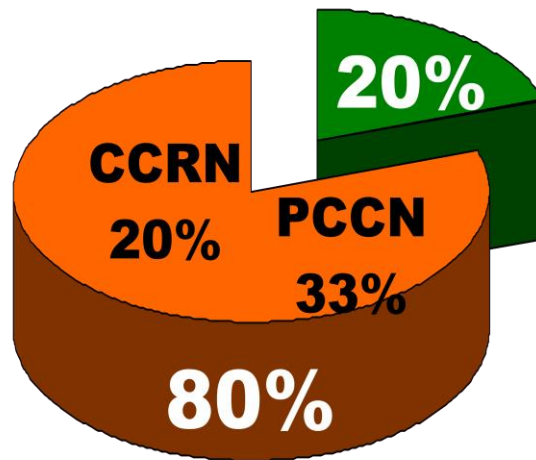
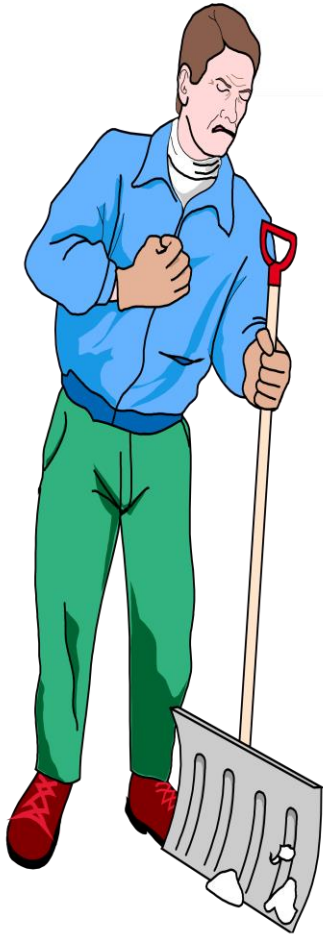
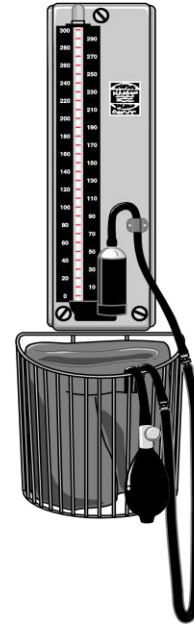
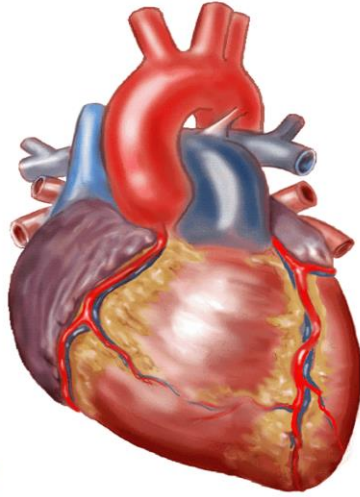


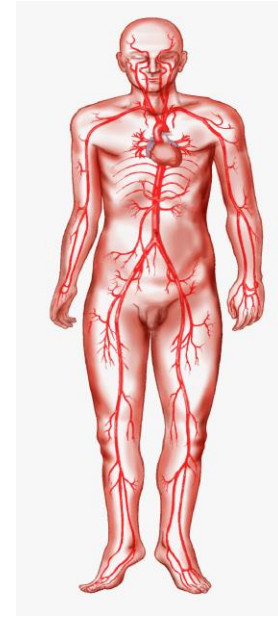
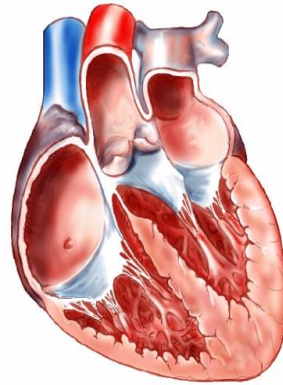
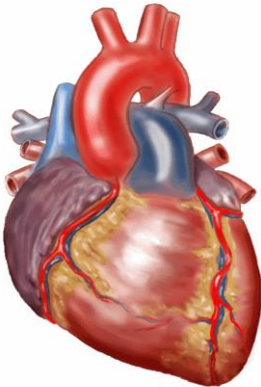
# Cardiovascular



# Cardiovascular

## Introduction

### A. Anatomy & Physiology



### B. Cardiac Assessment

1. Cardiac Risk Factors
  - ♥ Non Modifiable
    - ✓ Age
    - ✓ Gender
    - ✓ Family History
    - ✓ Race
  - ♥ Modifiable
    - ✓ Smoking
    - ✓ Hypertension
    - ✓ Diabetes
    - ✓ Obesity
    - ✓ Stress
    - ✓ Exercise
    - ✓ Hyperlipidemia
2. Medical & Surgical History
3. Social History
4. Medication History
5. Physical Exam

- ♥ Color
  - ♥ Pulses
  - ♥ Rate & Rhythm
  - ♥ PMI Location
  - ♥ Extremity Temperature
  - ♥ Dyspnea
  - ♥ Fatigue Level
  - ♥ Fluid Retention
  - ♥ Palpitations
  - ♥ Dizziness
6. Chest Pain Exam
- ♥ PQRST Assessment
    - ✓ P: Pain, Placement, Provocation
    - ✓ Q: Quality (sharp, stabbing, pressure) , Quantity
    - ✓ R: Radiation, Relief
    - ✓ S: Severity, Systems (nausea, sweaty, dizziness)
    - ✓ T: Timing (when it started, how long did it last, what makes it better or worse)

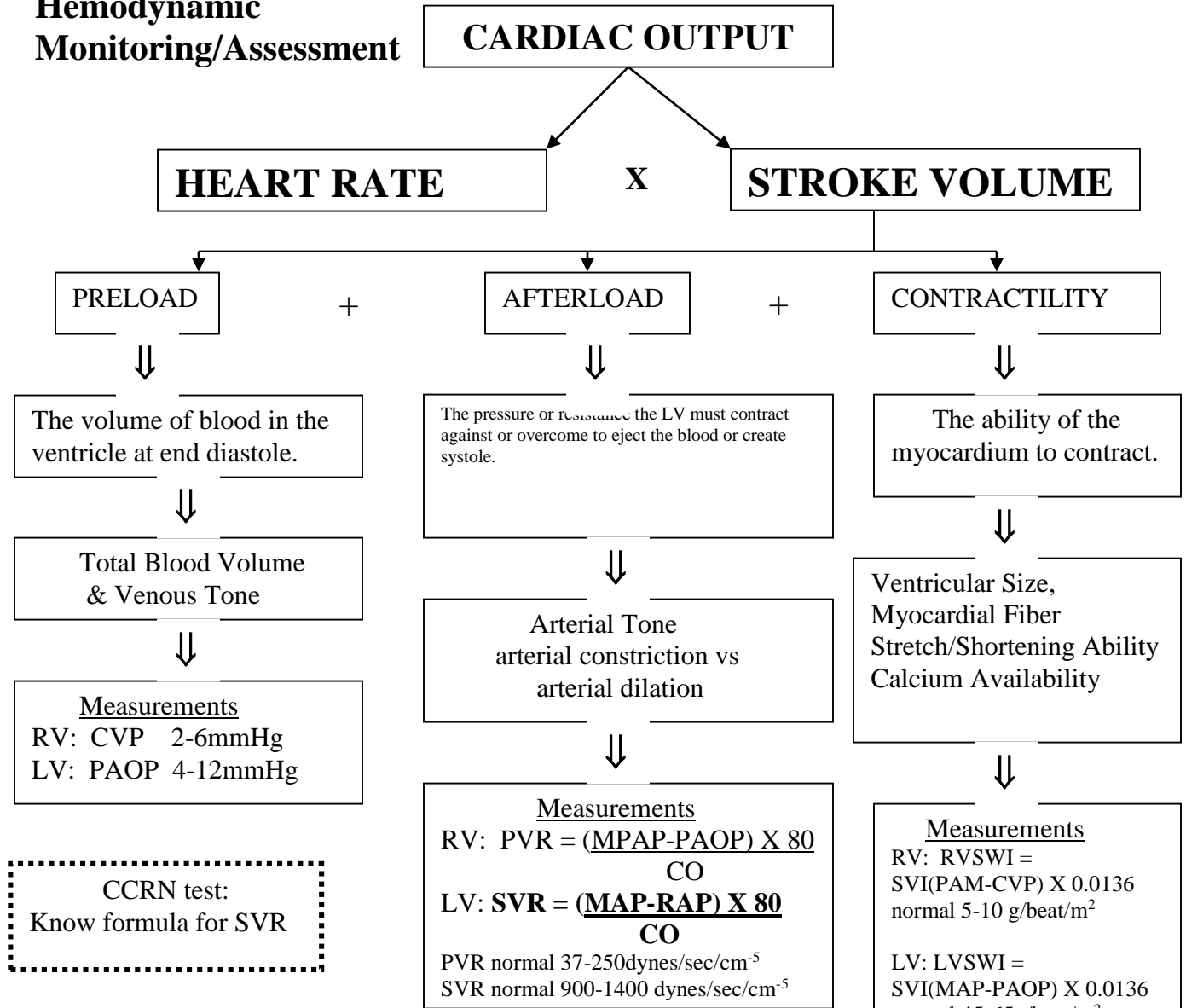
### C. **Diagnostic Tests & Procedures**

1. 12 Lead ECG
2. Echocardiography (Transthoracic and Transesophageal)
3. Stress Test
4. Cardiac Catheterization
5. Doppler Ultrasound
6. Blood Work
  - ♥ Acute Coronary Syndrome
    - ✓ Cardiac Enzymes: CK-MB,
    - ✓ Amino Acids: Troponins
    - ✓ Heme Proteins: Myoglobin
  - ♥ Lipid Profile
    - ✓ Triglycerides
    - ✓ Cholesterol
    - ✓ Low Density Lipoproteins
    - ✓ High Density Lipoprotein
  - ♥ Coagulation Profile
    - ✓ PT/INR
    - ✓ aPTT
    - ✓ ACT

♥ Misc

- ✓ B Type Natriuretic Peptide – BNP
- ✓ C Reactive Protein
- ✓ Homocysteine

**Hemodynamic Monitoring/Assessment**



CCRN test:  
Know formula for SVR

**CVP:** central venous pressure, **EF:** ejection fraction, **LV:** left ventricle, **LVEDV:** left ventricular end diastolic volume, **LVEF:** left ventricular ejection fraction, **MAP:** mean arterial pressure, **MPAP:** mean pulmonary arterial pressure, **PAOP:** pulmonary artery occlusion pressure, **PVR:** pulmonary vascular resistance, **RAP:** right atrial pressure, **RV:** right ventricular, **RVEDV:** right ventricular end diastolic volume, **RVEF:** right ventricular ejection fraction, **RVSWI:** right ventricular stroke work index, **SV:** stroke volume, **SVI:** stroke volume index, **SVR:** systemic vascular resistance.

**Measurements**  
 RV: RSWI = SVI(PAM-CVP) X 0.0136  
 normal 5-10 g/beat/m<sup>2</sup>  
 LV: LSWI = SVI(MAP-PAOP) X 0.0136  
 normal 45-65g/beat/m<sup>2</sup>  
 LVEF =  $\frac{LVEDV \times 100}{SV}$   
 RVEF =  $\frac{RVEDV \times 100}{SV}$   
 LVEF = 60-75%  
 RVEF = 45-50%

- A. **Preload:** The volume of blood creating a stretch on the muscle chamber at the end of diastole.
1. Decreases in Preload
    - ♥ Hypovolemia
    - ♥ Arrhythmia
    - ♥ Loss of “Atrial Kick”
    - ♥ Venous Vasodilatation
  2. Increases in Preload
    - Left Heart
      - ♥ LV Failure/Dysfunction
      - ♥ Mitral Valve Disease
      - ♥ Aortic Valve Disease
      - ♥ Cardiac Tamponade/Effusion
      - ♥ Volume Overload
      - ♥ Decreased Compliance
    - Right Heart
      - ♥ RV Failure Due to Ischemia
      - ♥ Increased Pulmonary Vascular Resistance
      - ♥ Cardiac Tamponade/Effusion
      - ♥ Volume Overload
      - ♥ LV Failure
- B. **Afterload:** The pressure or resistance the LV must contract against or overcome to eject the blood or create systole.
1. Decreases in Afterload
    - ♥ Vasodilation
    - ♥ Sepsis
    - ♥ Vasodilator Therapies
  2. Increases in Afterload
    - Right Heart
      - ♥ Pulmonary Hypertension
      - ♥ Hypoxemia
      - ♥ Pulmonic Stenosis
    - Left Heart
      - ♥ Vasoconstriction
      - ♥ Vasopressors
      - ♥ Hypothermia

♥ Aortic Stenosis

C. **Contractility:** The ability of the myocardium to contract.

1. Decreased Contractility

♥ Parasympathetic Stimulation

♥ Negative Inotropic Therapies

✓ Beta Blockers

✓ Calcium Channel Blockers

♥ Metabolic States

✓ Hyperkalemia

✓ Myocardial Ischemia/Infarct

✓ Acidosis

2. Increased Contractility

♥ Sympathetic Stimulation

♥ Inotropic Therapies

✓ Epinephrine

✓ Dopamine

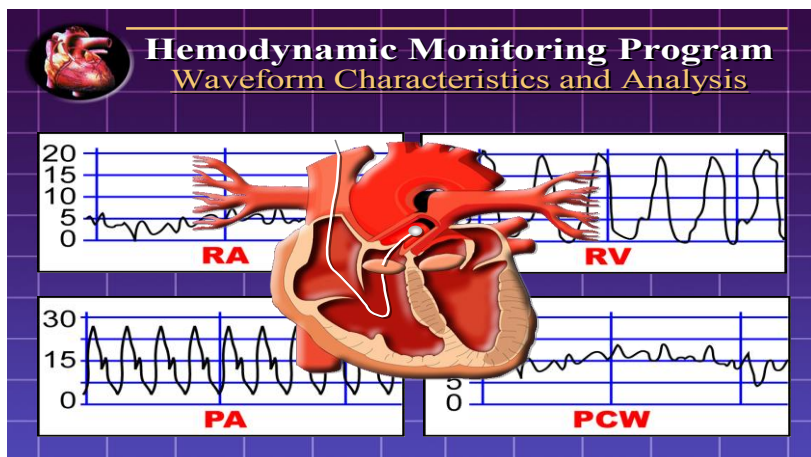
✓ Digoxin

✓ Calcium

♥ Metabolic States

✓ Hypercalcemia

## II. Pulmonary Artery Balloon Tipped Thermodilution Catheter



### Normal Values

CVP:

2-6mmHg

PA:

15-25/8-15mmHg

PAOP:

6-12mmHg

## Important to Remember

1. Spontaneous Breathing: Negative Pressure
2. Mechanical Ventilation: Positive Pressure
3. Review Waveforms for PA, PAOP and CVP
3. Mean Arterial Pressure:  
$$\text{MAP} = \frac{\text{Systolic BP} + (2 \times \text{Diastolic BP})}{3}$$
 Normal = 60-80mmHg

## III. SUMMARY

CCRN test:  
Know formula for MAP

# ACUTE HEART FAILURE

## I. PATHOPHYSIOLOGY OF HEART FAILURE

### A. Definition:

**Heart Failure** is the inability of the heart to adequately supply blood to meet the metabolic demands of the tissues resulting in inadequate tissue perfusion and volume overload.

**Acute Heart Failure** occurs when the inability to meet the demands of the tissues takes place abruptly, frequently without time for compensatory mechanisms to be activated. If the failure is severe or rapid enough the result will be cardiogenic shock.

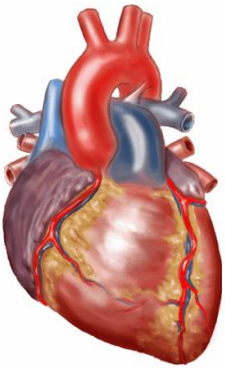
### Prevalence:

- ♥ 4.7 Million People Suffer from HF
- ♥ 550,000 New Cases Each Year
- ♥ Impact 1.5-2% of the Population
- ♥ 6-10% of those > 65 yo
- ♥ Only Major CV Disorder that is Increasing in Incidence and Prevalence
- ♥ 50% of Pts with Severe HF will Die within 1yr, 70% within 3 yrs
- ♥ 250,000 Deaths per year

C. **Cause:** Heart failure is a potential complication of most cardiac conditions and many organic and systemic problems. Acute failure is frequently the result of a new event or progression of a preexisting heart failure state.

### ♥ Cardiac Anatomical Causes

- ✓ Acute MI: loss of 40-50% of myocardial mass
- ✓ Mechanical Complications: perforated intraventricular septum, papillary muscle rupture
- ✓ Ventricular Aneurysm
- ✓ Congenital Heart Defects
- ✓ Pericardial Tamponade
- ✓ Valvular Heart Disease: severe/acute stenosis or regurgitation
- ✓ Constrictive Pericarditis
- ✓ Cardiomyopathies: congestive, hypertrophic or restrictive





### ♥ Cardiac Physiological Causes

- ✓ Coronary Artery Disease
- ✓ Hypertension
- ✓ Cardiac Transplant Rejection
- ✓ Postoperative Low Cardiac Output Syndrome
- ✓ Dysrhythmias
- ✓ Pericardial Disease
- ✓ Infective Endocarditis

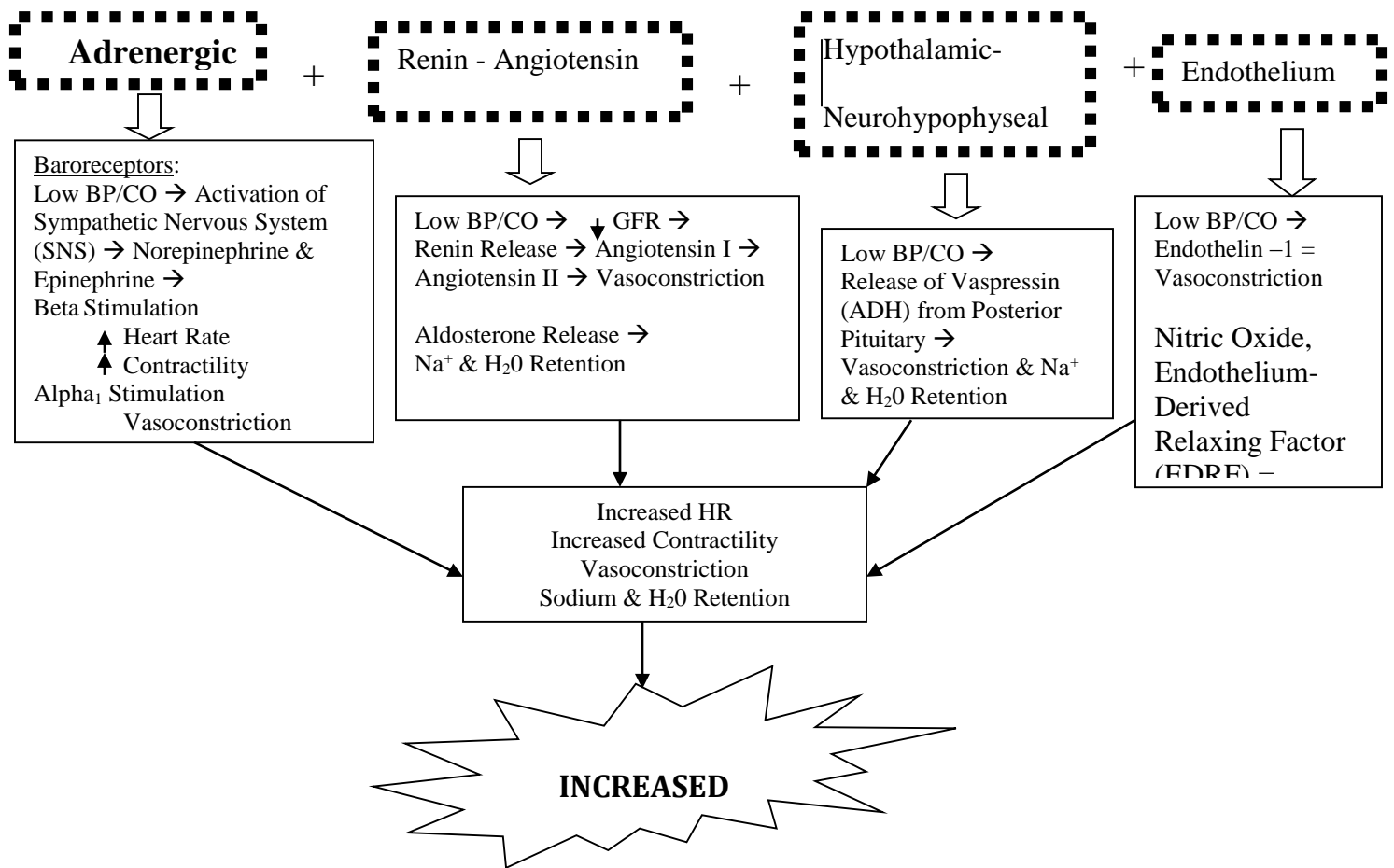
### ♥ Non-Cardiac Causes

- ✓ Emboli
- ✓ Hypovolemia
- ✓ Metabolic
- ✓ Sepsis
- ✓ Post-Partum Cardiomyopathy
- ✓ Viral Cardiomyopathy

**D. Mechanism of Failure:** Although failure may be caused by a variety of cardiac and non-cardiac pathologies, the outcome is the same - decline in cardiac function leads to a drop in cardiac output (CO). Low CO stimulates initial and progressive adaptation phases.

#### 1. Initial Adaptation to Low CO

- ♥ Drop in CO → Drop in Ejection Fraction (EF)
- ♥ Increase in End Diastolic Volume → Myocardial Fiber Stretch
- ♥ Increased Contractility  
(augmented sarcomere sensitivity to  $Ca^{++}$ )
- ♥ Activation of the Neurohormonal Systems (compensation)
  - ✓ Adrenergic System
  - ✓ Renin-Angiotensin-Aldosterone System
  - ✓ Hypothalamic-Neurohypophyseal System
  - ✓ Endothelium Activated Mediators



## 2. Progression of Heart Failure

- ♥ Continued Activation of the Sympathetic System Causes Increased Afterload
- ♥ Release of Natriuretic Peptides: ANP & BNP
  - Atrial Natriuretic Peptide (ANP): produced by the stretched atria → promotes diuresis, vasodilation, and inhibition of RAAS. Not strong enough to counteract the vasoconstricting mechanisms of the initial compensatory response
  - Brain Natriuretic Peptide (BNP): produced by the ventricles, is a marker for ventricular dysfunction and produces the same response as ANP
- ♥ Release of Cytokines

- Tumor Necrosis Factor (TNF- $\alpha$ ): produced secondary to hypervolemia, triggers both systemic and cardiac inflammatory responses
- ♥ Cardiac Hypertrophy & Remodeling: initially adaptive in nature, eventually leads to hypertrophy, ventricular dilation and increased O<sub>2</sub> demands leading to CO & ischemia
- ♥ Reflex Response from the Baroreceptors, Stretch Receptors
- ♥ Increase Demand and Decrease Function → Progressive Failure

## E. Classifications of Heart Failure

1. Systolic vs Diastolic
2. Right vs Left
3. High-Output vs Low-Output
4. Compensated vs Decompensated
5. New York Heart Association Classification of Congestive Heart Failure
  - ♥ Class I No Symptoms
  - ♥ Class II Symptoms on Maximal Exertion
  - ♥ Class III Symptoms on Minimal Exertion
  - ♥ Class IV Symptoms occur at Rest
6. ACC/AHA Evolution & Progression Classification System
  - ♥ Stage A At high risk for heart failure but without Structural heart disease or symptoms of HF
  - ♥ Stage B Structural heart disease but without symptoms of HF
  - ♥ Stage C Structural heart disease with prior or current symptoms of HF
  - ♥ Stage D Refractory HF requiring specialized interventions

## F. Signs & Symptoms of Heart Failure

1. Cardiac
  - ♥ Tachycardia
  - ♥ Weak pulses
  - ♥ Low CO & BP
  - ♥ Jugular Venous Distention

- ♥ S<sub>3</sub> Diastolic Gallop
- ♥ Displaced PMI
- ♥ Chest X-ray: cardiomegaly and vascular prominence
- ♥ 2D Echo: valvular abnormalities, cardiac enlargement
- ♥ Peripheral Edema
- ♥ Positive Hepatojugular Reflux

2. **Pulmonary**

- ♥ Dyspnea
- ♥ Bibasilar Rales
- ♥ Paroxysmal Nocturnal Dyspnea

3. **Neurological**

- ♥ Fatigue, Weakness &/or Dizziness
- ♥ Change in LOC
- ♥ Feeling of Impending Doom

## II. HEART FAILURE MANAGEMENT

### Goals of Therapy

- A. Prevent and/or Reverse Cause of Failure
- B. Decrease the Negative Spiral of the Compensatory Mechanisms
- C. Decrease Demands on the Heart
- D. Decrease Ectopy or Maintain Electrical Stability
- E. Focus on Quality of Life

**A. Prevent and/or Reverse Cause of Failure**

1. If the primary cause is poor coronary perfusion the tx should be directed towards opening the arteries and revascularization of the myocardium.
  - ♥ Thrombolytics
  - ♥ Percutaneous Coronary Interventions (PCI)
  - ♥ Coronary Artery Bypass Graft Surgery (CABG)
  - ♥ Cardiac Transplantation
2. Surgery to repair anatomical problem

3. Treat the physiological cause: CAD, HTN, Dysrhythmia

**B. Decrease the Negative Spiral of the Compensatory Mechanisms.**  
The major treatment modalities in this category are pharmacologic

1. **Vasodilators:** venodilators and arteriodilators are both helpful in the management of heart failure. They can decrease preload, decrease afterload, increase renal perfusion and improve symptoms of both failure and those related to the compensatory response.

a. **Angiotensin-Converting Enzyme (ACE) Inhibitors:**  
ACE inhibitors are now the flagship agent in drug management for heart failure. Utilized as a solo agent or in combination with other drugs.

♥ Dilate Arterioles

- ⇒ Decrease afterload
- ⇒ Increase stroke volume
- ⇒ Increase cardiac output
- ⇒ Improve regional blood flow

♥ Dilate Veins

- ⇒ Decrease preload
- ⇒ Decrease pulmonary congestion
- ⇒ Decrease cardiac dilation
- ⇒ Increase renal perfusion
  - Enhances  $\text{Na}^+$  &  $\text{H}_2\text{O}$  excretion
  - Decreases negative compensatory spiral

♥ Decrease Release of Aldosterone (decreasing the need for high doses of diuretics)

- ⇒ Excretion of  $\text{Na}^+$
- ⇒ Excretion of  $\text{H}_2\text{O}$
- ⇒ Retention of  $\text{K}^+$

b. **Angiotensin II Receptor Blockers (ARB):** block the actions of Angiotensin II. Same pharmacological affects as ACE inhibitors without adverse effects of cough,

hyperkalemia & angioedema. Used with ACE Inhibitors are not tolerated.

- c. **Hydralazine** (Apresoline) a selective arteriole dilator and **Isosorbide dinitrate** (Isordil, sorbitrate) a nitrate that is a selective venous dilator, are commonly used together to create a similar effect as the ACE inhibiting agents. ACE inhibitors should be used first.
- d. **Nitroglycerin** and the nitrate class drugs work directly on the vascular smooth muscle causing venodilation with only minimal arteriole dilation.
- e. **Calcium Channel Blockers:** It would seem logical that  $Ca^{++}$  blockers because of their vasodilating effect would be useful in the tx of heart failure. Clinical trials have shown just the opposite. These agents have either shown not to be helpful or in some cases to actually be harmful to pts with heart failure. They are not recommended for use in the tx of heart failure.
- f. **B-Type Natriuretic Peptide:** Nesiritide (Natrecor) simulates cGMP production and binds to the receptors in vasculature and kidneys. Increases cardiac output and GFR, decreases aldosterone levels, in order to promote diuresis. Main side effect is hypotension.

2. **Diuretics:** one of the first string management agents for heart failure that will decrease both preload and afterload by reducing water retention. The therapeutic goal is to decrease the work of the failing heart muscle. Diuretics are recommended for use with all patients who have symptomatic heart failure.

- a. **Potassium Sparing Diuretics:** Spironolactone (Aldactone, Novo-Sprion) blocks aldosterone in the renal tubule, causing a loss of  $Na^{+}$  and  $H_2O$  and retention of  $K^{+}$ . This agent is only a weak diuretic and can be used in combination with  $K^{+}$  losing diuretics to decrease the

loss of  $K^+$ . The positive effect on heart failure is related to its aldosterone antagonist properties.

- b. **Thiazide Diuretics:** Hydrochlorothiazide (HydroDiuril, Esidrix) is a weak diuretic (increases sodium excretion 5-10%) that can be effective in heart failure as long as the cardiac output and renal perfusion are adequate. It is ineffective when the GFR is low. Major side effect is hypokalemia.
  - c. **Loop Diuretics:** Furosemide (Lasix) inhibits  $Na^+$  &  $Cl^-$  reabsorption from the loop of Henle and is a potent diuretic (increases sodium excretion 20-25%) that is effective in low cardiac output and GFR states like severe heart failure. Side effects include hypotension and hypokalemia.
3. **Inotropic Agents:** increase the force of myocardial contraction enhancing stroke volume → increasing cardiac output.
- a. **Cardiac Glycosides:** Digoxin (Lanoxin) promotes the accumulation of  $Ca^{++}$  within the cardiac cell and therefore contractility by inhibiting  $Na^+/K^+$ ATPase. It also decreases heart rate by slowing conduction through the AV node.
    - ♥ Increases cardiac output
    - ♥ Decreases sympathetic tone
    - ♥ Decreases renin release
    - ♥ Increases filling time
    - ♥ Improves symptoms of heart failure & QOL but not outcome
  - b. **Sympathomimetics:** Dobutamine (Dobutrex) a synthetic catecholamine with selective beta-adrenergic agonist properties
    - ♥ Increases myocardial contractility → stroke volume → cardiac output (beta 1)
    - ♥ Increases sinus node automaticity & AV conduction → heart rate (beta 1)

- ♥ Mild vasodilator (beta 2)
- ♥ Increases myocardial oxygen demand

- c. **Phosphodiesterase Inhibitors:** Amrinone (Inocor) and Milrinone (Primacor) are non-catecholamine agents that increase contractility by increasing cyclic adenosine monophosphate (cAMP) → which enhances  $Ca^{++}$  entry into the cell. By blocking PDE III they block sympathetic vasoconstriction causing vasodilation.
- ♥ Increases contractility → Increases CO
  - ♥ Vasodilation → Decreases afterload → Increases CO
  - ♥ No effect on heart rate or myocardial oxygen demands
  - ♥ No tolerance problems

4. **Beta Blockers:** The normal response to beta blockage (in the non heart failure pt) is decreased heart rate, reduced force of contraction, and decreased velocity of impulse conduction through the AV node. Blocking the activation of the sympathetic nervous system has an effect on the negative spiral of compensatory responses in heart failure. Beta Blockade in the heart failure pt:
- ♥ Decreases cardio toxic effects of chronic sympathetic activation
  - ♥ Alleviates tachycardia
  - ♥ Increases ejection fraction and improves of resting cardiac function
  - ♥ Improves quality of life (less symptoms, increased exercise tolerance)
  - ♥ Prolongs survival

These benefits do not happen initially with treatment – in fact many patients actually experience a worsening of symptoms at the beginning of beta-blocker administration. The benefit is seen in patients with NYHA Class II – IV failure but not in patients with Class I failure. Recommended for Stage B & C and D if the patient will tolerate.

### **Third Generation Beta Blockers:**



Non-selective for  $\beta_1$  and  $\beta_2$  and also have ancillary cardiovascular actions.

Carvedilol (Coreg) – is also an  $\alpha_1$  adrenergic blocker and causes vasodilation – the ONLY FDA APPROVED beta blocker for use in heart failure

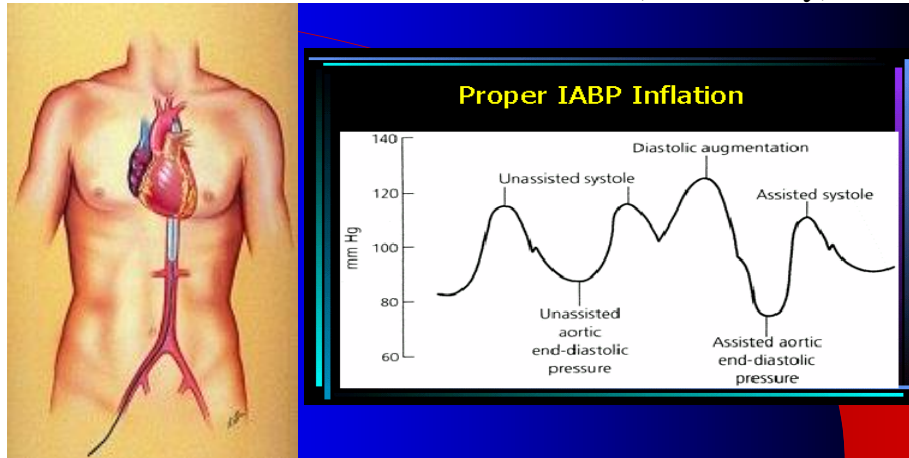
- ♥ Decreases HR
- ♥ Decreased PAOP
- ♥ Increases CI, EF
- ♥ Improves QOL

Bucindolol – is also a direct action vasodilator

Although the FDA has only approved carvedilol, survival benefits have been demonstrated in heart failure patients with metoprolol, bucindolol, bisoprolol, nebivolol.

### C. Decrease Demands on the Heart

1. Intra-Aortic Balloon Pump (IABP): Improves Coronary Perfusion and Decreases Afterload (CCRN only)



2. Ventricular Assist Devices (VAD) (CCRN Only)
  - ♥ Recovery/Rest Therapy: Post-Cardiotomy Shock, AMI
  - ♥ Bridge to Transplantation
  - ♥ Permanent Support as an Alternative to Transplantation

**D. Decrease Ectopy and Maintain Electrical Stability:** Approx half of deaths from heart failure occur suddenly and are most likely the result of a dysrhythmia.

Treatments:

1. Oral Antidysrhythmic Agents: Amiodarone, Beta Blockers, Digoxin
2. Pacemakers:
  - ♥ Atrial (A) Pacing: electrode in right atria and spike before P wave
  - ♥ Ventricular (V) Pacing: electrode in right ventricle and spike before QRS Complex
  - ♥ Atrial/Ventricular (AV) Pacing – Dual Chamber Pacing: electrode in both right sided chambers and spike before P and QRS complex
  - ♥ Biventricular or Cardiac Resynchronization Therapy: electrode in RA, RV and outside of LV



### **Cardiac Resynchronization Therapy (CRT)**

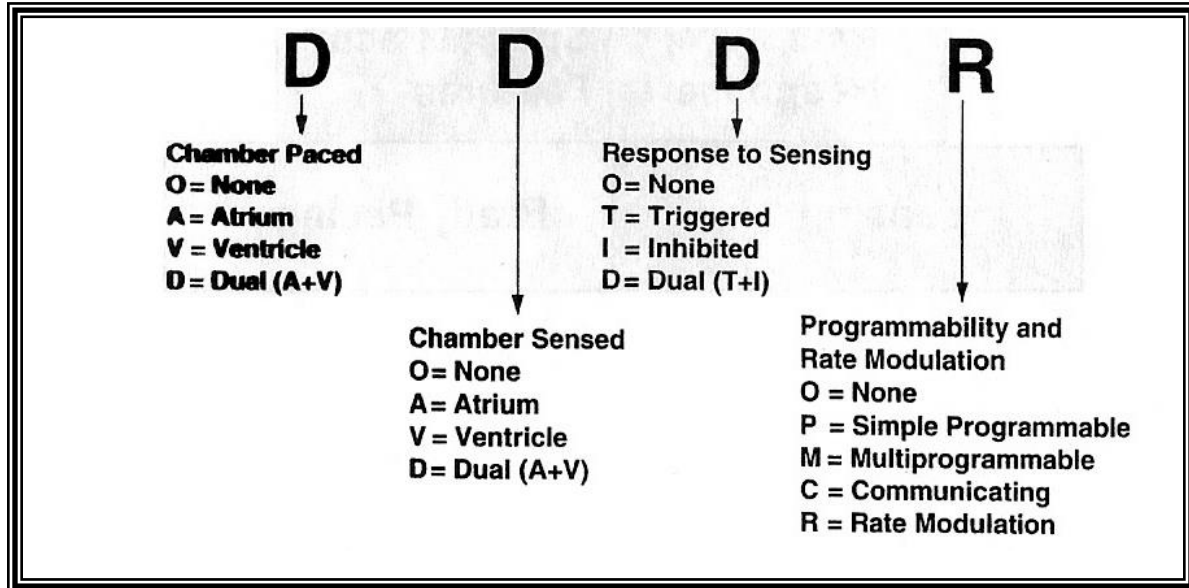
aka atrial synchronized biventricular pacing

Three lead system. One in the right atrium & right ventricle and one in the left ventricular. The LV lead is threaded into the coronary sinus.

FDA Criteria:

- ♥ Symptomatic heart failure even with optimal medical tx
- ♥ NYHA Class III or IV heart failure
- ♥ QRS interval at or greater than 0.13-0.15 sec
- ♥ Left ventricular ejection fraction of 35% or lower.

## Pacing Code



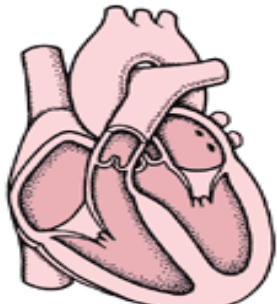
### ♥ Trouble Shooting Pacing:

- ✓ Failure to Capture: spike not followed by complex
- ✓ Failure to Sense: Pacer firing inappropriately
- ✓ Failure to Fire: no spike at all

### 3. Implantable Cardioverter Defibrillators (ICD)

### E. Cardiac Transplantation

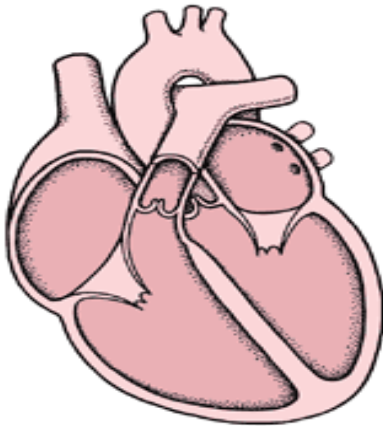
### F. Quality of Life Focus



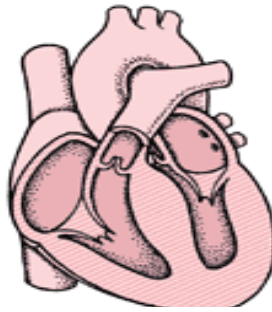
**Normal**

### Cardiomyopathy

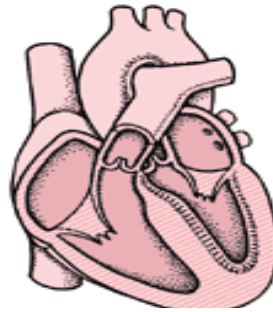
Cardiomyopathy is a disease of the cardiac muscle and impacts the muscle globally. There are many risk factors but the actual cause is frequently unknown (idiopathic cardiomyopathy). There are three classifications: dilated, hypertrophy and restrictive. All three can lead to systolic or diastolic heart failure or both, dysrhythmias (chronic and lethal) and many systemic problems.



**Dilated**



**Hypertrophic**



**Restrictive**

## **III. Cardiomyopathy**

Adapted from AACN Core Orientation Program

### **Classifications**

#### **A. Dilated (congestive) Cardiomyopathy (DCM): Most Common**

##### **1. Pathophysiology**

- ♥ Ventricular(s) Dilation → Decreased Contractility
- ♥ Systolic Dysfunction
- ♥ Drop in CO
- ♥ Activation of Compensatory Mechanism (don't work)
- ♥ Poor contractility and increases in left ventricular end diastolic volume (LVEDV)
- ♥ Dilated ventricles and increased LVEDV → dilated annulus of the valves and valve dysfunction
- ♥ Dilated ventricles and increased LVEDV → pulmonary edema

##### **2. Causes:**

- ♥ CAD

- ♥ Alcohol
- ♥ Pregnancy/Post Partum
- ♥ Viral Infections
- ♥ Chemotherapy
- ♥ Hypertension
- ♥ Chronic Overworked Heart (chronic morbid obesity)

## **B. Hypertrophic Cardiomyopathy (HCM)**

### 1. Pathophysiology

- ♥ Septum disproportionate enlarged creating narrow, long cavity
- ♥ Septum can obstruct outflow tract.
- ♥ Ventricular wall becomes rigid →
- ♥ Inc LVEDP, L atrial pressure →
- ♥ Backward flow and pulmonary congestion
- ♥ Dysrhythmias may occur and cause sudden death

### 2. Causes:

- ♥ Frequently Familial
- ♥ Average age mid 20s
- ♥ Hypertension
- ♥ Abnormal catecholamine levels
- ♥ Type I Diabetics

## **C. Restrictive Cardiomyopathy**

### 1. Pathophysiology

- ♥ Stiff ventricles reduce ventricular filling leading to drop in CO and bi-atrial dilation
- ♥ Causes pulmonary edema and systemic congestion

### 2. Causes:

- ♥ 90% from Amyloidosis – Deposits of Insoluble Protein into Muscle and Conductive Tissue
- ♥ Pathologies: Sarcoidosis, Endomyocardial Fibrosis, Radiation Therapy, Scleroderma, Cancer Metastasis

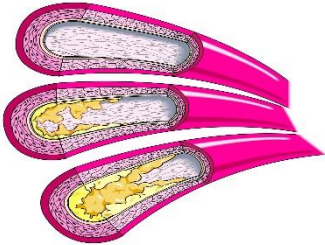
## **D. Treatment**

**Sudden Cardiac Death is the Major Cause of Death**

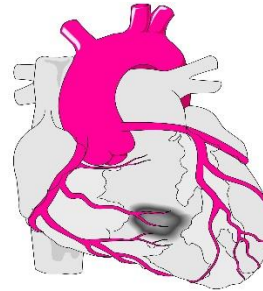
- ♥ Treat the Causative Factors
- ♥ Rest Heart
- ♥ Relieve Pulmonary and Systemic Congestion
- ♥ Prevent Thromboembolic Events (DCM)
- ♥ Antidysrhythmic Agents/Pacer/ICD
- ♥ Assist Devices
- ♥ Consider Transplant
- ♥ Pharmacology
  - Digitalis \*
  - Diuretics\*
  - Beta-Blockers, Ace Inhibitors
  - Vasodilators
  - Inotropic Agents\*
  - Antidysrhythmics                   \*caution with HCM

#### **IV. SUMMARY**

# ACUTE CORONARY SYNDROME



## UNSTABLE ANGINA & ACUTE MYOCARDIAL INFARCTION



### I. Introduction

### II. Initiating Acute Coronary Syndromes:

The rupture or disruption of the plaque is caused from internal and/or external factors or triggers.

**Angina:** Myocardial Anoxia

**Exertional Angina:** Pain that is brought on during times of increased myocardial oxygen demand like exertion, eating, extreme emotions and exposure to cold temperatures the four Es. These symptoms are typically caused by or a sign of atherosclerosis.

**Prinzmetal's Angina or Variant Angina:** Pain that occurs at rest, during sleep or without evidence of provocation. Symptoms are thought to be caused by coronary vasospasm.

**Stable Angina:** Exertional angina with consistent symptoms which is typically relieved with rest or cessation of cause and possibly nitroglycerine administration.

**Unstable Angina** (aka crescendo or pre-infarction angina): Angina that:

- ♥ Has a recent onset (within 2 months) and severely limits activity
- ♥ Newly occurs at rest
- ♥ Differs in characters or symptoms from the person's 'typical exertional angina' (it occurs with less exertion, has a greater

intensity or longer duration, requires more interventions before obtaining relief)

- B. Non-CAD Causes:** Non-ischemic causes of chest pain must be ruled out, such as:

**Cardiac Causes**

- ♥ Acute Pericarditis
- ♥ Cardiac Tamponade
- ♥ Acute Myocarditis
- ♥ Aortic Stenosis
- ♥ Myocardial Contusion
- ♥ Mitral Valve Prolapse
- ♥ Cardiomyopathies

**Non Cardiac Causes**

- ♥ Panic Attack/Anxiety
- ♥ Illicit Drug Use
- ♥ Gastrointestinal Disorders
- ♥ Spontaneous Pneumothorax
- ♥ Pulmonary Embolism
- ♥ Pulmonary Hypertension
- ♥ Esophageal Rupture
- ♥ Costochondritis
- ♥ Hypovolemia

### **III. UNSTABLE ANGINA**

- A. Pathophysiology:** “A partially occluding thrombus produces symptoms of ischemia, which are prolonged and may occur at rest. At this stage the thrombus is platelet-rich. Therapy with anti-platelet agents such as aspirin and GP IIb/IIIa receptor inhibitors is most effective at this time. Fibrinolytic therapy is *not* effective and may paradoxically accelerate occlusion by the release of clot-bound thrombin, which further activates platelets. An intermittently occlusive thrombus may cause myocardial necrosis, producing a non-Q wave MI.” (AHA ACLS Provider Manual).

**Questions:**

1. Is it Cardiac or is it Not Cardiac?
2. If it is Cardiac, is it Ischemic or is it Not Ischemic?
3. If it is Ischemic, is it Stable or is it Not Stable?



4. If it is Unstable Where is it: Platelet, Blood Clot or Done Deal?

**Time is Muscle and the Clock is Ticking!!!**

**B. Assessment:**

1. **History:**

- ♥ Assessment of Angina: PQRST Assessment
  - P: Pain, Placement, Provocation
  - Q: Quality, Quantity
  - R: Radiation, Relief
  - S: Severity, Systems (nausea, sweaty, dizziness)
  - T: Timing (when it started, how long did it last)
- ♥ Medical History
- ♥ Medications: Prescription, Over the Counter, Dietary Supplements
- ♥ Social History
- ♥ Family History
- ♥ Major Risk Factors of Atherosclerosis

2. **Physical Examination**

3. **12-Lead Electrocardiogram (ECG)**

- ♥ Ischemia: ST – Segment Depression
- ♥ Injury: ST – Segment Elevation
- ♥ Infarction: Q waves

**Questions:**

1. Is the J point at the Isoelectric line?
2. Does the T wave go in the same direction as the QRS in each lead?
3. If there is a Q wave - is it more than a box down or a box wide?
4. In which leads do you identify the abnormalities?

**Acute MI 12 lead**

<b>Location</b>	<b>Indicative Leads</b>	<b>Reciprocal Leads</b>	<b>Coronary Arteries</b>	<b>Major Complications</b>
Anterior	Leads V1, V2, V3, V4	Leads II, III, aVF	LAD	Cardiogenic Shock Bundle Branch Blocks Vent Dysrhythmias
Inferior	Leads II, III, aVF	Leads I, aVL	RCA	Bradycardia Heart Blocks

Lateral	Leads I, aVL, V5, V6	Leads II, III, aVF	Circumflex	Heart Blocks in some
Septal	Leads V1, V2	Leads V5, V6	LAD	Bundle Branch Blocks
Posterior	Leads V7, V8, V9	Leads V1, V2	RCA or Circumflex	Heart Blocks

#### 4. Biochemical Cardiac Markers

##### ♥ **Creatine Kinase (CK):**

**CK total: Male 60 – 170 U/L**  
**Female 40 – 140 U/L**

This enzyme is important in the breakdown of creatinine to creatinine. It increases in the serum when muscle damage has occurred. Three CK isoenzymes have been identified; CK-I BB from the brain tissue and smooth muscle, CK-II MB from heart tissue and CK-III MM from muscle tissue. CK serum levels will begin to rise 3-6hr after chest pain, peak in 12-24 hrs and return to normal in 2-3 days.

Isoenzymes: CK I BB 0 – 1%  
CK II MB < 3 - 6%  
CK III MM 95 – 100%

##### ♥ **Troponin: cTn T: < 0.1 mcg/L cTn I: < 3.1 mcg/L**

The troponin complex is found on cardiac and skeletal muscle. Troponin C, T, & I are proteins that work in synchrony to regulate the force and speed of muscle contraction. These proteins modulate the interaction of actin and myosin. The amino acid structures of cardiac troponin T & I (cTn T & cTn I) are uniquely different than skeletal muscle. During periods of cardiac ischemia, intracellular troponin will leak out of the cell. Troponin levels can be detected within 3-5hrs after chest pain, cTnT will peak in 4-6hrs and cTnI 14-18hrs, and cTnT will return to normal 21 days and cTnI in 5-7 days.

##### ♥ **Myoglobin: 50 – 120 mcg/mL**

**Carbonic Anhydrase III (CA-III): 13 – 29 mcg/L**

Myoglobin is a heme protein located on cardiac and skeletal striated muscle. Due to its low molecular wt. it is released very

rapidly from the muscle after an ischemic event (faster than troponin or CK-MB). Serum levels will rise within 2 hrs of chest pain, peak in 3-15hrs and return to normal levels in 2 days. Because there is not a cardiac specific myoglobin, many non-cardiac events may cause an elevation. Carbonic Anhydrase III (CA-III) is another cytoplasmic protein found primarily in skeletal muscle. In skeletal muscle damage both CA III and myoglobin rise. In cardiac muscle damage there is only a rise in myoglobin. Therefore a rise in the **myoglobin/CA III ratio** is more indicative of an AMI than just an elevated myoglobin. A ratio of  $\geq 3.21$  is considered abnormal and indicates for cardiac damage.

5. **Early Risk Stratification:** Early identification of the cause and severity of the pain is essential in determining triage and appropriate therapy. The five factors from the patient's history that increase the likelihood that the ischemia is from CAD are:

- ♥ Nature of Symptoms
- ♥ Prior History of CAD
- ♥ Gender & Age
- ♥ Number of CAD Risk Factors

- C. **Treatment:** Treatment should be initiated as quickly as possible, while assessment is being completed. Immediate general-treatment includes

- ♥ Oxygen at 4L/min
- ♥ Aspirin 160-325mg (chewed)



- ♥ Loading Dose of P2Y<sub>12</sub> Receptor Inhibitor

1. Clopidogrel 600mg po
2. Prasugrel 60mg
3. Ticagrelor 180mg

} 2013 STEMI Guidelines AHA  
Circulation 2013;127:529-555

- ♥ Nitroglycerin SL or Spray
- ♥ Morphine IV (if pain not relieved by NTG)
- ♥ "MONA" meets the patient (Morphine, Oxygen, Nitroglycerin, Aspirin) and now Clopidogrel "MONiCA"

Once assessment is complete, patient is identified as having characteristics for one of four categories

1. Non-Cardiac Diagnosis
2. Chronic Stable Angina
3. Possible Acute Coronary Syndrome (ACS)
4. Definite ACS

**Possible ACS:**

- ♥ Give Aspirin and Clopidogrel – may have already done so
- ♥ Consider Antithrombin Tx
  1. ASA
  2. Glycoprotein IIb/IIIa Inhibitor: Abciximab (ReoPro), Eptifibatide (Integrelin), Tirofiban (Aggrastat)
  3. Heparin
- ♥ Consider Beta Blocker
- ♥ NTG and MSO4

#### **IV. ACUTE MYOCARDIAL INFARCTION**

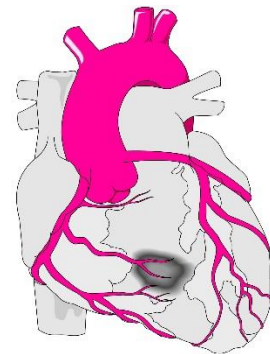
**A. Pathophysiology**

**B. Assessment**

1. History
2. Physical Examination
3. 12-lead Electrocardiogram: ST Elevation
4. Biochemical Cardiac Markers

**C. Treatment**

1. Give Triple Anti-Thrombin Tx
2. NTG if Pain Present
3. If ST-Segment Elevation - evaluate for reperfusion
  - ♥ Thrombolytics
  - ♥ Percutaneous Coronary Interventions (Stents, PTCA)
  - ♥ Coronary Artery Bypass Grafting
4. Give  $\beta$ -blocker
5. Once reperfused evaluate myocardial damage and provide post MI care



**Purpose:**

Revascularize the Heart

## CABG Surgery

**Procedure:**

With the use of cardiopulmonary bypass, hypothermia and cardioplegia the heart is motionless and bloodless. Grafts are used to supply blood distal to occlusion. Cannulation sites for bypass are typically the aortic & RA.

**Graft Option:**

Saphenous Vein, Internal Mammmary, Radial Artery

**Post Op Care:**

Pain, Volume Overload, MI, Stroke, Dysrhythmias  
Infection, Decrease CO, Impaired Gas Exchange  
Impaired Work of Breathing, Hypoperfusion Complications

**Additional Nursing Concerns:**

Pain, Immobility, Risk for Infection, Life Style Modification, Discharge Education, Nutrition

### 6. Adjunct Therapy for ACS

- ♥ Calcium channel Blockers: vasodilate, decrease HR
- ♥ Angiotensin Converting Enzyme Inhibitors: decrease afterload and preload

## THROMBOLYTIC AGENTS

Thrombolytic agents have been proven to decrease mortality and complications of acute MI. They initiate fibrinolysis by binding with the fibrin in the thrombus and converting plasminogen to plasmin. The early agents (streptokinase & anistreplase) bind with free or fibrin-bound plasminogen whereas the new agents (alteplase & reteplase) initiate local fibrinolysis (local to the clot) and therefore have a lower risk of systemic bleeding complications. These agents are very effective fibrinolytic drugs. Their relative effectiveness is determined by the

timing of the administration of the agent after ischemia has occurred. The common term used in emergency and cardiology practice is door-to-drug time. The sooner the occlusion is opened and cardiac muscle or brain is reperfused the less damage that will take place.

**Therapeutic Uses:** being given IV, directly into peripheral clot, intracoronary & intracerebral

- ♥ Acute Coronary Thrombosis
- ♥ DVT
- ♥ Massive Pulmonary Emboli
- ♥ Adjunct to PCI
- ♥ Thrombotic Stroke
- ♥ Combination tx of thrombolytic agents and GP IIb/IIIa, UFH, & LMWH have been shown to increase long term perfusion, mortality & morbidity

**Absolute Contraindications:**

- ♥ Active Bleeding
- ♥ Aortic Dissection
- ♥ Cerebral Neoplasm
- ♥ History of Intracranial Hemorrhage
- ♥ Recent (within 2 mo) Intracranial or Intraspinial Surgery or Trauma
- ♥ Cerebral Vascular Disease (aneurysm, arteriovenous malformation)
- ♥ Bleeding Diathesis
- ♥ Severe Uncontrolled Hypertension ( $\geq 180/110$ )

**Relative Contraindications:**

- ♥ Recent (within 10 mo) Major Surgery
- ♥ Recent (within 10 days) GI or GU Bleeding
- ♥ High likelihood of Left Heart Thrombus (mitral stenosis or A-fib)
- ♥ Acute Pericarditis or Sub Acute Bacterial endocarditis
- ♥ Significant Liver Dysfunction
- ♥ Pregnancy
- ♥ Diabetic Hemorrhagic Retinopathy

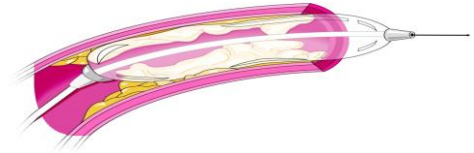
**Adverse Effects:**

- ♥ Major Risk is for Bleeding
- ♥ Should Major Bleeding Occur

- ✓ Stop infusion & other anticoagulants
- ✓ Anticipate immediate head CT if ICH suspected
- ✓ Administer cryoprecipitate, FFP, platelets
- ✓ Aminocaproic acid (Amicar)

## Interventional Cardiology

Percutaneous coronary interventions have increased in both number of procedures and success rates since the first balloon angioplasty was performed in 1977.



### Percutaneous Coronary Interventions (PCI)

- ♥ Diagnostic Coronary Angiography
- ♥ Percutaneous Transluminal Coronary Angioplasty (PTCA)
- ♥ Coronary Stents

### Nursing Care Concerns

#### ♥ Pre Procedure

- ✓ BUN/Creatinine Levels
- ✓ Dye Allergy
- ✓ Hydration Status
- ✓ Anticoagulation and Antiplatelet Medications
- ✓ Rate & Rhythm
- ✓ Electrolyte Balance: Especially Potassium
- ✓ Limb Circulation

#### ♥ Post Procedure/Potential Complications

- ✓ Myocardial Ischemia
- ✓ Stroke
- ✓ Groin Site Bleeding
- ✓ Distal Circulation
- ✓ Dysrhythmias
- ✓ Coronary Artery Spasm
- ✓ Abrupt Closure/Restenosis
- ✓ Coronary Artery Dissection

- ✓ Peripheral Vascular Complication
- ✓ Discharge Education

**D. Complications of AMI**

1. Cardiogenic Shock: infarction of  $\geq 40\%$  of the left ventricle

- ♥ Hypotension: SPB  $< 100\text{mmHg}$
- ♥ Pulmonary Edema
- ♥ Cardiac Index  $< 2.5$
- ♥ PAOP  $> 18\text{mmHg}$
- ♥ S&S of Poor Peripheral Perfusion
- ♥ Fibrinolytic tx has not been shown to be helpful
- ♥ PCI is Advocated

2. Arrhythmias Associated with Ischemia, Infarction & Reperfusion

**E. Treatment Goals for Cardiogenic Shock**

- ♥ Assist Contractility
- ♥ Alleviate Cause of Failure
- ♥ Fluid
- ♥ Pharmacological Agents
- ♥ Coronary Reperfusion
- ♥ Mechanical Assist

**V. SUMMARY**



# Cardiac Pharmacology

## Vasoactive & Inotropic Agents

### PHARMACOLOGICAL THERAPIES

#### Physiological Principles

#### Physiology of the Autonomic Nervous System

	<b>SYMPATHETIC</b>	<b>PARASYMPATHETIC</b>
<b>Purpose to Regulate Autonomic Function</b>	Activation: Flight or Fight Responses	Conservation: Maintain Organ Function and Conserve Energy
<b>Motor Neurons</b>	Large and Diffuse Number of Postganglionic Stimulation	Narrow and Specific Postganglionic Stimulation
<b>Neurotransmitters</b>	Norepinephrine Epinephrine	Acetylcholine
<b>Receptors</b>	Adrenergic: Alpha Beta (throughout body)	Cholinergic: Nicotinic Muscarinic (specific areas)
<b>Innervation</b>	Heart, Blood Vessels, Glands, Visceral Organs & Smooth Muscles	Heart, Glands, & Visceral Organs

## Autonomic Receptor Stimulation

ORGAN	SYMP	ADRENERGIC		PARA	CHOLINERGIC
	RECEPTOR	RESPONSE		RECEPTOR	RESPONSE
Heart	Beta 1	Increase Conduction Velocity (rate) & Increase Contractility		Muscarinic 2	Decrease Conduction Velocity & Contractility
Lungs	Beta 2	Bronchial Dilation decrease secretions		Muscarinic 2	Bronchial constriction increase secretions
Vessels	Alpha 1 Beta 2	Constriction Dilation			
Skeletal Muscle	Beta 2	Increase Contractility			
Bladder Sphincter	Alpha 1	Contraction		Muscarinic 3	Relaxes
GI: Motility Sphincter	Alpha1,2 B2 Alpha 1	Decrease Contraction		Muscarinic 3	Increase Relax
Kidney	Beta 1	Rennin Secreted			
Liver	Alpha 1, B2	Increase Glucose			

## Common Vasopressor & Inotropic Agents

### 1. Dopamine Hydrochloride (Dopamine)

✎ **Therapeutic Use:** Naturally occurring catecholamine and precursor to norepinephrine, also serves as a central and peripheral neurotransmitter. First line agent for many types of shock states. Versatile drug secondary to different actions depending on delivered concentration. The stimulation of dopaminergic receptors is a unique property of this agent.

✎ **Pharmacokinetics:** IV administration only with short half life

✎ **Pharmacodynamics:**

- ✓ Central and peripheral nervous system neurotransmitter and precursor of norepinephrine
- ✓ Low concentration: vascular DA<sub>2</sub> – dopaminergic receptors primarily in renal, mesenteric, coronary and cerebral beds – cause vasodilation. D<sub>1</sub> receptors mediate a mild natriuresis. Current research has

demonstrated that even with 1mcg most people will also get some alpha or beta stimulation.

- ✓ Moderate concentrations: beta<sub>1</sub> adrenergic receptor agonist positive inotropic effect
- ✓ High concentrations: alpha<sub>1</sub> adrenergic receptor agonist potent vasoconstriction

✎ **Hemodynamics** (dose dependent)

- ✓ Low concentration: increase in UO, maybe some increase in HR or SBP (current research has not shown this to be renally protective)
- ✓ Moderate concentrations: increase in HR, SBP, CO (mild)
- ✓ High concentrations: increase in SBP, DBP, SVR

✎ **Mixing and Dosing**

- ✓ Typical 400mg/250 D5W or NS
- ✓ Dosed in mcg/kg/min
- ✓ 1-3mcg/kg/min low dose
- ✓ 3-5mcg/kg/min mid dose
- ✓ 5-10mcg/kg/min high dose
- ✓ > 10 pure alpha dose

## 2. **Norepinephrine Bitartrate (Levophed)**

✎ **Therapeutic Use**: Endogenous catecholamine with powerful inotropic and peripheral vasoconstriction effects. Typically not utilized as first line drug due to strong vasoconstrictive properties.

✎ **Pharmacokinetics**: IV administration only with short half life

✎ **Pharmacodynamics**:

- ✓ Potent  $\alpha_1$  &  $\alpha_2$  agonist
- ✓ Mild  $\beta_1$  agonist
- ✓ No effect on  $\beta_2$
- ✓ Systemic arterial and venous constriction
- ✓ Coronary flow increases slightly

✎ **Hemodynamics**

- ✓ Increase in SBP & DBP
- ✓ Increase in SVR and PVR
- ✓ Cardiac output unchanged or decreased (increase in afterload)
- ✓ Heart rate may slow from compensatory vagal reflex

✎ **Dosing and Mixing**

- ✓ Typical 4mg/250 D5W
- ✓ Dosed in mcg/min
- ✓ 2-10mcg/min

### 3. **Epinephrine Hydrochloride**

✓ **Therapeutic Use:** Endogenous catecholamine with powerful inotropic, peripheral vasoconstriction effects and inotropic properties. Typically not utilized as first line drug due to profound vasoconstrictive and subsequent side effects.

✓ **Pharmacokinetics**

- ✓ Short half life with rapid onset

✓ **Pharmacodynamics**

- ✓ Alpha and Beta agonist
- ✓ Increases myocardial contractility
- ✓ Vasoconstriction (all beds)
- ✓ Increases myocardial O<sub>2</sub> consumption

✓ **Hemodynamics**

- ✓ Increases HR, MAP, CO, SVR, PVR
- ✓ Pro-arrhythmic

✓ **Mixing and Dosing**

- ✓ Typical 2mg/250 D5W or NS up to 8mg/250 Dosed in mcg/min
- ✓ 1-4mcg/min

### 4. **Vasopressin**

✓ **Therapeutic Use:** is a naturally occurring antidiuretic hormone. In unnaturally high doses it functions as a non-adrenergic peripheral vasoconstrictor. Major use is as a first line agent in ACLS for pulseless VT/V-fib. Shown to reduce or eliminate the need for catecholamine administration.

✓ **Pharmacokinetics**

- ✓ IV administration only
- ✓ Half life 10-20 min

✓ **Pharmacodynamics**

- ✓ Direct stimulation of smooth muscle V<sub>1</sub> receptors
- ✓ Smooth muscle constriction: pallor of skin, nausea, intestinal cramps, desire to defecate, bronchial constriction, uterine contraction

- ✓ Less constriction of coronary and renal vascular beds and vasodilation of cerebral vasculature
- ✓ No skeletal muscle vasodilation or increased myocardial O<sub>2</sub> consumption during CPR because there is no Beta-adrenergic activity
- ✓ May enhance platelet aggregation in septic shock

✎ **Hemodynamics**

- ✓ Increase in SBP, MAP and SVR
- ✓ Increase UO

✎ **Mixing and Dosing**

- ✓ Typical 200U/250 D5W or NS
- ✓ Dosed in unit/min
- ✓ 0.2-0.9U/min

## 5. **Phenylephrine (Neo-synephrine)**

- ✎ **Therapeutic Use:** Pure and powerful alpha agonist. Used when no beta stimulation is wanted or needed.

✎ **Pharmacokinetics**

- ✓ Short half life

✎ **Pharmacodynamics**

- ✓  $\alpha_1$  receptor agonist

✎ **Hemodynamics**

- ✓ Increase BP, SVR, PVR
- ✓ Strong increase in afterload
- ✓ Coronary vasoconstriction

✎ **Mixing and Dosing**

- ✓ Typical 10mg/250 D5W or NS up to 8mg/250
- ✓ Dosed in mcg/min
- ✓ 40-60mcg/min

## **Dobutamine**

- ✎ **Therapeutic Use:** Synthetic catecholamine which has selective beta adrenergic agonist properties. Effective as a positive inotropic for both preload and afterload reduction. Used for its positive inotropic properties when vasoconstriction is not preferable. Also used commonly as a combination therapy with another catecholamine or vasodilator

✓ **Pharmacokinetics**: IV administration only, half life 2 minutes – rapid onset

✓ **Pharmacodynamics**

- ✓  $\beta_1$  adrenergic receptor agonists: increases contractility and stroke volume, increases sinus node automaticity and AV conduction, increases in myocardial oxygen demand
- ✓ Mild  $\beta_2$  adrenergic receptor agonist: mild vasodilation, increased perfusion
- ✓ Mild  $\alpha_1$  vasoconstriction properties are counter balanced by  $\beta_2$  properties
- ✓ Does not cause release of endogenous norepinephrine
- ✓ Infusions of > 72 hrs have shown tolerance 2<sup>0</sup> to down regulation of  $\beta$  adrenergic receptors
- ✓ Less effective in patients receiving  $\beta$  blocking agents or with chronic heart failure

✓ **Hemodynamics**

- ✓ Increase CO
- ✓ Mild decrease in SVR
- ✓ Mild increase in HR (sometimes more than mild)

✓ **Mixing and Dosing**

- ✓ Typical 500mg/250 D5W or NS
- ✓ Dosed in mcg/kg/min
- ✓ 2-10mcg/kg/min

**7. Milrinone (Primacor)**

✓ **Therapeutic Use**: Synthetic noncatecholamine agent that does not stimulate or block adrenergic receptors. Inhibits the phosphodiesterase III enzyme. Effective as a positive inotrope and vasodilator.

✓ **Pharmacokinetics**

- ✓ IV administration only
- ✓ Hepatically cleared
- ✓ Half life 2-3 hours

✓ **Pharmacodynamics**

- ✓ Phosphodiesterase III enzyme inhibitor – increases cyclic adenosine monophosphate (cAMP) which enhances calcium entry into the cell and improves myocardial contractility, and inhibiting vasoconstriction (vasodilator).

- ✓ Increased cardiac output by positive inotropic action and reduction in preload and afterload
- ✓ Most effective with patients who have over stimulated sympathetic system
- ✓ Effective in patients with beta receptor down regulation

✎ **Hemodynamics**

- ✓ Increase in CO
- ✓ Decrease in CVP, SVR, PAOP
- ✓ No significant effect on HR or BP (unless compensatory)

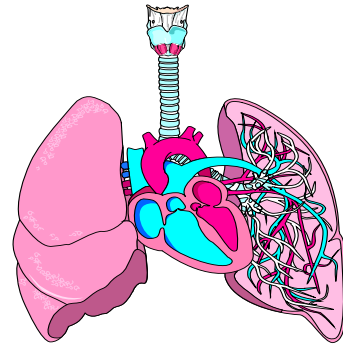
✎ **Mixing and Dosing**

- ✓ Mix with NS ONLY
- ✓ Dosed in mcg/kg/min
- ✓ Loading dose 50mcg/kg over 10 min
- ✓ 0.375-0.75mcg/kg/min

**Review ALL ACLS Drugs and Algorithms When Studying for the CCRN/CCRN-E**

# ACUTE

## PULMONARY



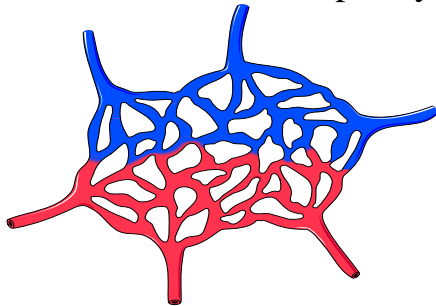
### I. INTRODUCTION

A fundamental loss in alveolar-capillary membrane integrity causes an increased membrane permeability and subsequent pulmonary interstitial edema. The initiating pathology can be either cardiac or non-cardiac in origin.

### II. PATHOGENESIS OF PULMONARY EDEMA

#### A. Alveolar-Capillary Membrane

##### 1. Capillary Endothelial Layer: Microvascular Barrier



A massive network of microvascular membranes (capillaries) that allow for water, lipid-insoluble molecules, macromolecules, and most importantly, gas exchange to take place. The overlapping spaces between the endothelial cells are termed clefts and are considered 'loose junctions' because their width can be increased easily allowing for more extravasation.

##### 2. Interstitial and Lung Lymphatics:

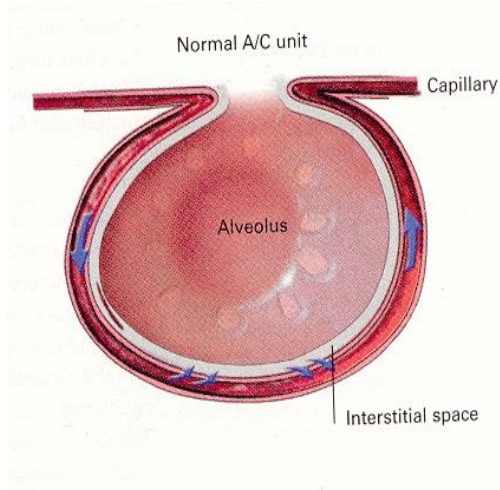
The pulmonary interstitium and lung lymphatic channels drain extravasated fluid and proteins from the lung along the lymphatic ducts. The drainage is then pumped via thoracic ducts into the superior vena cava. This system, which is estimated to drain approximately 20ml/hr in the average adult, functions to decrease fluid accumulation in the lung.

##### 3. Alveolar Epithelial Layer: Alveolar Barrier

The alveolar epithelial cells form a barrier between the microvascular network and the lung unit. This layer of cells, unlike the capillary endothelial cells, form very tight junctions not allowing much transport of anything, except gas, across the membrane.



## B. Fluid Dynamics of the Alveolar-Capillary Membrane

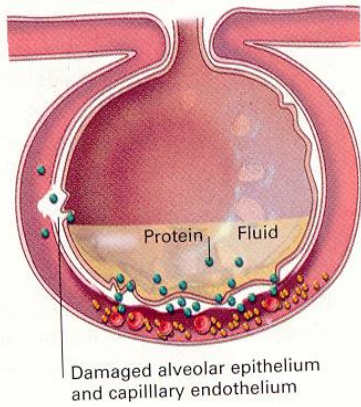


### – 1. Hydrostatic Pressure

The hydrostatic pressure is the pressure exerted within a compartment and typically causes fluid to move out. The transvascular hydrostatic pressure is the difference between the microvascular and interstitial hydrostatic pressures and this gradient determines whether fluid will move from the capillary to the interstitium or in the opposite direction. Under normal physiological conditions there is a higher hydrostatic pressure in the microvascular bed than the interstitium allowing for a net loss of fluid from the capillaries. This fluid is then drained by the lymphatic system. The fluid extravasation is greater in the basilar regions because pulmonary blood flow is gravity dependent. The pulmonary artery occlusion pressure (PAOP) or wedge pressure is a reflection of the microvascular hydrostatic pressure.

### 2. Colloidal Osmotic Pressure

The colloidal osmotic (oncotic) pressure is the 'holding in' pressure gradient. The presence of plasma proteins and macromolecules helps to 'hold' fluid within a particular compartment. Because these large molecules cannot cross cell membranes easily, they tend to stay within the vascular space. Therefore, the colloidal osmotic pressure is typically greater within the pulmonary microvascular circulation than within the interstitium creating a net flow of fluid toward the capillary.



### 3. Membrane Permeability

The movement of fluid across a cell membrane is regulated by the permeability of the membrane. The loose junctions of the microvascular endothelial layer allow for high permeability. The tight junctions of the alveolar epithelial layer allow for low permeability. There are many factors that can change the relative permeability of a membrane. When membrane permeability changes, so will fluid movement. Alveolar injury, from a direct cause such as gastric aspiration or indirect cause such as sepsis, can dramatically change alveolar epithelial membrane permeability.

## III. ETIOLOGY OF PULMONARY EDEMA

- A. Cardiogenic Pulmonary Edema:** Cardiogenic pulmonary edema, which is the most common type of acute pulmonary edema, occurs when there is an increase in hydrostatic pressure within the pulmonary capillary bed as a result of heart failure.

#### Causes Include:

- ♥ Heart Failure
- ♥ Myocardial Infarction
- ♥ Cardiac Ischemia
- ♥ Acute Mitral Regurgitation
- ♥ Cardiac Tamponade
- ♥ Tachy Dysrhythmias
- ♥ Hypertensive Crisis

- B. Non-Cardiogenic Pulmonary Edema:** Non-cardiogenic pulmonary edema results from one of four primary abnormalities (or a combination thereof):

1. Impaired endothelial integrity
2. Decreased colloidal oncotic pressure
3. Elevated capillary hydrostatic pressure
4. Lymphatic obstruction

The impaired endothelial integrity (change in permeability) is typically caused by a direct or indirect injury to the lung tissue. Acute respiratory distress syndrome (ARDS) is a form of non-cardiogenic pulmonary edema.

### **Causes Include:**

- ♥ Sepsis (#1 cause)
- ♥ Shock (Hypoperfusion States)
- ♥ Systemic Inflammatory Response Syndrome (SIRS)
- ♥ Pulmonary Contusion
- ♥ Trauma
- ♥ Intravenous Fluid Overload
- ♥ Pulmonary, Fat or Amniotic Embolism
- ♥ Smoke or Toxic Chemical Inhalation
- ♥ Pulmonary Aspiration
- ♥ Near Drowning
- ♥ Narcotic Overdose
- ♥ Pancreatitis
- ♥ Severe Anemia
- ♥ Disseminated Intravascular Coagulation
- ♥ High-Altitude Sickness
- ♥ Eclampsia
- ♥ Cardiopulmonary Bypass
- ♥ Anesthesia

## **IV. DIAGNOSIS**

### **A. History**

#### Cardiogenic Pulmonary Edema

- ♥ Acute Cardiac Event
- ♥ Chest Pain
- ♥ Tachy-Palpitations
- ♥ New Dysrhythmia
- ♥ History of Ischemic Heart Disease
- ♥ Acute CP and/or SOB in the absence of any other pathologies
- ♥ Absence of Cardiac Hx does not rule out CPE

### **B. Physical Exam**

- ♥ Dyspnea, Tachypnea & Apprehension: assessed in both types
- ♥ Presence of S<sub>3</sub> Heart Sound: more commonly assessed in cardiogenic
- ♥ Jugular Venous Distension: more commonly assessed in cardiogenic
- ♥ Breath Sounds: Bibasilar rales more commonly assessed in cardiogenic

- ♥ Increased Frothy Sputum Production: more commonly assessed in cardiogenic
- ♥ Laterally Displaced Point of Maximal Impulse (PMI): more commonly assessed in cardiogenic
- ♥ New or Louder Cardiac Murmur: more commonly assessed in cardiogenic
- ♥ Unilateral Lung Adventitious Sounds: more commonly assessed in non-cardiogenic
- ♥ Diffuse Decreased Breath Sounds: more commonly assessed in non-cardiogenic
- ♥ Peripheral Edema is non specific

**C. Chest X-Ray**

There are chest x-ray changes that are unique to cardiogenic and non-cardiogenic pulmonary edema. If the pattern changes from day to day or significantly after treatment it is more indicative of cardiogenic.

Chest X-Ray alone cannot be utilized to distinguish cardiogenic from non-cardiogenic pulmonary edema.

<b>CARDIOGENIC</b>	<b>NON CARDIOGENIC</b>
▲ Opacity in PA area due to enlargement of pulmonary vasculature	Diffuse bilateral, fluffy, infiltrates. Periphery and central portions are equally involved
Upper lobe diversion	'White-out' diffuse airspace disease pattern, large diffuse infiltrates
Presence of Kerley B lines in lung periphery from interstitial edema (PAOP > 19mmHg) and swollen lymphatics	Normal cardiac size
Left ventricular enlargement displacing cardiac apex downwards and laterally	X-Ray changes lag behind (12-24 hr) clinical symptoms
Right sided pleural effusion	
Hilum may appear distended and nearby vessels may be blurred. 'Central involvement'	

From Lohrman, J. (1999). Chest and Abdominal X-ray Interpretation. Barbara Clark Mims Seminar. Reprinted with Permission

**D. ECG Changes:** Tachycardia or acute ST-T segment changes are more commonly assessed in cardiogenic pulmonary edema

**E. Echocardiography:** A Transthoracic echo may be helpful to identify myocardial ischemia, wall motion abnormalities, ventricular dysfunction, valvular disease, and LV hypertrophy, all of which will suggest cardiogenic pulmonary edema.

**F. Pulmonary Artery Catheter Pressure Readings:**

The PA readings are very helpful in evaluating cardiac and pulmonary pressures. In cardiogenic causes the picture will be that of heart failure and pulmonary congestion. In non-cardiogenic causes no heart failure is present but pulmonary vasoconstriction or hypertension is usually evident. Care must be taken to evaluate the waveforms/pressures according to whether the patient is intubated or not. Also, the patient on increased levels of PEEP will have higher capillary hydrostatic pressures and potential cardiac compromise.

**G. Laboratory Data:**

1. Arterial Blood Gas:

- ♥ Low Oxygen Saturation
- ♥ Respiratory Alkalosis
- ♥ Refractory Hypoxemia

**V. TREATMENT OPTIONS**

**A. Cardiogenic Pulmonary Edema (ACLS algorithm):** while treating the underlying cardiac condition

- ♥ Diuretics: Furosemide IV 0.5 to 1.0 mg/kg
- ♥ Analgesics: Morphine IV 2 – 4mg
- ♥ Preload Reduction: Nitroglycerin SL
- ♥ Oxygen/Intubation
- ♥ Afterload Reduction if SBP > 100mmHg
  - ✓ IV Nitroglycerin 10-20 µg/min or consider IV Nitroprusside 0.1 – 5.0 µg/kg/min
- ♥ Vasoconstriction if Hypotensive
  - ✓ Severe (SBP < 70mmHg) and S&S of shock  
IV Norepinephrine 0.5 – 30 µg/min,
  - ✓ Moderate (SBP 70– 100mmHg) with S&S of shock  
IV Dopamine 5 – 15 µg/kg/min
  - ✓ Moderate without shock  
IV dobutamine 2 – 20 µg/kg/min
- ♥ Further Diagnostic Considerations
  - ✓ PA Catheter
  - ✓ Intra-Aortic Balloon Pump
  - ✓ Cardiac Angiography

**B. Non Cardiogenic Pulmonary Edema (ARDS)**

- ♥ Treat the Underlying Cause

- ♥ Block the Specific Mechanism of Alveolar Capillary Membrane Injury
- ♥ Minimize Pathologic Consequences of Acute Lung Injury
- ♥ Supportive Treatments

## **VI. SUMMARY**

# AORTIC ANEURYSMS & ACUTE AORTIC DISSECTION

## I. ARTERIAL ANATOMY

### A. Arteries

**Intima:** inner layer of vessel, primarily endothelium and connective tissue

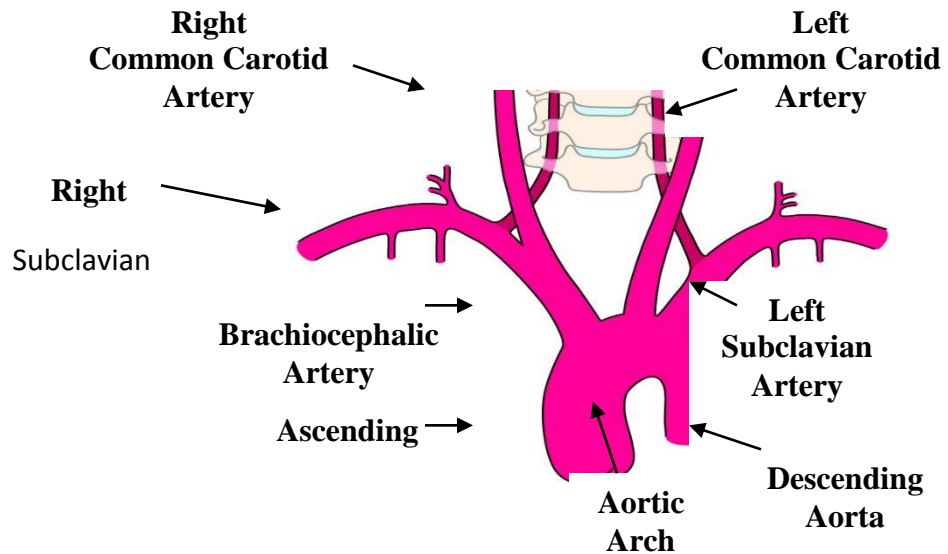
**Media:** thick middle layer of connective tissue, elastic and smooth muscle. Major portion of arterial wall.



**Adventitia:** thin outer layer of connective tissue

**Vasa Vasorum:** small blood vessels that supply blood to the walls of the arteries. A network of capillaries embedded in the adventitia supplying blood to the media layer.

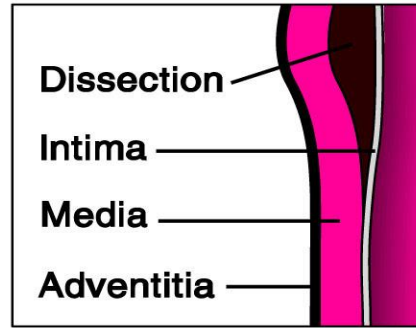
### B. Aorta



**II. DEFINITIONS:** these conditions can overlap at times with one leading to or increasing the risk of the other.

**A. Aortic Dissection:** a longitudinal separation of the aortic wall between the intima and the adventitia. An acute dissection is one that is diagnosed within 14 days of the onset of symptoms. The risk of

death is greatest during this acute period. A chronic dissection is one that is diagnosed after two weeks of the onset of symptoms.



**B. Aortic Aneurysm:** a localized dilation of the arterial wall that can be saccular, fusiform or cylindrical. The dilation frequently renders the aorta weak in that region.



### Complication of Aneurysms

### Management of Aneurysms

**C. Aortic Transection:** a laceration of the entire arterial wall, typically caused by a traumatic event.

## III. CLASSIFICATION SYSTEMS

**A. Stanford Classification System:**

1. **Type A Aortic Dissection:** the dissecting area involves the ascending aorta. It may be confined to only the ascending aorta or may also involve the descending as well. Typically occur in a younger patient population with a congenital weakening of

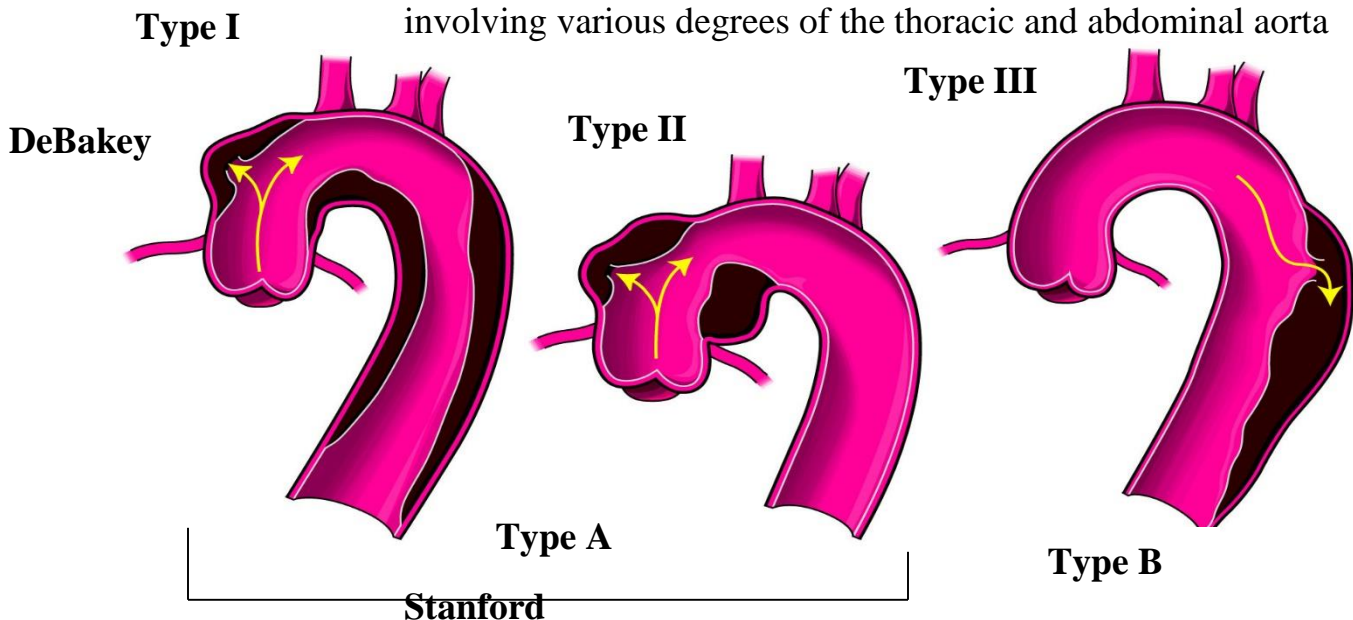


the ascending aorta. Type A dissection account for 2/3 of all dissections.

2. **Type B Aortic Dissection**: the dissecting area involves only the descending aorta distal to the left subclavian artery. Typically occurs in the older patient population with a history of hypertension and atherosclerosis.

### B. DeBakey Classification System:

1. **Type I Aortic Dissection**: the dissection involves the ascending aorta but also extends beyond the left subclavian artery.
2. **Type II Aortic Dissection**: the dissection involves only the ascending aorta.
3. **Type III Aortic Dissection**: the dissection involves only the descending aorta. IIIa limited to the thoracic aorta, IIIb involving various degrees of the thoracic and abdominal aorta



## IV. COMMON RISK FACTORS

Although hypertension does not appear to be the sole contributor to the occurrence of AAD, it appears to play a major role in the development and/or propagation of a dissection. The etiology of AAD is believed to be a combination of something that has caused a weakening in the vessel that 'allows' the original tear to occur and that, in combination of HTN (70-90% of the victims have a history of HTN) triggers the filling of the false lumen and dissection of the arterial layers.

- ♥ Cystic Medial Necrosis
- ♥ Marfan's Syndrome (a chromosomal mutation with many genotypes and phenotypes)
- ♥ Annuloaortic Ectasia
- ♥ Aortic Dilatation and Wall Thinning
- ♥ Unicommissural or Bicuspid Aortic Valve (congenital malformation)
- ♥ Spontaneous Rupture of Vasa Vasorum
- ♥ Aortic Coarctation
- ♥ Trauma (blunt, penetrating or iatrogenic)
- ♥ Aging
- ♥ Arterial Hypertension
- ♥ Cocaine Use

## V. DIAGNOSIS

### A. Presenting Signs & Symptoms:

AAD is Known as the Great Imitator

- ♥ Sudden Severe Pain Not Relieved with Analgesics
  - ✓ Chest Pain (sharp): retrosternal or interscapular chest pain is more commonly associated with ascending dissections
  - ✓ Severe Flank Pain: commonly associated with descending dissections
  - ✓ Epigastric Pain: commonly associated with descending dissections
- ♥ Initially Normal or High Blood Pressure
- ♥ Hypotension
- ♥ Acute Aortic Valvular Insufficiency: High-Pitched, Blowing Diastolic Murmur
- ♥ Audible S<sub>3</sub> Heart Sound
- ♥ Abrupt Onset of a Pulseless Extremity
- ♥ Peripheral Vascular Insufficiency
- ♥ End-Organ Ischemia (brain, kidney, intestines, spinal, lower extremities)
- ♥ Pericardial Effusion
- ♥ Cardiac Tamponade
- ♥ Acute Myocardial Ischemia

### B. Tests

- ♥ Chest X-ray
  - ✓ Wide mediastinum
  - ✓ Wide aortic silhouette
  - ✓ Pleural effusion

- ✓ CHF
- ✓ Pericardial effusion (cardiomegaly)
- ♥ ECG: Nonspecific Changes
  - ✓ Left ventricular hypertrophy
  - ✓ Acute myocardial ischemia
- ♥ Chest CT: Sensitive test. Need scan with contrast medium to clearly identify location of tear
- ♥ **Transthoracic** or Transesophageal (TEE) Echocardiography: Rapid, noninvasive, bedside test that can reveal dissection, aortic regurgitation, pericardial effusion and cardiac dyskinesia. The TEE is not as helpful in viewing the descending aorta and branches.
- ♥ Magnetic Resonance Imaging (MRI) Scan: a highly sensitive test but difficult in the critically ill due to time and metal restrictions.
- ♥ Aortography: historically the gold standard, a very sensitive and specific test has been replaced with less invasive procedures for most diagnoses.

## VI. TREATMENT

### A. Adequate Blood Pressure Management

- ♥  $\alpha$  blocker &  $\beta$  blockers: Labetalol
- ♥ Calcium Channel Blocker: Nicardipine Hydrochloride/ Cardene
- ♥ Negative Inotropic Agents:  $\beta$  blockers (Esmolol)
- ♥ Pharmacological management may be the primary treatment for a dissection involving the descending aorta

### B. Pain Relief: typically done with Morphine

### C. Reduction of Environmental and Emotional Stresses: May need anti-anxiety agents

### D. Surgical Repair

- ♥ All patients with a dissection involving the ascending aorta should be immediately referred to surgery
  - ✓ Resection of the tear and false lumen and placement of a prosthetic graft

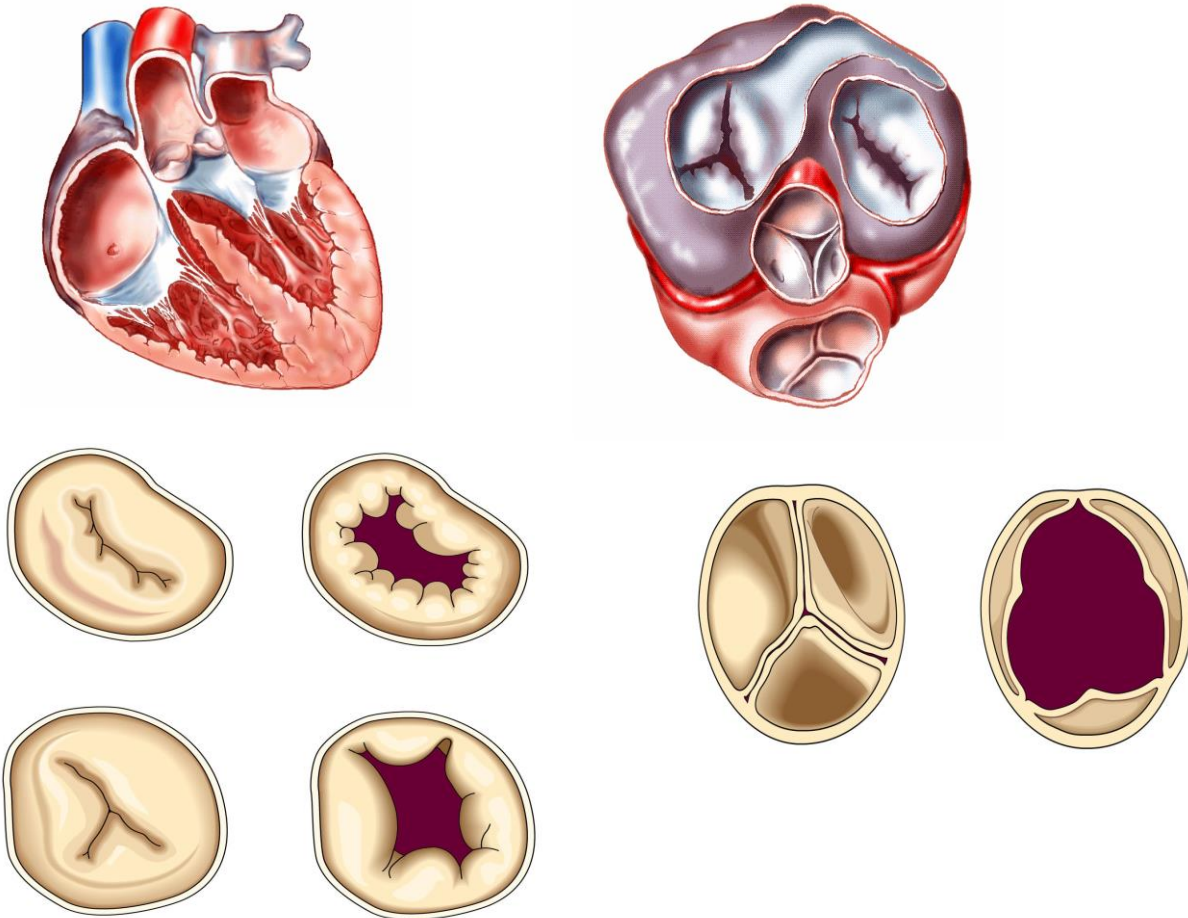
- ✓ Aortic arch repair
  - ✓ Resuspension or replacement of the aortic valve
  - ✓ Bentall procedure is the replacement of the aortic valve and arch – commonly done with a cadaver graft
- 
- ♥ Surgical repair is recommended for all Marfan's patients with a dissection of the ascending or descending aorta
  
  - ♥ Descending dissections are commonly treated with pharmacological management because resection and even repair of the descending aorta carries very high mortality and morbidity. Elective surgical repairs are recommended in patients with an aortic diameter that exceeds five cm, the presence of uncontrolled hypertension and/or when occlusion of the major aortic branches has occurred (renal or mesenteric)
  
  - ♥ Ascending repairs are accomplished via a medialstinal approach and descending via a left thoracotomy incision.
  
  - ♥ Cardiopulmonary bypass is needed for ascending repairs but an aortic cross-clamp method is typically utilized for descending repairs.
  
  - ♥ A percutaneous placement of an endovascular stent and balloon fenestration of the intimal flap has been used for descending dissections with success

## **VII. SUMMARY**

# Structural Heart Defects

## Valvular Defects

### I. Introduction



### II. Pathophysiology

- A. Congenital Malformations
- B. Connective Tissue Disorders
- C. Degenerative Disease
- D. Rheumatic Heart Disease
- E. Infective Endocarditis
- F. Dysfunctional Ruptures

### III. Specific Valvular Dysfunction

- A. Mitral Stenosis
- B. Mitral Insufficiency/Regurgitation

- C. Aortic Stenosis
- D. Aortic Insufficiency/Regurgitation

#### **IV. Management of Valve Disorders** (pre op and post op)

- A. Oxygenation
- B. Hemodynamic Stability
- C. Dysrhythmias
- D. Activity
- E. Anticoagulation
- F. Antibiotic Prophylaxis
- G. Patient/Family Education

#### **V. Surgical Management of Valve Defects**

##### **A. Indications**

##### **B. Valve Repairs**

- ♥ Valve Leaflet Reconstruction
- ♥ Chordae Tendinae Reconstruction

##### **C. Prosthetic Valve Replacement**

- ♥ Mechanical
- ♥ Biological

#### **VI. Summary**

# ENDOCARDITIS

## I. Pathophysiology/Definition

Infective Endocarditis (IE) is a microbial infection of the endothelial surface of the heart.

## II. Risk Factors

- ♥ RHD
- ♥ Congenital Heart Disease
- ♥ Any Valve Disease
- ♥ Marfan's Syndrome
- ♥ Cardiac or Valve Surgery
- ♥ Aortic Grafts
- ♥ Alcoholism
- ♥ Chronic Hemodialysis
- ♥ Immunosuppression
- ♥ Severe Burns
- ♥ IV Drug Abuse

## III. Clinical Assessment

- ♥ Fever
- ♥ Splenomegaly
- ♥ Hematuria
- ♥ Petechiae
- ♥ Cardiac Murmurs
- ♥ Fatigue
- ♥ Osler Nodes: tender, reddish or purplish subcutaneous nodules on the ends of fingers or toes (soft tissue).
- ♥ Splinter Hemorrhage in nail Beds

## IV. Treatment

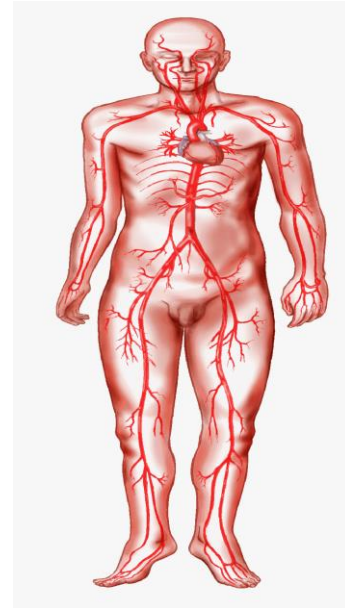
- ♥ Prevention
- ♥ Resolve Infection
- ♥ Long ABX Course
- ♥ Surgery

# Peripheral Vascular Disease & Surgery

## I. Introduction

## II. Peripheral Vascular Disease

- A. Pathophysiology
- ✓ Smoking
  - ✓ HTN
  - ✓ DM
  - ✓ Lipid Disorders
  - ✓ Hyperhomocysteinemia
- B. Assessment
- ✓ Intermittent Claudication
  - ✓ Resting Pain
  - ✓ Cool Temperature
  - ✓ Diminished Pulses
  - ✓ Leg/Skin Changes
- C. Diagnosis
- D. Acute Arterial Occlusion
- 5Ps
- ✓ Pain
  - ✓ Pulselessness
  - ✓ Pallor
  - ✓ Paresthesia
  - ✓ Paralysis
- E. Management/Treatment
- ✓ Risk Factor Modification
  - ✓ Vasodilators
  - ✓ Antiplatelets
  - ✓ Exercise
  - ✓ Angioplasty
  - ✓ Vascular Bypass Surgery
- F. Compartmental Syndrome
- ✓ Compartments are closed spaces containing muscles, nerves, and vascular structures

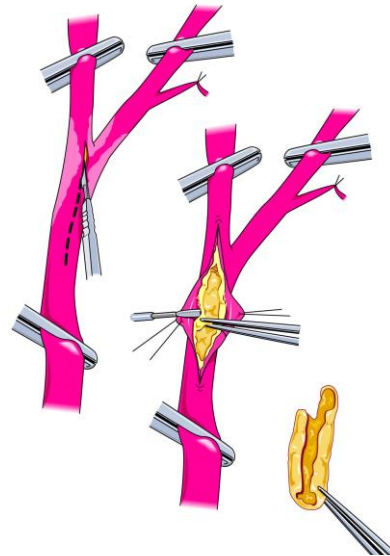




- ✓ Internal and external causes can increase pressure within a compartment
- ✓ Increased pressure can lead to ischemia, injury and necrosis to the contents within the compartment
- ✓ Signs and Symptoms of CS
  - ⇒ Throbbing Pain (localized)
  - ⇒ Firmness of area
  - ⇒ Altered Sensation: Numbness, tingling, sticking feelings
  - ⇒ Pulselessness
  - ⇒ Decreased Voluntary Limb Movement
- ✓ Treatment for CS
  - ⇒ Eliminate the Cause
  - ⇒ Pain Management
  - ⇒ Fasciotomy

### III. Carotid Artery Disease

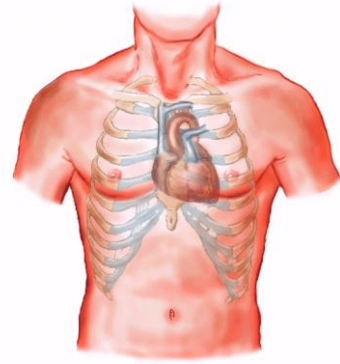
- A. Pathophysiology
- B. Assessment
- C. Diagnosis
- C. Management/Treatment
  - ✓ Risk Factor Modification
  - ✓ Vasodilators
  - ✓ Antiplatelets
  - ✓ Neuro Monitoring
  - ✓ Carotid Endarterectomy



### IV. Summary

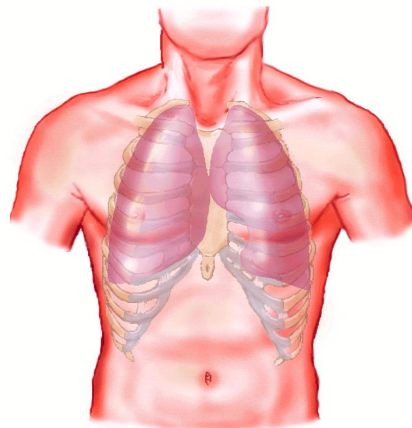
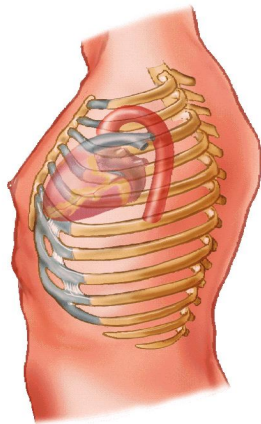
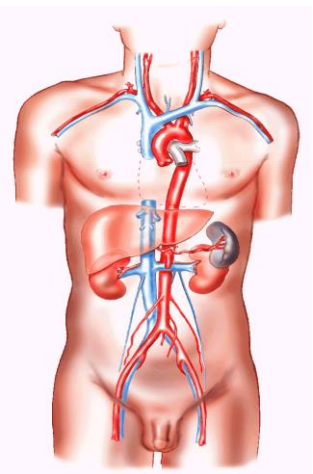


# CHEST TRAUMA



## I. INTRODUCTION

### Thoracic Anatomy



## II. MECHANISM OF INJURY

### A. Penetrating

#### ♥ High-Energy: Ballistic-Type

- ✓ Gunshot Wounds
- ✓ Arrow Wounds
- ✓ Explosions
- ✓ Impalements

#### ♥ Low-Energy: Stabbings and Slashings

- ✓ Knives
- ✓ Swords
- ✓ Ice Picks & Wire

### B. Blunt

- ♥ Motor Vehicle Crashes
- ♥ Falls
- ♥ Assaults
- ♥ Pedestrians Struck

#### ♥ Deceleration

#### ♥ Acceleration

#### ♥ Acceleration/Deceleratio

#### ♥ Shearing

#### ♥ Compression

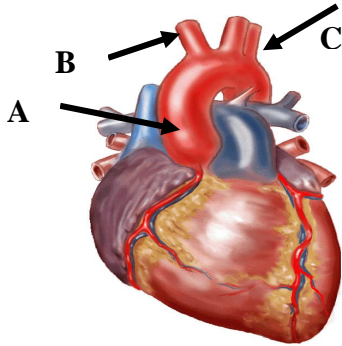
### III. COMMONN CARDIAC INJURIES

- A. **Penetrating Injuries**: The injury is directly related to the penetrating object, its velocity and the anatomical structures penetrated. The Right Ventricle has the highest incidence of injury followed by the Left Ventricle, Right Atrium and Left Atrium because of their respective locations within the chest cavity.
- B. **Cardiac Rupture**: Pt may present with or without a pulse, in cardiogenic shock, or with a new onset loud murmur depending on the degree and location of the rupture. These injuries are more likely to occur if the blunt force is encountered near the apex of the heart during late diastole or early systole. These injuries require surgical repair – if the patient is found alive.
- ♥ Free Ventricular Wall: Left > Right
  - ♥ Septum
  - ♥ Chordae Tendineae or Papillary Muscles
- C. **Cardiac Contusion**: Bruising of the myocardium due to rupture or hemorrhage of small vessels. The contusion is epicardial rather than transmural injury like an MI. This is a very difficult diagnosis and one without standard medical criteria. There is no ‘typical’ picture of a contusion. There is myocardial dysfunction, temporary or permanent, ischemia or necrosis without evidence of coronary artery occlusion. The right ventricle is the most common location for the injury. A pulmonary contusion may accompany cardiac contusion.
- ♥ History of blunt trauma
  - ♥ Chest Pain that does not travel to left arm or jaw, unrelieved by vasodilators
  - ♥ SOB
  - ♥ Low CO, BP, and tachycardia
  - ♥ Muffled heart sounds
  - ♥ Dysrhythmias: occur within first 3 days
  - ♥ Right heart failure “picture”
  - ♥ ECG changes: may be nonspecific ST-T abnormalities – not necessarily in contiguous leads, may also see common ACS changes
  - ♥ CK-MB levels typically rise but are not helpful, the absence of troponin elevation helps to rule out ACS
  - ♥ Transthoracic Echo may reveal dysfunctional area and also any additional injuries
  - ♥ Treatment

- ✓ Small Contused Area: may not need treatment
- ✓ Medium Contused Area: Cardiac dysfunction usually responds to fluid and inotropic therapy
- ✓ Large Contused Area: may need mechanical support, IABP or even possibly reperfusion surgery (rare)

D. **Cardiac Tamponade**: Can be caused by blunt or penetrating injuries. Covered extensively in next lecture.

E. **Traumatic Aortic Dissection**: Aortic disruption is the leading cause of death from thoracic trauma. There are four common sites of injury.



**A. Ascending Aorta**

**B. Innominate Artery from Aortic Arch**

**C. Left Subclavian Artery**

**D. Lower Thoracic Aorta Above the Diaphragm**

**A complete transection can occur or a dissection may develop. With a transection the person will most likely be dead the scene.**

# HYPERTENSIVE EMERGENCIES

## I. INTRODUCTION

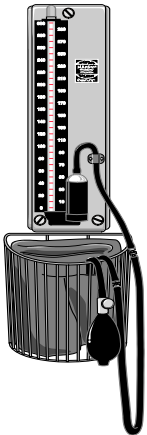
## II. PATHOPHYSIOLOGY OF HYPERTENSION

### A. Definition:

**2104 Evidence-Based Guideline for Management of High BP** 8<sup>th</sup> Joint Nation committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (2013). JAMA published on line 12/18/13

Population	Goal BP mm/hg	Initial Drug Tx Options
General $\geq$ 60 yo	< 150/90	Nonblack: thiazide-type diuretic, ACEI, ARB, or CCB
General $\leq$ 60 yo	< 140/90	Black: thiazide-type diuretic or Calcium channel blocker (CCB)
Diabetes	< 140/90	Thiazide-type diuretic, ACEI, ARB or CCB
Chronic Kidney Dis	< 140/90	ACEI or Angiotensin receptor blocker (ARB)

Table 6, pg E12 JAMA and AMA



### B. Causes:

1. **Primary Hypertension:** a chronic progressive disorder without an identifiable cause. Accounts for 92% of hypertension.  
Populations at greatest risk

- ♥ Age > 60 years
- ♥ Obese
- ♥ Physical Inactivity
- ♥ Minority Groups (African American, Mexican Americans & Native American)
- ♥ Family History
- ♥ Cigarette Smoking

2. **Secondary Hypertension:** identifiable source/cause of hypertension

\* major risk factor

- ♥ Dyslipidemia\*
- ♥ Diabetes Mellitus\*
- ♥ Pheochromocytoma
- ♥ Cushing Syndrome
- ♥ Primary Aldosteronism
- ♥ Hyperparathyroidism
- ♥ Renal Parenchymal Disease
- ♥ Medications

- ♥ Renovascular Disease
- ♥ Polycystic Kidneys
- ♥ Aortic Coarctation

**C. Target Organ Damage (TOD)  
Clinical Cardiovascular Disease (CCD)**

- ♥ Heart Disease
  - ⇒ Left Ventricular Hypertrophy
  - ⇒ Angina or Prior Myocardial Infarction
  - ⇒ Prior Coronary Revascularization
  - ⇒ Heart Failure
- ♥ Stroke or Transient Ischemic Attack
- ♥ Nephropathy
- ♥ Peripheral Arterial Disease
- ♥ Retinopathy

**III. MANAGEMENT OF HYPERTENSION**

**A. Lifestyle Modification**

- ♥ Weight Reduction
- ♥ Moderation of Alcohol Intake (< one drink daily – defined as 1oz of ethanol, 24oz of beer, 10oz of wine, or 2oz of 100 proof whiskey)
- ♥ Increase Physical Activity (30 – 45 min most days)
- ♥ Moderation of Sodium Intake (< 2.4 g Na)
- ♥ Maintaining Adequate Potassium Intake ( $\geq 90$  mmol/d)
- ♥ Maintaining Adequate Calcium & Magnesium Intake
- ♥ Smoking Cessation
- ♥ Moderate Caffeine Intake
- ♥ Stress Reduction

**B. Pharmacological Management**

1. **Diuretics** – first line agents for new HTN either as solo therapy or with beta-blockers. Demonstrated with control studies to decrease BP with lowest mortality and morbidity stats. Drug ex. thiazide diuretics Chlorthalidone (Hygroton) and loop diuretics Furosemide (Lasix).
2. **Beta Blockers** – first line agent in HTN, decreases both heart rate and velocity (stroke volume). Drug ex. Metoprolol (Lopressor), Propranolol (Inderal)

4. **Centrally Acting Alpha<sub>2</sub> Agonists** – reduce firing of sympathetic neurons by selective stimulation of alpha<sub>2</sub> receptors in CNS, causes a decrease in alpha and beta-adrenergic stimulation in the periphery. Drug ex. Clonidine (Catapres) & Methylopa (Aldomet).
5. **Adrenergic Neuron Blockers** – reduces the release of norepinephrine (NE) from postganglionic sympathetic neurons, decreases stored NE and blocks synthesis of NE. Drug ex. Reserpine.
5. **Alpha<sub>1</sub> Adrenergic Blockers**: direct vasodilators - block alpha<sub>1</sub> receptors on both arterioles and veins causing vasodilation. Drug ex. Doxazosin (Cardura), Prazosin (Minipress) & Perazosin (Hytrin)
6. **Alpha & Beta Adrenergic Blockers**: Carvedilol & Labetalol have combined alpha and beta-adrenergic blockade.
7. **Direct Vasodilators**: Hydralazine & Minoxidil are selective to arteries and Sodium Nitroprusside relaxes smooth muscle of arterioles and veins.
8. **Calcium Channel Blockers**: there are three classifications of Ca<sup>++</sup> Channel Blocking agents. Each block Ca<sup>++</sup> entrance into cells and depending on selectivity of the agent it will either cause vasodilation or decrease heart rate and contractility or both. Drug ex. Amlodipine (Norvasc), Nifedipine (Procardia), & Nifedipine (Procardia).
9. **Angiotensin Converting Enzyme Inhibitors**: decrease blood pressure by preventing the conversion of AT I to AT II. Drug ex. Benazepril (Lotensin), Captopril (Capoten), & Enalapril (Vasotec).
10. **Angiotensin II Receptor Blockers**: decrease blood pressure by preventing the actions (primarily vasoconstriction and water retention) of AT II at the receptor sites. Drug ex. Losartan (Cozaar) & Candesartan (Atacand).
11. **Combination Agents**: compounds that combine the above actions and work synergistically to decrease blood pressure



with the added benefit of decreasing the number of medications the individual takes daily.

## IV. HYPERTENSIVE CRISIS

### A. Definitions

1. **Hypertensive Crisis**: A diastolic blood pressure greater than 120mmHg. Global term does not denote physiologic response or need for immediate treatment.
2. **Hypertensive Emergency**: A diastolic blood pressure of greater than 120mmHg with acute or ongoing end organ (neurological, cardiac or renal) damage. Immediate blood pressure reduction is required within a few hours to prevent or limit target organ damage. The reduction does not necessarily need to be back to normal pressure just out of the dangerous range.
3. **Hypertensive Urgency**: A diastolic blood pressure of greater than 120mmHg without end organ damage. Reduction of blood pressure is important to limit the risk of potential end organ damage but not emergent. The goal is to bring down the blood pressure within 24 – 48 hours.
4. **Malignant Hypertension**: Described by Volhard and Fahr in 1914, MHT is characterized by severe accelerating hypertension with evidence of renal, neurological, vascular and retinal damage/dysfunction that can be rapidly fatal ending in heart attack, stroke or heart and renal failure. The modern criteria for MHT are severe hypertension (DBP > 120mmHg) associated with retinal hemorrhages, exudates and papilledema (group 4 Keith-Wagener-Barker retinopathy) (Laragh, 2001). Some authors define it simply as elevated BP accompanied by encephalopathy or nephropathy (Varon, 2000).
5. **Accelerated Hypertension**: A more ‘mild’ form of MHT without the presence of papilledema and a group 3 Keith-Wagener-Barker retinopathy.
6. **Post Operative Hypertension**: Defined as systolic blood pressure of greater than 190mmHg and/or diastolic blood pressure of greater than or equal to 100mmHg on two consecutive readings following surgery. Because of the unique



and transient physiological factors following surgery and anesthesia this clinical syndrome is separated from the other hypertensive crises.

7. **Gestational Hypertension:** There are multiple names for this syndrome. A blood pressure is considered an emergency in a pregnant woman and requires immediate pharmacologic management when the systolic pressure is greater than 169mmHg or diastolic greater than 109mmHg.

**B. Etiologies:** There is not one cause of HTN Crisis. A history of preexisting hypertension is the common denominator regardless of the secondary causative factor(s). (List adapted from Chase, 2000 & Vaughan, 2000)

- ♥ Preexisting Hypertension
- ♥ Post Operative
- ♥ Cardiac Pathology
  - ✓ Abrupt Increase in Chronic HTN
  - ✓ Acute Aortic Dissection
  - ✓ Acute Left Ventricular Failure
  - ✓ Acute or Impending MI
  - ✓ S/P Coronary Bypass Graft Surgery
- ♥ Renal Parenchymal Disease
  - ✓ Acute Glomerulonephritis
  - ✓ Vasculitis
  - ✓ Hemolytic Uremic Syndrome
  - ✓ Thrombotic Thrombocytopenic Purpura
  - ✓ Scleroderma and other Collagen Vascular Diseases
- ♥ Renovascular Disease
  - ✓ Renal-Artery Stenosis
- ♥ Pregnancy
  - ✓ Eclampsia
- ♥ Severe Burns
- ♥ Endocrine
  - ✓ Pheochromocytoma
  - ✓ Cushing's Syndrome
  - ✓ Thyroid Storm
  - ✓ Renin-Secreting Tumors
  - ✓ Mineral corticoid Hypertension (rarely)
- ♥ Drugs
  - ✓ Cocaine, Amphetamines, PCP, LSD
  - ✓ Sympathomimetics
  - ✓ Erythropoietin

- ✓ Cyclosporin
- ✓ Antihypertensive Withdrawal (usually centrally acting agents such as clonidine)
- ✓ Interactions w/ Monoamine-Oxidase Inhibitors (Tyramine)
- ✓ Lead Intoxication
- ♥ Autonomic Hyper-Reactivity
  - ✓ Guillain-Barre Syndrome
  - ✓ Acute Intermittent Porphyrin
  - ✓ Autonomic Dysreflexia (from spinal cord injury)
- ♥ Cerebrovascular Conditions
  - ✓ Head Injury
  - ✓ Cerebral Infarction
  - ✓ Intracerebral or Subarachnoid Hemorrhage
  - ✓ Brain Tumors

**D. Pathophysiology:** Although the exact physiological mechanism(s) of hypertensive crisis are unknown, there appears to be a vicious cycle of increased vasoconstriction which leads to increasing pressure. **Pressure-induced natriuresis** (excessive loss of Na<sup>+</sup> in the urine) occurs secondary to the increased pressure leading to volume depletion, which stimulates more vasoconstriction.

**E. Assessment:** In addition to the blood pressure, the presence and relative degree of end organ damage/dysfunction is important to assess for and essential to identify before selecting the appropriate treatment option.

- ♥ Previous Diagnosis of HTN
  - ✓ How long?
  - ✓ Prescribed Medications?
  - ✓ Adherence to Prescription Medication?
  - ✓ General Level of Control or Typical Blood Pressure?
- ♥ All Other Medications: Prescription, Over the Counter, Dietary Supplements and/or Illicit Drugs
- ♥ Cardiac Assessment
  - ✓ Hx of CAD
  - ✓ Hx of Heart Failure
  - ✓ Signs and Symptoms of Chest Pain, SOB, Bibasilar Crackles, S<sub>3</sub>, JVD, Dyspnea, Abdominal Bruits
  - ✓ 12 Lead ECG: look for Ischemia, Injury, Infarction, Rhythm Disturbances or Ventricular Hypertrophy
  - ✓ Measure BP Supine and Standing, Evaluating Fluid Status (pressure induced natriuresis) and in Both Arms
  - ✓ S&S of Aortic Dissection

- ✓ New or Increased Murmur (mitral insufficiency)
- ✓ Chest X-ray Looking for Widened Mediastinum, Pulmonary Edema
- ✓ 2D Echocardiography: Differentiate Diastolic from Systolic Cardiac Dysfunction
- ♥ Renal Assessment
  - ✓ Hematuria
  - ✓ Oliguria
  - ✓ Urine Analysis
  - ✓ Plasma Renin Activity and Aldosterone Levels
  - ✓ S&S of Renal Insufficiency or Failure
- ♥ Neurological Assessment – Hypertensive Encephalopathy
  - ✓ Headache, Restlessness, Weakness, Dizziness
  - ✓ Seizures
  - ✓ Altered LOC
  - ✓ Head CT
  - ✓ Signs of Meningeal Irritation
  - ✓ Visual Fields
  - ✓ Arm Drift
  - ✓ Funduscopic Examination: Retinal Edema, Hemorrhage, Exudate and/or Papilledema
- ♥ Laboratory Data
  - ✓ Complete Blood Count
  - ✓ Peripheral Blood Smear: detect presence of microangiopathic hemolytic anemia - schistocytes
  - ✓ BUN and Creatinine
  - ✓ Blood Chemistry: Metabolic Acidosis, Hypocalcemia, Hypokalemia, Hyponatremia
  - ✓ Thyroid Stimulating Hormone
  - ✓ Blood Glucose
- ♥ Evaluate Secondary Causes
  - ✓ Renovascular Hypertension: Captopril Challenge Test
  - ✓ Pheochromocytoma
  - ✓ Primary Hyperaldosteronism

**F. Treatment for Hypertensive Emergency:** the goal is to reduce the mean arterial pressure by 25% within the first two hours (preferably within the first few minutes) in a controlled, predictable and safe fashion and then toward 160/100mmHg within two to six hours.

**Nitroprusside Sodium (Nipride):** OLD first line drug. Rapid onset of action, arterial and venodilator. Significant side effects and caution use with CNS complication (blurred vision, confusion, tinnitus, or seizures) due to potential for increased intracranial pressure.

**Fenoldopam Mesylate (Corlopam)**: A selective dopamine receptor agonist causes peripheral vasodilatation by stimulating dopamine-1 receptors and increasing renal blood flow and GFR. Caution use in patients with glaucoma because it can increase intraocular pressure. Also need to monitor for dose-related tachycardia.

**IV Vasodilators**: direct dilators, ACE inhibitors, and calcium channel blockers.

Diazoxide (Hyperstat) has a rapid onset but can trigger hyperglycemia, and salt and water retention.

Hydralazine hydrochloride (Apresoline) historically very popular but has been replaced with more controllable and predictable agents.

Enalapril maleate (Vasotec)

**\*\*Nicardipine hydrochloride (Cardene)\*\* first line drug**

Sublingual nifedipine (Procarda) is strongly discouraged because of studies revealing increased mortality.

**IV Adrenergic Inhibitors**: esmolol hydrochloride (Brevibloc),

**\*\*Labetalol hydrochloride (Normodyne, Trandate)\*\* first line drug**

**Diuretics**: should be avoided in HTN emergencies because of the likelihood of hypovolemia from the pressure-induced natriuresis.

Fluid replacement has been shown to actually lower pressure in these patients (Vaughan, 2000).

**G. Treatment for Hypertensive Urgency:** blood pressure should be lowered within 24-48 hours and frequently oral agents are adequate in this patient population.

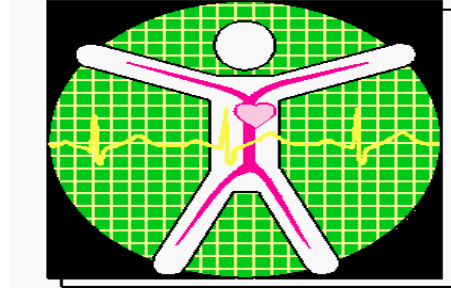
1. ACE inhibitors
2. Calcium Channel Blockers
3. Alpha<sub>2</sub> Adrenergic Stimulators (Clonidine)

## V. SUMMARY

# Dysrhythmias: Self Review

## I. INTRODUCTION

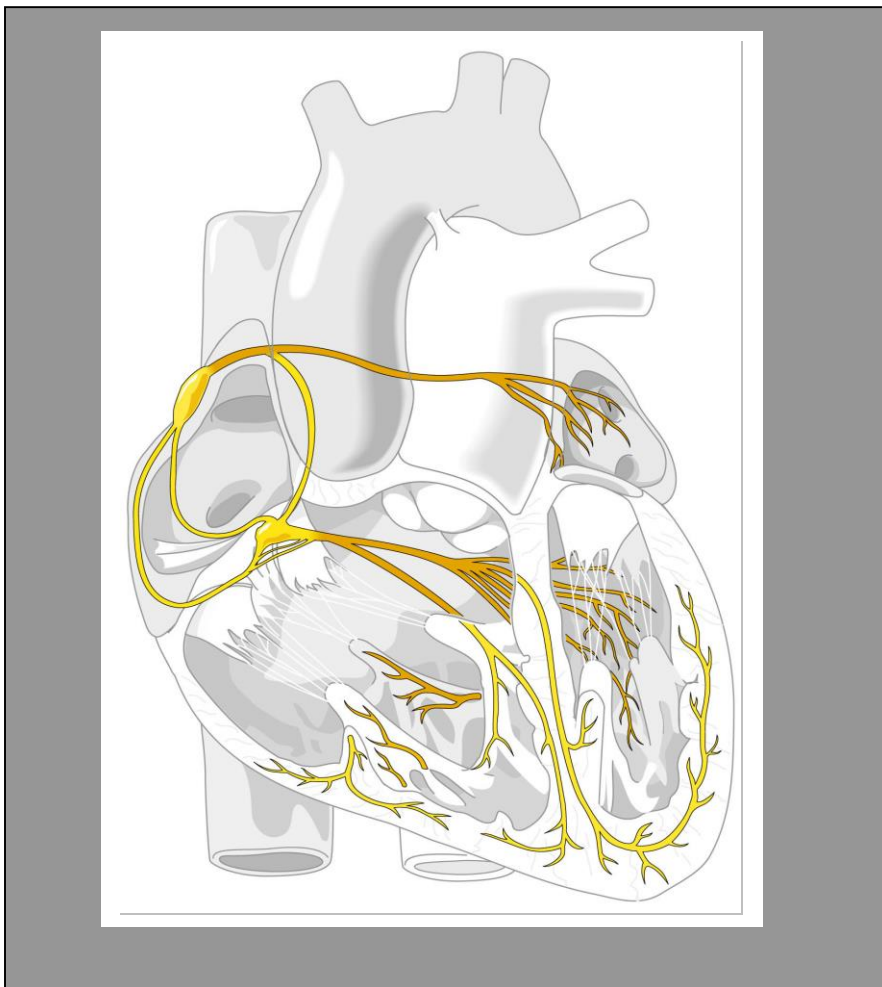
Review basis dysrhythmias. Questions on the exam will be related to rhythm identification, cause or appropriate tx



## II. Cardiac Electrophysiology

### Impulse Conduction & Pathways

SA Node → Internodal Pathways (atrial contraction) → AV Node (delay) → His-Purkinje System (ventricular contraction)



### III. Dysrhythmias

#### A. Common Causes

- ♥ Decreased Coronary Perfusion (CAD)
- ♥ Impaired Myocardial Oxygen Delivery (hypoxia)
- ♥ Electrolyte Disturbances
- ♥ Cardiac Muscle Injury
- ♥ Ischemia or Infarction
- ♥ Defects in the Heart Muscle or Electrical System
- ♥ Cardiac Surgery
- ♥ Electrical Stimulation to the Heart Muscle
- ♥ Medications

#### B. Treatment Options

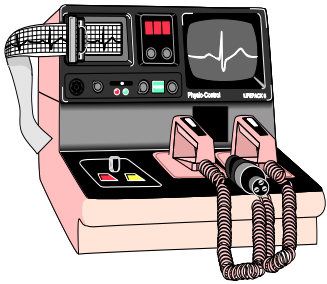
1. Identify & Treat the Underlying Cause

2. Defibrillation/Cardioversion:

The passage of electrical current through the cardiac muscle (cells) causes a massive depolarization allowing the cells to 'reset' themselves and hopefully creating an environment where the SA node can 'take back' the pacemaker function.

3. Pacing: For bradycardic rhythms, electrical stimulation of the heart might be necessary with a transcutaneous, transvenous or permanent pacemaker.

4. Pharmacology: Drugs are the primary treatment if the dysrhythmia is NOT life-threatening. Meaning there is not a significant drop in blood pressure or level of consciousness. The Antidysrhythmic agents are classified using the VaughanWilliams classification system.



DRUG CLASSIFICATION	PRIMARY ACTION	DRUG OPTIONS/ INDICATIONS
<p><b>CLASS I</b></p> <p><b>Sodium Channel Blockers</b></p>	<p>Membrane-Stabilizing</p> <ul style="list-style-type: none"> <li>♥ Slow/Block influx of Na into cell,</li> <li>♥ Effect Stimulus Automaticity, Conduction &amp; Excitability</li> </ul> <p><b>IA</b></p> <ul style="list-style-type: none"> <li>♥ Slows Conduction Velocity</li> <li>♥ Negative Inotrope</li> <li>♥ Prolong Refractory Period (wide QRS &amp; QT)</li> <li>♥ Suppress Ectopic &amp; Reentry Foci</li> </ul> <p><b>IB</b></p> <ul style="list-style-type: none"> <li>♥ Shorten Duration of Action Potential</li> <li>♥ Shorten Refractory Period</li> </ul> <p><b>IC</b></p> <ul style="list-style-type: none"> <li>♥ Slow Conduction Velocity (depress phase 0)</li> <li>♥ Increase Refractory Period</li> <li>♥ Increase QRS Duration</li> </ul>	<p><b>IA</b></p> <p>Quinidine (Quinaglute)  Procaïnamide (Procan, Proestyl)  Disopyramide (Norpace)</p> <p>Atrial Flutter, Atrial Fib, SVT, VT</p> <p><b>IB</b></p> <p>Lidocaine (Xylocaine)  Phenytoin (Dilantin)  Mexiletine (Mexitil)  Tocainide (Tonocard)</p> <p>Ventricular Rhythms</p> <p><b>IC</b></p> <p>Flecainide (Tambocor)  Propafenone (Rythmol)</p> <p>Ventricular Rhythms</p>
<p><b>CLASS II</b></p> <p><b>Beta Blockers</b></p>	<p><b>Antisymphathetic Effects</b></p> <ul style="list-style-type: none"> <li>♥ Inhibit Sympathetic Activity Associated with Beta-Adrenergic Stimulation</li> <li>♥ Decrease Force of Ventricular Contraction</li> <li>♥ Slow SA Node and AV Conduction</li> <li>♥ Decrease in Myocardial Oxygen Consumption</li> </ul>	<p>Propranolol (Inderal)  Acebutolol (Sectral)  Esmolol (Brevibloc)  Sotalol (Betapace)</p> <p>Supraventricular Rhythms</p>
<p><b>CLASS III</b></p> <p><b>Potassium Channel Blockers</b></p>	<p><b>Antiadrenergics</b></p> <ul style="list-style-type: none"> <li>♥ Prolong duration of action potential</li> <li>♥ Delay repolarization</li> <li>♥ Prolong QT interval, AP &amp; effective refractory period</li> </ul>	<p>Bretylium (Bretylol) dropped from ACLS  Amiodarone (Cordarone)  Sotalol (Betapace)  Ibutilide (Corvert)  Dofetilide (Tikosyn)</p> <p>Ventricular and Supraventricular Rhythms</p>
<p><b>CLASS IV</b></p> <p><b>Calcium Channel Blockers</b></p>	<p><b>Calcium Antagonist</b></p> <ul style="list-style-type: none"> <li>♥ Decreased SA node firing</li> <li>♥ Slower conduction → AV node</li> <li>♥ Decrease myocardial O<sub>2</sub> demand &amp; contractility</li> </ul>	<p>Diltiazem (Cardizem)  Verapamil (Isoptin, Calan)</p> <p>Supraventricular Tachycardias</p>

## C. Specific Dysrhythmias

**Tachycardias:** The major problem with the tachy dysrhythmias is that the heart chambers do not have enough time to completely fill or empty. This leads to a drop in stroke volume and subsequently cardiac output. Depending on the exact rhythm, there may also be loss of synchrony between atrial and ventricular contractions (A-fib, V-Tach), which causes a loss of atrial kick and up to 30% of cardiac output. Another potential problem is clot formation in a chamber that has incomplete emptying. In clinical terms tachycardic rhythms can cause anything from dizziness to heart failure and cardiac arrest.

### 1. Narrow QRS Complex Tachycardias (supraventricular)

#### a. Rhythms

- ♥ Sinus Tachycardia (ST)
- ♥ Atrial Fibrillation (A-Fib)
- ♥ Atrial Flutter (AF)
- ♥ Atrial Tachycardia (ectopic and reentrant) (AT)
- ♥ Multifocal Atrial Tachycardia (MAT)
- ♥ Junctional Tachycardia (JT)
- ♥ Accessory Pathway-Mediated
- ✓ Atrial tachycardia w/ accessory pathway
- ✓ AV reentry tachycardia

#### b. Treatment (remember to evaluate ventricular function) Based on 2010 ACLS guidelines

- ♥ Stable?
- ✓ A-Fib or AF: Identify length of time in rhythm and consider WPW and LV impairment before determining treatment. Control Rate, Convert Rhythm, Provide Anticoagulation
- ✓ Vagal Stimulation
- ✓ Adenosine
- ✓ PSVT:  $\beta$ -Blockers,  $Ca^{++}$  Channel Blockers, Dig, Antiarrhythmics and Cardioversion. If EF < 40% Start with Cardioversion
- ✓ JT:  $\beta$ -blockers,  $Ca^{++}$  Channel Blockers, Amiodarone, NO Cardioversion



- ✓ MAT:  $\beta$ -blockers,  $\text{Ca}^{++}$  Channel Blockers, Amiodarone, NO Cardioversion

♥ Unstable? Immediate Cardioversion, Followed by Drugs

## 2. Wide QRS Complex Tachycardias

### a. Criteria for Wide QRS

- ♥ Rate > 120 bpm
- ♥ Uniform QRS  $\geq$  120 ms
- ♥ No S&S or  $\Delta$  in Consciousness

### b. Rhythms

- ♥ Ventricular Tachycardia (VT)
- ♥ Ventricular Fibrillation (VF)
- ♥ SVT with Aberrancy (identify and treat as SVT)

### c. Treatment (remember to evaluate ventricular function) Based on 2010 ACLS guidelines

- ♥ Ventricular Tachycardia Stable w/ Pulse
- ✓ Monomorphic: Procainamide, Sotalol, Amiodarone, Lidocaine. Amiodarone 1<sup>st</sup> if ventricle impaired
- ✓ Polymorphic: normal QT -  $\beta$ -blockers, Lidocaine, Amiodarone, Procainamide, Sotalol, Amiodarone 1<sup>st</sup> if ventricle impaired. Long QT –  $\text{Mg}^{+}$ , overdrive pacing, Isoproterenol, Phenytoin, Lidocaine
- ♥ Ventricular Tachycardia Unstable w/ Pulse: Immediate Cardioversion
- ♥ Ventricular Tachycardia Without Pulse – Treat as VF
- ♥ Ventricular Fibrillation/Pulseless VT:
  - ✓ Assess ABCs
  - ✓ Basic Life Support
  - ✓ Defibrillation: 120-200J biphasic or 360J monophasic (one shock)
  - ✓ CPR
  - ✓ Defibrillation: 120-200J biphasic or 360J monophasic (one shock)
  - ✓ Vasopressin or Epinephrine
  - ✓ Defibrillation: 200J biphasic or 360J monophasic

- ✓ Antiarrhythmics: Amiodarone, Lidocaine, Magnesium, Procainamide
- ✓ Defibrillation: 200J biphasic or 360J monophasic

3. **Long QT Syndrome:** The QT represents the repolarization of the ventricle. Repolarization is an electrically unstable time. VT is a likely outcome if the next R wave were to fall on the T wave. In situations where the QT interval is long, there is an increased likelihood of an R on T to occur. Conditions that can lead to this situation include:

- ♥ Congenital Long QT Syndrome
- ♥ Exercise Induced QT Syndrome
- ♥ Drug Induced QT Syndrome (many drugs lengthen QT)
  - ✓ Antiarrhythmic Agents: Class IA, IB & III
  - ✓ Tricyclic Antidepressants
  - ✓ Phenothiazine
  - ✓ Antimicrobials (specifically Erythromycin)
  - ✓ Nicardipine (Cardene)
  - ✓ Cisapride (Propulsid)
  - ✓ Haloperidol (Haldol)
  - ✓ Tamoxifen (Nolvadex)

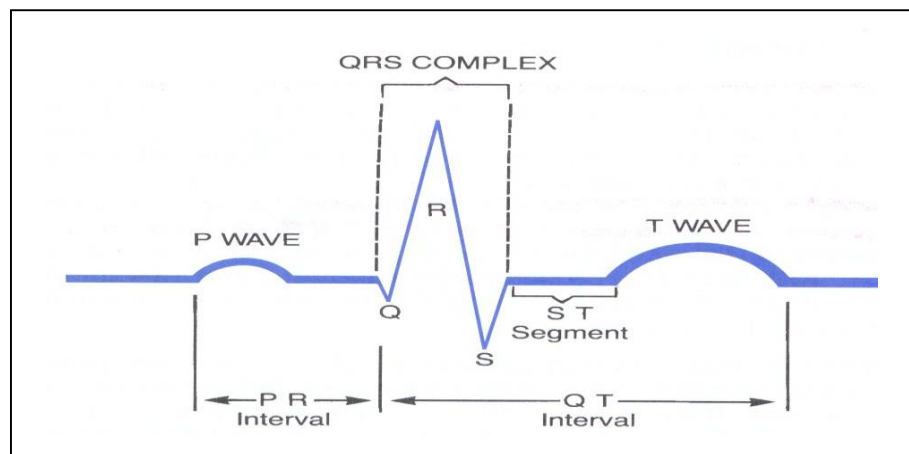
D. **Bradycardias:** The major problem with slow rhythms is a lack of stroke volume to sustain an adequate cardiac output. Treatment is dependent on rhythm and cause of slow rate. In the unstable patient with a slow rate:

- ♥ ABCs & BLS
- ♥ Atropine
- ♥ Transcutaneous Pacing
- ♥ Dopamine or Epinephrine
- ♥ If the rhythm is Type II 2<sup>nd</sup> Degree or 3<sup>rd</sup> Degree HB and the pt is unstable pace ASAP (transcutaneous → transvenous)

### E. **Conduction Defects**

**Normal Parameters:**

PR = 0.12-0.20  
 QRS = > 0.12  
 QT rate dependent



1. First Degree Heart Block

- ♥ Rate: 60 - 100 bpm
- ♥ Rhythm: Regular
- ♥ P Waves: One P for Every QRS with PRI > 0.20
- ♥ QRS Complexes: Normal
- ♥ Symptoms/Concerns: Symptoms will depend on HR  
Concern for reason this is occurring and will it progress to higher level block
- ♥ Tx: Depends on Symptoms, tx rarely required. Rhythm very common in elderly

2. Second Degree Heart Block (two types)

Mobitz Type I, also known as Wenckebach

- ♥ Rate: atrial rate 60 - 100 bpm, ventricular rate varies
- ♥ Rhythm: Irregular with Pattern
- ♥ P Waves: All QRSs are preceded by Ps
  - ⇒ But not all Ps are followed by QRSs
  - ⇒ The PRI progressively gets longer
  - ⇒ Until there is a dropped beat (a P wave not followed by a QRS)
  - ⇒ Pattern then starts over
- ♥ QRS Complexes: Normal
- ♥ Symptoms/Concerns: Symptoms will depend on Ventricular HR
- ♥ Tx: Depends on Symptoms, tx rarely required

Mobitz Type II, also known as Classical

- ♥ Rate: atrial rate 60 - 100 bpm, ventricular varies
- ♥ Rhythm: P-P regular, R-R regular or irregular
- ♥ P Waves: All QRSs are preceded by Ps
  - ⇒ But not all Ps are followed by QRSs
  - ⇒ The PRI consistent and typically > 0.20
  - ⇒ More than one P wave for every QRS
  - ⇒ Typically a consistent pattern ex. 2Ps:1QRS
- ♥ QRS Complexes: Normal
- ♥ Symptoms/Concerns: Symptoms will depend on Ventricular HR
- ♥ Tx: Depends on Symptoms, typically treated
  - ⇒ Consider External Pacemaker

- ⇒ Consider Cause
- ⇒ Stop Digoxin
- ⇒ Atropine or Epinephrine

### 3. Complete Heart Block aka AV Dissociation

- ♥ Rate: < 60 bpm
- ♥ Rhythm: P-P regular, R-R regular
- ♥ P Waves: P waves “march” out regular but have no discernible relationship to the QRS
- ♥ QRS Complexes: Slow, Wide > 0.12, “march” out, regular
- ♥ Symptoms/Concerns: Symptoms will depend on Ventricular HR and LOC
- ♥ Tx: Depends on Symptoms, tx typically required
  - ⇒ External pacemaker
  - ⇒ Atropine (not typically helpful because it will increase sinus node firing (P waves) but not ventricular conduction)
  - ⇒ Epinephrine

## 4. Bundle Branch Blocks

### QRS Complex:

Represents:

Shape:

Duration (time):

Ventricular Depolarization

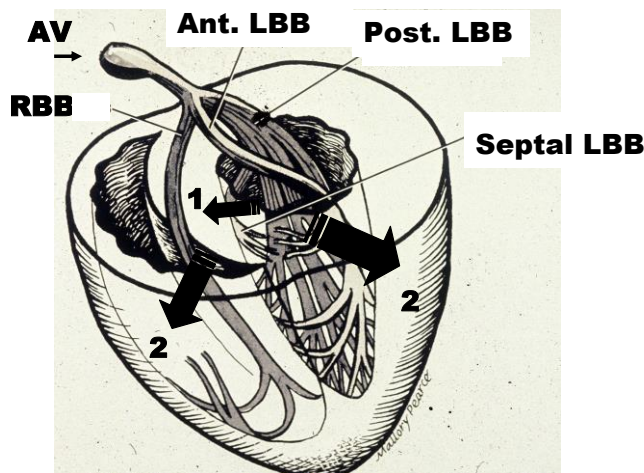
Q First Negative Deflection

R First Positive Deflection

S Second Negative Deflection

QRS < 0.12sec (3mm)

Q < 0.03sec (< 1mm)



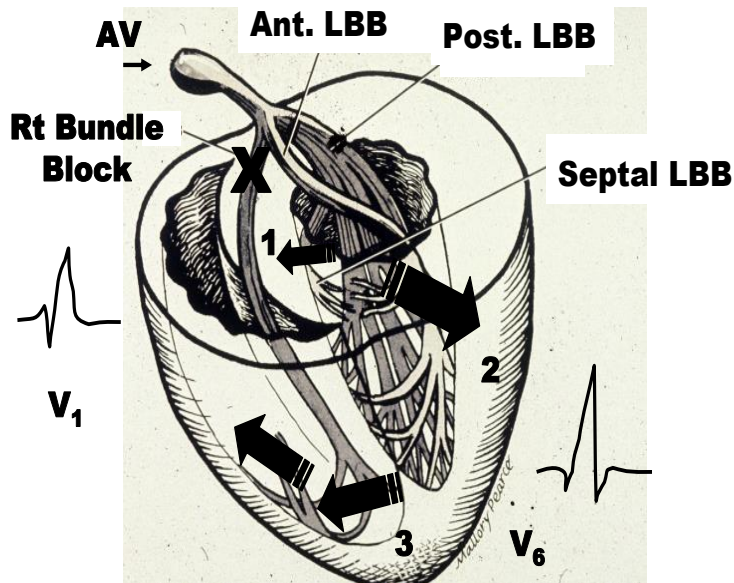
AV Node → Bundle of His →

1. Septal Depolarization L → R

2. Biventricular Depolarization

The ventricles depolarize simultaneously. Because the LV mass is larger than the RV the mean vector of electrical current is the LV depolarization.

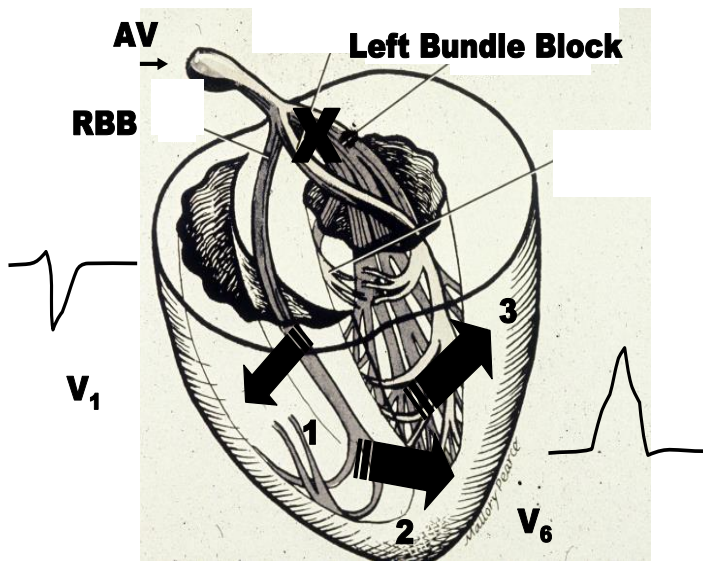
When there is an electrical block in the normal conduction pathway for ventricular depolarization it is called a bundle branch block. This block can be permanent or intermittent and has a variety of causes. Depolarization occurs because of the principle of conductivity. This depolarization takes longer (QRS duration > 0.12sec) and the configuration is slightly different than the normal QRS pattern. Bundle Branch Block patterns are best evaluated in precordial leads V<sub>1</sub> and V<sub>6</sub>.



Right  
Bundle Branch Block

V<sub>1</sub> rsR' > 0.12sec

V<sub>6</sub> qRs > 0.12sec



Left  
Bundle Branch Block

V<sub>1</sub> rS > 0.12sec

V<sub>6</sub> R > 0.12sec

## V. Summary

