSELESS ARREST

- ABC'S- VF/VT OR ASYSTOLE OR PEA?
- RX: VF/VT: DEFIB 2J/KG - 4J/KG - 4J/KG
- USE ADULT PADDLE IF >10KG CHILD
  - EPI 1ST DOSE .2MG/KG (1:10,000)
  - LIDOCAINE 1MG/KG
  - DEFIB 4J/KG - EPI 2ND & SUBSEQ. DOSES HIGH .1-.2MG/KG IV/IO Q3-5MIN
  - BRETILUM 5MG/KG THEN 10MG/KG
- RX: ASYSTOLE: EPI .01MG/KG 1ST THEN HIGH DOSE Q3-5MIN
- RX: PEA: ABOVE + TREAT UNDERLYING CAUSE, IE IVF, O2, HEAT, PERICARDIOCENTESIS

# PEDIATRIC SEPTIC SHOCK

- COMPLICATED: COMBINATION OF HYPOVOLEMIC , CARDIOGENIC (MYOCARDIAL DEPRESSANT FACTOR) & DISTRIBUTIVE
- RX: AGRESSIVE NS VOLUME IN 20 CC/KG BOLUSES W/ EXCESS OF 40 CC/KG IN THE 1ST HOUR IN ED = IMPROVED SURVIVAL, DECREASED OCCURRENCE OF PERSISTENT HYPOVOLEMIA & NO INCREASE IN THE RISK FOR PULM. EDEMA/ARDS
- RX: BROAD SPECTRUM ANTIBIOTICS/PRESSORS

# NORMAL NEWBORN ECG

- RELATIVELY SMALL QRS VOLTAGES IN LIMB LEADS
- T WAVES ARE OF LOW VOLTAGE
- RIGHT VENTRICULAR DOMINANCE IN R PRECORDIAL LEADS
- RIGHT AXIS UP TO +180 DEGREES
- At Birth flipped T waves: UPRIGHT T WAVES IN THE RIGHT PRECORDIAL LEADS IN THE FIRST WEEK OF LIFE

# PREMATURE INFANTS

## ECG

- LOWER VOLTAGES OF QRS & T WAVES IN LIMB LEADS
- LESS RIGHT VENTRICAL DOMINANCE AND MORE LEFTWARD FORCES:  
**MORE LEFT-AXIS**
- **RELATIVELY SHORTER PR, QRS, & QT INTERVALS**
- MORE VARIABILITY COMPARED TO TRACINGS IN TERM NEWBORNS

# **PEDIATRIC ECG: T WAVE**

- **T WAVES ARE NORMALLY INVERTED IN THE R PRECORD. LEADS(V1-V3) THROUGHOUT MOST OF CHILDHOOD**
- **THE TRANSITION TO AN UPRIGHT PATTERN OCCURS IN ADOLESCENCE**
- **T WAVES ARE NORMALLY UPRIGHT IN FIRST WEEK OF LIFE**
- **POSITIVE OR UPRIGHT T WAVES IN THE R PRECORD. LEADS IS A RELIABLE INDICATION OF MILD TO MODERATE ELEVATION IN RIGHT VENTRICULAR PEAK SYSTOLIC PRESSURE**
- **LVH W/STRAIN(CHF): UPRIGHT T WAVES V1-V3**

# PEDIATRIC BRADYCARDIA

- ABC'S; SECURE AIRWAY; 100% O<sub>2</sub>; START IV/IO; VITALS
- IF SEVERE CARDIORESP COMPRIMISE: POOR PERFUSION(MOTTLED SKIN), HYPOTENSION, RESP DISTRESS: PERFORM CHEST COMPRESSIONS IF HR<80-INFANT & <60-CHILD
- EPI IV/IO 0.01MG/KG (1:10,000) or ET-tube 0.1mg/kg (1:1000) q3-5min
- ATROPINE 0.02MG/KG IV/IO; MINIMUM DOSE 0.1MG, MAX .5MG CHILD, 1MG ADOLESCENT

