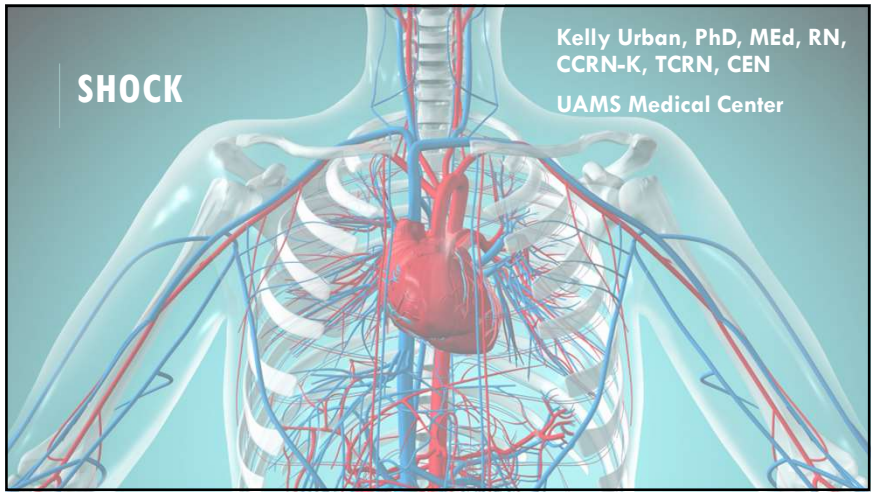

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OBJECTIVES

Identify the 3 stages of shock.

Verbalize the treatment of hypovolemic shock.

2

SHOCK - DEFINED

Inadequate tissue perfusion

Caused when oxygen intake, absorption, or delivery fails, or when the cells are unable to take up and use the delivered oxygen to generate sufficient energy to carry out cellular functions.

3

SHOCK - DEFINED

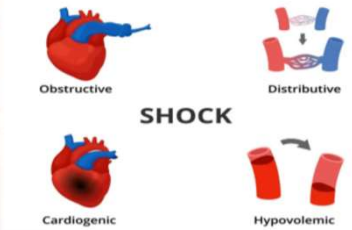
Inadequate tissue perfusion

Volume Problem
- ↓ intravascular volume

Pump Problem
- Heart Failure

Pipe Problem
- ↓ vascular tone

Obstruction
- Mechanical barrier to blood flow



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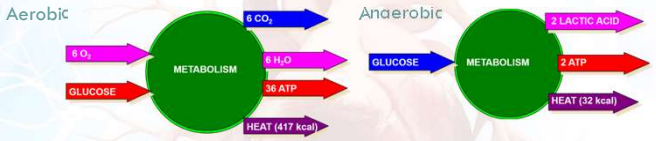
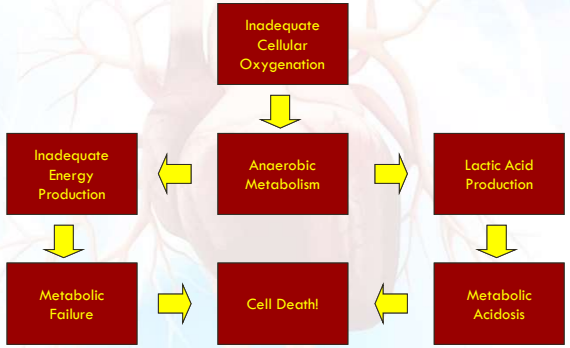
METABOLISM REVIEW



- No oxygen = lactic acid production
- Reduction in number of ATP = reduced energy

5

ANAEROBIC METABOLISM – SO WHAT?



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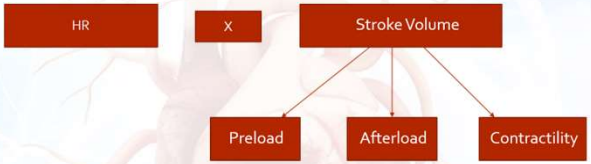
CARDIAC OUTPUT REVIEW



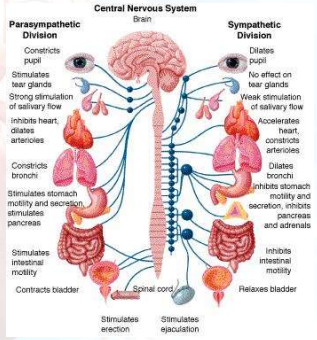
- Preload: amount of stretch at end of diastole (volume ready to be ejected)
- Afterload: resistant in which ventricle has to overcome to contract (vasoconstriction)
- Contractility: ability of the heart to contract

7

AUTONOMIC NERVOUS SYSTEM REVIEW

Effects of Sympathetic Nervous System Stimulation

Organ	Effect
Heart (muscle)	↑ force of contraction (+ inotropy)
Heart (rate)	↑ heart rate (+ chronotropy)
Peripheral vessels	Vasoconstriction
Pupils	Dilation
Sweat glands (cholinergic)	↑ secretion
Adrenal glands	↑ cortisol and medullary secretion
Bronchi	Dilation
Kidneys	↑ Renin secretion (↓ urine output)
Liver	Glycogenolysis (↑ blood sugar)



8

STAGES OF SHOCK

- Compensated
- Progressive
- Irreversible

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COMPENSATED SHOCK

(COMPLEX SERIES OF NEURO-ENDOCRINE RESPONSES TO ↑ CO)

Decreased cardiac output compensatory mechanisms
- Tachycardia

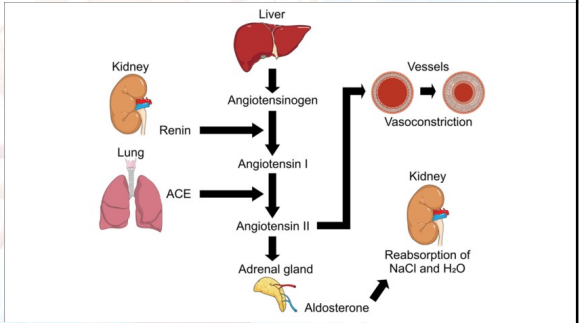
Activation of autonomic nervous system
- Tachycardia
- Vasoconstriction

Activation of renin-angiotensin system (due to reduced blood flow to kidneys)
- Vasoconstriction
- Na/Water retention

Increased rate and depth of respirations

10

ACTIVATION RENIN-ANGIOTENIN-ALDOSTERONE SYSTEM (RAAS)



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COMPENSATED SHOCK – CLINICAL FINDINGS

- Normal BP, narrow pulse pressure
- Sinus tachycardia
- Fast, deep respirations
- ↓ Urine Output
- ↑ Urine Specific Gravity
- Cool, clammy skin
- ↓ LOC
- Dilated pupils
- ↑ blood sugar
- Respiratory alkalosis with hypoxemia

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DECOMPENSATED (PROGRESSIVE) SHOCK

Decreased oxygen delivery to cells

- Shift to anaerobic metabolism
- Decreased ATP production
- Production of lactic acid = metabolic acidosis
- Failure of Na+/K+ pump
- Arrhythmias
- Alteration of capillary fluid dynamics
- Further decrease in cardiac output
- DIC

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DECOMPENSATED (PROGRESSIVE) SHOCK – CLINICAL FINDINGS

- ↓ BP with narrow pulse pressure
- Continued tachycardia
- Acute renal failure
- Continued decreasing LOC
- Interstitial pulmonary edema
- Peripheral edema
- Metabolic and respiratory acidosis with hypoxemia

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IRREVERSIBLE SHOCK – MULTIORGAN DYSFUNCTION SYNDROME

Microvascular and organ damage are now irreversible

There is often a "last ditch" effort from the ischemic midbrain with an enormous discharge of endogenous catecholamines and this can create a last spike of sinus tachycardia

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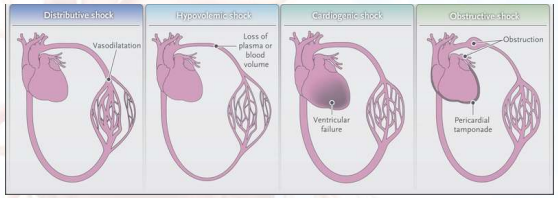
CLASSIFICATION OF SHOCK

Hypovolemic

Distributive

Cardiogenic

Obstructive



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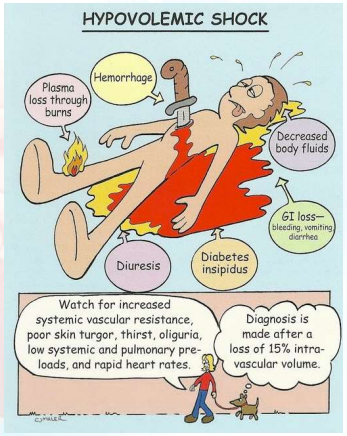
HYPOVOLEMIC SHOCK

Causes

Absolute Fluid Volume Deficit
- Hemorrhage or Blood Loss
  - Surgery
  - Trauma
  - GI Bleed
- Severe Dehydration
  - Sweat
  - Vomiting/Diarrhea
- Skin Loss via Burns

Relative Fluid Volume Deficit
- Severe Ascites
- Severe Burns (interstitial)
- Severe hypoalbuminemia



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HYPOVOLEMIC SHOCK - PATHOPHYSIOLOGY

Decreased Intravascular Volume

Decreased Venous Return

Decreased Ventricular Filling

Decreased Stroke Volume/ Cardiac Output

Inadequate Tissue Perfusion

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HYPOVOLEMIC SHOCK POINTS TO CONSIDER

Increases in HR may be blunted in patients taking beta-blockers

Pulse pressure and mean arterial pressure (MAP) are better than looking at systolic or diastolic pressure alone

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HYPOVOLEMIC SHOCK TREATMENT

Airway management

Control Hemorrhage

IV access

Fluid Resuscitation

Prevent Hypothermia

20

HYPOVOLEMIC SHOCK TREATMENT – CONTROL HEMORRHAGE

Apply direct pressure

Apply splints/tourniquets

Tranexamic Acid

Timely operative intervention

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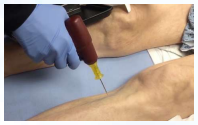
HYPOVOLEMIC SHOCK TREATMENT – IV/IO ACCESS



Peripheral IV access preferred

Obtain IO access if PIV unavailable.

- Locations include: sternum, distal femur, distal tibia, humerus, and proximal tibia
- Fractures of the bone or previous attempts in the same bone are contraindications
- Considered short term (< 24 hours)

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HYPOVOLEMIC SHOCK TREATMENT – FLUID RESUSCITATION

Ideally, the volume that is lost is replaced

Crystalloids

Colloids

Blood/Blood Products

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HYPOVOLEMIC SHOCK TREATMENT - FLUID RESUSCITATION

Crystalloids should be considered if patient has volume loss through means other than hemorrhage

Types: Normal Saline, Lactated Ringers, Plasmalyte

Colloids may be considered instead of crystalloids

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BLOOD AND BLOOD PRODUCTS

Product	Notes	Test ABO Status?	Dosage
Whole Blood	Contains all components	Yes	500 ml/unit
PRBCs	Each unit ↑ Hgb by 1 g/dl & HCT by 3%	Yes	250 ml/unit
Platelets	Each unit of platelets ↑ platelet count 5000-10000 u/dL	No	Mult units ordered (i.e. "6 pack")
FFP/Plasma	20 minutes to thaw and must be used within 6 hours 1 unit provides 7% of coag factors	Yes	Variable

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HYPOVOLEMIC SHOCK TREATMENT – FLUID RESUSCITATION

Permissive Hypotension

- Minimal fluid resuscitation allowing blood pressure to remain low

Damage Control Resuscitation

- Minimizes the amount of crystalloid used
- Utilizes PRBCs, plasma, and platelets (1:1:1 ratio)

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PERMISSIVE HYPOTENSION

Advantages

↓ likelihood of coagulopathy

Less hypothermia

Less risk factors for acidosis

Diminishes dislodgement of clots due to elevated pressure

Risk Factors

Exacerbates head injuries

Not safe for pediatric or elderly patients

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END POINTS TO FLUID RESUSCITATION

Traditional

Invasive Hemodynamic Monitoring

Metabolic Parameters

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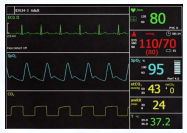
END POINTS TO FLUID RESUSCITATION - TRADITIONAL



Vital Signs
- Blood pressure is **not** a good predictor of tissue perfusion

Urinary Output
- < 0.5 ml/kg/hour is an early sign of inadequate perfusion

Mental Status
- May also be affected by pre-existing conditions, alcohol, or drugs

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END POINTS TO FLUID RESUSCITATION – INVASIVE HEMODYNAMIC MONITORING

CVP
- Measures right ventricular preload (norm 2-6 mmHg)

Wedge Pressure
- Measures left ventricular preload (norm 8-12 mmHg)

Cardiac Index
- Normal 2.5-4 L/min/m^2

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END POINTS TO FLUID RESUSCITATION – METABOLIC PARAMETERS

Lactate
- Byproduct of inadequate tissue perfusion
- Patients who lactate levels do not normalize have a higher mortality rate
- Lactate > 4 mmol/L indicates widespread tissue hypoperfusion

Base Deficit
- Measures buffering capacity of the blood reflecting metabolism and depth of hemorrhagic shock
- Base deficit > 6 mmol/L is a marker of severe injury

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HYPOVOLEMIC SHOCK HEMODYNAMICS

Shock Type	CVP	PAWP	SVR	C.O.	HR	Comments
Hypovolemic	↓	↓	↑	↓	↑	Too little volume

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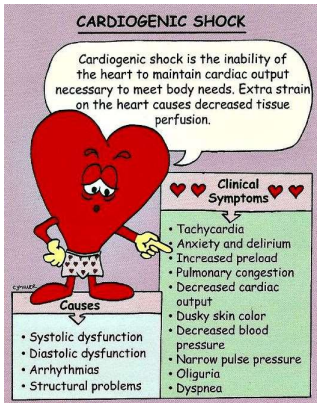

CARDIOGENIC SHOCK

Severe dysfunction of the right or left ventricle that results in inadequate pumping

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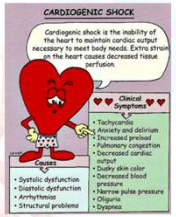
CARDIOGENIC SHOCK



Causes:

- Myocardial Infarction
- Structural Variations
  - Cardiomyopathy
  - Papillary Muscle Dysfunction
- Blunt Cardiac Injury (cardiac contusion)
- Ventricular Ischemia

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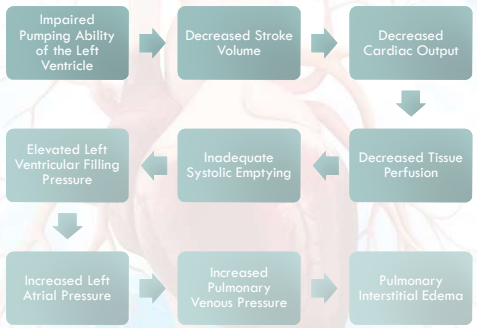
CARDIOGENIC SHOCK - PATHOPHYSIOLOGY



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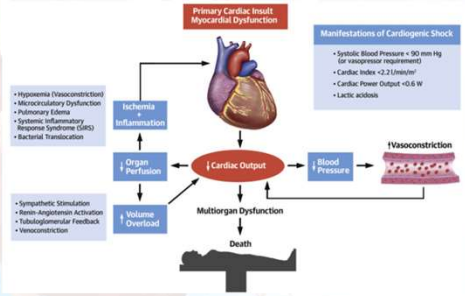
CARDIOGENIC SHOCK

Symptoms:

- ↓ BP (may be normal initially)
- ↑ HR
- ↑ RR/Dyspnea
- Bilateral Crackles
- ↓ Urine Output
- Normal Temp
- Cool, Pale Skin
- ↓ CO/CI
- ↑ PCWP/PA Pressures



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CARDIOGENIC SHOCK - TREATMENT

Hemodynamic Support
- ABCs (Supplemental Oxygen)

Decrease Preload	Increase Contractility	Decrease Afterload	Increase Afterload
• Diuretics • Nitrates • Morphine	• Dobutamine • Milrinone	• Nitroprusside • Nitrates • ACE Inhibitors	• Norepinephrine • Epinephrine

Identify & Treat Cause
- ECG/Cardiac Enzymes (MI/dysrhythmias)
- Chest Xray
- Echo

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CARDIOGENIC SHOCK HEMODYNAMICS

Shock Type	CVP	PAWP	SVR	C.O.	HR	Comments
Cardiogenic	↑	↑	↑	↓	↑	Ineffective pump

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OBSTRUCTIVE SHOCK

Hypoperfusion of tissue due to an obstruction in either the vasculature or heart

Cause	Treatment
Pregnancy	Roll patient to her side
Tension Pneumothorax	Chest Tube/Needle Decompression
Cardiac Tamponade	Pericardiocentesis
Pulmonary Embolism	Thrombolytics
Aortic Aneurysm	Surgical Intervention
Aortic Stenosis	Surgical Intervention
Excessive Positive End Expiratory Pressure	Readjust Ventilator Settings

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OBSTRUCTIVE SHOCK - PE

Source

DVT

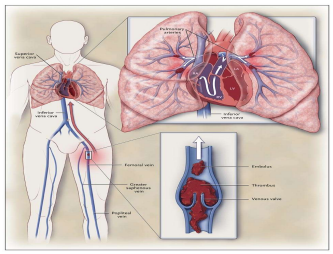
Air embolism

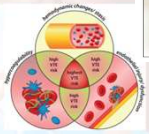
Fat embolism

Amniotic fluid embolism

Septic embolism

Tumor embolism





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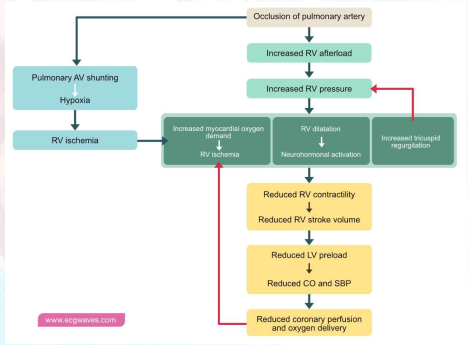
PE PATHOPHYSIOLOGY



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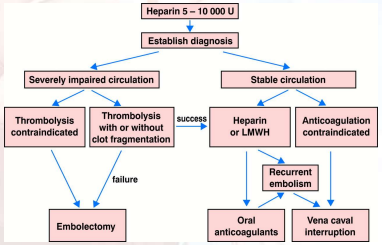
OBSTRUCTIVE SHOCK TREATMENT - PE

ABCs

Thrombolytics

Embolectomy



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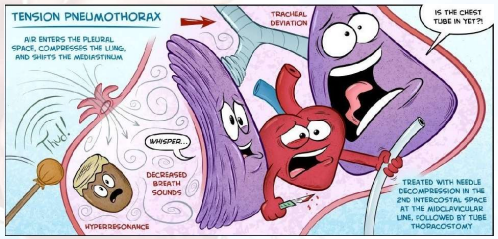
OBSTRUCTIVE SHOCK – TENSION PNEUMOTHORAX

Signs/Symptoms:

- Tachycardia
- ↑ RR
- JVD
- Absent Breath Sounds on affected side
- Tracheal Deviation (late)
- Cyanosis (late)



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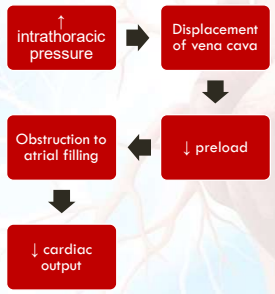
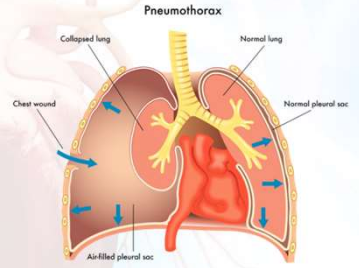
OBSTRUCTIVE SHOCK – TENSION PNEUMOTHORAX





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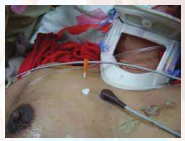
OBSTRUCTIVE SHOCK TREATMENT – TENSION PNEUMOTHORAX

Needle Decompression

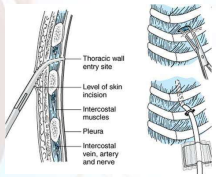
Insert 14 g x 3.25 in angiocath into chest wall

- 2nd intercostal space
- Midclavicular line
- Above 3rd rib (to avoid nerves, vein, artery that are located under ribs)



Chest Tube Placement

Thoracostomy tube placed 4th - 5th intercostal space, midaxillary line



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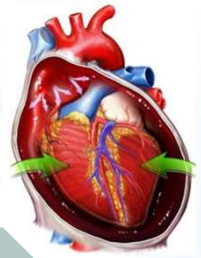
OBSTRUCTIVE SHOCK – CARDIAC TAMPONADE



Accumulation of fluid in the (inflexible) cardiac sac → Impediment of diastolic expansion and filling of ventricle → Low preload, SV, CO, and End-Organ Perfusion

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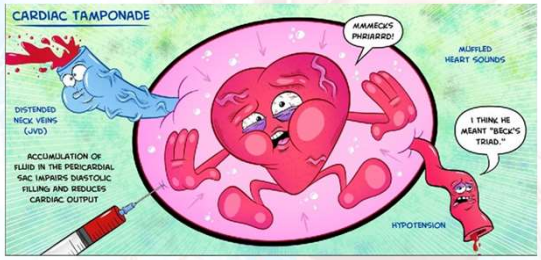
OBSTRUCTIVE SHOCK – CARDIAC TAMPONADE



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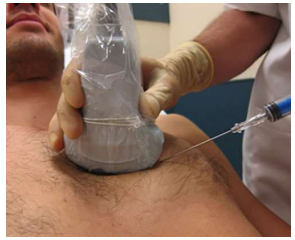
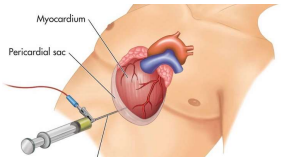
OBSTRUCTIVE SHOCK TREATMENT – CARDIAC TAMPONADE

Pericardiocentesis: procedure in which fluid is removed from the pericardium

Removal of 5-10 ml may ↑ stroke volume by 25-50%





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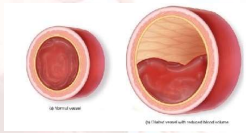
DISTRIBUTIVE SHOCK

Abnormality in the vascular system that produces a maldistribution of blood flow.

3 Types:
- Septic
- Neurogenic
- Anaphylactic



Occurs when blood vessels dilate without subsequent increase in volume

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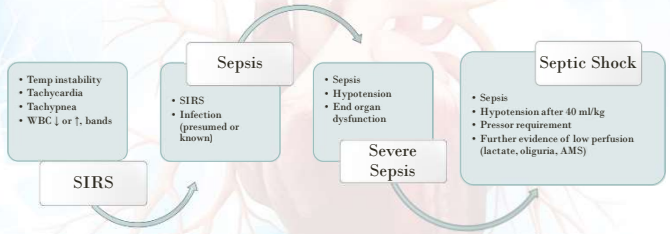
SEPTIC SHOCK (DISTRIBUTIVE)



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SEPSIS GUIDELINES (DISTRIBUTIVE)

Initial Resuscitation
Screening for Sepsis
Diagnosis
Antimicrobial Therapy
Source Control
Fluid Therapy
Vasoactive Medications
Corticosteroids
Blood Products
Immunoglobulins
Blood Purification
Anticoagulants

Mechanical Ventilation
Sedation & Analgesia
Glucose Control
Renal Replacement Therapy
Bicarbonate Therapy
VTE Prophylaxis
Stress Ulcer Prophylaxis
Nutrition
Goals of Care

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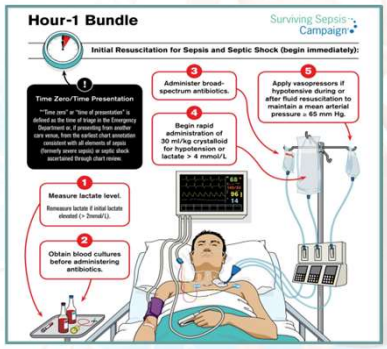
SEPTIC SHOCK (DISTRIBUTIVE) – INITIAL RESUSCITATION



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SEPTIC SHOCK (DISTRIBUTIVE) – INITIAL RESUSCITATION

Intervention	Goal	Notes
Fluid	30 ml/kg crystalloid within 1st 3 hours	• Additional fluids guided by frequent reassessment • Albumin may be considered
Hemodynamic Assessment	Maintain MAP ≥ 65 mmHg if requiring vasopressors (use arterial line if need vasopressors)	• Norepinephrine – 1st choice • Vasopressin or Epinephrine next • Dobutamine for persistent hypoperfusion (adequate volume and vasopressors)
Lactate	< 4	• Marker to guide evidence of tissue hypoperfusion
Antibiotics	Empiric broad-spectrum therapy (typically 7-10 days)	• Therapy narrowed once pathogen identified
Corticosteroids	200 mg / day	• Only to be used if hemodynamic stability not achieved with adequate fluid/vaso
Hgb	≥ 7.0 g/dL	

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ANAPHYLACTIC SHOCK (DISTRIBUTIVE)

Immune system overreaction that results in a host of vasoactive reactions

Causes:

- Repeated exposure to an antigen (antibiotics, other drugs, contrast media, food, insect stings, snake bites)

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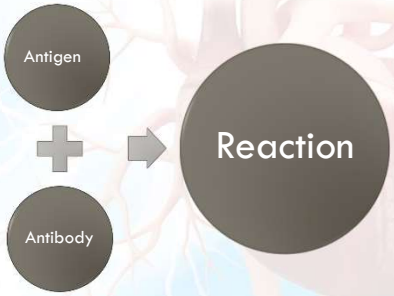
ANAPHYLACTIC SHOCK - PATHOPHYSIOLOGY



Massive Vasodilation
- Veins
- Arterioles

Increased Capillary Permeability
- Interstitial Edema
- Relative Hypovolemia

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ANAPHYLACTIC SHOCK

Symptoms

- Urticaria/Pruritis
- "Sense of Impending Doom"
- Bronchoconstriction
- Increased Capillary Permeability
- Decreased CO
- ↑ HR/RR
- ↓ PAP/PCWP

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ANAPHYLACTIC SHOCK – TREATMENT GOALS

ID and remove causative agent

Fluids

Epinephrine

Antihistamines

Corticosteroids

Bronchodilators

Patient Education

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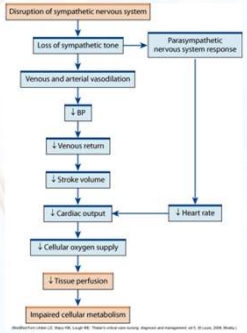
NEUROGENIC SHOCK (DISTRIBUTIVE)

Causes:

- Spinal Cord Injuries above T4-T6
- Brain Injury (if the central sympathetic system has been affected)



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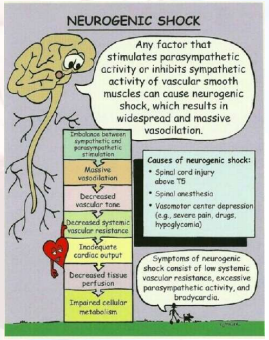
NEUROGENIC SHOCK (DISTRIBUTIVE)

Symptoms:

- Hypotension (due to vasodilation)
- Glyconeogensis
- Dry skin
- Bradycardia
- ↓ Contractility, Automaticity, Respiratory Rate



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NEUROGENIC SHOCK (DISTRIBUTIVE)

Treatment

- 1-2 L fluid bolus
- Vasopressors
- Goal: MAP 85-90 mmHg
- Atropine
- Assist with respirations as needed

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DISTRIBUTIVE SHOCK HEMODYNAMICS

Shock Type	CVP	PAWP	SVR	C.O.	HR	Comments
Neurogenic	↓	↓	↓	↓	↓	Loss of sympathetic tone
Septic	↓	↓	↓	↑	↑	Endotoxins/exotoxins result in vasodilation
Anaphylactic	↓	↓	↓	↓	↑	Histamine release results in vasodilation

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AGE-RELATED SHOCK CONSIDERATIONS

Geriatric Shock Patient

- Cardiac function ↓ by almost 50% with age
- ↓ CO and SV
- ↓ perfusion to end organs
- Less pulmonary reserve
- Pre-existing chronic diseases and medications used for these conditions (i.e. cardiovascular disease)
- Less able to tolerate changes in end-organ perfusion and develop multiple organ failure more quickly

Consider a geriatric patient to be hypoperfused if HR > 90 and/or SBP < 110

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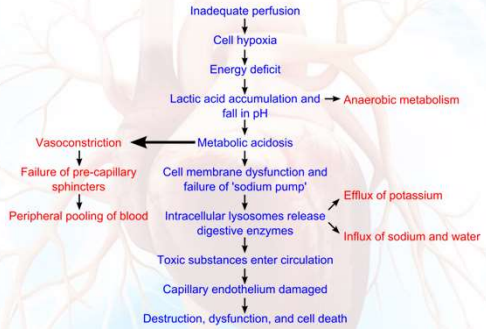
IN SUMMARY:



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REVIEW OF TYPES OF SHOCK

Shock Type	CVP	PAWP	SVR	C.O.	HR	Comments
Hypovolemic	↓	↓	↑	↓	↑	Too little volume
Cardiogenic	↑	↑	↑	↓	↑	Ineffective pump
Neurogenic	↓	↓	↓	↓	↓	Loss of sympathetic tone
Septic	↓	↓	↓	↑	↑	Endotoxins/exotoxins result in vasodilation
Anaphylactic	↓	↓	↓	↓	↑	Histamine release results in vasodilation

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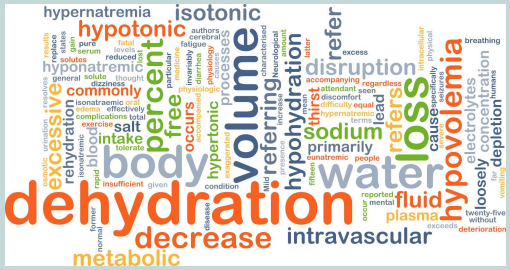

QUESTIONS?

Contact Kelly Urban @
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Acute Coronary Syndrome

Lynnette Flynn
CCRN-CMC/CSC, RCIS, CHFN, CNOR, MBA-HA

SW Regional ECC Secretary
Dir. of Education, Arkansas Heart Hospital
Education Quality Advisor-DoH&F NT

1

Objectives

- Define & delineate acute coronary syndrome
- Review Management Guidelines
  - Unstable Angina / NSTEMI
  - STEMI
- Review secondary prevention initiatives

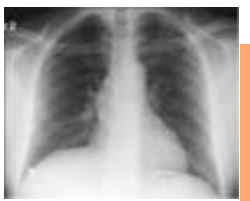
2

Scope of Problem
(2004 stats)

- CHD single leading cause of death in US
  - 452,327 deaths in the U.S. in 2004
  - 840,768 in 2016
- 1,055,000 new & recurrent coronary attacks /yr
- 38% of those who with coronary attack die in 1yr
  - 30% mortality at time of event (3.4%)
  - 52% of these are pre-hospital
    - 10.4 % survived to discharge
  - 5-10% more within first year (7% 1st yr then 2%/yr)
  - ½ are rehospitalized within 1yr (Zafari, 2019)
- Annual cost > $351.2 billion

3

Cardiac Risk Factors



- Diabetes
- HTN
- Smoking
- Hypercholesterolemia
- Family history
- Prior personal CAD

TESTING
Troponin I or T
CK-MB
Serial Ck-MB and Troponin
Q8 hrs until peak or 24 hours
CBC
Chemistry
Coag.
PA chest
12 lead initial, 4 hr and 12 hr
RX stress
Echo to r/o structural disease
Cath if unstable or sig. ischemia
Consider non cardiac causes…

4

5

5

Expanding Risk Factors

- Diabetes Mellitus
- Hypertension
- Smoking
- Obesity
- Dyslipidemia
  - Low HDL < 40
  - Elevated LDL / TG
- Lack of regular physical activity

- Family History—event in first degree relative
  - ≥55 male
  - 65 female
- Age- ≥45 for male
  - 55 for female
- Chronic Kidney Disease
- Lack of diet rich in fruit, veggies, fiber
- Lack of ETOH intake

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GI Complaints…

Gall Stones
Acid Reflux
Indigestion
Hiatal Hernia

Esophageal Spasm

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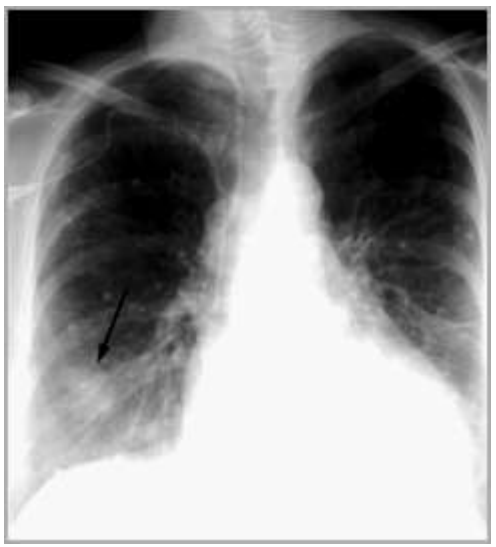
Apex of Right lower lobe



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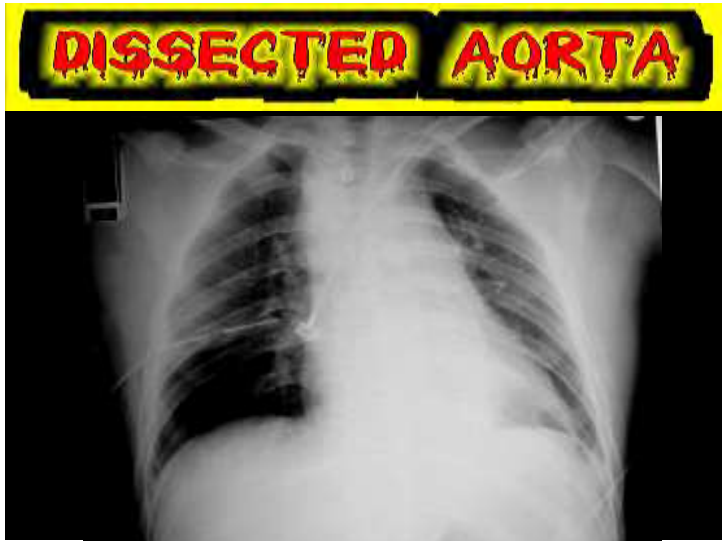

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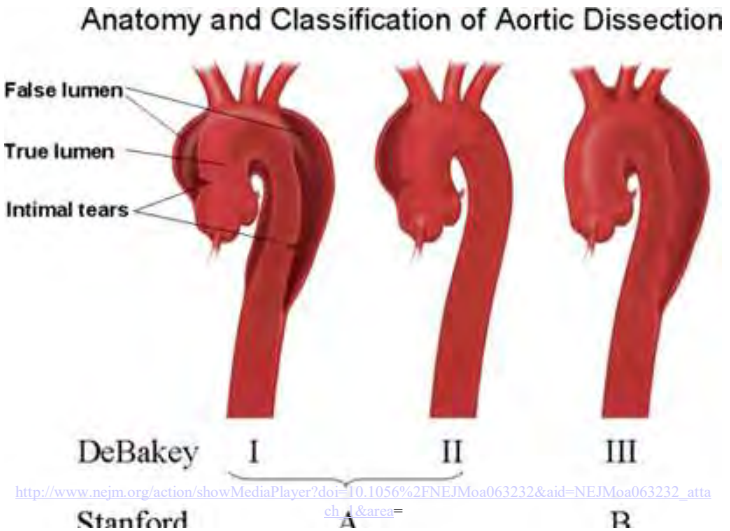

http://www.nejm.org/action/showMediaPlayer?doi=10.1056%2FNEJMoa063232&aid=NEJMoa063232_attach_1&area=

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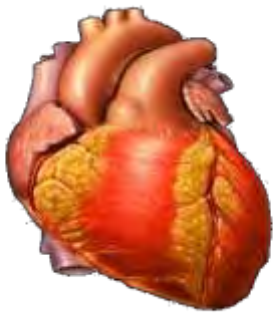
Cardiac – non CAD ischemia



Non CAD ischemia……

Anemia
Dehydration
HTN crisis
tachycardia
spasm

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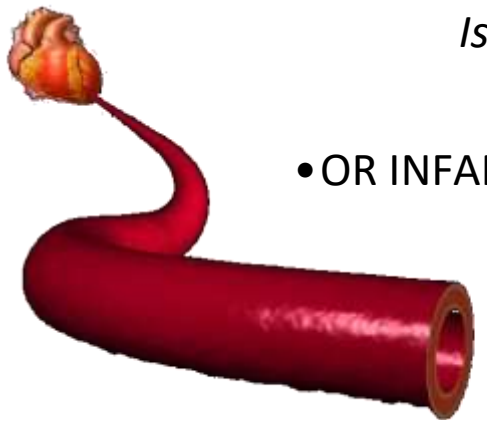
Cardiac

- MAY BE ANGINA

Ischemia



- OR INFARCTION

Injury

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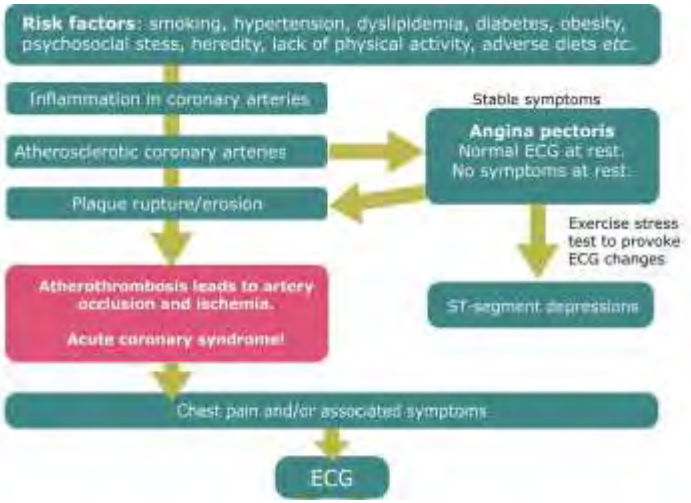

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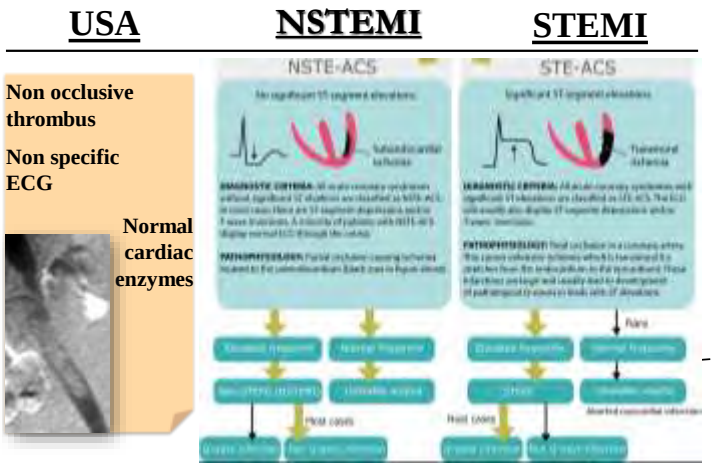

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Diagnosis of Acute MI
STEMI / NSTEMI

- At least 2 of the following
  - Ischemic symptoms
  - Diagnostic ECG changes
  - Serum cardiac marker elevations

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Diagnosis of Angina

- **Typical angina - All** three of the following
  - Substernal chest discomfort
  - Onset with exertion or emotional stress
  - Relief with rest or nitroglycerin
- **Atypical angina**
  - 2 of the above criteria
- **Noncardiac chest pain**
  - 1 of the above

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Diagnosis of Unstable Angina

- **Patients with typical angina** - An episode of angina
  - Increased in severity or duration
  - Has onset at rest or at a low level of exertion
  - Unrelieved by the amount of nitroglycerin or rest that had previously relieved the pain
- **Patients not known to have typical angina**
  - First episode with usual activity or at rest within the previous two weeks
  - Prolonged pain at rest

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Acute Management

- Initial evaluation & stabilization
- Efficient risk stratification
- Focused cardiac care

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Evaluation

- Efficient & direct history
- Initiate stabilization interventions

} Occurs simultaneously

Plan for moving rapidly to indicated cardiac care

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Description

Pain

- Provoking factors/ Palliation
- Quality
- Radiation
- Severity 1-10 scale
- Time of onset

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Chest pain suggestive of ischemia

Immediate assessment within 10 Minutes

Initial labs and tests

- 12 lead ECG
- CXR
- Lab
  - Cardiac enzymes
  - Electrolytes
  - CBC
  - Lipids
  - BUN/Cr
  - Glucose
  - Coags

Emergent care

- IV access
- Cardiac monitor
- Oxygen
- Aspirin
- Nitrates
- Morphine

History & Physical

- Establish diagnosis
- Read ECG
- Identify complications
- Assess for reperfusion

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Focused History

- Aid in diagnosis and rule out other causes
  - Palliative/Provocative factors
  - Quality of discomfort
  - Radiation
  - Symptoms associated with discomfort
  - Cardiac risk factors
  - Past medical history - especially cardiac
- Reperfusion questions
  - Timing of presentation
  - ECG c/w STEMI
  - Contraindication to fibrinolysis
  - Degree of STEMI risk

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Targeted Physical

- **Examination**
  - Vitals
  - Cardiovascular system
  - Respiratory system
  - Abdomen
  - Neurological status
- **Recognize factors that increase risk**
  - Hypotension
  - Tachycardia
  - Pulmonary rales
  - Pulmonary edema
  - JVD
  - New murmurs/heart sounds
  - Diminished peripheral pulses
  - Signs of stroke

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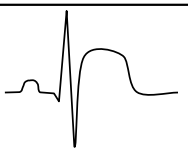
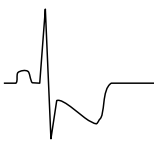
ECG assessment

ST Elevation or new LBBB
STEMI



ST Depression or dynamic T wave inversions
NSTEMI



Non-specific ECG
Unstable Angina

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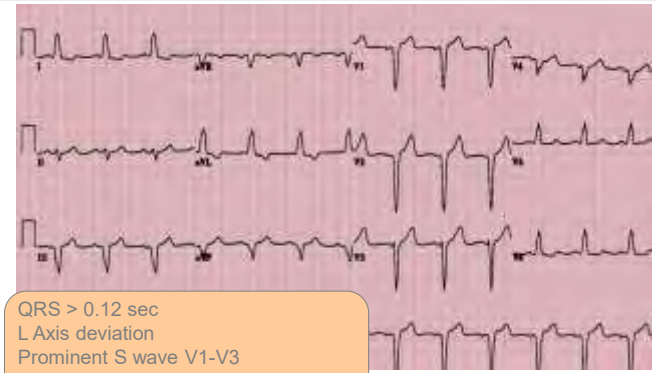
New LBBB



QRS > 0.12 sec
L Axis deviation
Prominent S wave V1-V3
Prominent R wave 1, aVL, V5-V6
with t-wave inversion

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Cardiac markers

Troponin (T, I)

- Very specific and more sensitive than CK
- Rises 4-8° after injury
- May remain elevated for up to two weeks
- Can provide prognostic information
- Troponin T may be elevated with renal dz, poly/dermatomyositis

CK-MB isoenzyme

- Rises 4-6° after injury and peaks at 24°
- Remains elevated 36-48°
- Positive if CK/MB > 5% of total CK & 2 x nml
- Elevation can be predictive of mortality
- False positives with exercise, trauma, muscle dz, DM, PE

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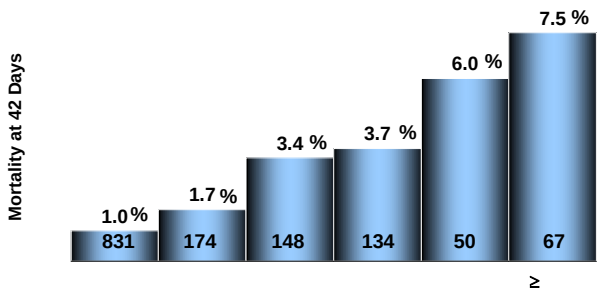
Prognosis with Troponin



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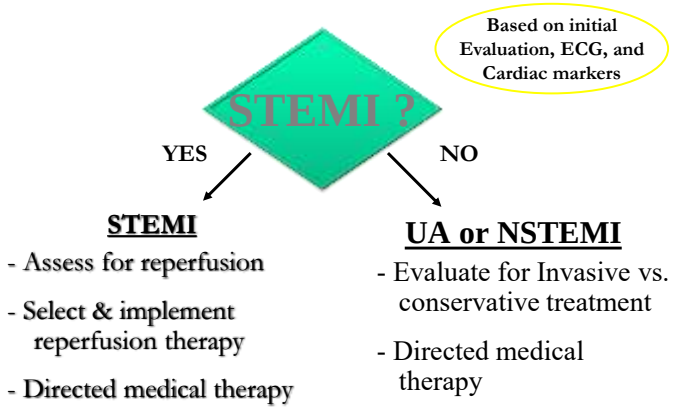
Risk Stratification



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Cardiac Care Goals

- Decrease amount of myocardial necrosis
- Preserve LV function
- Prevent major adverse cardiac events
- Treat life threatening complications

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STEMI cardiac care

- **STEP 1: Assessment**

Time since onset

– 90" for PCI / 12 hrs for fibrinolysis

Is this high risk STEMI?

– KILLIP classification, TIMI score

– If higher risk may manage with more invasive treatment

Determine if fibrinolysis candidate

– Meets criteria with no contraindications

Determine if PCI candidate

– Based on availability and time to balloon r

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Killip Class

- **The numbers below were accurate in 1967. Now, they have diminished by 30 to 50% in every class.**

https://acls.com/free-resources/acute-coronary-syndrome/myocardial-infarction-prognosis-and-predictors-of-mortality

Class	Symptoms	1967	today
I:	no clinical signs	6%	4%
II:	Rales, crackles, S3, JVD	17%	11%
III:	Frank pulmonary edema	38%	25%
IV:	Cardiogenic Shock	81%	54%

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TIMI Score for USA/NSTEMI

Antman et al JAMA 2000; 204:0.35.042

Historical

- Age ≥ 65 (1 point)
- 3 or more CAD risk factors (PHx, HTN, ↑chol, active smoker) (1 point)
- Known CAD with more than 50% stenosis (1 point)
- Aspirin use in the past 7 days (1 point)

Presentation

- Severe angina in the preceding 24 hours (1 point)
- Elevated cardiac markers (1 point)
- ST deviation greater than 0.5mm (1 point)

- **TIMI risk of Death or MI to urgent revascularization**
  - 0-1 points (3% to 5%)
  - 2 points (3% to 8%)
  - 3 points (5% to 13%)
  - 4 points (7% to 20%)
  - 5 points (12 % to 26%)
  - 6-7 points (19% to 41%).

*** Entry criteria UA or NSTEMI defined as ischemic pain at rest within past 24H with evidence of CAD (ST deviation or + marker)**

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Fibrinolysis indications

- ST segment elevation >1mm in two contiguous leads
- New LBBB
- Symptoms consistent with ischemia
- Symptom onset <12 hrs prior to presentation

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STEMI cardiac care

- **STEP 2**: Determine preferred reperfusion strategy

Fibrinolysis preferred if:
- ≤3 hours from onset
- PCI not available/delayed
  - door to balloon > 90min
  - door to balloon minus door to needle > 1hr
- Door to needle goal <30min
- No contraindications

PCI preferred if:
- PCI available
- Door to balloon < 90min
- Door to balloon minus door to needle < 1hr
- Fibrinolysis contraindications
- Late Presentation > 3 hr
- High risk STEMI
  - Killip 3 or higher
- STEMI dx in doubt

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ACS/STEMI Pharmacotherapy

Medical Therapy
MONA + BAH

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Oxygen Used in Acute Coronary Syndromes

- **Oxygen** (2-4 liters/minute) (class I, level C)
  - Up to 70% of ACS patient demonstrate hypoxemia
  - May limit ischemic myocardial damage by increasing oxygen delivery/reduce ST elevation

Why?
– Increases supply of oxygen to ischemic tissue

When?
– Always when AMI is suspected

How?
– Start with nasal cannula at 4 L/min
– Remember one word: oxygen-IV-monit

Watch Out!
- Rarely COPD patients with hypoxic ventilatory drive will hypoventilate

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Morphine Sulfate: Actions, Indications

- **Morphine** (class I, level C)
- ***Why? (Actions)***
  - Analgesia
    - To reduce pain of ischemia
  - To reduce anxiety
    - decrease sympathetic tone, SVR and O_2 demand
  - To reduce extension of ischemia by reducing oygen demands
- ***When? (Indications)***
  - Continuing pain
  - Evidence of vascular congestion (acute pulmonary edema)
  - Systolic blood pressure >90 mm Hg
  - No hypovolemia

Careful with hypotension, hypovolemia, respiratory depression

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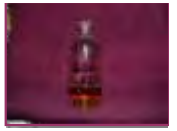
Nitroglycerin: Actions



- **Nitroglycerin** (class I, level B)
  - Analgesia—titrate infusion to keep patient pain free
  - Dilates coronary vessels—increase blood flow
  - Reduces systemic vascular resistance and preload
- Increases venous dilation
- Decreases cardiac oxygen consumption
- Dilates coronary arteries
- Increases cardiac collateral flow
- ** Sildenafil within 24 hours**

Careful with recent ED meds, hypotension, RV infarction, Aortic stenosis, peric effusion

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Aspirin: Actions

- **Aspirin** (160-325mg chewed & swallowed) (class I, level A)
  - Irreversible inhibition of platelet aggregation
  - Stabilize plaque and arrest thrombus
  - Reduce mortality in patients with STEMI
- Blocks formation of thromboxane A2 (thromboxane A2 causes platelets to aggregate and arteries to constrict)
- These actions will reduce
  - Overall mortality from AMI (23% @ 30 days)
  - Nonfatal reinfarction
  - Nonfatal stroke

Careful with active PUD, hypersensitivity, bleeding disorders

http://circ.ahajournals.org/content/92/10/2841.full#content-block

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ß-Blockers

Beta-Blockers (class I, level A)

- 14% reduction in mortality risk at 7 days
- 23% long term mortality reduction in STEMI
- 13% reduction in risk of progression to MI in patients with threatening or evolving MI symptoms
- Contraindications (CHF, Heart block, Hypotension)
- Reassess for therapy as contraindications resolve

Mechanism of action

- Blocks catecholamines from binding to $ß_1$ receptors
- Reduces HR, BP, myocardial contractility
- Decreases AV nodal conduction
- Decreases incidence of primary VF

(Lopressor 5mg IV q5 min x3 ot 25-50 mg po bid - without contraindication)

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ß-Blockers

Absolute Contraindications

- Severe CHF/PE
- SBP <100 mm Hg
- Acute asthma (bronchospasm)
- 2nd- or 3rd-degree AV block

Cautions

- Mild/moderate CHF
- HR <60 bpm
- History of asthma
- IDDM
- Severe PVD

Metoprolol	15 mg IV × 1 then 200 mg/day PO in div doses	MIAMI[19]
Atenolol	5-10 mg IV × 1, then 100 mg/day PO	ISIS-1[20]
Carvedilol	6.25 mg bid titrated to 25 mg BID	CAPRICORN

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- **ACE-Inhibitors / ARB** (class I, level A)
  - Start in patients with anterior MI, pulmonary congestion, LVEF < 40% in absence of contraindication/hypotension
  - Start in first 24 hours
  - ARB as substitute for patients unable to use ACE-I

Agent	Dosing (PO)	Original Trial
Captopril	6.25 mg tid titrated to 50 mg tid	SAVE: 3-16 days post-MI in asymptomatic patients with EF <40%[22]
Ramipril	1.25 mg bid titrated to 5 mg bid	AIRE: 3-10 days post-MI with symptoms of heart failure[23]
Captopril	6.25 mg bid titrated to 50 mg bid	ISIS-4: started within 24 hr of MI[24]
Lisinopril	5 mg/day titrated to 10 mg/day	GISSI-3: started within 24 hr of MI[25]

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Heparin

- **Heparin** (class I, level C to class IIa, level C)
  LMWH or UFH (max 4000u bolus, 1000u/hr)
  - Adjunct to surgical revascularization and thrombolytic / PCI reperfusion
  - 24-48 hours of treatment
  - Coordinate with PCI team (UFH preferred)
  - Used in combo with aspirin and/or other platelet inhibitors
  - Changing from one to the other not recommended
- **Indications**
  - PTCA or CABG
  - With fibrin-specific lytics
  - High risk for systemic emboli
    - large anterior MI, atrial fibrillation, or LV thrombus

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Unfractionated Heparin Dosing

Loading Dose

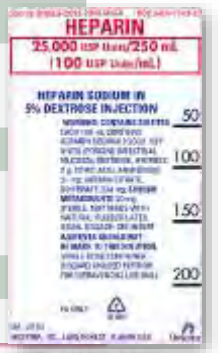
- 60 U/kg IV bolus
- Max 5000 U if >65 kg or 4000 U if <65 kg

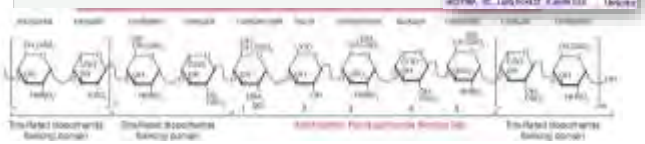
Maintenance Dose

- 12 U/kg/hr IV
- Max 1000 U/hr if >65 kg or 800 U/hr if <65 kg

Titration Goal

- PTT 50-70 sec





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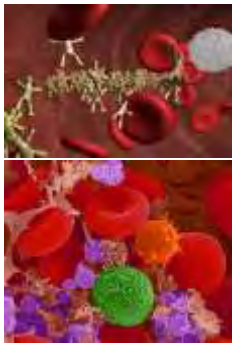
Antiplatelet Agents

- **Glycoprotein IIb/IIIa inhibitors** (class IIa, level B)
  - Inhibition of platelet aggregation at final common pathway
  - In support of PCI intervention as early as possible prior to PCI
  - Blocks glycoprotein IIb/IIIa receptors on platelets
  - Blocked receptors cannot attach to fibrinogen
  - Fibrinogen cannot aggregate platelets to platelets
- Indications: ACS with ***NO ST-segment elevation*:**
  - *–Unstable angina managed medically*
  - *–Non–Q-wave MI*
  - *–UA undergoing PCI*
- Examples:
  - –Abciximab (*reopro*)
  - –Eptifibitide (*integrilin*)
  - –Tirofiban (*Aggrastat*)
  - –Prasugrel
  - Ticagrelor
  - Vorapaxar
  - Cangrelor



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III. Dosing: Abciximab (ReoPro)

Load: 0.25 mg/kg IV bolus

Infuse: 0.125 ug/kg/min up to 10 ug/min IV x12 hours

IV. Dosing: Eptifibatide (Integrilin)

Acute Coronary Syndrome

Load: 180 ug/kg

Infuse: 2 ug/kg/min IV x72 hours

Angioplasty

Load: 135 ug/kg IV before procedure

Infuse: 0.5 ug/kg/min IV x24 hours

V. Dosing: Tirofiban (Aggrastat)

Load: 0.4 ug/kg/min for 30 minutes

Infuse: 0.1 ug/kg/min

Infusion duration

Acute Coronary Syndrome: 48 to 108 hours

Angioplasty: 12 to 24 hours

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Additional medication therapy

- **Clopidodrel** (class I, level B)
  - Irreversible inhibition of platelet aggregation
  - Used in support of cath / PCI intervention or if unable to take aspirin
  - 3 to 12 month duration depending on scenario

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Additional medication therapy

- **Aldosterone blockers** (class I, level A)
  - Post-STEMI patients
    - no significant renal failure (cr < 2.5 men or 2.0 for women)
    - No hyperkalemia > 5.0
    - LVEF < 40%
    - Symptomatic CHF or DM
  - **Inspra®**
  - **Spironolactone**

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Post Stent Dual Antiplatelet Tx,

Treatment Modality	Aspirin	Clopidogrel
Medical management	75-162 mg/day indefinitely	Optional: 75 mg/day × 1 month
Bare Metal stent	162-325 mg/day × 1 month, then 75-162 mg/day indefinitely	300 mg loading dose,* then 75 mg/day × 1 month
Sirolimus eluting stent (Cypher)	162-325 mg/day × 3 months, then 75-162 mg/day indefinitely	300 mg loading dose,* then 75 mg/day × 1 year
Paclitaxel eluting stent (Taxus)	162-325 mg/day × 6 months, then 75-162 mg/day indefinitely	300 mg loading dose,* then 75 mg/day × 1 year

Table 1: Antiplatelet Medications
Note: No loading dose in patients older than 75 years.

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STEMI care CCU

- Monitor for complications:
  - recurrent ischemia, cardiogenic shock, ICH, arrhythmias
- Review guidelines for specific management of complications & other specific clinical scenarios
  - PCI after fibrinolysis, emergent CABG, etc…
- Decision making for risk stratification at hospital discharge and/or need for CABG

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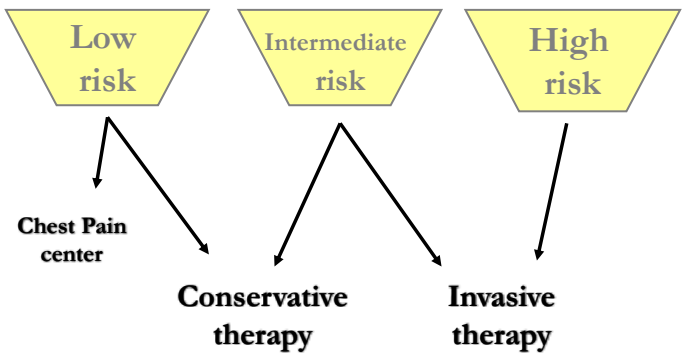
Cases to Ponder…

- 64 y/o female collapses in the cafeteria with c/o new onset chest pain. She has no cardiac history, her troponin is negative and her ECG has non-diagnostic, fixed changes.
- A 75 y/o male visitor is sagging against the wall in the hallway, he is diaphoretic, with weak rapid pulses. You notice the top of a sternotomy scar at his neckline.
- Your diabetic co-worker just returned from a prolonged break and states she had a bit of chest pain but it's gone now. You hook her up to a monitor and notice inverted T waves in II and III

63



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Invasive therapy option UA/NSTEMI

- Coronary angiography and revascularization within 12 to 48 hours after presentation to ED For high risk ACS (class I, level A)
- MONA + BAH *(UFH)*
- Clopidogrel
  - 20% reduction death/MI/Stroke – CURE trial
  - 1 month minimum duration and possibly up to 9 months
- Glycoprotein IIb/IIIa inhibitors

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Conservative Therapy for UA/NSTEMI

- Early revascularization or PCI not planned
- MONA + BAH *(LMW or UFH)*
- Clopidogrel
- Glycoprotein IIb/IIIa inhibitors
  - Only in certain circumstances (planning PCI, elevated TnI/T)
- Surveillance in hospital
  - Serial ECGs
  - Serial Markers

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Secondary Prevention

- Disease
  - HTN, DM, HLP
- Behavioral
  - smoking, diet, physical activity, weight
- Cognitive
  - Education
  - cardiac rehab program

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Secondary Prevention disease management

- Blood Pressure
  - Goals < 140/90 or <130/80 in DM /CKD
  - Maximize use of beta-blockers & ACE-I
- Lipids
  - LDL < 100 (70) ; TG < 200
  - Maximize use of statins; consider fibrates/niacin first line for TG>500; consider omega-3 fatty acids
- Diabetes
  - A1c < 7%

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Secondary prevention behavioral intervention

- **Smoking cessation**
  - Cessation-class, meds, counseling
- **Physical Activity**
  - Goal 30 - 60 minutes daily
  - Risk assessment prior to initiation
- **Diet**
  - DASH diet, fiber, omega-3 fatty acids
  - <7% total calories from saturated fats



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Thinking outside the box…

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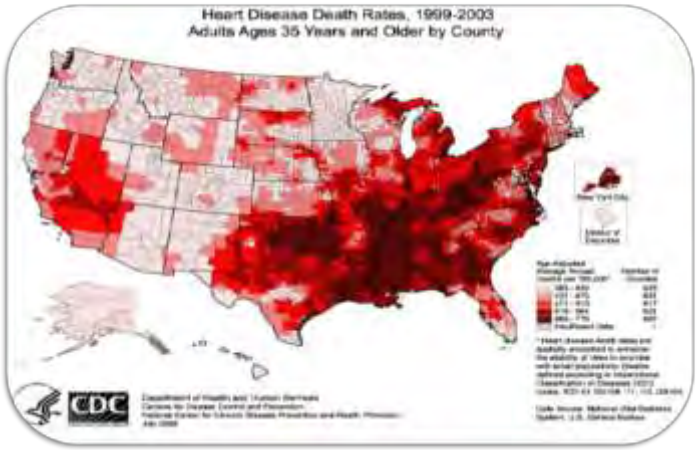
Or maybe just move….



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Secondary prevention cognitive

- **Patient education**
  - In-hospital – discharge –outpatient clinic/rehab
- **Monitor psychosocial impact**
  - Depression/anxiety assessment & treatment
  - Social support system

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Medication Checklist after ACS

- **Antiplatelet agent**
  - Aspirin* and/or Clopidogrel
- **Lipid lowering agent**
  - Statin*
  - Fibrate / Niacin / Omega-3
- **Antihypertensive agent**
  - Beta blocker*
  - ACE-I*/ARB
  - Aldactone (as appropriate)

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Summary

- ACS includes UA, NSTEMI, and STEMI
- Management guideline focus
  - Immediate assessment/intervention (MONA+BAH)
  - Risk stratification (UA/NSTEMI vs. STEMI)
  - RAPID reperfusion for STEMI (PCI vs. Thrombolytics)
  - Conservative vs Invasive therapy for UA/NSTEMI
- Aggressive attention to secondary prevention initiatives for ACS patients
  - Beta blocker, ASA, ACE-I, Statin

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- ***Reserve Calcium channel blocker therapy for patients who do not respond to or do not tolerate either nitrates or beta-blockers***
- ***If LDL greater than 100, begin statin therapy 24-96 hrs after admission***
- ***If HDL < 40 use a fibrate or niacin***
- ***Consider that fatty fish and fish oil supplements have been proven to reduce death after MI***
- ***Consider that herbal products and dietary supplements including L-arginine, L-carnitine and B complex vitamins have been used to relieve angina***
- ***Maintain B/P < 130/85 while avoiding antiHTNsives adverse effects.***

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Unraveling the web of 12-Lead Interpretation

Lynnette Flynn
Corporate Trainer MedCath Inc
Informatics Coordinator WRMC
CCRN, RCIS, LRT, LNC

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Unraveling the web of 12-Lead Interpretation

Foundations
- Waveforms
- Lead placement
- ECG layout
- Normals
  - Ischemia
  - Injury
  - Necrosis
- **Localizing**
  - Anterior
    - antero septal
    - antero lateral
  - Lateral
  - Inferior
  - Posterior

Extended Lead ECG
- RVMI
- Posterior

•Non diagnostics
- Pericarditis
- LV aneurysm
- Captured Pacer
- Bi-Ventricular Pacer

•Electrolyte disturbances
- Hyperkalemia
- Hypokalemia
- Hypercalcemia
- Hypocalcemia

Conduction abn
- Bundle Branches
  - –LBBB
  - –RBBB
- fasicular blocks
  - – LAFB
  - – LPFB
- Bifascicular
- Trifascicular

•Hypertrophies
- LVH
- RVH
- LAE
- RAE

Other conditions
- Pulmonary Emb.
- Cerebral Events
- Brugada Syndrome
- Long QT syndrome
- Wolfe-Parkinson White syndrome

Aberrant conduction
- Ashman's
- SVT with aberrant conduction versus VT
- LBBB with acute MI

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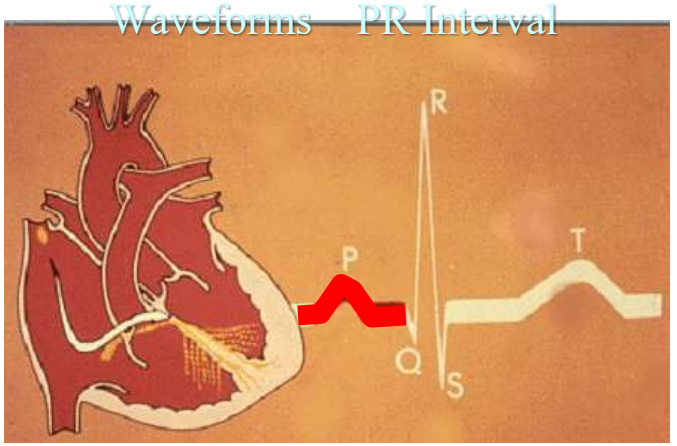

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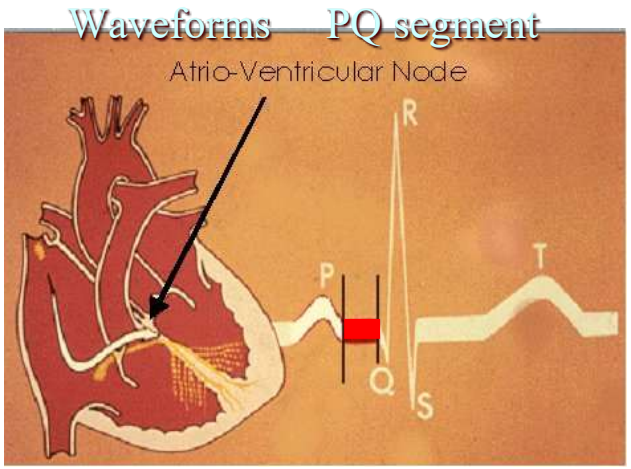

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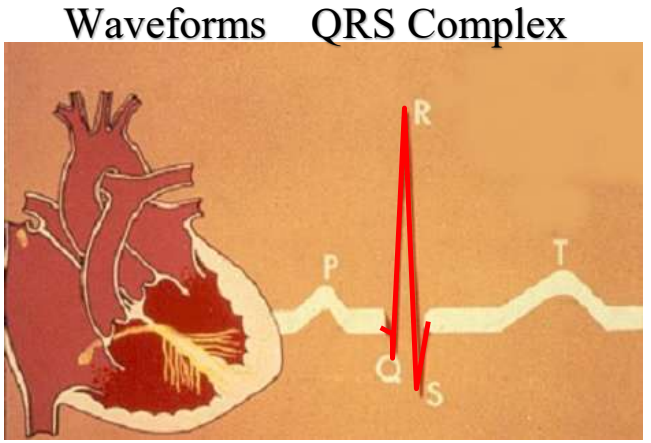

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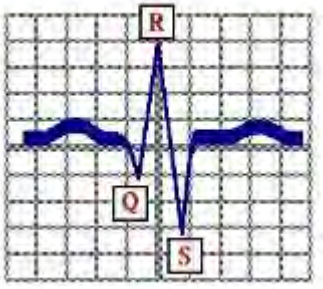
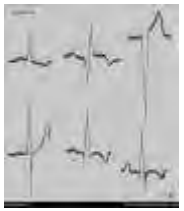
QR&S Waves





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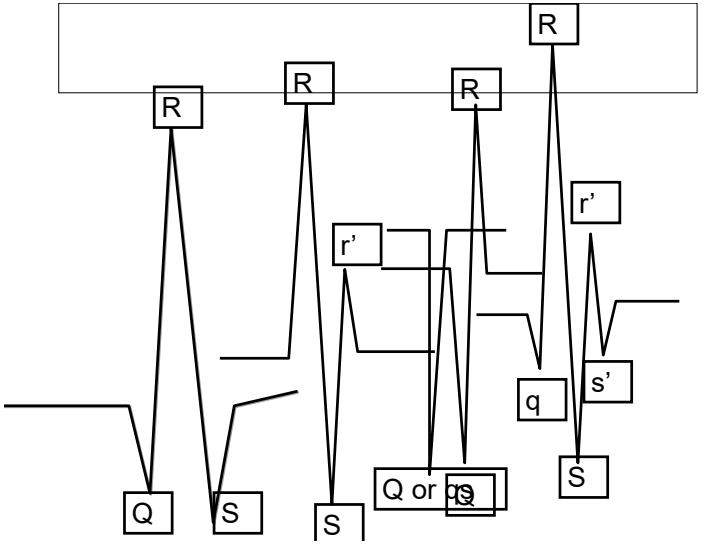

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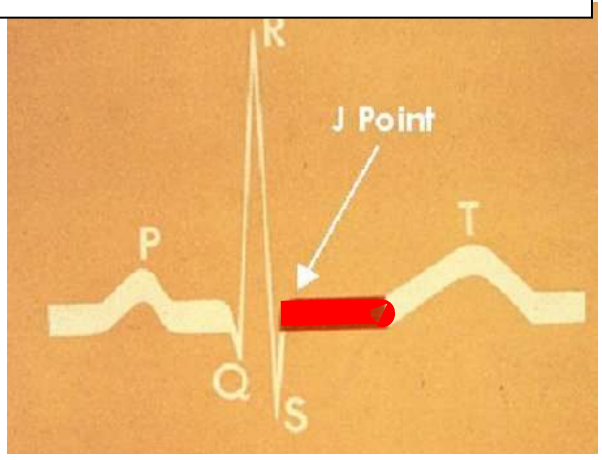
Waveforms ST Segment



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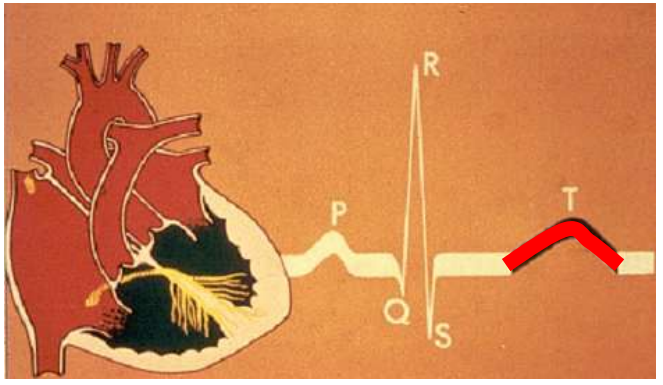
Waveforms T-Wave



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Standard Lay-out

Lead I	aVR	V1	V4
Lead II	aVL	V2	V5
Lead III	aVF	V3	V6

Rhythm Strip one
Rhythm Strip two
Rhythm Strip three

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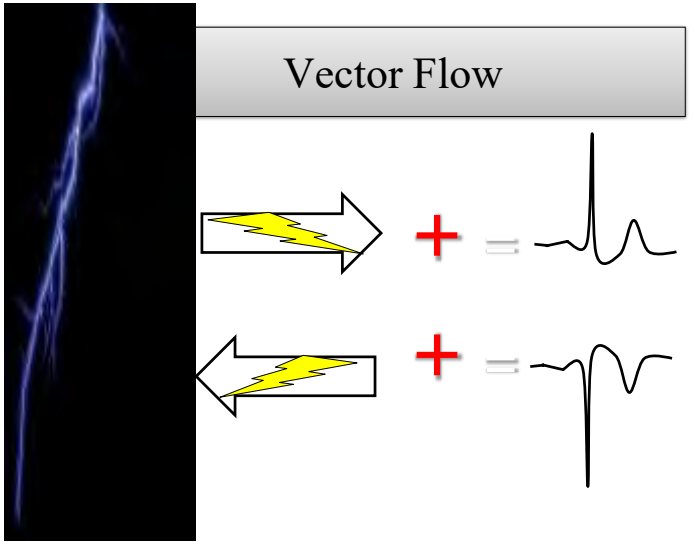

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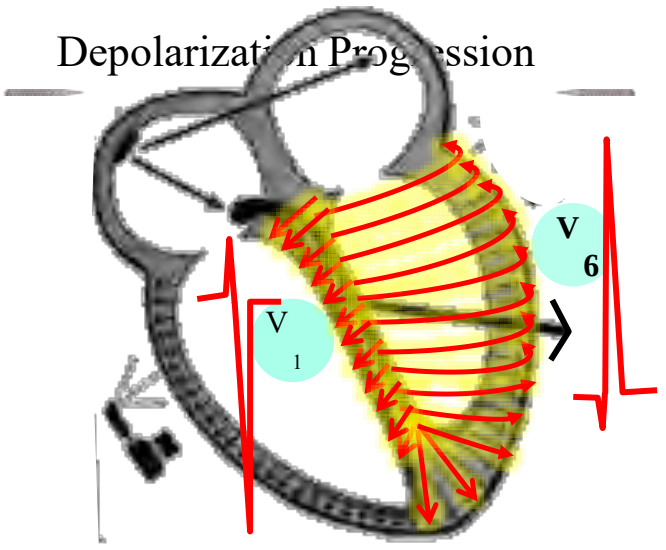

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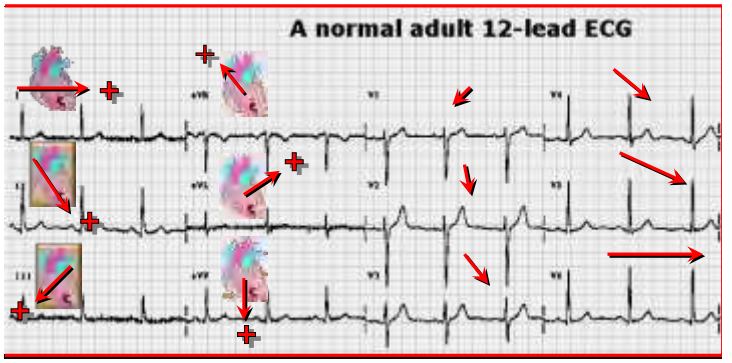
Normal 12-Lead



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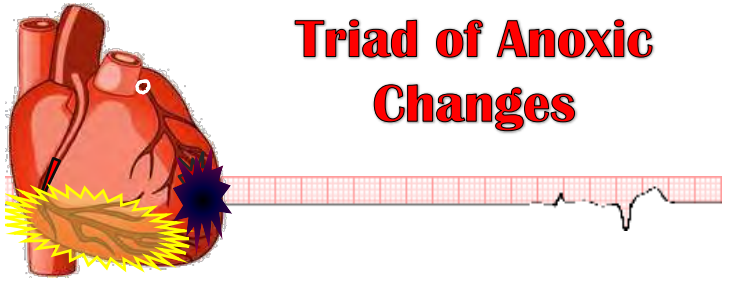

Triad of Anoxic Changes

- **ST Elevation** **Injury** **MI**
- **T-Wave Inversion** **Ischemia** **Angina**
- **Abnormal Q-wave** **Necrosis** **Tissue Death**

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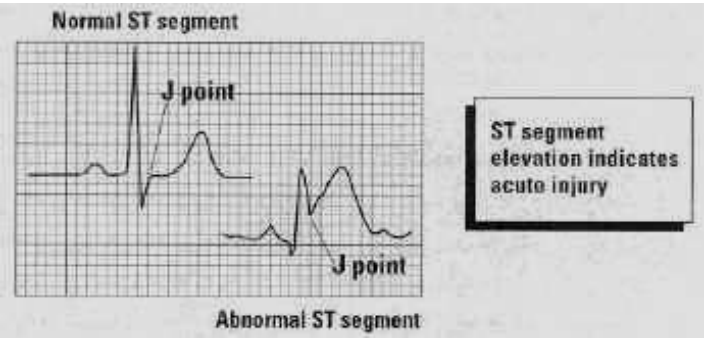
Triad of Anoxic Changes
ST Elevation



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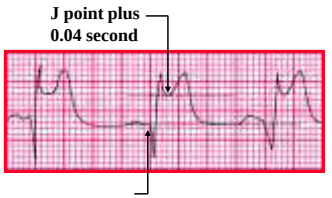
12-Lead ECG

- ST elevation
- = Injury
- = Acute MI

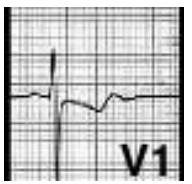


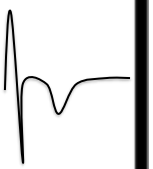
- STE >1mm indicates MI
- Must be present in at least 2 contiguous leads

96

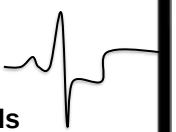
12-Lead ECG

- **T-Wave Inversion**
- **= Ischemia**
- **= Angina**





- **Resolves when perfusion restored**
- **Global may be pericarditis**
- **Must be present in 2 contiguous leads**



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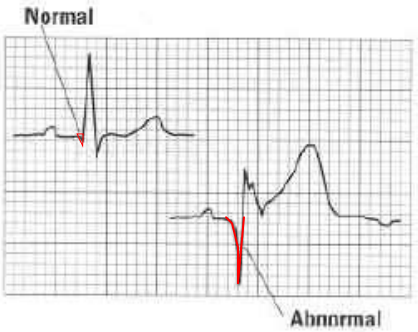
Abnormal Qs

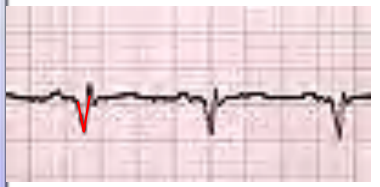


Abnormal Q wave accompanied by ST segment elevation indicates both infarct and injury stages are present.

98

12-Lead ECG

- Abnormal Q-Wave
- = Necrosis
- = Old MI
- Permanent indicator of previous infarct
- > 2 mm wide
- >2 mV deep
- III, aVL, and V6 may have small normal q



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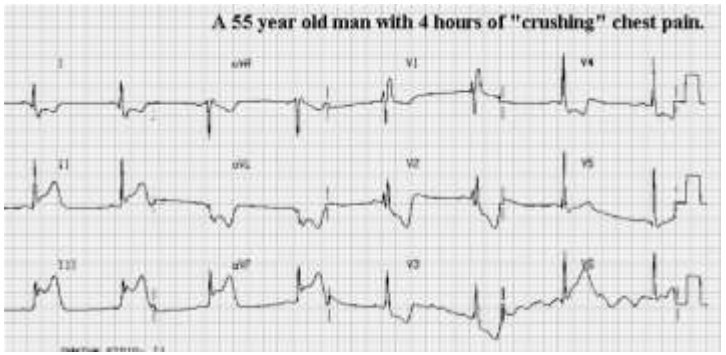

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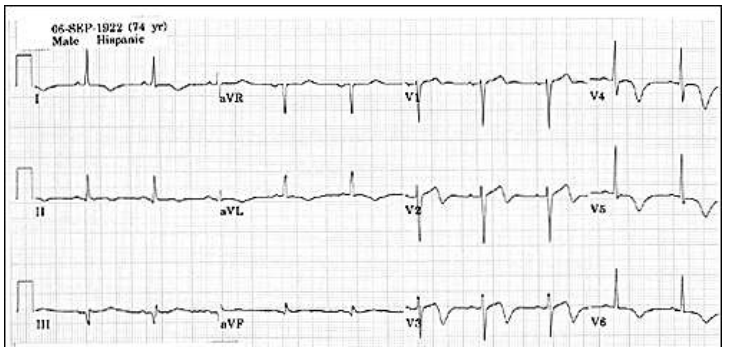

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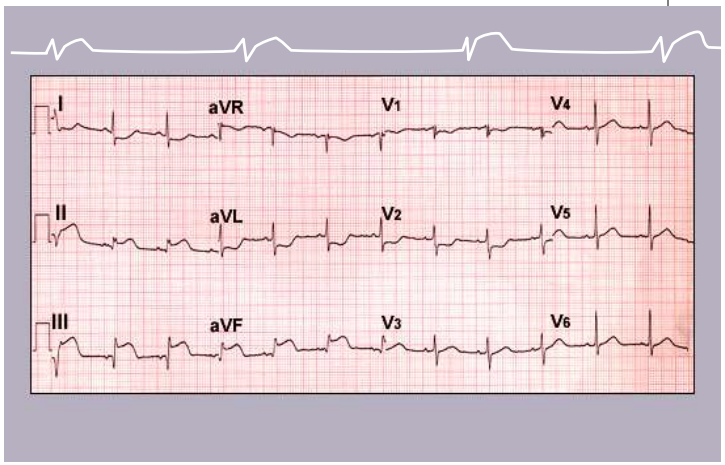

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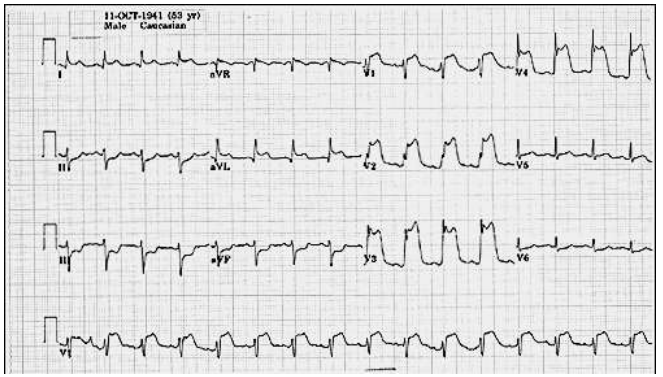

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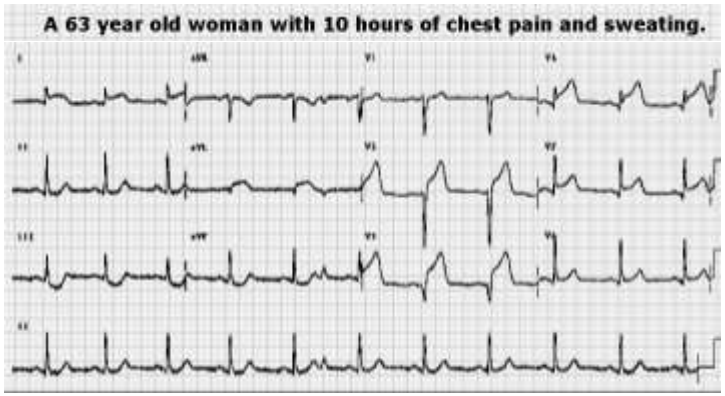

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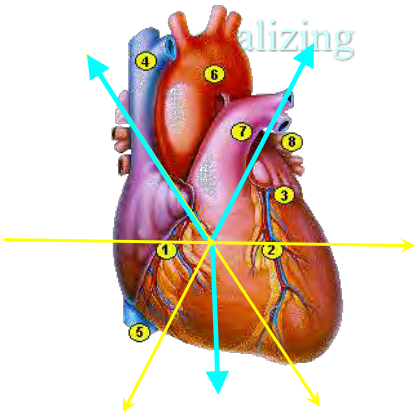

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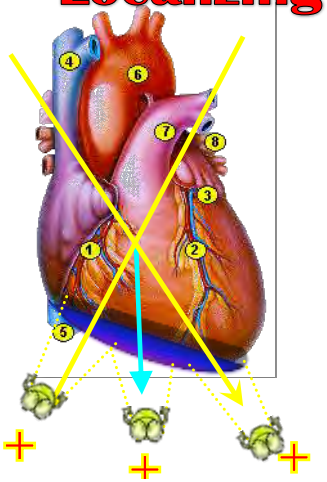
Localizing

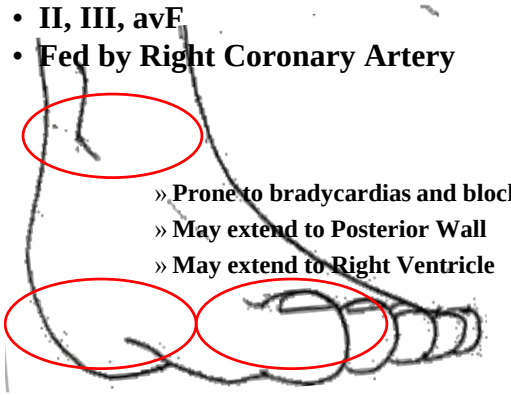


106

Localizing

Changes

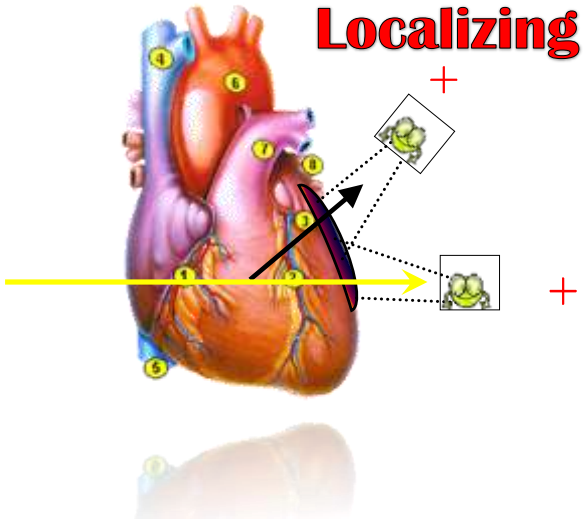
- **Inferior Wall**
- **II, III, avF**
- **Fed by Right Coronary Artery**



» **Prone to bradycardias and blocks**
» **May extend to Posterior Wall**
» **May extend to Right Ventricle**

107

Localizing

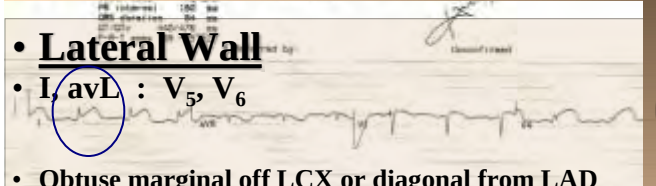


108

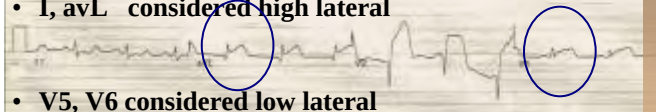
Localizing

Changes

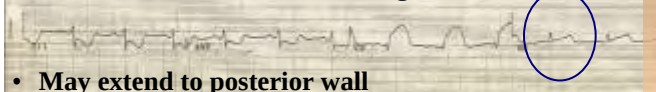
- **Lateral Wall**
- **I, avL : V_5, V_6**



- **Obtuse marginal off LCX or diagonal from LAD**
- **I, avL considered high lateral**



- **V5, V6 considered low lateral**
- **Most common cause of non-diagnostic ECG MI**



- **May extend to posterior wall**

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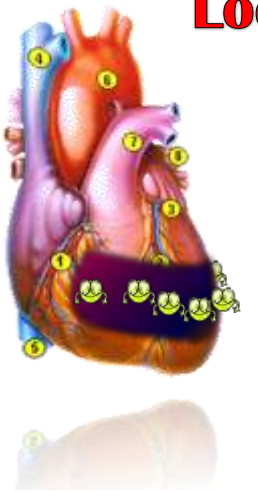
Localizing



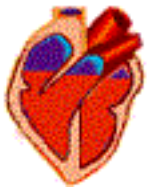
110

Localizing

Changes

- Anterior Wall
- V Leads
- Left Anterior Descending Artery
- Prone to VT and VF

111



Localizing

Changes

More Specific V Lead:

- V_1-V_3 = Antero-Septal
  - Highest mortality MI
  - Prone to bundle branch, and fasicular blocks
  - High-risk for septal rupture and PEA
- V_5-V_6 = Antero-Lateral
  - Tied to Inferior, Lateral or Anterior MI
  - Indicate LAD dominance

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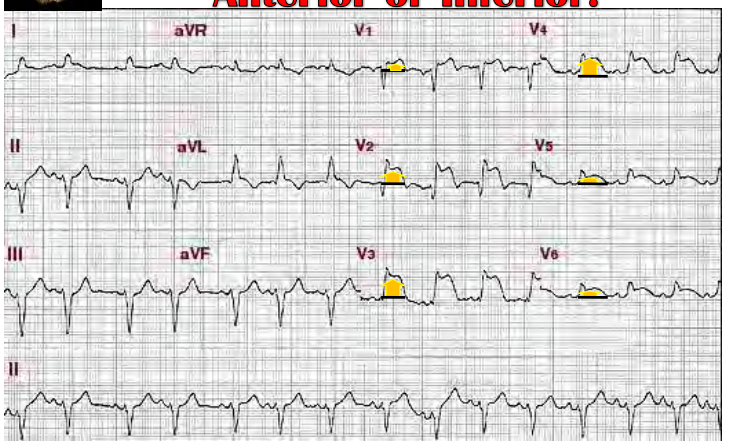
ECG #1

Anterior or Inferior?



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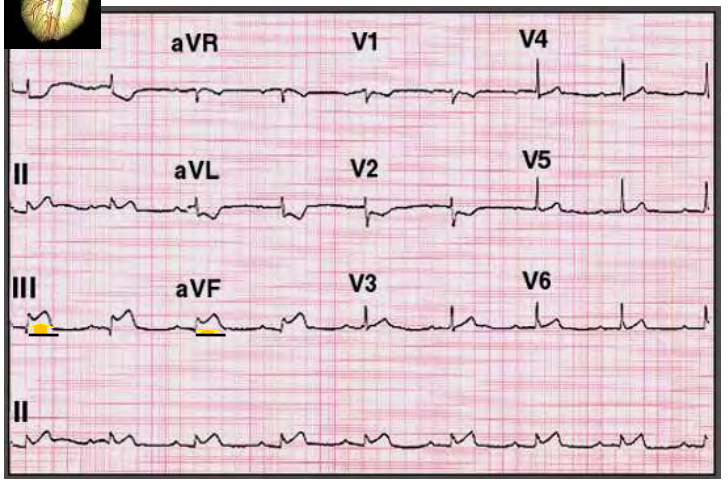
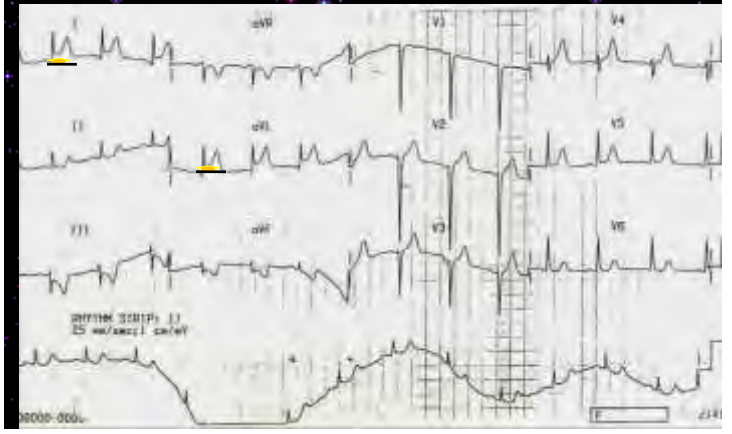
ECG #2



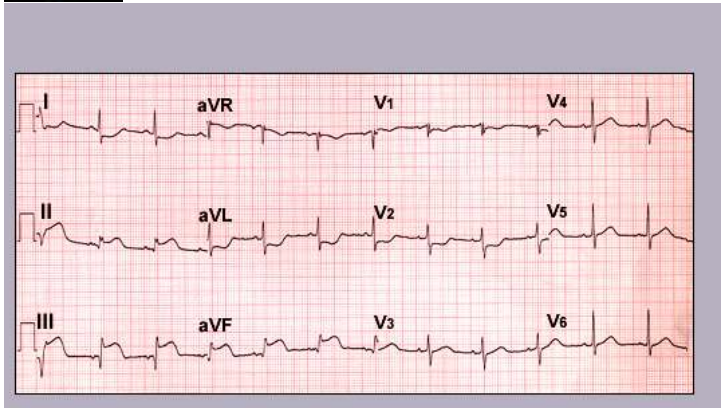
114

Jose Cuervo



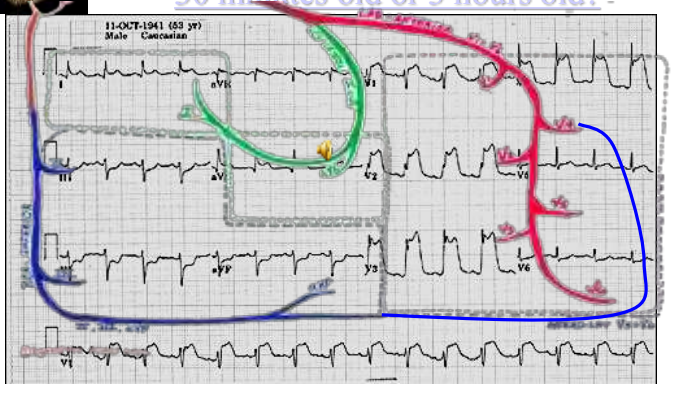
115

ECG #3
Anterior, Inferior?
30 minutes old or 3 hours old?



116

ECG #5
Anterior, Inferior?
30 minutes old or 3 hours old?



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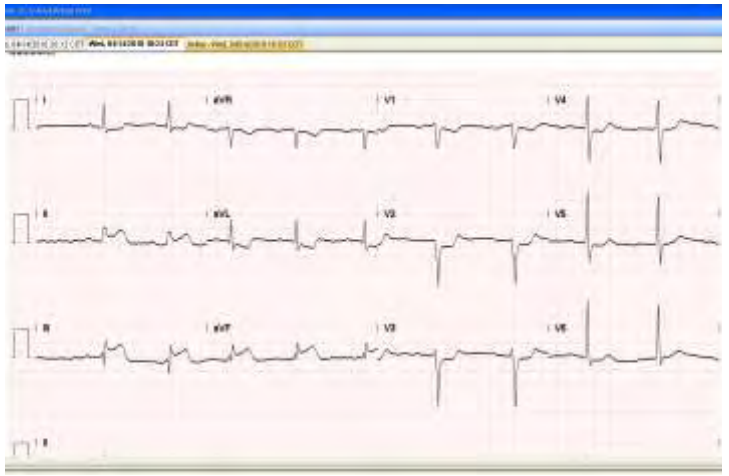

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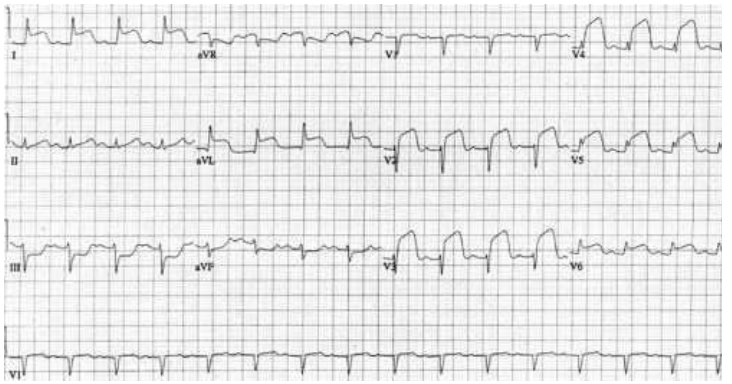
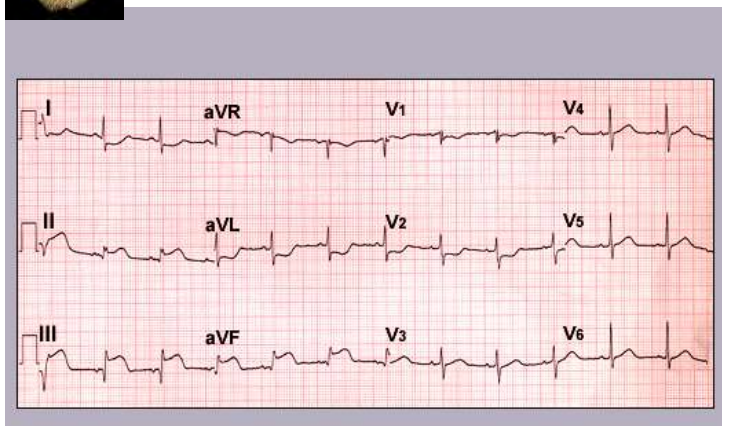
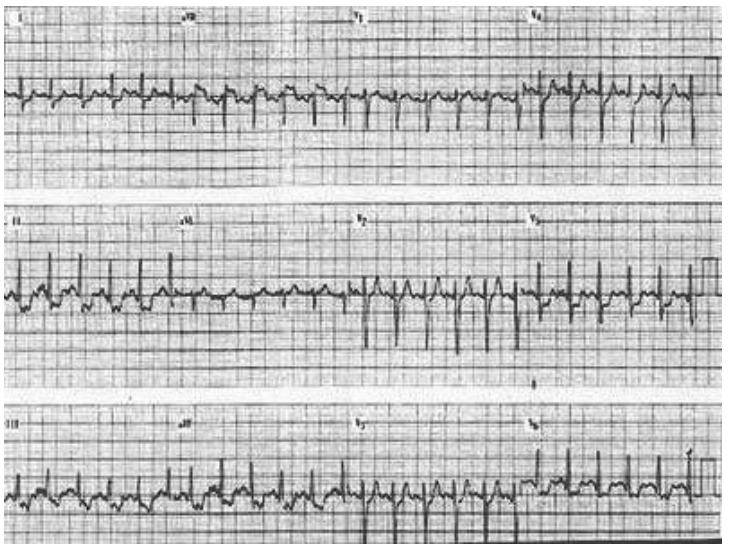


119

ECG #3
Anterior, Inferior?
30 minutes old or 3 hours old?

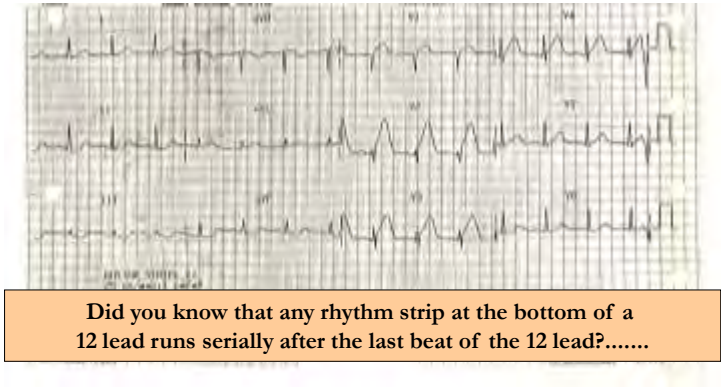


120



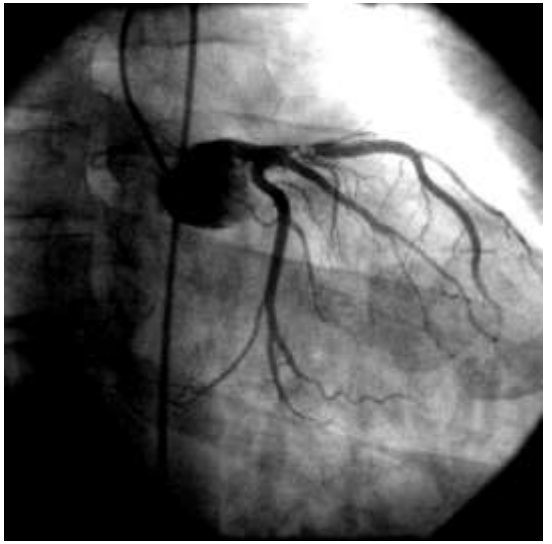
122

Patient being evaluated for USA



Did you know that any rhythm strip at the bottom of a 12 lead runs serially after the last beat of the 12 lead?.....

123



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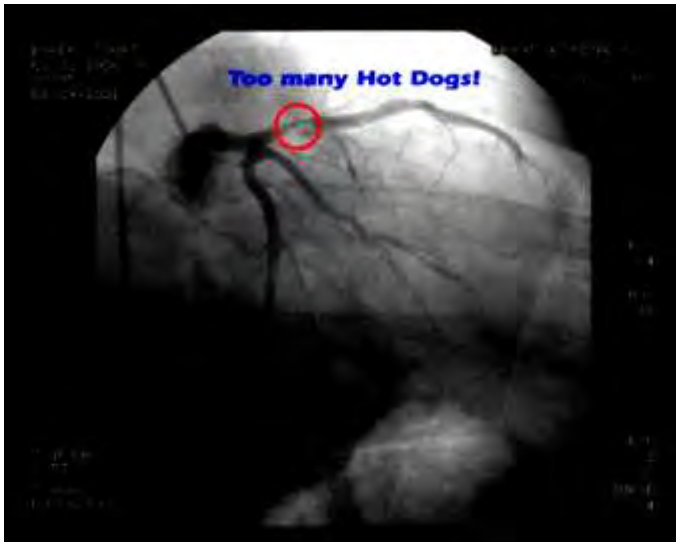

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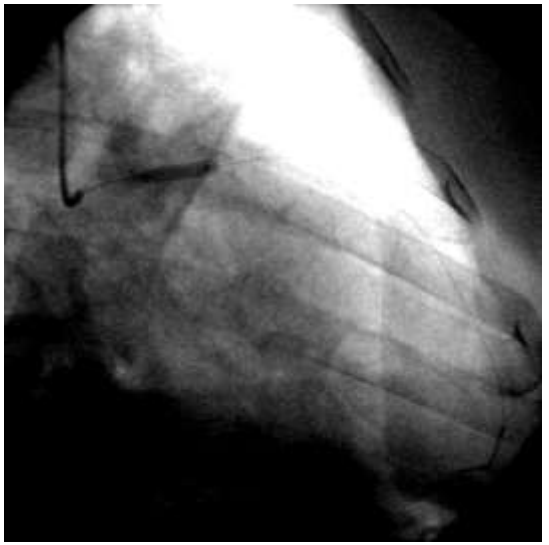

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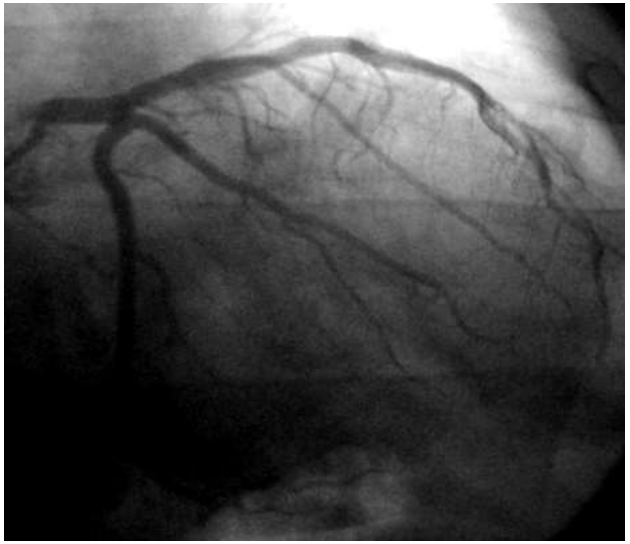
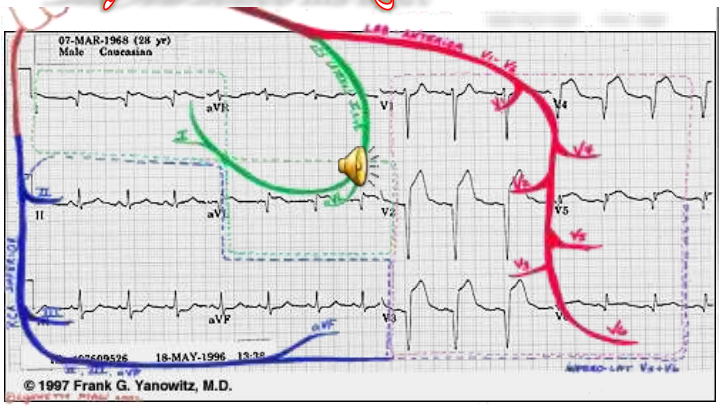


127

Did you notice his age!

ECG #6



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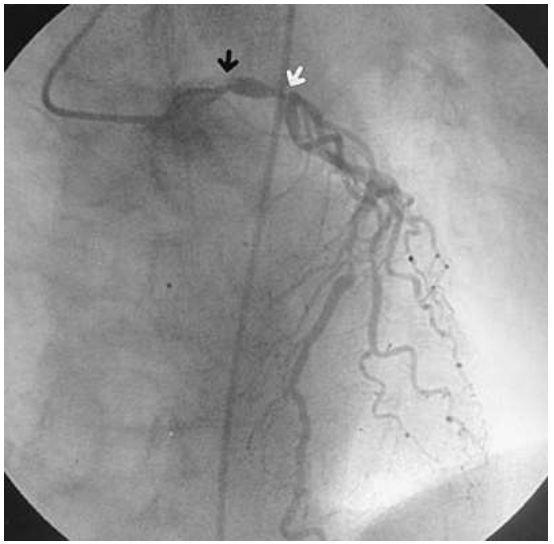


129

Worksheets

- Review 1 and 3
- Find all STElevation
- Find any pathological Q waves
- Localize all changes to Anterior, Inferior or Lateral

130

Reciprocals

131

Reciprocal MI

Mirror image changes to acute STElevation
Assists in estimating size of injury

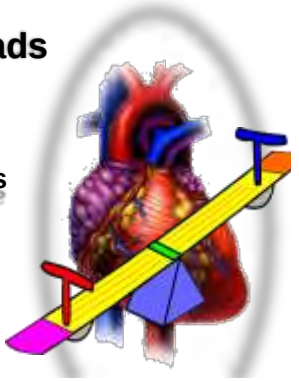
Inferior leads
II III aVF

Mirror leads
I aVL

Anterior Leads
V1-6

Limb leads

Posterior Wall
V1-V3



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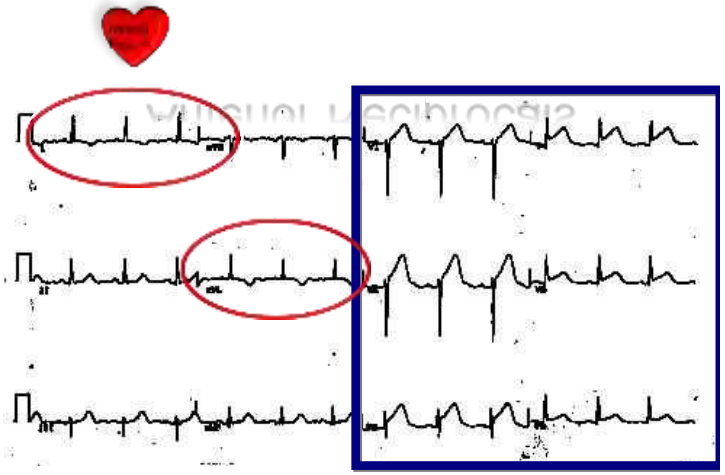

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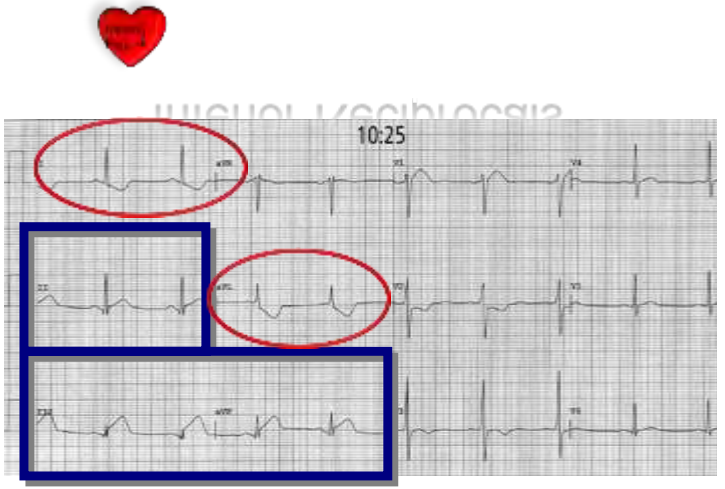
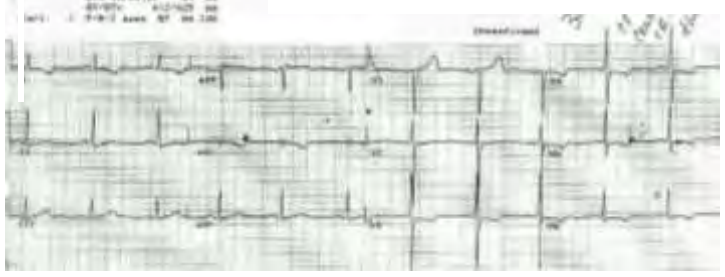


134

49 y/o 3ppd smoker
CP x 3 days intermittently
Constant @ 0700

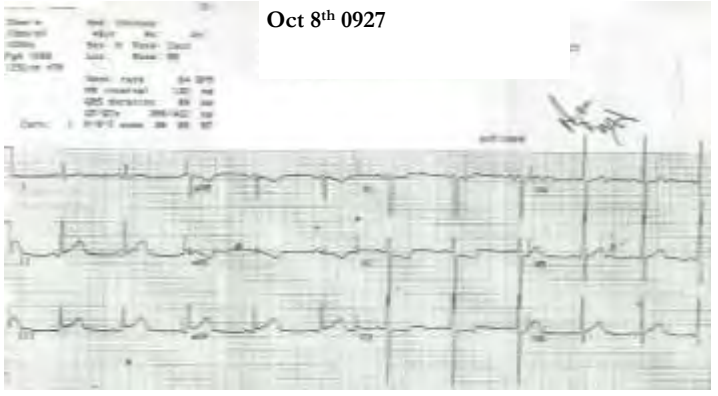
Oct 8th 0842



135

TPA started @ 0927

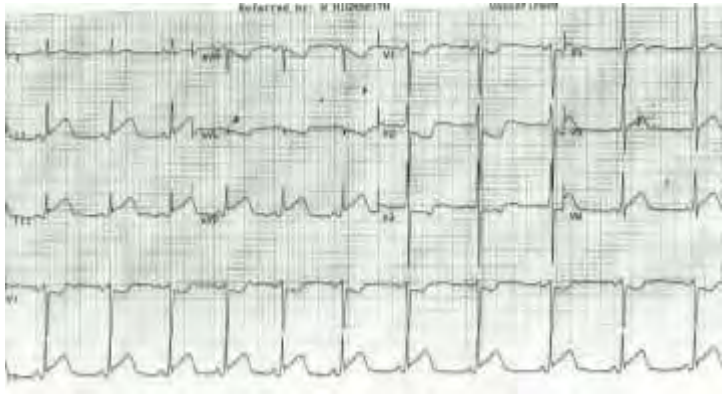
Oct 8th 0927



136

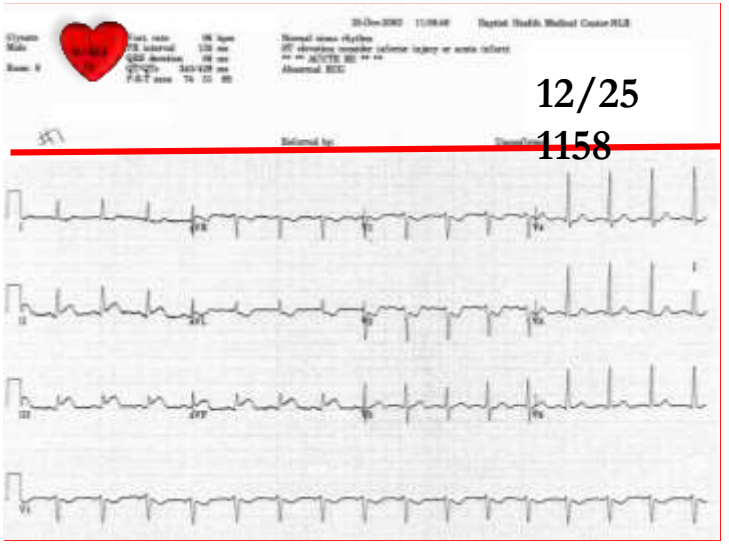
October 8th
1002

SCA next morning.
Thrombotic mid LCX
+ >75% 3VD



137

12/25
1158



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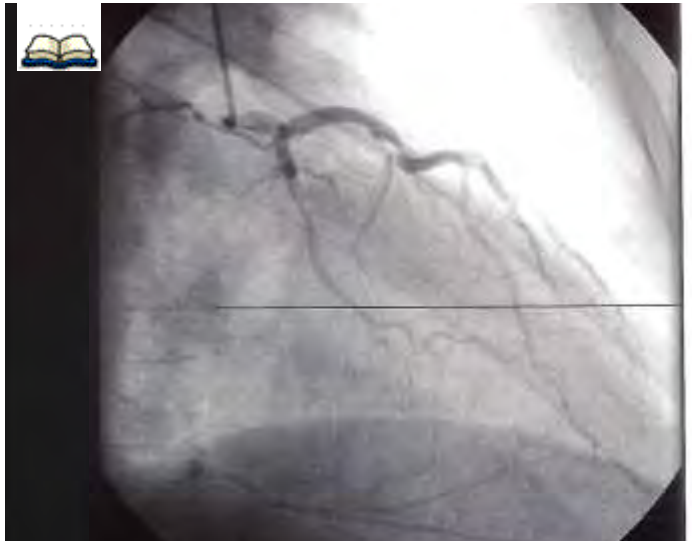

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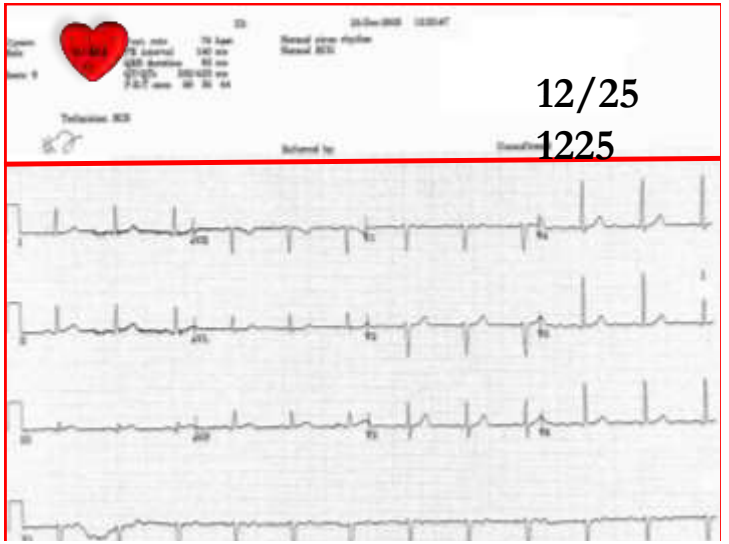
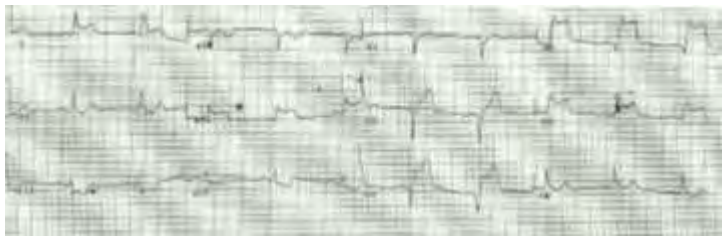


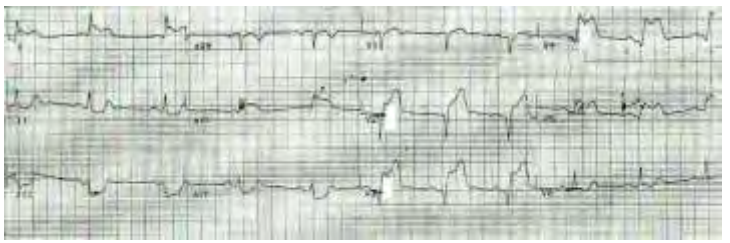
140

78 y/o male arrives at the ER c/o CP 8/10 of 6 hours duration
9/25 @23:25



141

20 minutes later. After ASA O2, Heparin, and MSO4 patient is asleep and felt to be pain free…….. 9/25 @ 23:44



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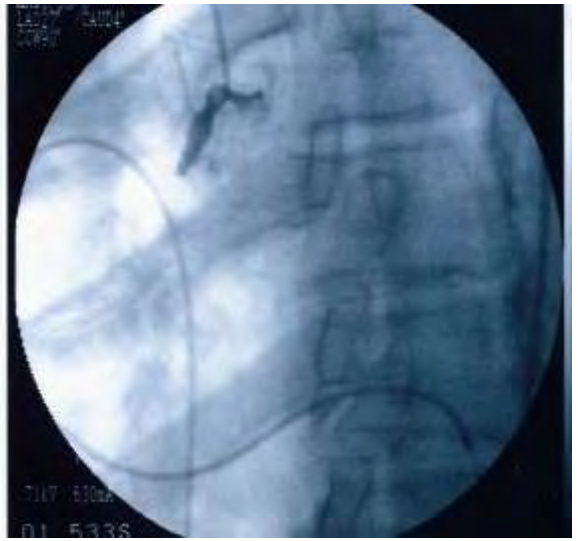

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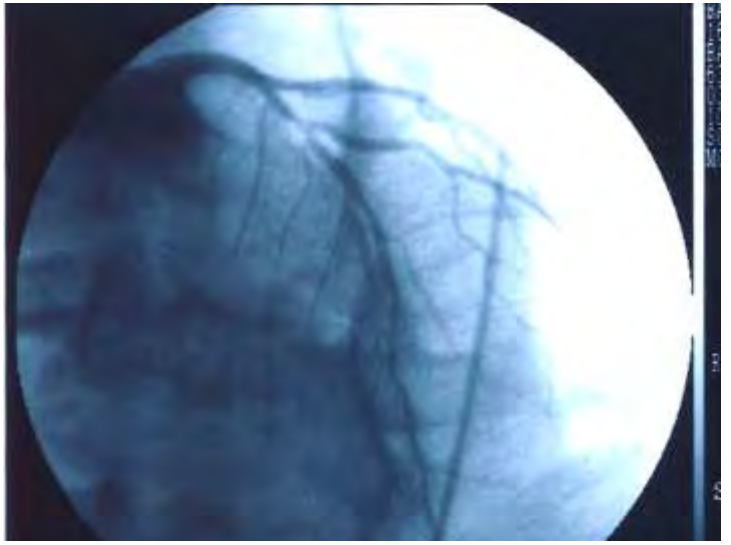


144

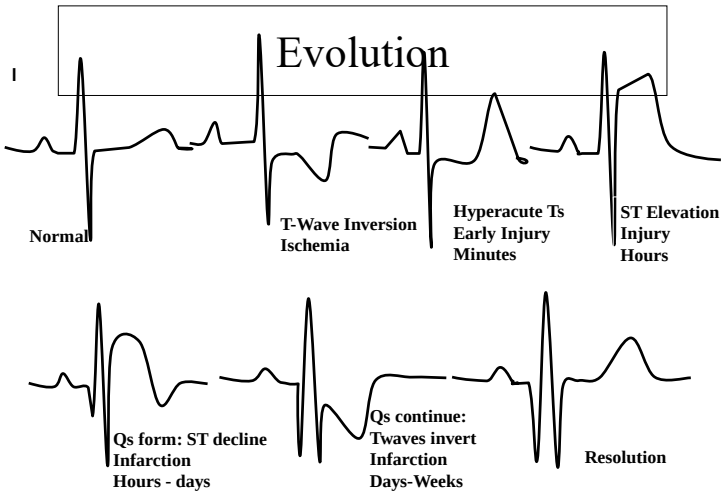
Worksheets

- Review 1
- Review 3
- Find all reciprocal changes

145

Evolution

146



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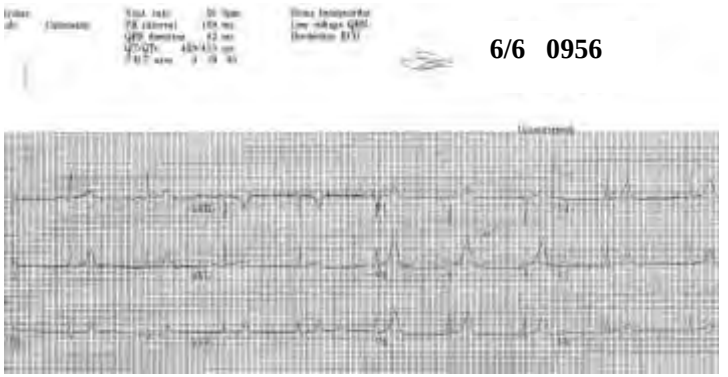

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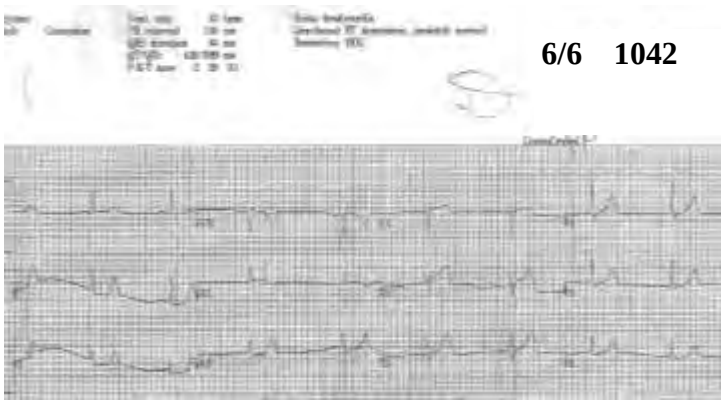


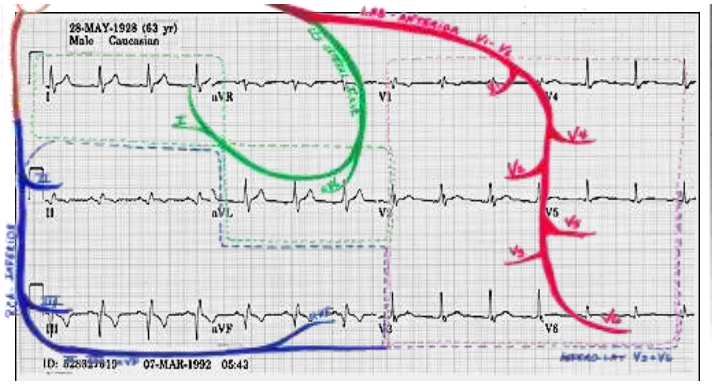
149

The attached ECG was a 56 y/o male farmer who came to the ED with complaints of SOB as you describe thinking he had a touch of the flu.

On questioning he admitted that about 5 days ago he had a weak spell while out in the garden and had to lie down for several hours before regaining the strength to walk into the house.

He has been laying around on the couch since them with progressive weakness and this presenting SOB which he thinks must be the flu

150



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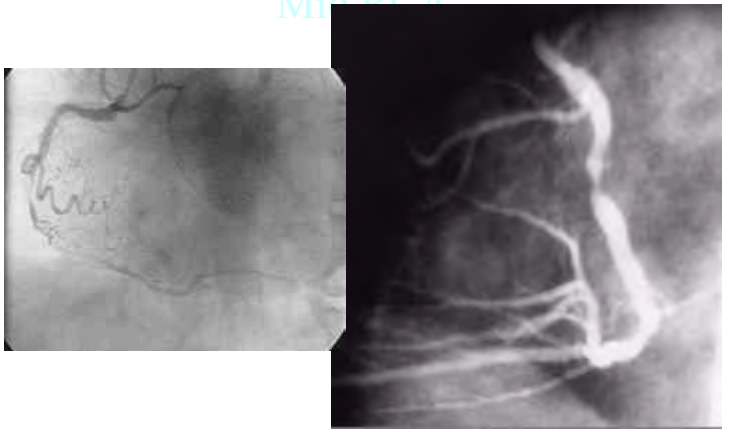


152

- List all possible differentials
- What lab will help define process?
- What emergent steps should be taken?
  - (refer to prompt protocol)

153

Worksheets

- Compare 1 and 2
- Compare 3 and 4

154

155

CHIEF COMPLAINT:

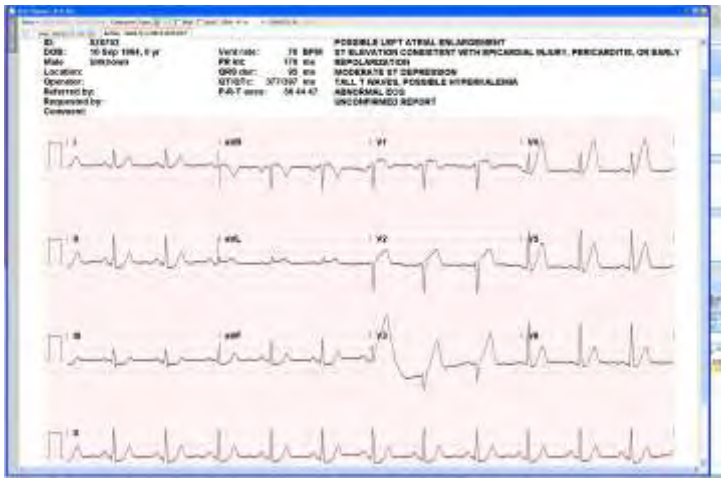
Interscapular aching.

HISTORY OF PRESENT ILLNESS:

The patient is a 48-year-old man without known ischemic heart disease who developed the acute onset of severe, unrelenting interscapular pain at 8:00 this morning. Pain persisted, and he sought medical attention. In the emergency room triage, his ECG showed anterior ST elevation, consistent with an evolving infarction. He had noted exertional chest tightness for the past several weeks, consistent with angina. He has a positive family history of ischemic heart disease with his father having had an MI at age 49. He has been a light smoker with less than 5-pack-year smoking history. He has had borderline hypertension and hyperlipidemia treated with pravastatin 40 mg daily.

Troponin 8.4

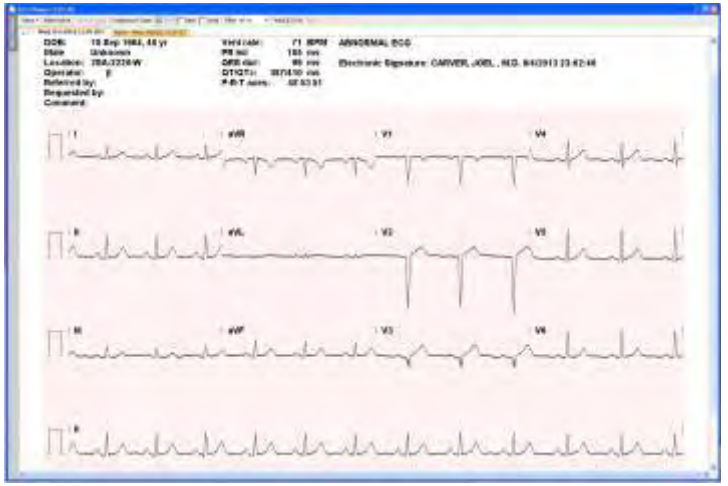
156



157

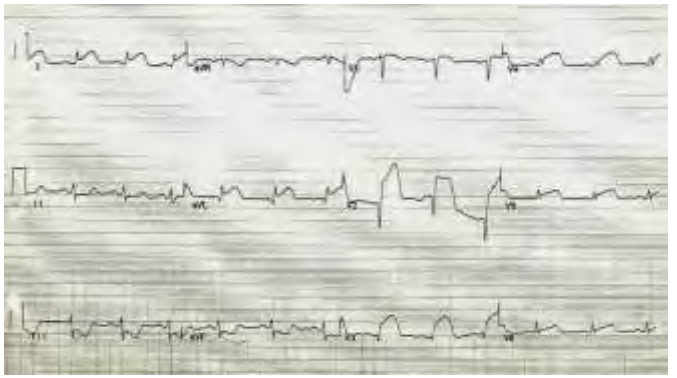
- SELECTIVE CORONARY ANGIOGRAPHY
- A. LEFT MAIN CORONARY ARTERY: The left main is free of disease.
- B. LEFT ANTERIOR DESCENDING CORONARY ARTERY: The left anterior descending coronary artery is a large wrap around vessel. It was totally occluded in its mid segment. This is reduced to no appreciable narrowing with PTCA and drug eluting stent deployment.
- C. CIRCUMFLEX CORONARY ARTERY: The circumflex system consists of a large OM 1. The circumflex system is normal.
- D. RIGHT CORONARY ARTERY: The right coronary artery is large, dominant, and normal.

158



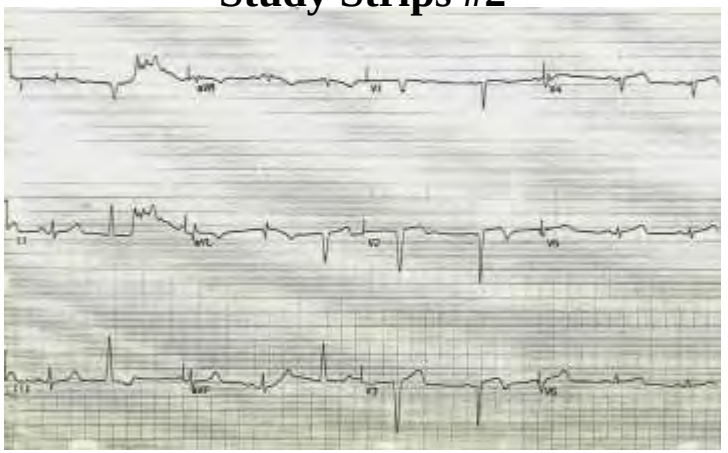
159

Study Strips #1



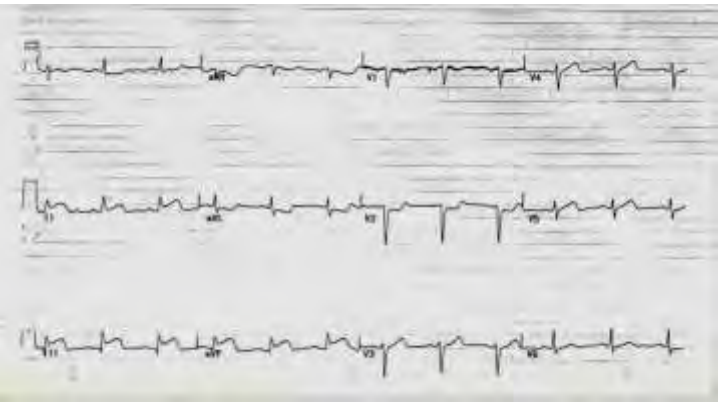
160

Study Strips #2



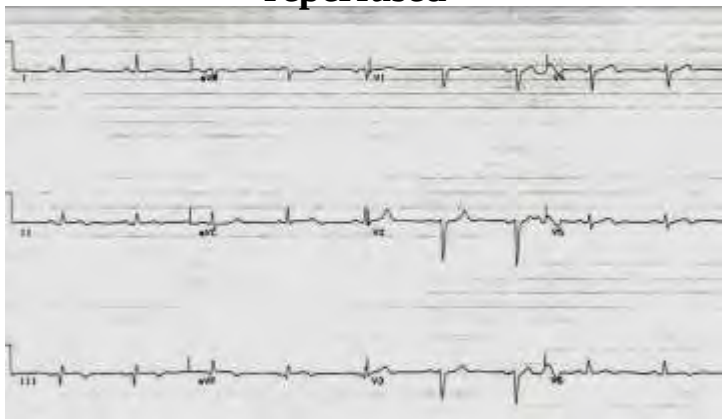
161

Study Strips #3 05:48



162

Study Strips #4
reperfused



163

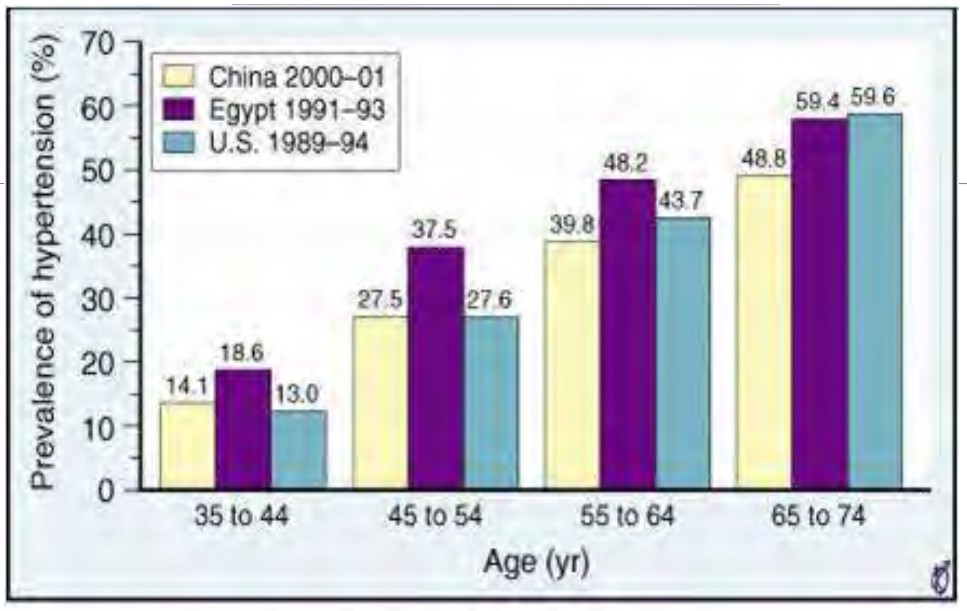
Vasopressors
Hypertensive Crisis

Based on AACN CCRN-PCCU core curriculum
Lynnette Flynn
MB-HA, CHFN, CCRN-CMC, RCIS, CNOR
Director of Education
Arkansas Heart Hospital

Key Concept

HYPERTENSIVE CRISIS IS A LIFE-THREATENING ELEVATION IN BP NECESSITATING EMERGENCY TREATMENT WITHIN 1 HOUR TO PREVENT SEVERE END ORGAN DAMAGE AND DEATH.

URGENT PHARMACOLOGIC THERAPY IS NEEDED TO PREVENT DEATH.



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Physical Effects

Hypertensive pathophysiology and its effects on the heart, brain, and kidneys:

- Enhanced sympathetic stimulation
- Effects of renin-angiotensin system
  - *increased fluid retention*
  - *increased systemic vasoconstriction)*
- Necrosis of arterioles
- Decreased blood flow to end organs

Heart Effects

Heart

Tachycardia

↑ CO

↓ perf. →angina→MI

CAD

LV hypertrophy

LV failure

Angina

Brain Effects

Brain

Loss of autoregulatory mechanisms

Arterial spasm→ischemia→TIA

Weakened vessels →aneurysms→ hemorrhage→CVA

Kidney Effects

Kidney

↓ Renal perf

↓ Ability to concentrate

↑ BUN, CR

↑ Proteinuria

Kidney failure

Uremia

Diagnostics

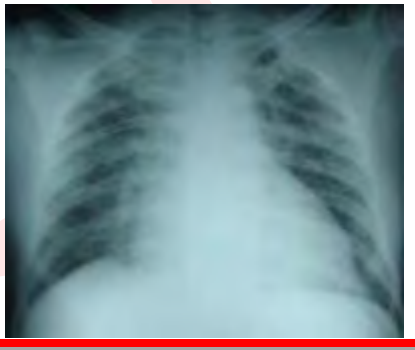
Laboratory

- BUN and creatinine
- Electrolyte levels
  - Hypocalcemia, hyponatremia, hypokalemia
- Enzyme levels for MI

ECG

Radiologic

- CXR
- Echocardiogram
- MRI or CT
- Renal ultrasound



Management

Anticipated patient trajectory:
Immediate BP reduction is essential for the prevention or minimization of end organ damage

Goals of care

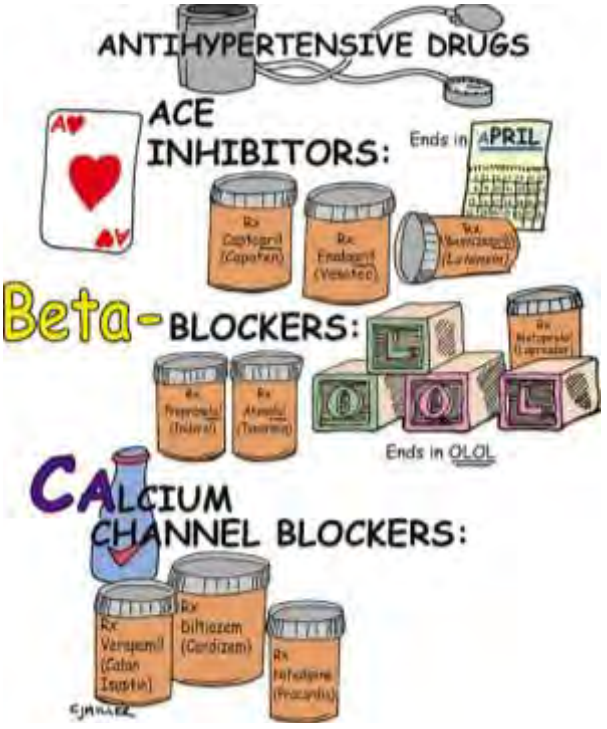
- Rapid treatment of elevated BP
- MAP is lowered in small decrements
- Cause of the hypertension is identified and treated

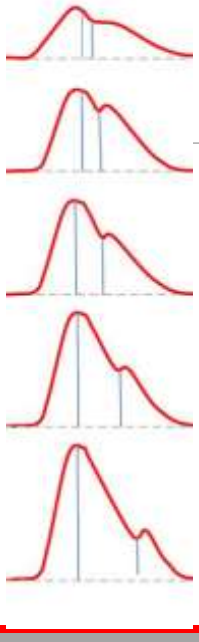
Pharmacology

Nipride: gold standard for malignant HTN therapy.

Med of choice for HTN encephalopathy, cerebral infarction or bleeding, dissecting aortic aneurysm. Ø in pregnancy

- 0.25 to 0.5 mcg/kg/min IV Titrate every 5 min
  - max 10 mcg/kg/min.
  - titrate to lowest dose.
  - Run at Max no more than 10 min
- Acts in seconds, reversed by stopping (1-5 min)
- Protect bag and lines from light
- Watch for cyanide toxicity after 48 hours or with RI.
  - Blurred vision, tinnitus, confusion
  - Thiocynate blood level at 48 hours. should not exceed 1.7 mmol/L.
- Closely monitor the patient's response to therapy





Rx Management

Nicardipine (CaCh blocker): Safer & similar effect

- Dose: 5mg/hr; titrated to a max dose of 15 mg/hr
- Half-life 3-6 hours
- Longer onset of action (9.5-10 min), longer half-life

Fenoldopam (selective dopamine receptor agonist); potent vasodilator; as effective as nipride in lowering BP

- Dose: 0.1 mcg/kg/min; titrated every 15 min to response
- Half-life is 10 min
- SEs hypokalemia, headache, flushing, dizziness, reflex tachy
- Increases intraocular pressure

Labetalol med of choice for ICH

- Dosage: 20 mg IV bolus, then 20-80 mg q 10 min or IV infusion
- An ά & ß blocking agent, esp. for adrenergic crisis.
- Does not increase heart rate (good in CAD)

Rx Management

ACE inhibitors - **Enalapril**: 1.25 - 5 mg IV every 6 hours
- Medication of choice for LV failure and pulmonary edema
- Onset of action: 10-15 minutes

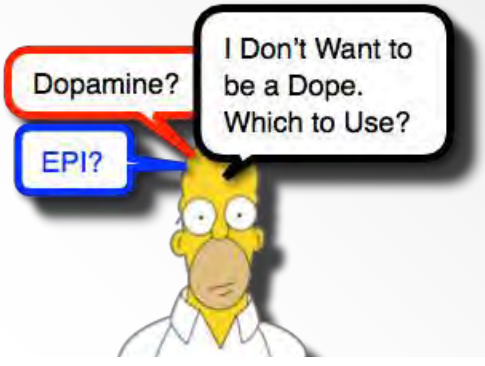
Beta blockers: reduce mortality and morbidity
- **Metoprolol**: 5 mg IV every 5 min up to 15 mg total
- **Esmolol**: 500 mg/kg/min for 4 min, then 50-300 mg/ig/min IV

IV **NTG** for hypotension due to cardiac (AMI, failure)
Loop diuretics (torsemide, furosemide, ethacrynic acid) for LV failure, pulmonary edema.
- Watch for volume depletion.

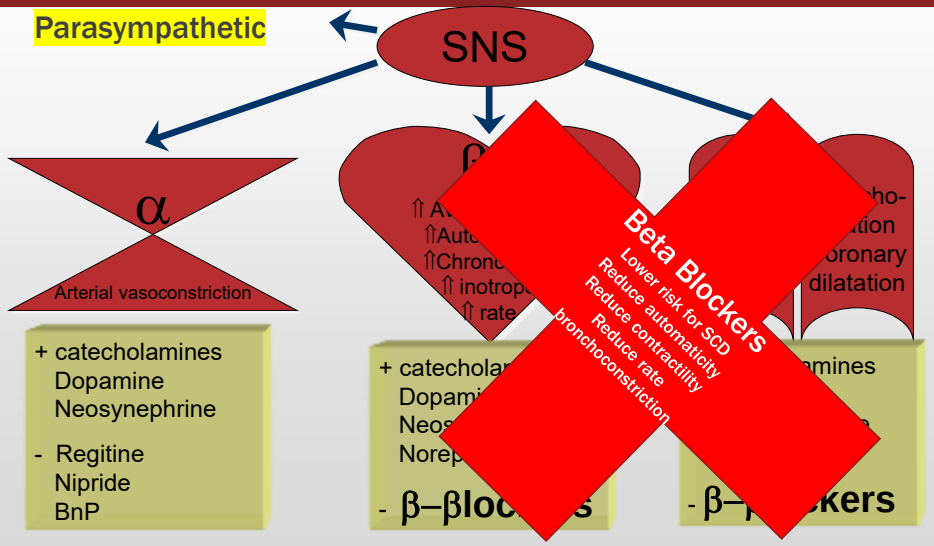
ARKANSAS HEART HOSPITAL®

Vasopressors

LYNNETTE FLYNN
DIRECTOR OF EDUCATION
ARKANSAS HEART HOSPITAL



Extrinsic Regulation



Terms

Agonist

Mimetic

*Properties

Antagonist

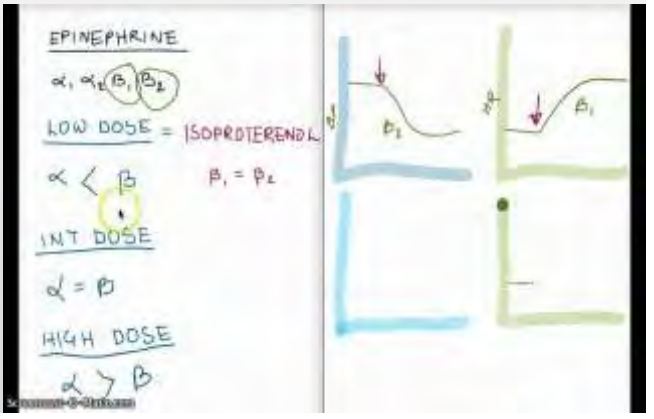
Blocker

Inhibitor

Effects of Various Vasopressors

Epinephrine

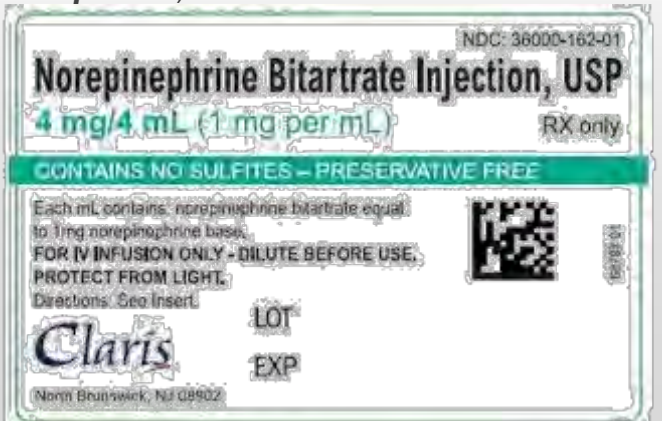
- ***Receptors**: Moderate beta-2, strong beta-1 and alpha adrenergic*
- Increased (CO) and (HR)
- Decreased renal perfusion
- Increased (PVR) minimal
- Increased (SVR)
- Significant increase in systolic function
- No effect in diastolic function
- Increased oxygen demand, significantly
- Variable blood pressure (BP)



Effects of Various Vasopressors

Norepinephrine

- ***Receptors**: Strong alpha-1 and alpha-2, moderate beta-1*
- Increased PVR, minimally
- Increased BP
- Increased SVR, significantly
- No effect on diastolic function
- Increased oxygen demand
- Increased systolic function, minimally
- Decreased renal perfusion
- Variable CO



Effects of Various Vasopressors

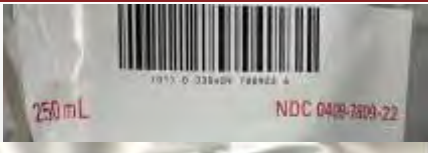
Phenylephrine

- ***Receptors**: Strong alpha-1*
- Increased SVR, significantly
- No effect on PVR
- Increased BP
- No effect on HR
- No effect on systolic or diastolic function
- No effect on myocardial oxygen demand
- Decreased CO and renal perfusion

Effects of Various Vasopressors

Dopamine, low dose (1-5 µg/kg/min)

- ***Receptors**: Dopaminergic agonist*
- Renal and mesenteric vasodilation
- Increased HR
- Increased systolic function, minimal
- No effect in diastolic function
- Increased oxygen demand, minimal
- Increased SVR, minimal
- No effect on PVR



Effects of Various Vasopressors

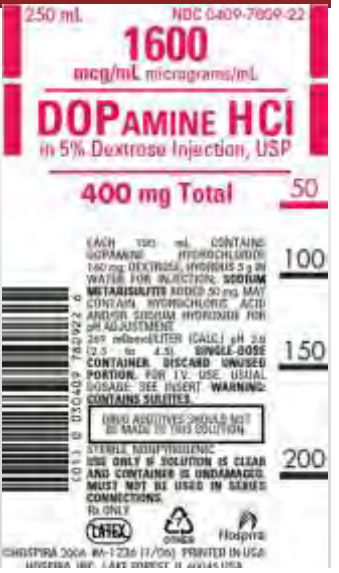
Dopamine, medium dose (6-10 µg/kg/min)

- ***Receptors**: Beta-1 agonist*
- Increased systolic function
- Increased HR and CO
- No effect in diastolic function
- Increased myocardial oxygen demand
- Increased SVR
- Increased PVR, minimal
- Renal vasodilation

Effects of Various Vasopressors

Dopamine, large dose (11-20 µg/kg/min)

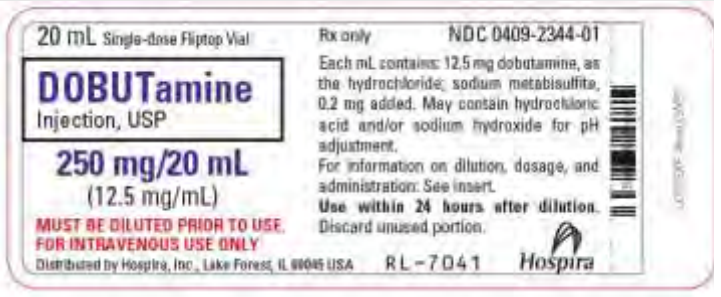
- ***Receptors**: Alpha-1 agonist*
- Increased HR, CO, PVR
- No effect on diastolic function
- Increased myocardial oxygen demand
- Increased PVR, minimal
- Increased SVR, significantly



Effects of Various Vasopressors

Dobutamine

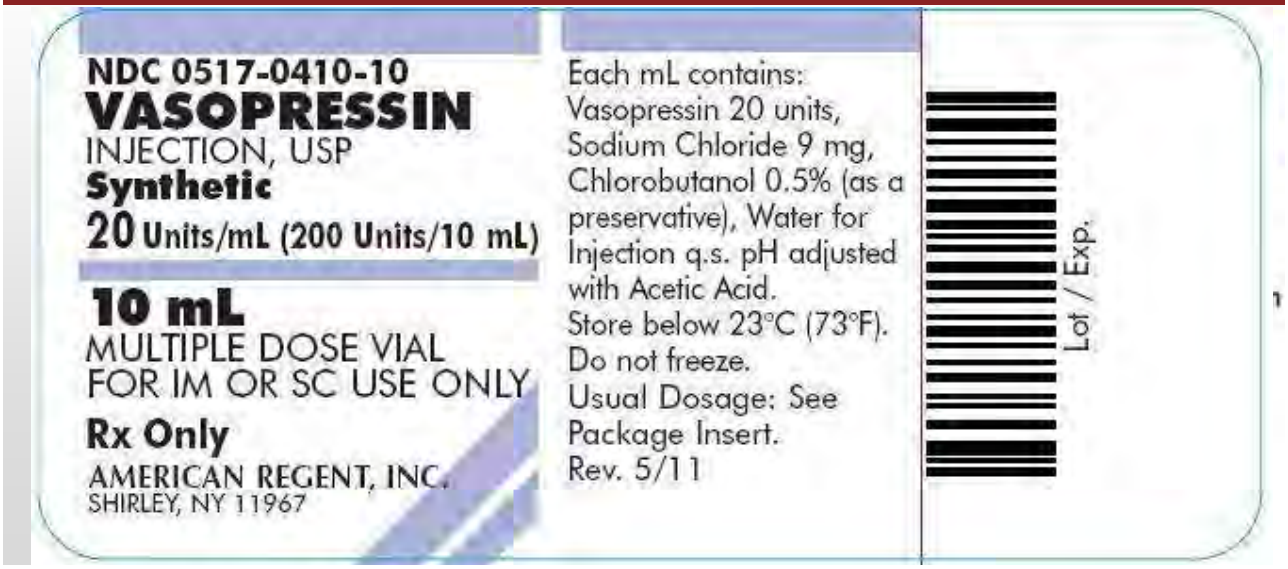
- ***Receptors**: Strong beta-1, weak beta-2 and alpha receptors*
- Increased in myocardial oxygen demand
- Increased HR, CO
- Increased HR
- Increased systolic function
- No effect on diastolic function
- Decreased SVR
- Decreased PVR, minimally



	Dopamine	Dobutamine
Arrhythmogenicity	More (Less than dobutamine)	More ,(Generally safe)
Inotropic action	Less	More ,Consistent,
Clinical utility* Shock Cardiac failure Ischemic LVF Renal failure	 • Yes • No • No • Yes	 • No • Yes • Yes • No

*Combination of dopamine & dobutamine infusion is very ideal in many situations but it needs diligent monitoring. Dobutamine is more often used as stress echocardiography now.

Effects of Various Vasopressors



PRESSOR	RECEPTOR	MAIN EFFECT	MAIN SHOCK USE	OTHER
EPINEPHRINE	α1 α2 β1 β2	VASOCONSTRICTION IONOTROPY DROMOTROPY CHRONOTROPY	AAA - Anaphylaxis, Asthma, Arrest (Cardiovascular)	DIRTY - Nonselective α and β receptors FLOGs the Heart
NOREPINEPHRINE	α1 β1	VASOCONSTRICTION IONOTROPY	SEPTIC SHOCK Most commonly used in US for most kinds of shock	First line for most kinds of shock
PHENYLEPHRINE	α1	VASOCONSTRICTION	Hypotension (often used as "push dose" pressor in anesthesia and ED)	BRADYCARDIA - may cause reflex bradycardia
DOBUTAMINE	β1 β2	IONOTROPY MILD VASODILATION	CARDIOGENIC SHOCK	Minimal changes in heart rate FLOGs the Heart
DOPAMINE	α1 α2 β1 β2 DA	VASOCONSTRICTION (higher doses) IONOTROPY DROMOTROPY CHRONOTROPY	Hypotension but second line, per surviving sepsis in septic shock	TACHYDYSRHYTHMIAS - main side effect Commonly used prehospitally
VASOPRESSIN	V1	VASOCONSTRICTION	ADJUNCT- Norepinephrine sparing effect at low dose Used by some in cardiac arrest	
MILRINONE	Phosphodiesterase inhibitor	IONOTROPY Reduces preload and afterload	Decompensated heart failure	May cause DYSRHYTHMIAS, HYPOTENSION

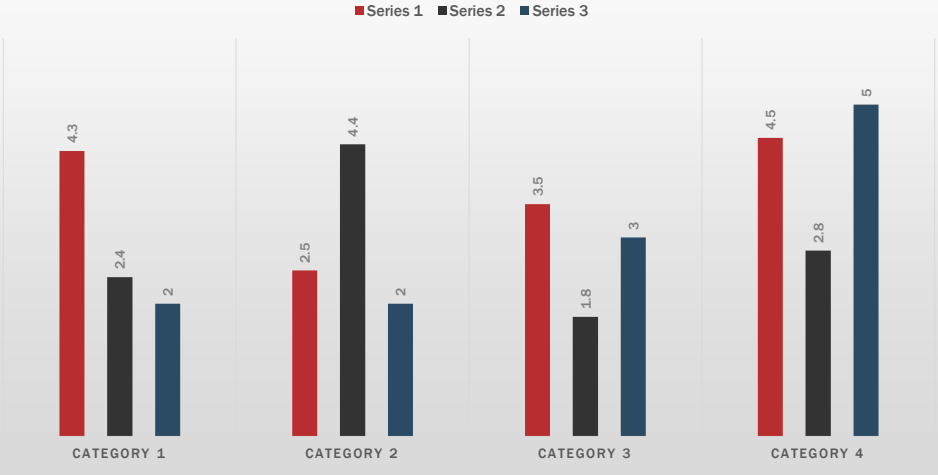
Patient A	Patient B	Patient C
B/P 80/60	BP 80/p	BP 80/60
HR 130	HR 135	HR 135
SVR 1800	SVR 600	SVR 1800
Sat 78%	Sat 78%	Sat 78%
Lungs Wet	Lungs Dry	Lungs Dry
CVP 35	CVP 4	CVP 4
PCWP 40	PCWP 2	PCWP 2
CO 1.8	CO 7	CO 3.2

- www.Pie.med.utoronto.ca

Online simulations

- Goal – CO >3.0 Use Positive Inotrope when CO < 3.5
- MAP > 65 Use fluids or pressors when < 65
- CVP > 6 Use fluids when less than 6
- SVR > 1500 Use Vaso or arterial dilator to maintain 800-1200
- SVR < 800 Use arterial constrictor to maintain 800-1200
- Watch Output and Bleeding

Title and Content Layout with Chart



Two Content Layout with Table

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- Third bullet point here

Class	Group A	Group B
Class 1	82	95
Class 2	76	88
Class 3	84	90

Two Content Layout with SmartArt

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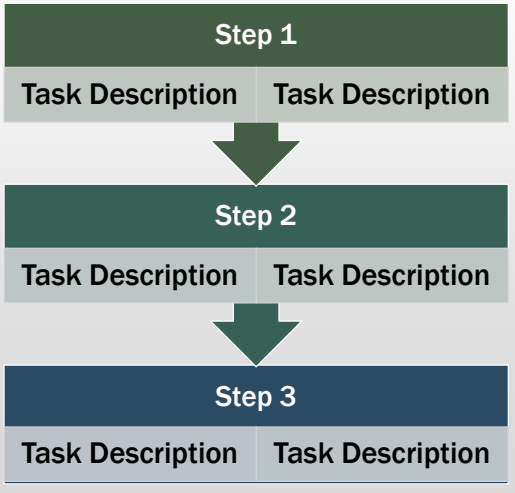
- Third bullet point here



Add a Slide Title - 1

Add a Slide Title - 2

Add a Slide Title - 3

Add a Slide Title - 4

Add a Slide Title - 5

Heart Failure

March, 2019
LRCCP
Basic Critical Care Course
Lynnette Flynn CCRN-CMC, CHFN, RCIS

2013 ACCF/AHA Guideline for the Management of Heart Failure

by Clyde W. Yancy, Mariell Jessup, Biykem Bozkurt, Javed Butler, Donald E. Casey, Mark H. Drazner, Gregg C. Fonarow, Stephen A. Geraci, Tamara Horwich, James L. Januzzi, Maryl R. Johnson, Edward K. Kasper, Wayne C. Levy, Frederick A. Masoudi, Patrick E. McBride, John J.V. McMurray, Judith E. Mitchell, Pamela N. Peterson, Barbara Riegel, Flora Sam, Lynne W. Stevenson, W.H. Wilson Tang, Emily J. Tsai, and Bruce L. Wilkoff

Circulation
Volume 128(16):e240-e327
October 15, 2013

Objectives - *By the end of this session, learners will demonstrate understanding of*:

The management of HFrEF and HFpEF acute exacerbations

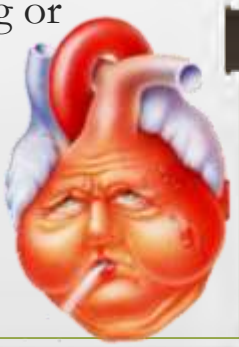
The indications for device therapy in the treatment of HF

Stages A-D Guideline Directed Medical Therapy for Heart Failure & NYHA symptom class I-IV

Definition

A complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood

- **Cardinal manifestations:**
  - Dyspnea & fatigue
  - Limited exercise tolerance
  - Fluid retention



Epidemiology

- Lifetime risk for Americans over 40yrs is 20%
- > 650,000 new cases diagnosed annually
- 5.1 million Americans clinically manifesting HF
- Mortality rate - 50% within 5 yrs of diagnosis.
- One month readmission rate is 25%
- $30 billion annually (1/2 of which is hospitalizations)

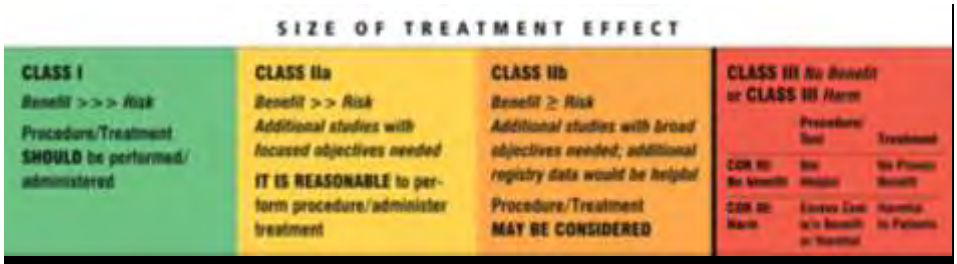
CATEGORIZING HEART FAILURE

NYHA CLASS I-IV

ACCF/AHA STAGE A-D

PRESERVED EF HEART FAILURE

REDUCED EF HEART FAILURE



The *Class of Recommendation (COR)*

- Estimates of the size of the treatment effect
- Considers risks versus benefits
- Determines usefulness of a given treatment or procedure

The *Level of Evidence (LOE)* is an estimate of the certainty or precision of the treatment effect.

The ACCF/AHA stages of HF *emphasize the development and progression of the disease to describe individuals and populations*

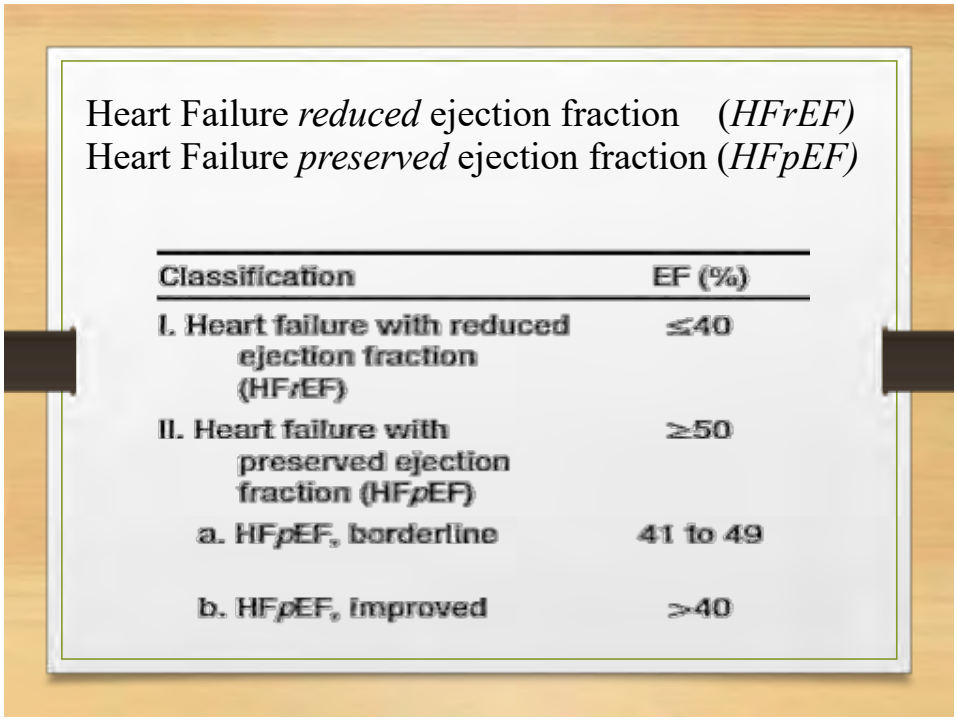
The NYHA classes *focus on exercise capacity and the symptomatic status f the disease and is an independent predictor of mortality.*

AHA/ACCF HEART FAILURE STAGES A-D

Stage A	Stage B	Stage C	Stage D
5 yr survival A 97%	B-96%	C – 75%	D-20%
High risk for developing HF No structural disorder of the heart	Structural disorder of heart Never developed symptoms of HF	Past or current symptoms of HF Symptoms assoc. with underlying heart disease	End-stage disease Requires specialized treatment strategies

NYHA CLASSES 1-IV

Class I	Class II	Class III	Class IV
No limitation of activity Ordinary activity does not cause symptoms	Slight limitation of activity Comfortable at rest Ordinary activity results in symptoms.	Marked limits of activity Comfortable at rest Less than ordinary activity = symptoms IIIa – no SOB at rest IIIb – recent SOB at rest	Inability to carry on any activity without symptoms Symptoms present at rest and exacerbated by any activity



HFrEF

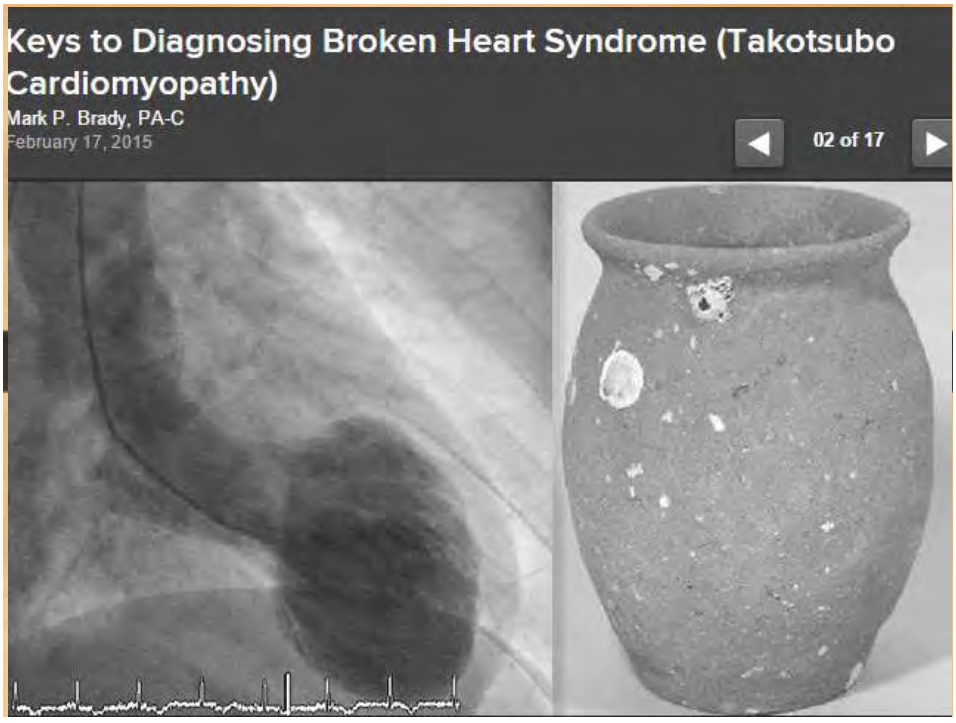
Definition: the clinical diagnosis of HF and an EF of ≤ 40%.

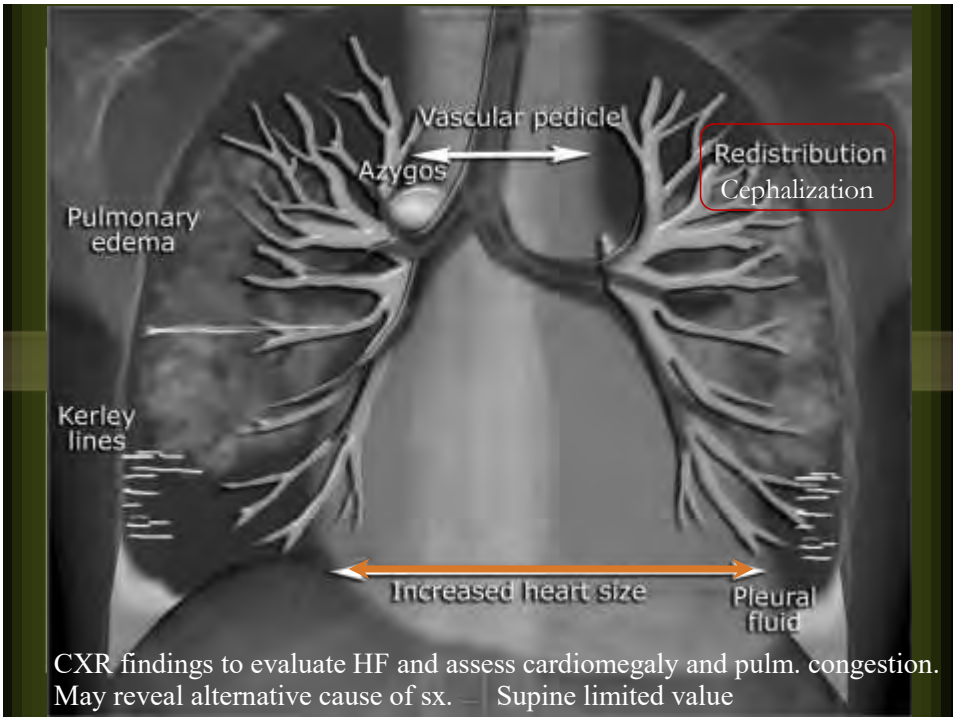
Prevalence: 50% of all HF cases with ½ having variable degrees of LV enlargement.

- Commonly have elements of diastolic dysfunction

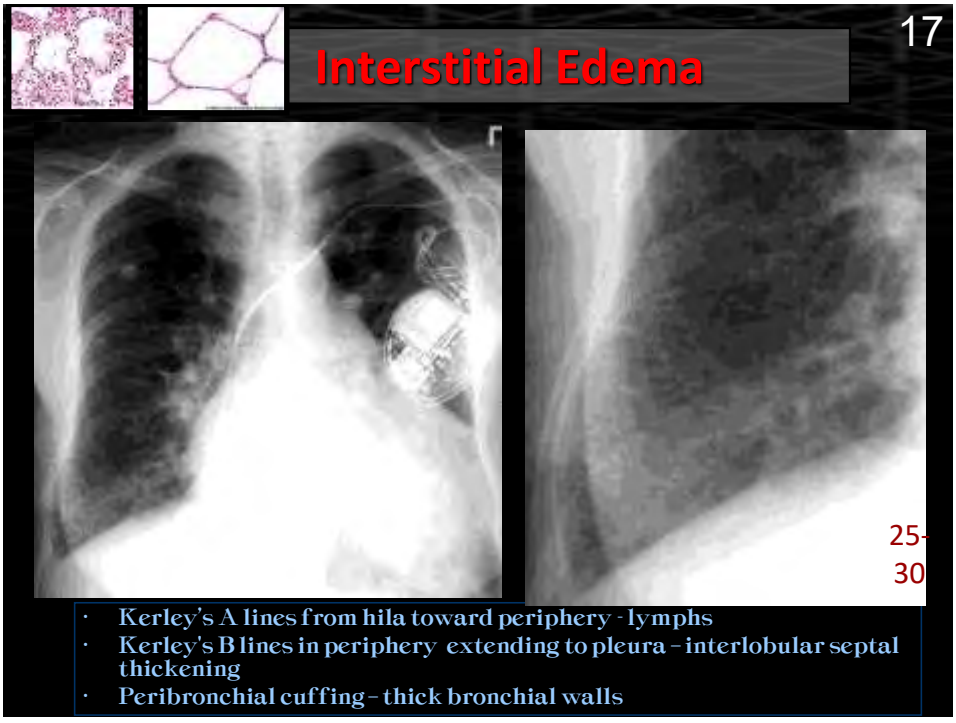
HFrEF

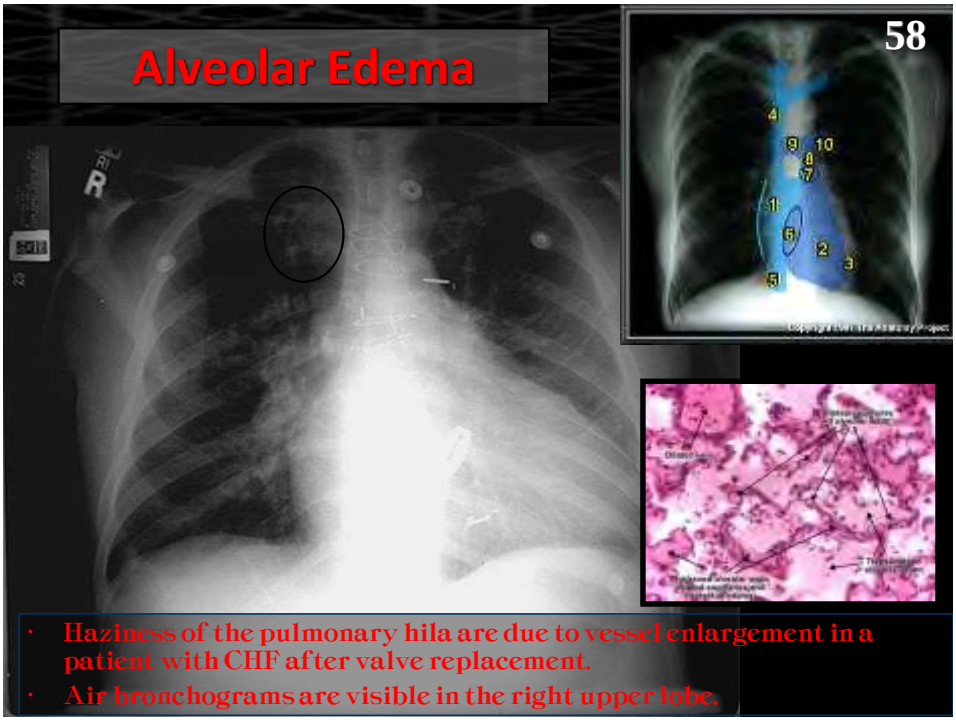
- Familial
- Obesity
- Diabetic
- Thyroid disease
- Acromegally and GH Def.
- Alcoholic
- Cocaine
- Cardiotoxic chemotherapy
- Tachycardia induced
- Myocarditis
- Acq. Immunodeficiency Synd
- Chagas Disease
- Hypersensitivity Myocarditis
- Rheumatologic/Connective tissue disorders
- Peripartum cardiomyopathy
- Iron overload
- Amyloidosis
- Sarcoidosis
- Stress (Takotsubo)





CXR findings to evaluate HF and assess cardiomegaly and pulm. congestion. May reveal alternative cause of sx. Supine limited value


Interstitial Edema
17
25-30
· Kerley's A lines from hila toward periphery - lymphs
· Kerley's B lines in periphery extending to pleura – interlobular septal thickening
· Peribronchial cuffing – thick bronchial walls


Alveolar Edema
58
· Haziness of the pulmonary hila are due to vessel enlargement in a patient with CHF after valve replacement.
· Air bronchograms are visible in the right upper lobe.

HFpEF

Definition: Clinical diag of HF & EF >40%

- LV diastolic dysfunction by doppler echo or cardiac catheterization
- Diagnosis is one of exclusion of other non-cardiac causes of the symptoms suggestive of HF

EF of 40-50% - intermediate group

- treated for risk factors and comorbidities with GDMT similar to that used with HFrEF.

HFpEF

Prevalence: 50% (40-71%)

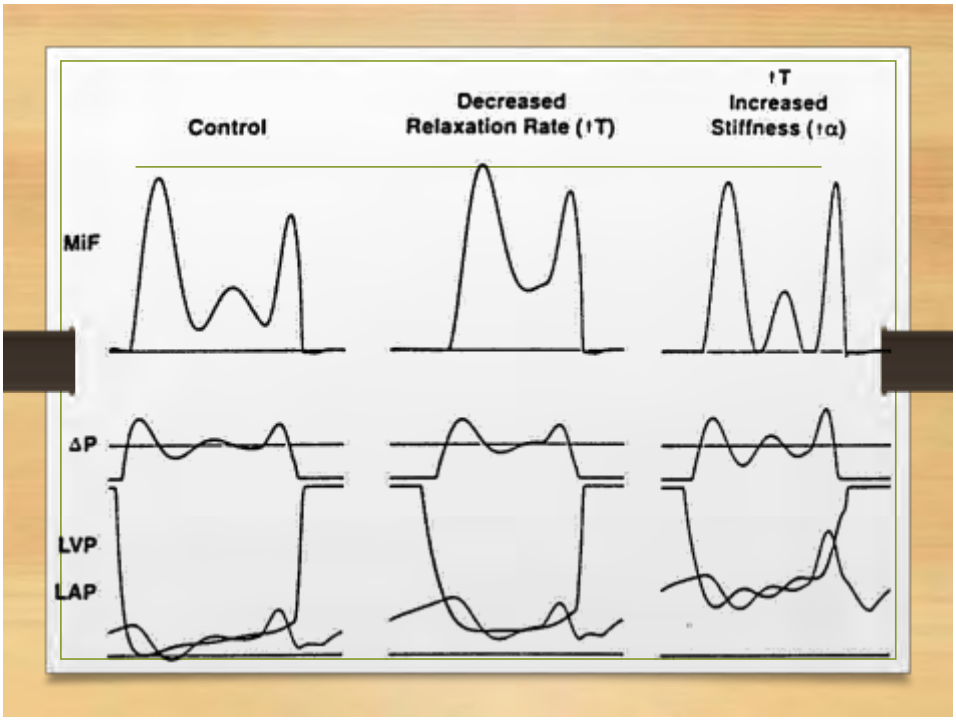
Concentrically remodeled left ventricle 2º arterial hypertension, obesity, and diabetes, with no evidence of CAD (Barry A. Borlaug, 2011)

HFpEF

Abnormal mitral flow velocity on echo suggestive of diastolic LV dysfunction are non-specific for HFpEF, occurring also in elderly and in HFrEF pts

Recently, invasive studies of HFpEF have found a uniform presence at rest of:

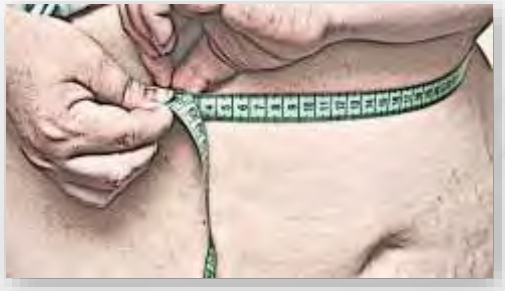
- slow LV relaxation
- elevated diastolic LV stiffness which limited cardiac performance during atrial pacing and exercise



Patient Characteristics

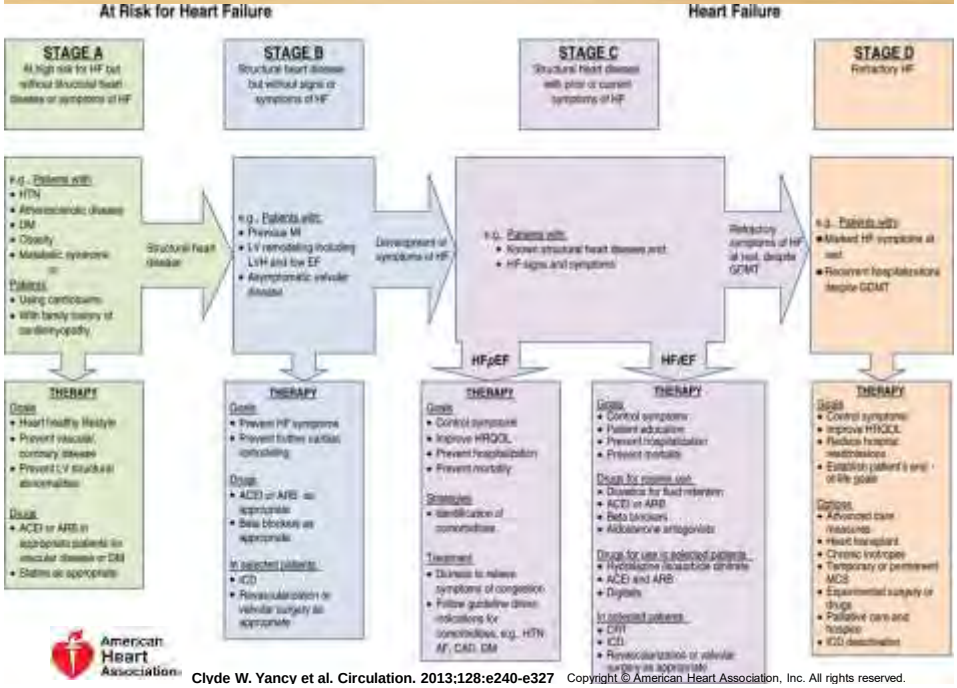
Older women with a hx of:

- HTN
- Obesity
- CAD
- DM
- AF
- Hyperlipidemia



HTN is the most important cause having a prevalence of 60-89%

Stages in the development of HF and recommended therapy by stage.



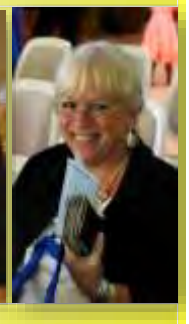
Clyde W. Yancy et al. Circulation. 2013;128:e240-e327

Stage A

At high risk for HF

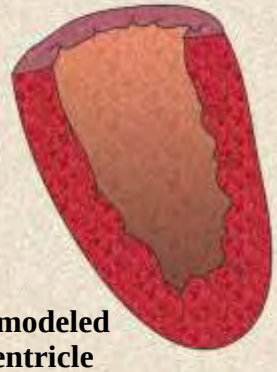
but no structural heart disease or symptoms

Patients factors	HTN ASHD DM Obesity Metabolic syndrome Using cardiotoxins Family history of cardiomyopathy
GOALS	Heart healthy lifestyle Prevent vascular, Coronary disease Prevent LV remodeling
DRUGS	ACEi/ARB with vascular disease or DM Statins as appropriate *(trig < 150, tot chol < 200, LDL < 100)*



Left Ventricular Remodeling

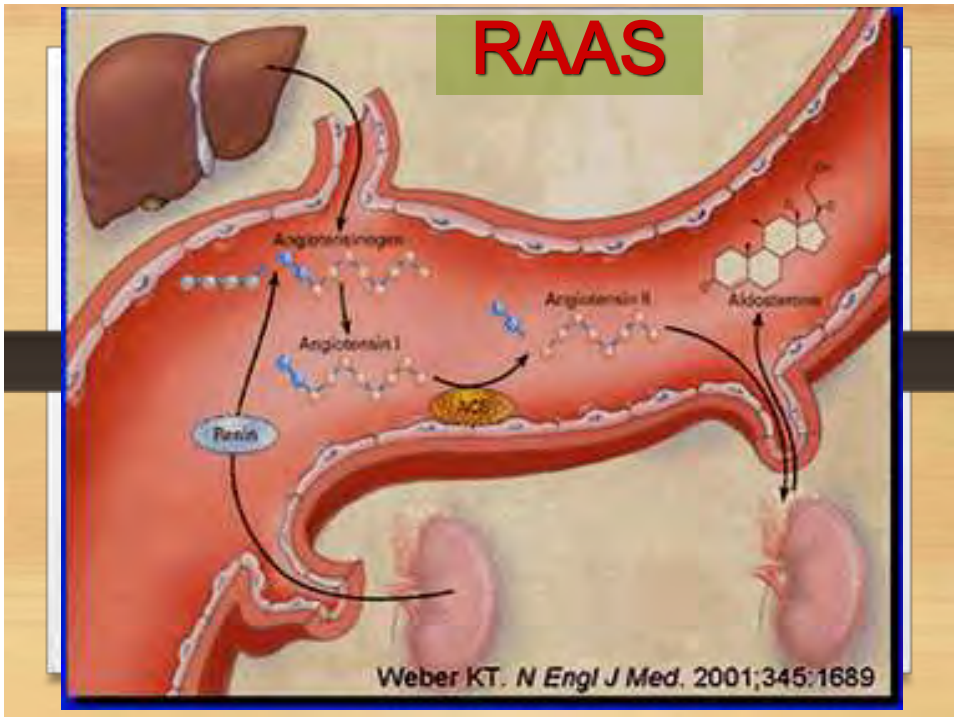
Relatively normal chamber size & geometry

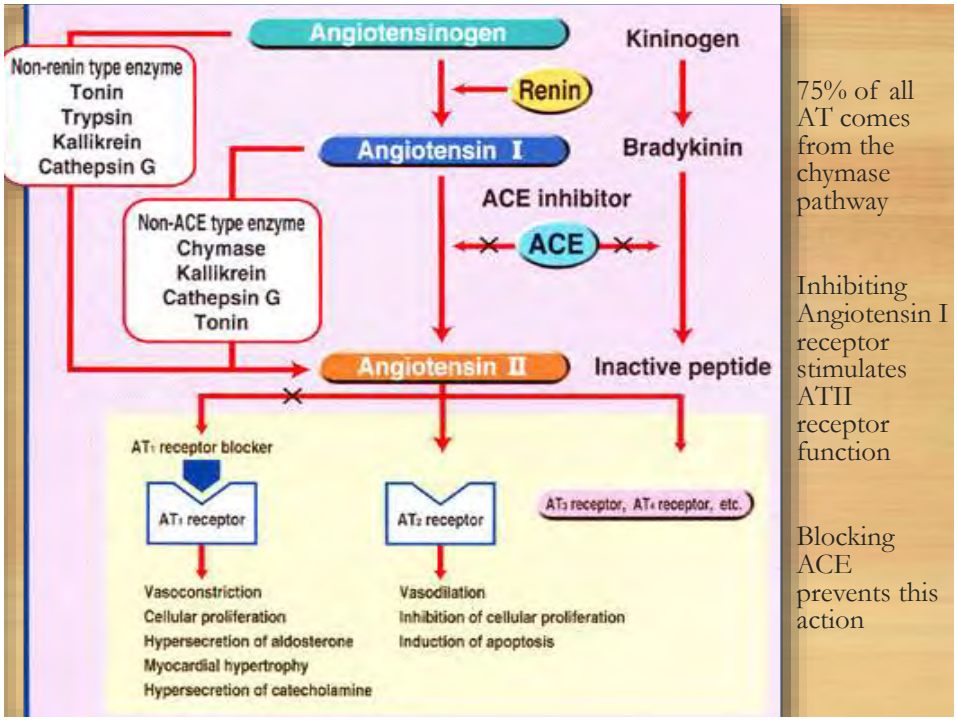


Remodeled ventricle

Angio II synthesis of fibroblasts
Aldosterone collagen synthesis
Norepinephrine directly toxic to myocytes

Cohn JN, et al. *J Am Coll Cardiol.* 2000;35:569-582.

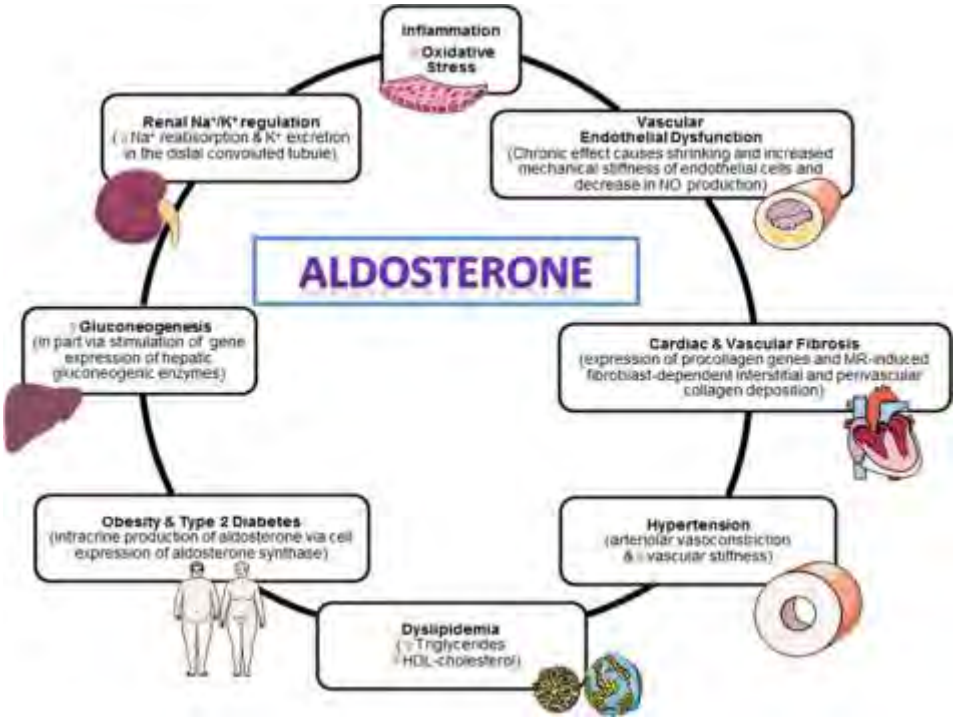


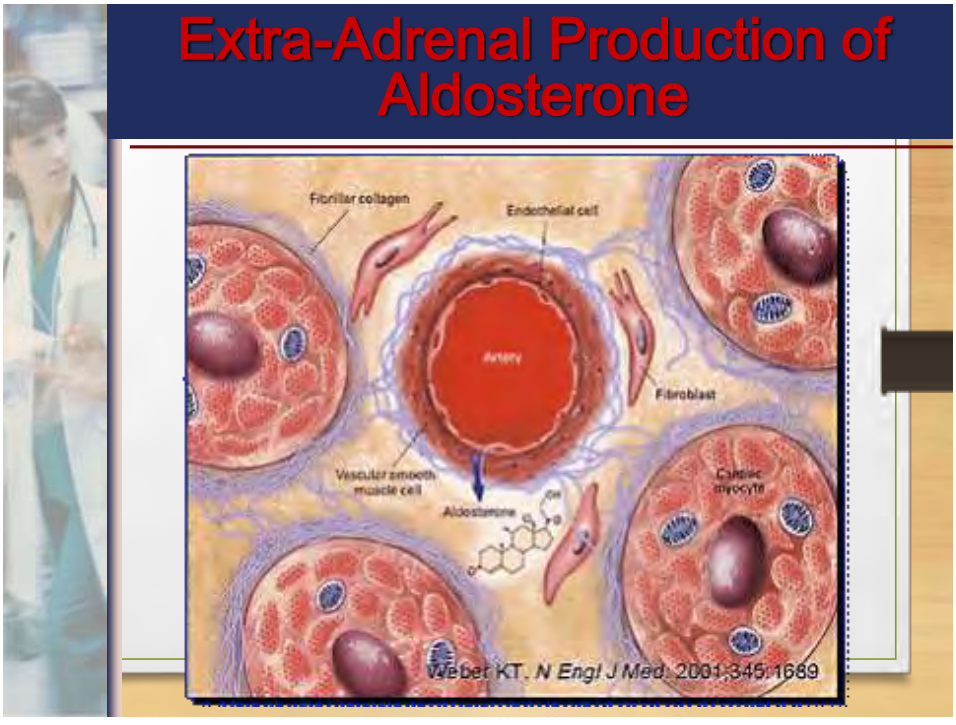


75% of all AT comes from the chymase pathway

Inhibiting Angiotensin I receptor stimulates ATII receptor function

Blocking ACE prevents this action


Inflammation
↑ Oxidative Stress
Renal Na+/K+ regulation
(↑ Na+ reabsorption & K+ excretion in the distal convoluted tubule)
Vascular Endothelial Dysfunction
(Chronic effect causes shrinking and increased mechanical stiffness of endothelial cells and decrease in NO production)
ALDOSTERONE
↑ Gluconeogenesis
(in part via stimulation of gene expression of hepatic gluconeogenic enzymes)
Cardiac & Vascular Fibrosis
(expression of procollagen genes and MR-induced fibroblast-dependent interstitial and perivascular collagen deposition)
Obesity & Type 2 Diabetes
(intracrine production of aldosterone via cell expression of aldosterone synthase)
Hypertension
(arteriolar vasoconstriction & ↑ vascular stiffness)
Dyslipidemia
(↑ Triglycerides
↓ HDL-cholesterol)


Extra-Adrenal Production of Aldosterone
Fibrillar collagen
Endothelial cell
Artery
Fibroblast
Vascular smooth muscle cell
Aldosterone
Cardiac myocyte
Weber KT. N Engl J Med. 2001;345:1689

Extrinsic Regulation

Parasympathetic

SNS

α

Arterial vasoconstriction

+ catecholamines
Dopamine
Neosynephrine
- Regitine
Nipride
BnP

β1

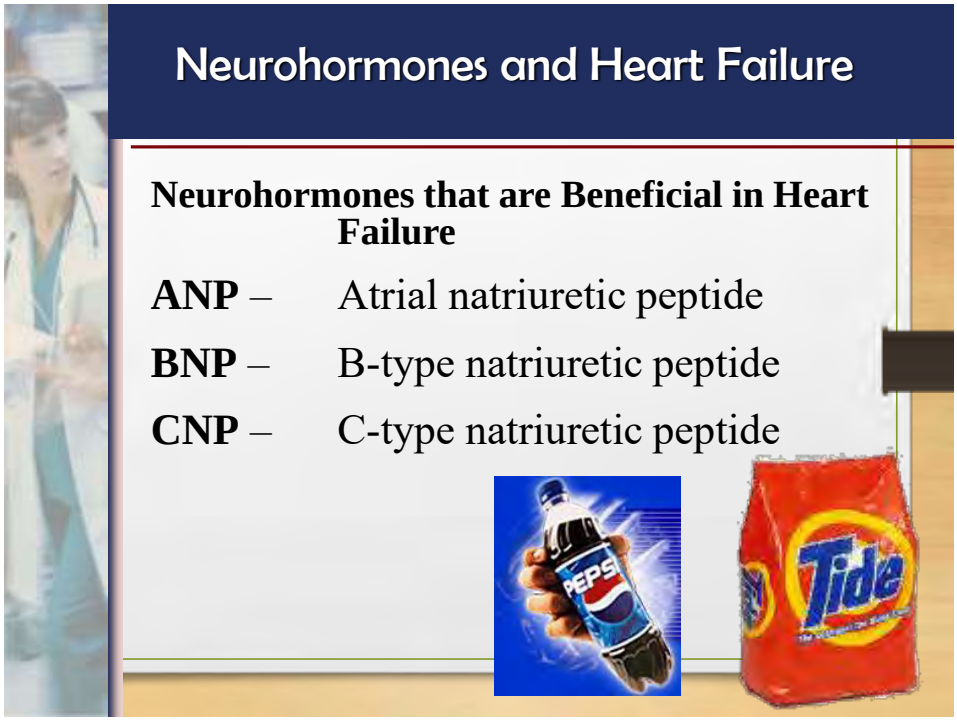
⇑ AV conduction
⇑Automaticity
⇑Chronotrope
⇑ inotrope
⇑ rate

+ catecholamines
Dopamine
Neosynephrine
Norepi/Epi
- **β–βlockers**

β2

Broncho-dilation
Coronary dilatation

+ catecholamines
Dopamine
Neosynephrine
Norepi/Epi
- **β–βlockers**



Stage B	
Structural heart disease but without S&S of HF	
Patient factors	Previous MI LV remodeling including LVH and low EF Asymptomatic valvular disease
GOALS	Prevent HF symptoms Prevent further remodeling
DRUGS	ACEi/ARB BBlkrs **Selected patients:** ICD Revascularization or valve OR

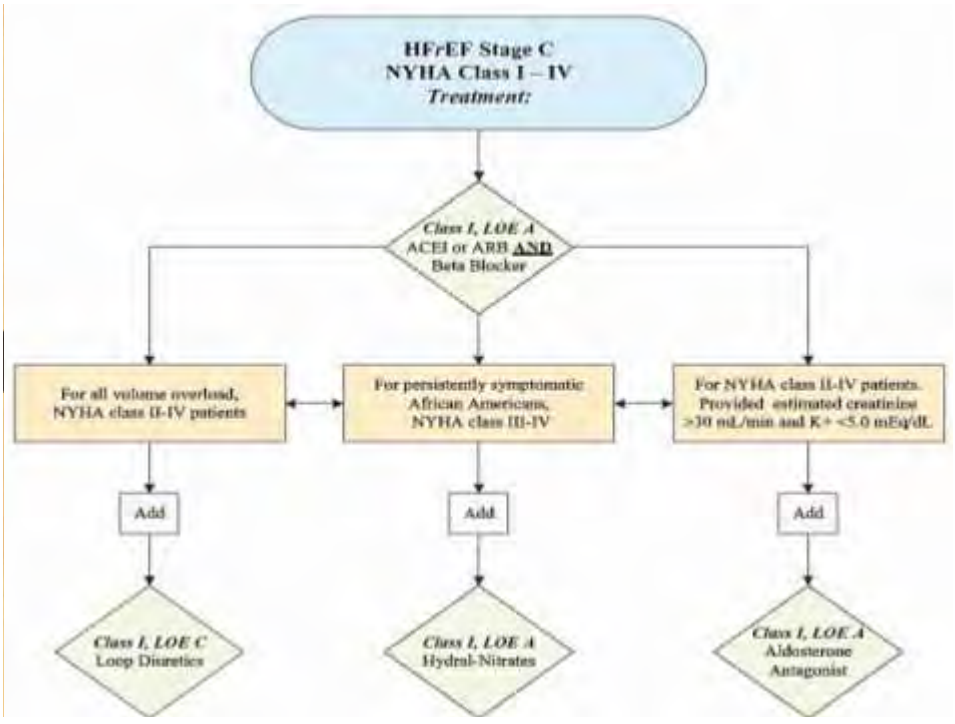
Stage C		
Structural heart disease with prior or current sympt of HF		
Known structural heart disease and HF S&S		
GOALS	**HFpEF** Control symptoms Improve HRQOL Prevent hospitalization Prevent mortality ID co-morbidities	**HFrEF** Control symptoms Patient education Prevent hospitalization Prevent mortality
DRUGS	Diuretics Follow GDMT for comorbidities HTN, AF, CAD, DM	Diuretics ACEi or ARB BBlkrs Aldosterone antagonists **Selected patients:** Hydralazine/isosorbide Digitalis CRT - ICD Revascularization/valve OR

Pharmacologic Tx. Of HFrEF

Drug	Mortality	HF Adm	Use for
ACEi/ARB	17%	31%	Stage A – control HTN Stages BCD – everyone
Beta Blkrs	34%	41%	Stage AB - control HTN Stages CD - everyone
Aldosterone Blkrs	30%	35%	Stage CD Class II-IV with EF < 35% and CR < 2.5/2.0 *Avoid ACEi, ARB and Ald blkr 2°to ↑K+*
Hydral/Isosorbide	43%	33%	Symptoms despite GDMT in African Americans
Digoxin			Symptoms despite GDMT
Anticoagulants	No proven	No proven	consider
Omega-3 850-882mg	10-20%	Significant	consider
Calcium Channel Blkr			Except amlodipine
NSAIDS			Causes NA and H2O retention and blunt effects of diuretics

Pharmacologic tx: HFpEF

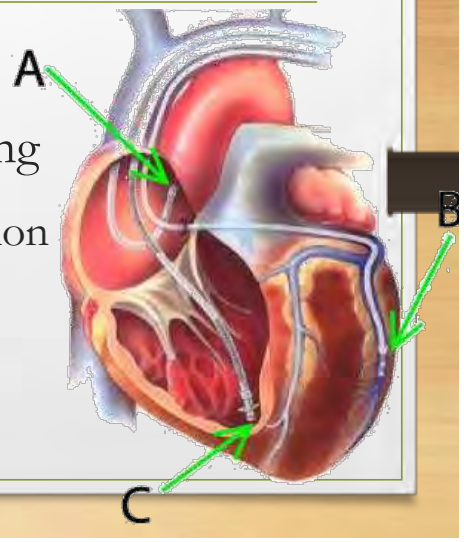
Intervention	Drug
Control HTN	ACEi/ARB/Beta Blkrs
Control Symptoms	Loop Diuretics Furosemide Torsemide Bumetamide
Manage CAD/AF	Revascularize/Rate control


HFrEF Stage C
NYHA Class I – IV
Treatment:
Class I, LOE A
ACEI or ARB AND
Beta Blocker
For all volume overload, NYHA class II-IV patients
For persistently symptomatic African Americans, NYHA class III-IV
For NYHA class II-IV patients. Provided estimated creatinine >30 mL/min and K+ <5.0 mEq/dL
Add
Add
Add
Class I, LOE C
Loop Diuretics
Class I, LOE A
Hydral-Nitrates
Class I, LOE A
Aldosterone Antagonist


Device Therapy
ICD & CRT

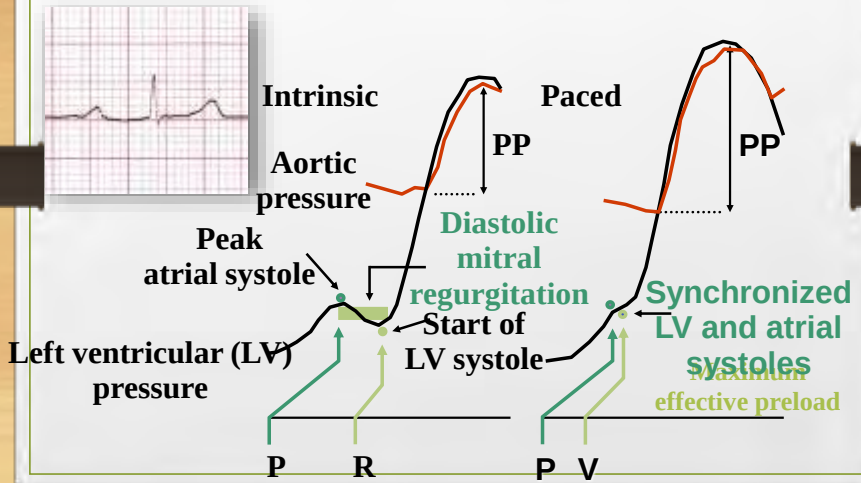
Non-Pharmacologic Therapies

- IABP
- Biventricular Pacing
- AV Resynchronization
- ICD
- Stem Cells



AV Dysynchrony

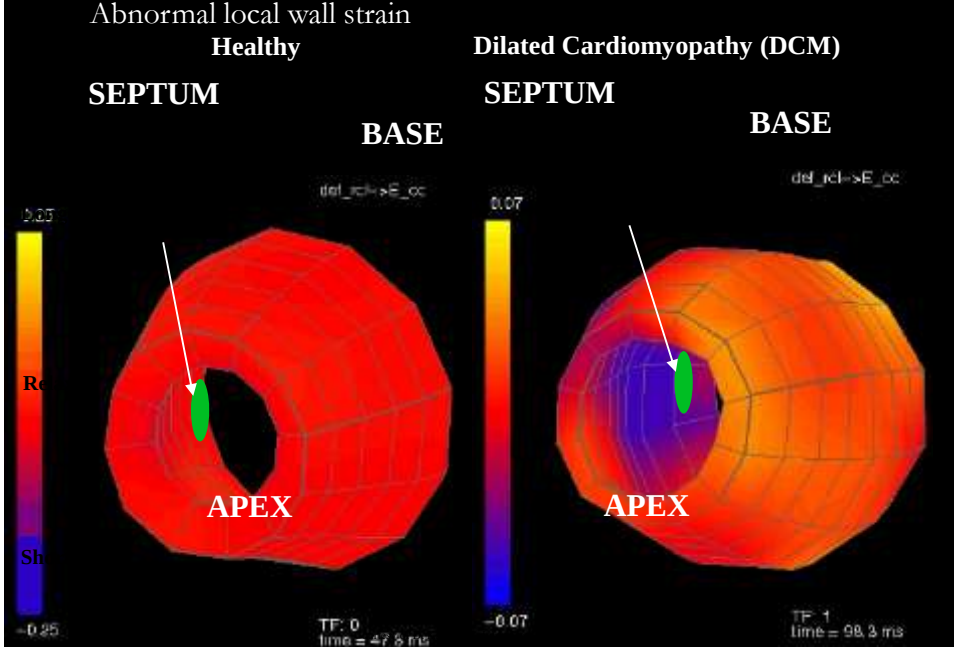
Mechanism I–optimal AV delay improves AV synchrony



Resynchronization

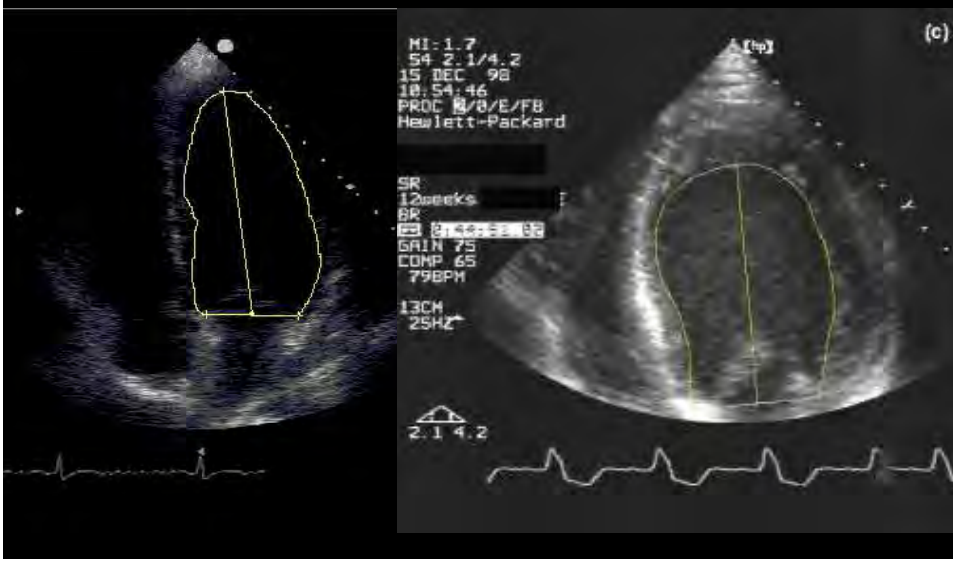
Ventricular Wall Motion Abnormalities

Abnormal local wall strain



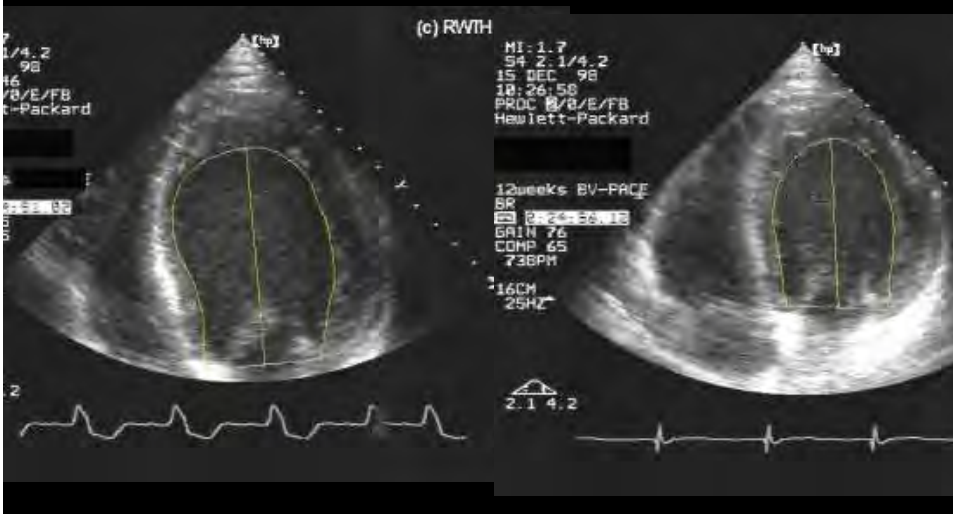
Ventricular Dysynchrony by Echo

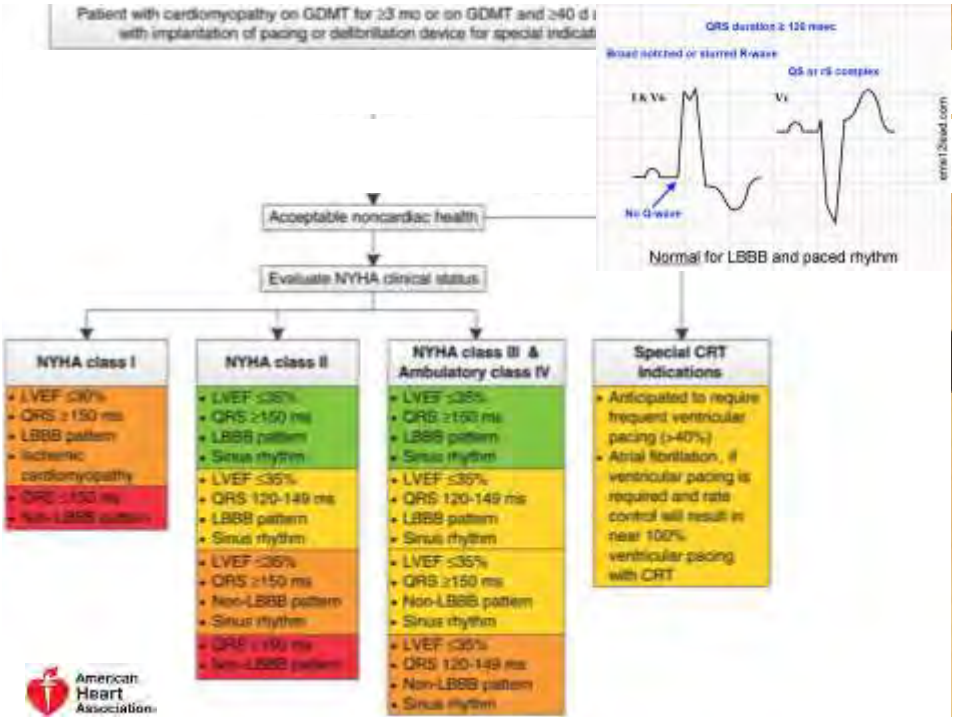
Abnormal wall motion



After Cardiac Resynchronization Therapy

Cardiac resynchronization therapy (CRT)– global synchrony





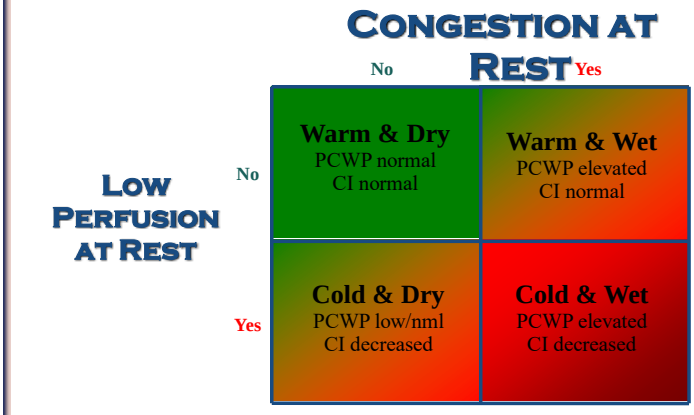
Stage D

Refractory HF

Marked HF symptoms at rest
Recurrent hospitalizations despite GDMT

GOALS	**Control symptoms** **Improve HRQOL** **Reduce admissions** **Establish EOL goals**
DRUGS	**Heroics** Heart transplant Chronic inotropes Temporary or permanent MCS Experimental surgery or drugs **Hospice** and Palliative care ICD deactivation

Rapid Assessment of Hemodynamic Status



S&S OF CONGESTION

Orthopnea/PND
JV distension
Ascites
Edema
Rales (rare in CHF)

POSSIBLE EVIDENCE OF LOW PERFUSION

Narrow pulse pressure
Sleepy/obtunded
Low serum sodium
Cool extremities
Hypotension with ACE inhibitor
Renal dysfunction (one cause)

Fonarow GC. *Rev Cardiovasc Med*. 2001;2(suppl 2):S7-S12.

Cardiogenic shock pending definitive therapy or resolution

Short-term support for threatened end-organ dysfunction in stage D and severe HF/EF

Long-term support with continuous infusion palliative therapy in select stage D HF

Routine intravenous use, either continuous or intermittent, is potentially harmful in stage D

Short-term intravenous use in hospitalized patients without evidence of shock or threatened end-organ performance is potentially harmful

Recommendations	COR
Inotropic support	
Cardiogenic shock pending definitive therapy or resolution	I
BTT or MCS in stage D refractory to GDMT	IIa
Short-term support for threatened end-organ dysfunction in hospitalized patients with stage D and severe HF/EF	IIb
Long-term support with continuous infusion palliative therapy in select stage D HF	IIb
Routine intravenous use, either continuous or intermittent, is potentially harmful in stage D HF	III: Harm

Recent Literature

SWEDISH

YALE

UK UNIVERSITY OF KENTUCKY College of Medicine

Exercise Impact

Palliative Care

Cardiac Rehab

Corlanor (ivabradine) - *HFrEF*

New drug indicated to reduce the risk of hospitalization for worsening heart failure in patients with stable, symptomatic chronic heart failure with LVEF less or equal to 35% who are in sinus rhythm with a resting heart rate of over 70 bpm and who are either on maximally tolerated doses of beta-blockers or have a contraindication to beta-blocker use.

The SHIFT trial showed a highly significant drop (18%) in risk for cardiovascular death or hospitalization for worsening heart failure over an average of 23 months.

Mechanism: Blocks the HCN channel responsible for the cardiac pacemaker or "funny" current which regulates heart rate. Effects are most pronounced in the SA node and no effect was seen on ventricular repolarization or myocardial contractility.

Dosage: 5 MG po bid with meals initially adjusting after 2 weeks to achieve a resting HR of 50—60 bpm not to exceed 7.5 mg BID.

Adverse Reaction: ≥ 1% bradycardia, hypertension, atrial fibrillation and luminous phenomena.

Drug Interactions:

CYP3A4 inhibitors increase Corlanor plasma concentrations & inducers decrease plasma concentrations of Corlanor.

Negative chronotropes: Increased risk of bradycardia – monitor heart rate

Pacemakers: Not recommended for use with demand pacemakers set to rates ≥ 60 bpm.

Entresto (sacubitril/valsartan) - *HFrEF*

New drug combination to reduce the risk of CV death and hospitalization for HF in patients with chronic HF class II-IV and reduced ejection fraction.

Trials showed that CV death or HF hospitalizations was reduced by 20% as compared with *enalapril* alone. It was also shown that significantly greater reduction in NT proBNP levels with Entresto as compared to *Valsartan* alone.

Mechanism(s):

Sacubitril is a neprilysin inhibitor which prevents the degradation of ANP and BNP allowing prolonged beneficial effects of these peptides.

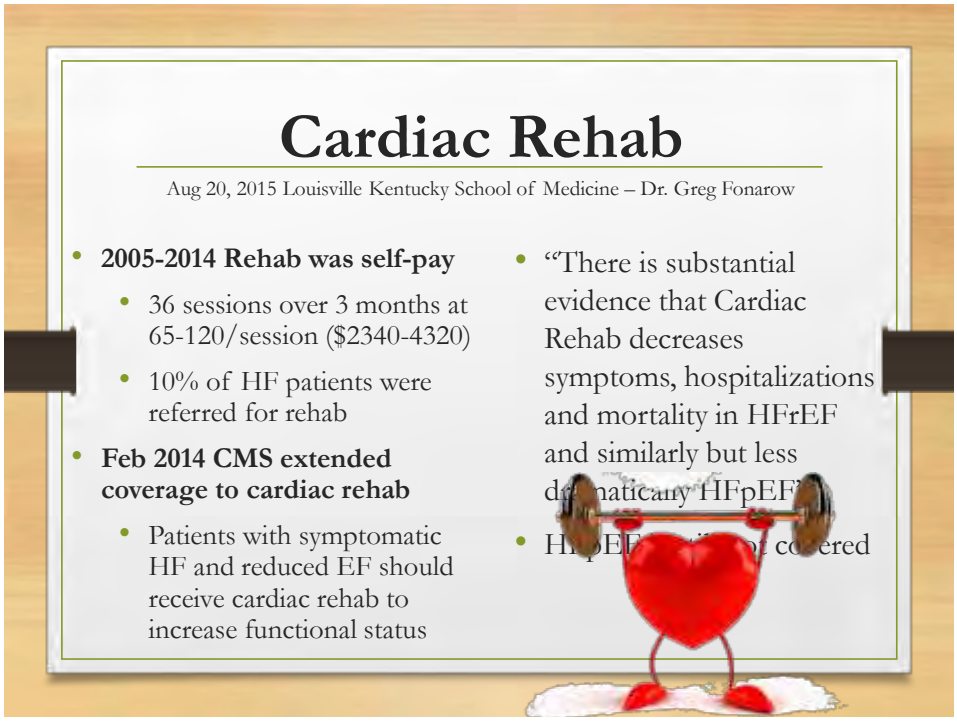
Valsartan is an angiotensin II receptor type I inhibitor (ARB)

Dosage: 49 mg/51 mg PO BID initially increasing after 2-4 weeks to maintenance dose of 97 mg/103 mg PO BID as tolerated.

Adverse Reaction: ≥ 5% experienced hypotension, hyperkalemia, cough, dizziness and renal failure.

Drug Interactions:

Dual blockade of the renin-angiotensin system: Do not use with an ACEi, do not use with *aliskiren* in patients with diabetes, and avoid use with an ARB.
Potassium-sparing diuretics: May lead to increased serum potassium.
NSAIDs: May lead to increased risk of renal impairment.
Lithium: Increased risk of lithium toxicity.



Objectives - *By the end of this session, learners will demonstrate understanding of*:

The management of HFrEF and HFpEF acute exacerbations

The indications for device therapy in the treatment of HF

Stages A-D Guideline Directed Medical Therapy for Heart Failure & NYHA symptom class I-IV

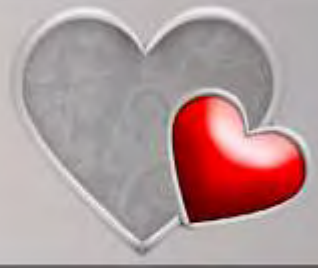
A growing medical challenge

Compliance

- 50% have three or more comorbidities
- Average of six medications
- 78% had at least two admissions per year
- Only 10% completed their annual prescription regimen
- One-third never refilled any heart failure prescription

Cardiomyopathy

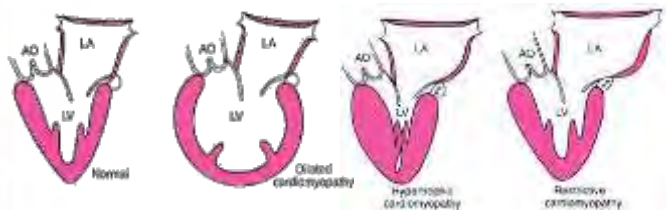
Lynnette Flynn
CCRN-CMC, CHFN, CFN, LRT, LNC, RCIS

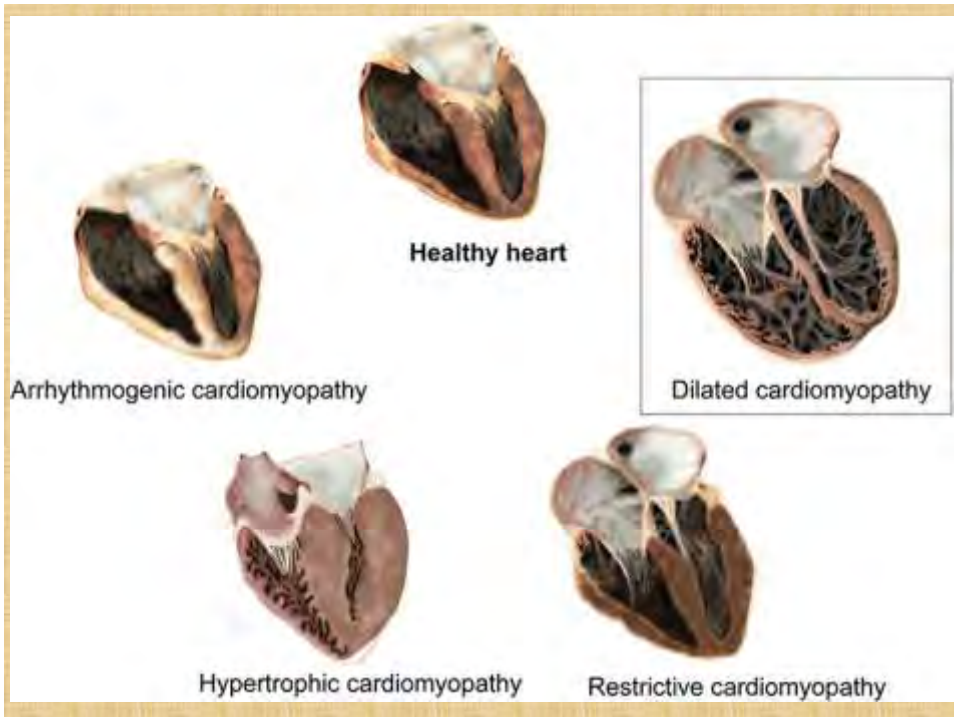


Types

Definition:
A Heterogenoeous group of diseases of the myocardium associated with mechanical and or electrical dysfunction. Usually but not invariably they exhibit inappropriate ventricular hypertrophy or dilation due to a variety of causes

- Dilated cardiomyopathy
- Restrictive cardiomyopathy
- Hypertrophic cardiomyopathy
- Stress-induced cardiomyopathy
  - Tako Tsubo cardiomyopathy





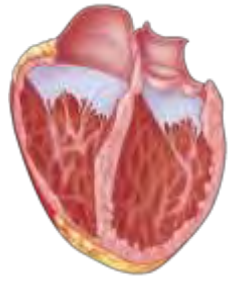
Dilated Cardiomyopathies

*Characterized by **ventricular dilation** and **depressed myocardial contractility** in the absence of abnormal loading such as HTN or valvular disease*

- Categorized as **ischemic** or **nonischemic**
- **African Americans** have a nearly 3 fold increased risk of developing DCM
- **Mortality is high** when presenting with HF
  - 25% at 1 year
  - 50% 5 year

Dilated Cardiomyopathies

- **Most common form of cardiomyopathy**
  - Idiopathic
  - Genetic disorders
  - Viral/bacterial infection
  - Hyperthyroidism
  - Chemotherapy
  - Peripartum syndrome related to toxicity
  - Cardiotoxic effects of drugs or alcohol



Dilated Cardiomyopathy Treatment Strategies

As per description in Heart Failure systolic dysfunction:

- ACEi/ARB
- Beta Blocker
- If AA – add nitrate and hydralazine
- Digoxin
- Pacer/ICD/CRT
- Revascularize
- Valve repair
- Mechanical support

Restrictive Cardiomyopathy

Restrictive Cardiomyopathy

- **Rigidity of myocardial wall**
  - NOT secondary to:
    - untreated hypertension
    - aortic stenosis or
    - hypertrophy seen with HCM

- **Results in decreased ability of chamber walls to expand during ventricular diastole**
- **Diastolic dysfunction with normal systolic function**
- **Least common form of cardiomyopathy**
  - 5% of all primary heart muscle diseases

Restrictive Cardiomyopathy

Primary Causes

Endomyocardial Dzs

- Eosinophilic Endomyocardial Fibrosis
- Endocardial Fibrosis
- Cardiac Transplant
- Anthracycline Toxicity
- Idiopathic
- Loffler's Endocarditis

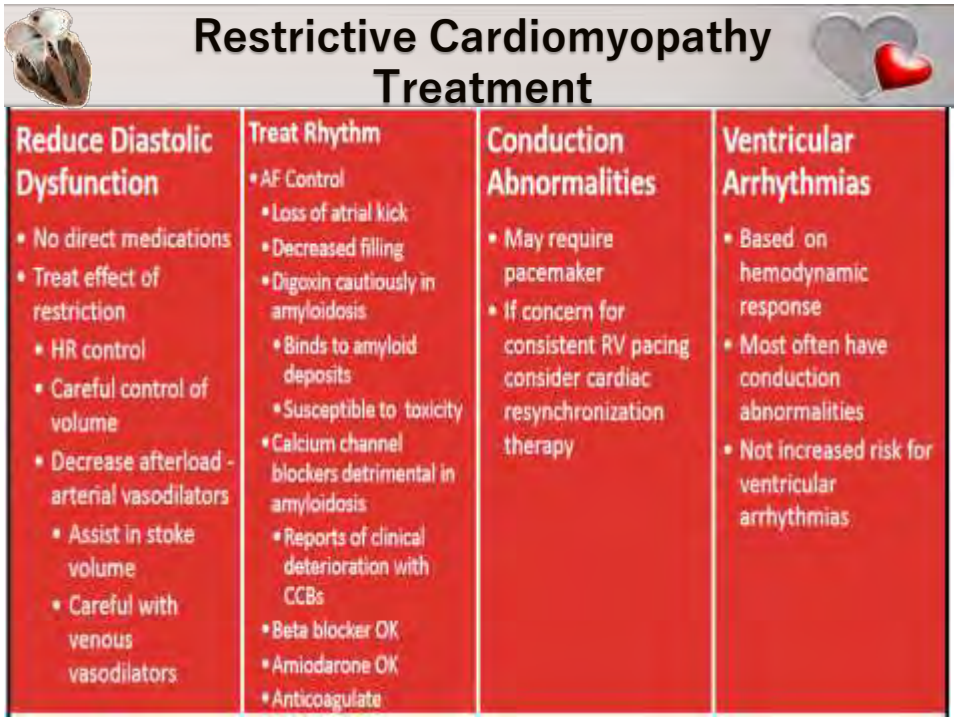
Secondary Causes

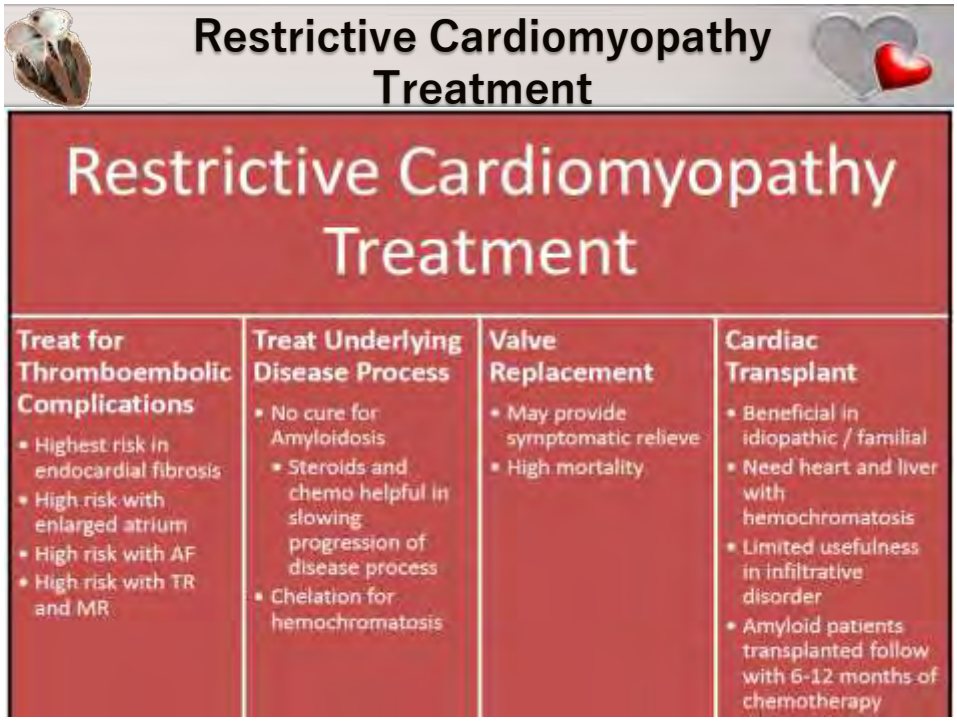
Infiltrative disorders

- Amyloidosis
  90% of RCM in North America
- Sarcoidosis
- Radiation carditis

Storage Diseases

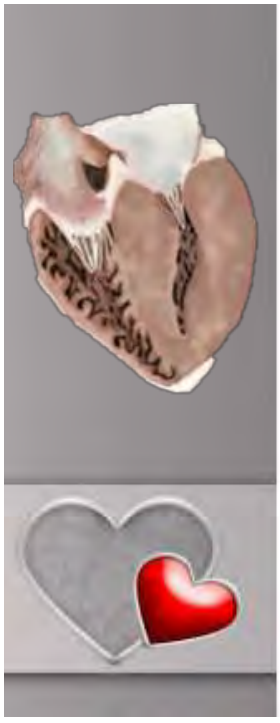
- Hemochromatosis
- Glycogen storage disease
- Fabry's Disease


Restrictive Cardiomyopathy Treatment
Reduce Diastolic Dysfunction
• No direct medications
• Treat effect of restriction
• HR control
• Careful control of volume
• Decrease afterload - arterial vasodilators
• Assist in stoke volume
• Careful with venous vasodilators
Treat Rhythm
• AF Control
• Loss of atrial kick
• Decreased filling
• Digoxin cautiously in amyloidosis
• Binds to amyloid deposits
• Susceptible to toxicity
• Calcium channel blockers detrimental in amyloidosis
• Reports of clinical deterioration with CCBs
• Beta blocker OK
• Amiodarone OK
• Anticoagulate
Conduction Abnormalities
• May require pacemaker
• If concern for consistent RV pacing consider cardiac resynchronization therapy
Ventricular Arrhythmias
• Based on hemodynamic response
• Most often have conduction abnormalities
• Not increased risk for ventricular arrhythmias


Restrictive Cardiomyopathy Treatment
Restrictive Cardiomyopathy Treatment
Treat for Thromboembolic Complications
• Highest risk in endocardial fibrosis
• High risk with enlarged atrium
• High risk with AF
• High risk with TR and MR
Treat Underlying Disease Process
• No cure for Amyloidosis
• Steroids and chemo helpful in slowing progression of disease process
• Chelation for hemochromatosis
Valve Replacement
• May provide symptomatic relieve
• High mortality
Cardiac Transplant
• Beneficial in idiopathic / familial
• Need heart and liver with hemochromatosis
• Limited usefulness in infiltrative disorder
• Amyloid patients transplanted follow with 6-12 months of chemotherapy

Restrictive Cardiomyopathy
Outcomes

- Poorest mortality of all cardiomyopathies
- 90% mortality rate at 10 years
- Amyloid heart
  - 80% mortality at 2 years
  - Senile systemic amyloidosis
    - Median survival 60 months
  - AL amyloidosis: 5.4 months
  - Idiopathic:
    - 64% 5 year survival
    - 37% 10 year survival



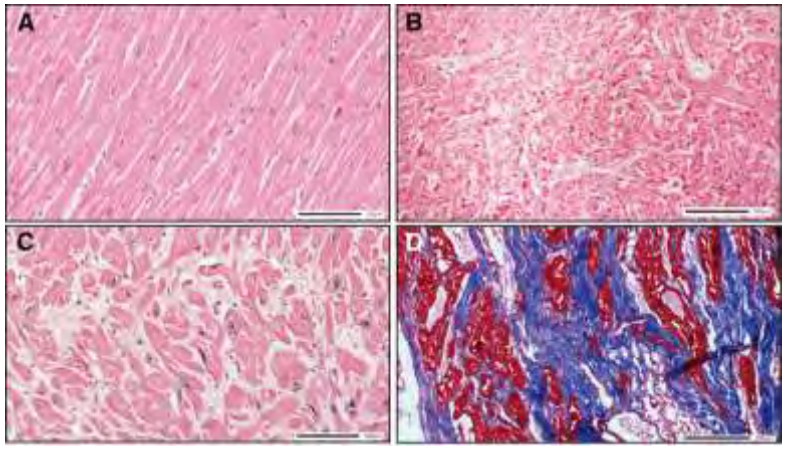
Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy

- 1 of every 500
- Primary genetic cardiomyopathy Effects men and women equally
- Hypertrophy of myocardial muscle mass in the absence of increased ventricular afterload
- Associated with decreased ventricular filling (diastolic dysf.) and decreased CO
- Most common cause of sudden death in young adults
- Cause unknown
  - 50% transmitted genetically

Hypertrophic Cardiomyopathy

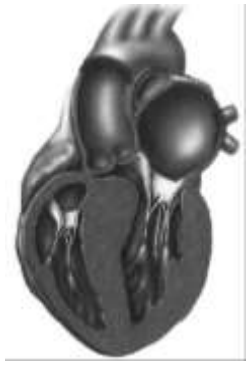
- Disarray of myofibrils with hypertrophy of myocytes
- Cells take on a variety of shapes
- Myocardial scarring and fibrosis occurs



Hypertrophic Cardiomyopathy

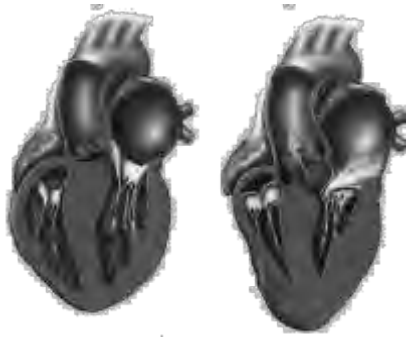
- Usually only effects the LV
- Changes may be symmetrical
- Asymmetrical septal hypertrophy is more common

May involve entire septum or only a portion of the septum

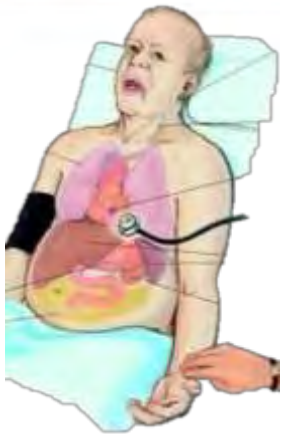


Hypertrophic Cardiomyopathy with OBSTRUCTION (HOCM)

- 35% of HCM patients have obstruction of outflow tract at rest
- 35% additionally have obstruction with provocation
- Obstruction Septal wall enlarges into ventricular cavity
- Anterior leaflet of mitral valve drawn towards the septum during ejection
- Early closure of aortic valve, decreased ejection time, decreased CO



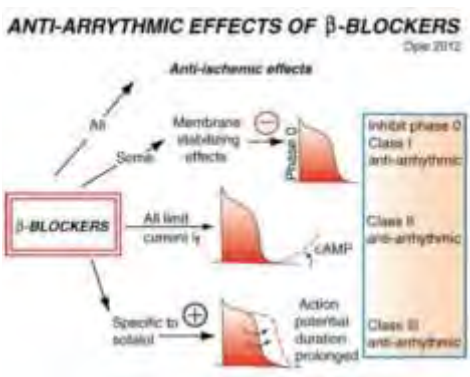
Hypertrophic Cardiomyopathy Presentation



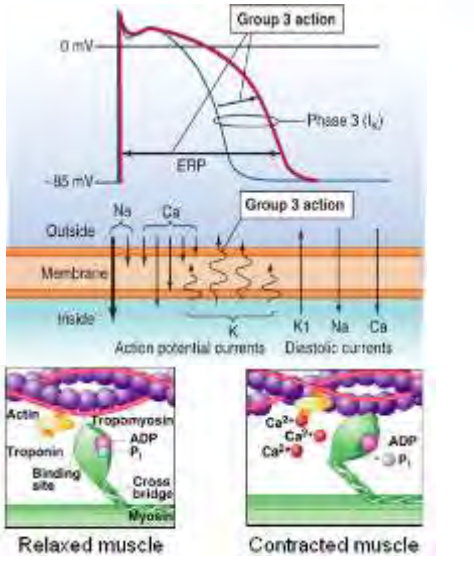
- Many asymptomatic for years
- Incidence of SCD often first presentation
  - ID during screening of relative with HCM
- Heart failure
  - Dyspnea #1 sign
  - Syncope/palpitations with activity
  - Chest pain
  - SVT
- Development of MR
- Symptoms r/t to severity of diastolic dysfunction

Hypertrophic Cardiomyopathy Treatment

Beta Blockers



Calcium Channel Blockers



Treatment

- **Other Medications**
  - Diuretics – with caution
  - ~~– ACEi and NTG~~ – avoid with HOCM
  - ~~– Positive inotropes~~ – NEVER with HOCM
- **Pregnancy**
  - Not restricted in non-obstructive disease
- **Endocarditis Prophylaxis**
  - NO LONGER INDICATED
- **Non-Obstructive Disease Rx.**
  - More difficult to treat if no symptoms
  - Ultimately evolves into dilated cardiomyopathy

Surgical Myectomy

- Marked outflow obstruction
- On maximum medical therapy
- NYHA Class III or IV
- MV replacement or repair at same time (increased Op mortality)
- Improvement noted immediately and lasts 20-30 yrs
- Survival rates 80% at 10 years
- May need pacemaker (2%)

Percutaneous Alcohol Septal Ablation

- Symptomatic with full therapy
- NYHA Class III or IV
- Not appropriate if MVR needed
- Cath lab procedure
- Catheter in septal perforator
- Ethyl alcohol injected
- Myocardial infarction occurs
- Enlarged septum eventually shrinks
- May need pacemaker (20%)

Outcomes

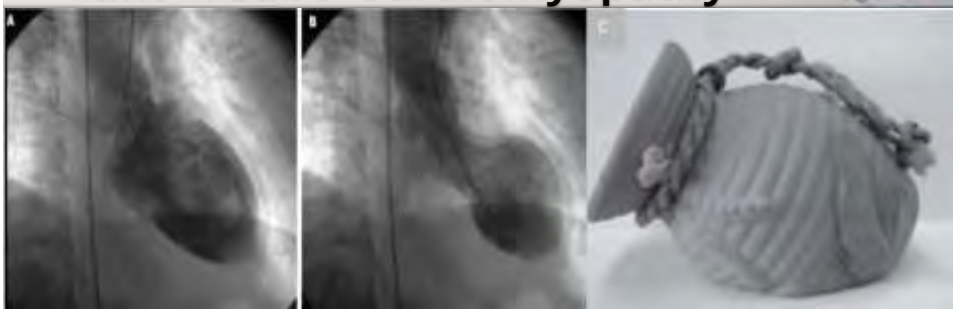
Normal life span

Routine f/u every 12-18 mos.

SCD primary cause of shortened life span

Prevent SCD

Tako-Tsubo Cardiomyopathy



- Transient LV apical ballooning
- Abrupt onset of ballooning or dilatation of LV
- Post menopausal women
- Occurs after psychosocial or physical stressors
- Also referred to as stress cardiomyopathy
- Cause unknown
  - Related to excessive catecholamines

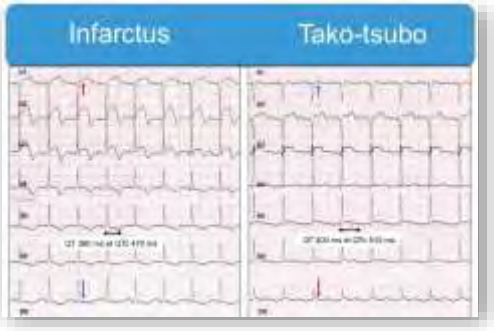
Presentation Tako-Tsubo

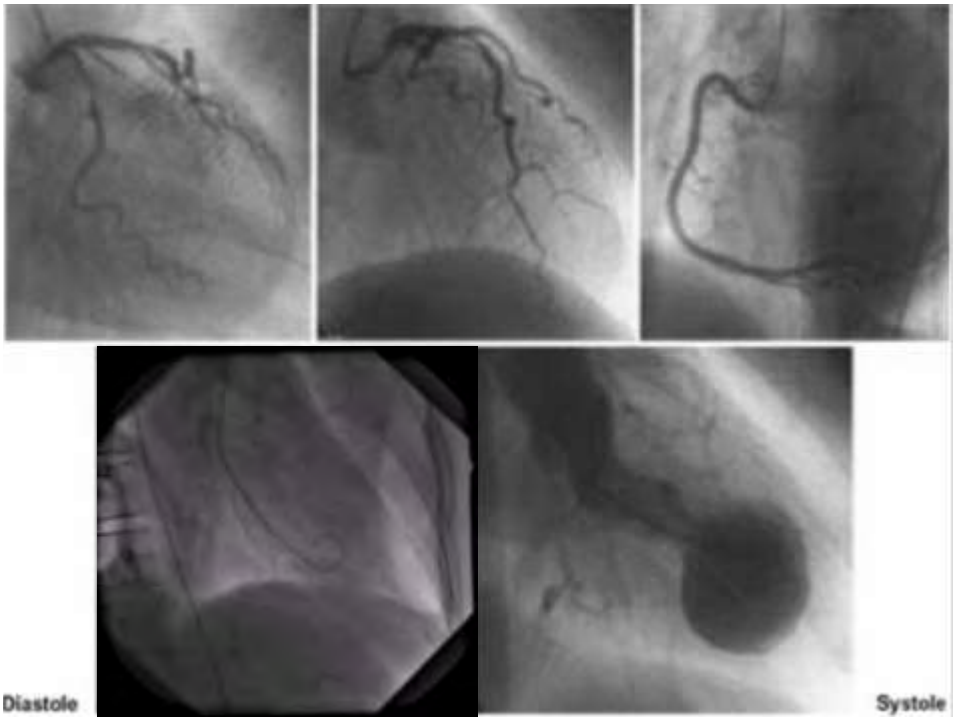
- Chest pain mimicking acute MI
- ST segment changes similar to AAMI
- Elevated cardiac biomarkers
- Dyspnea
- Hypotension
- Signs of LV failure

Diagnosis Tako Tsubo

- **ECG**
  - STE mimicking AAMI
  - Prolonged AT interval
- **Cardiac Biomarkers**
  - Do not follow same rise and fall as AMI
  - Mildly elevated
- **Cardiac Cath**
  - No significant CAD
  - Visualize ballooning of LV
- **Echo**
  - LV dysfunction with ↓ EF
  - Visualize ballooning of LV

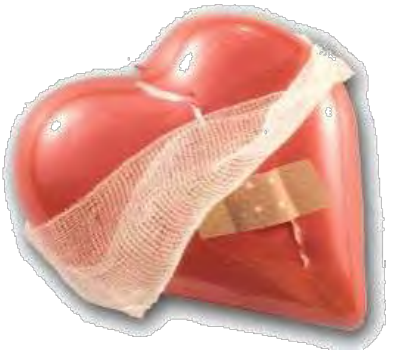
Investigation	Takotsubo	STEMI
Initial TnI	1.1	1.9
Peak TnI	4.9	7.3
Ejection fraction	33	25





Treatment

- **Goals**: Similar to patients with acute MI
- Treat LV failure
  - Cardiogenic shock
  - IABP
  - Arrhythmias
  - Hypotension
  - Avoid inotropes
- Cardiac Rehab
- Stress reduction



Outcomes

- **Recovery is good in this population**
- **Improvement of LV dysfunction occurs rapidly with much improvement seen within the hospital stay**
- **Full resolution of LV dysfunction seen in 1-3 months**
- **Most common complications associated with TCM include:**
  - Heart failure
  - Mitral regurgitation
  - Cardiogenic shock
- **In hospital mortality rates are low - reported at 1-2%**
  - Deaths result from: Cardiogenic shock, malignant arrhythmias, free wall rupture, systemic embolization
  - Post-discharge mortality nearly 13% at 7 years with over 52% dying from cancer and others from other non-cardiac related causes.
- **Incidence of recurrence is low - between 2-10%**
  - Most repeat events are stimulated by an event similar to the initial

Summary

- Heterogenous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction
- Usually exhibit inappropriate ventricular hypertrophy or dilation

THINK FUNCTIONAL CARDIOMYOPATHY

Summary Cardiomyopathy