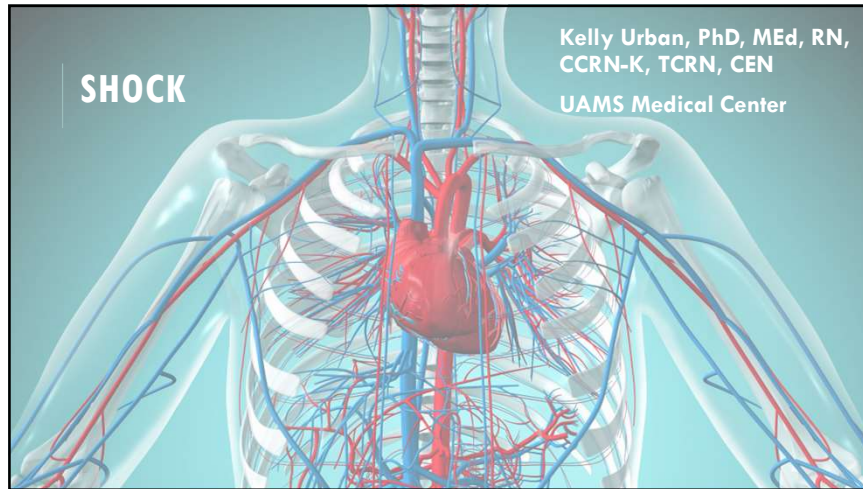
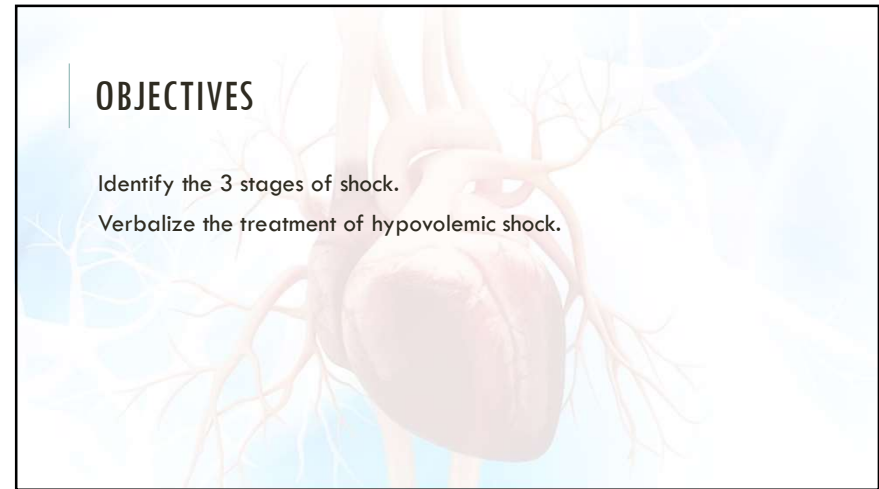
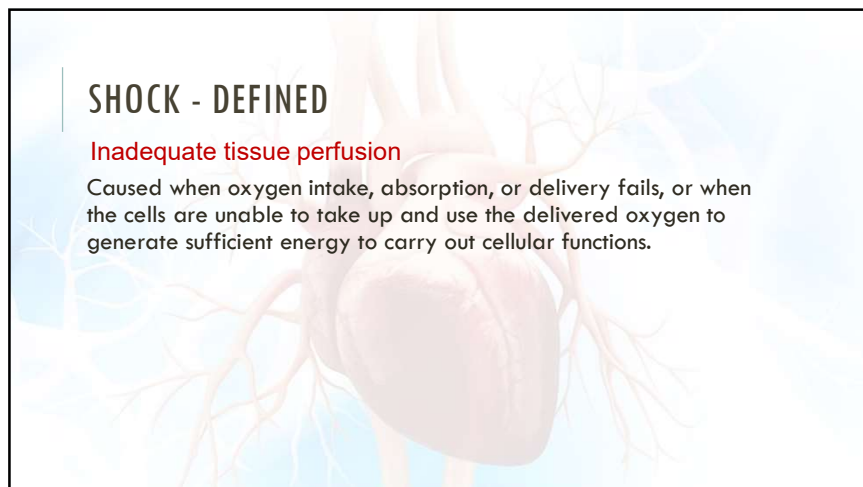




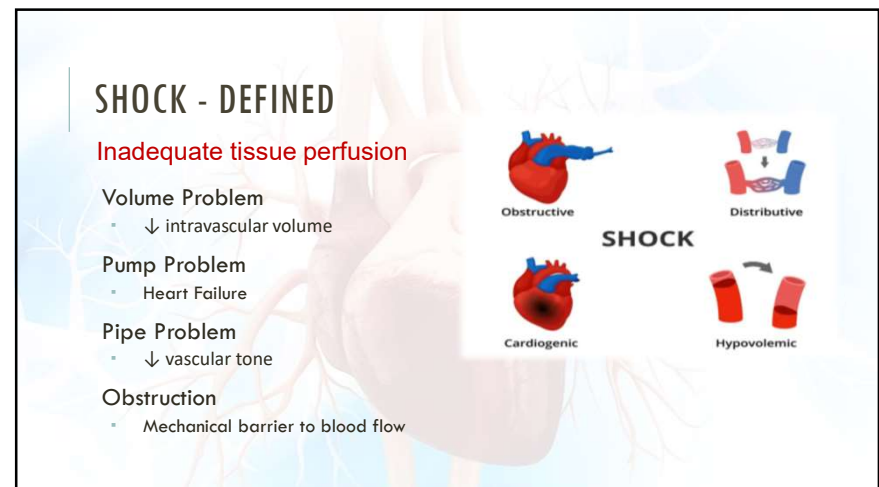
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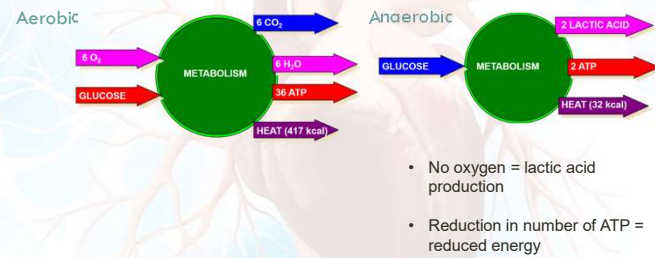


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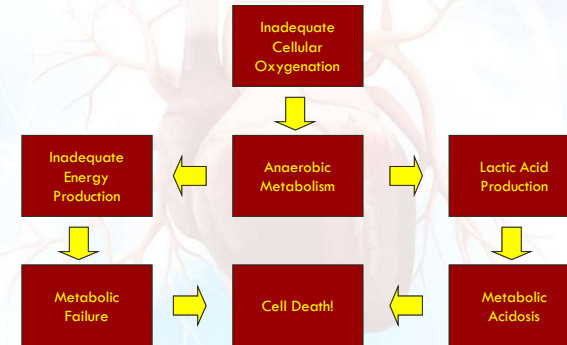
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METABOLISM REVIEW



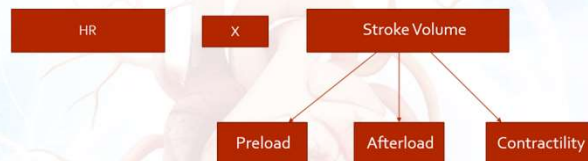
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ANAEROBIC METABOLISM — SO WHAT?



6

CARDIAC OUTPUT REVIEW



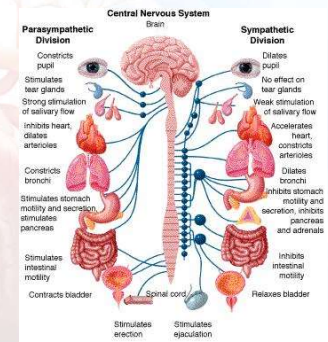
- Preload: amount of stretch at end of diastole (volume ready to be ejected)
- Afterload: resistant in which ventricle has to overcome to contract (vasoconstriction)
- Contractility: ability of the heart to contract

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

9

COMPENSATED SHOCK

(COMPLEX SERIES OF NEURO-ENDOCRINE RESPONSES TO $\uparrow$ CO)

Decreased cardiac output compensatory mechanisms

- Tachycardia

Activation of autonomic nervous system

- Tachycardia
- Vasoconstriction

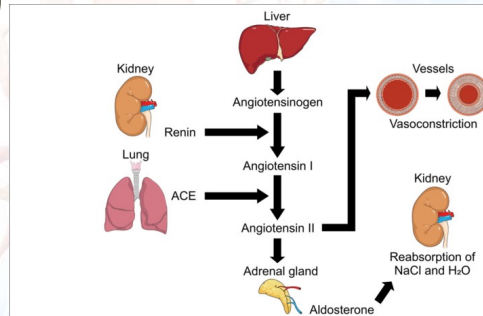
Activation of renin-angiotensin system (due to reduced blood flow to kidneys)

- Vasoconstriction
- Na/Water retention

Increased rate and depth of respirations

10

ACTIVATION RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM (RAAS)



11

COMPENSATED SHOCK — CLINICAL FINDINGS

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

12

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

13

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

14

IRREVERSIBLE SHOCK — MULTIORGAN DYSFUNCTION SYNDROME

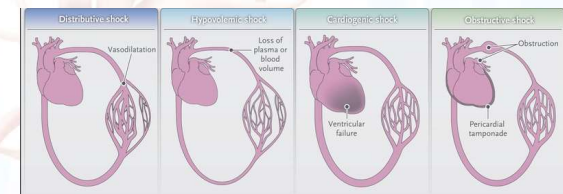
Microvascular and organ damage are now irreversible

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

15

CLASSIFICATION OF SHOCK

Hypovolemic
Distributive
Cardiogenic
Obstructive



16

HYPOVOLEMIC SHOCK

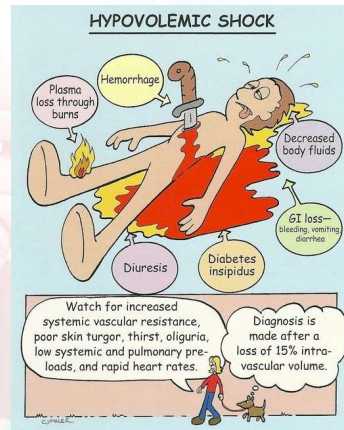
Causes

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



17

HYPOVOLEMIC SHOCK - PATHOPHYSIOLOGY

Decreased Intravascular Volume

Decreased Venous Return

Decreased Ventricular Filling

Decreased Stroke Volume/ Cardiac Output

Inadequate Tissue Perfusion

18

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

19

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



21

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)

22

HYPOVOLEMIC SHOCK TREATMENT — FLUID RESUSCITATION

Ideally, the volume that is lost is replaced

- Crystalloids
- Colloids
- Blood/Blood Products



23

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

24

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



25

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)

26

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

27

END POINTS TO FLUID RESUSCITATION

Traditional

Invasive Hemodynamic Monitoring

Metabolic Parameters

28

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



29

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/m²



30

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

31

HYPOVOLEMIC SHOCK HEMODYNAMICS

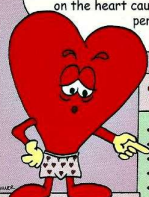
Shock Type	CVP	PAWP	SVR	C.O.	HR	Comments
Hypovolemic	↓	↓	↑	↓	↑	Too little volume



32

CARDIOGENIC SHOCK

Cardiogenic shock is the inability of the heart to maintain cardiac output necessary to meet body needs. Extra strain on the heart causes decreased tissue perfusion.



Causes

- Systolic dysfunction
- Diastolic dysfunction
- Arrhythmias
- Structural problems

Clinical Symptoms

- Tachycardia
- Anxiety and delirium
- Increased preload
- Pulmonary congestion
- Decreased cardiac output
- Dusky skin color
- Decreased blood pressure
- Narrow pulse pressure
- Oliguria
- Dyspnea

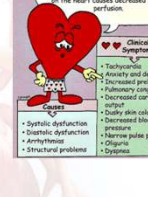
CARDIOGENIC SHOCK

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

33

CARDIOGENIC SHOCK

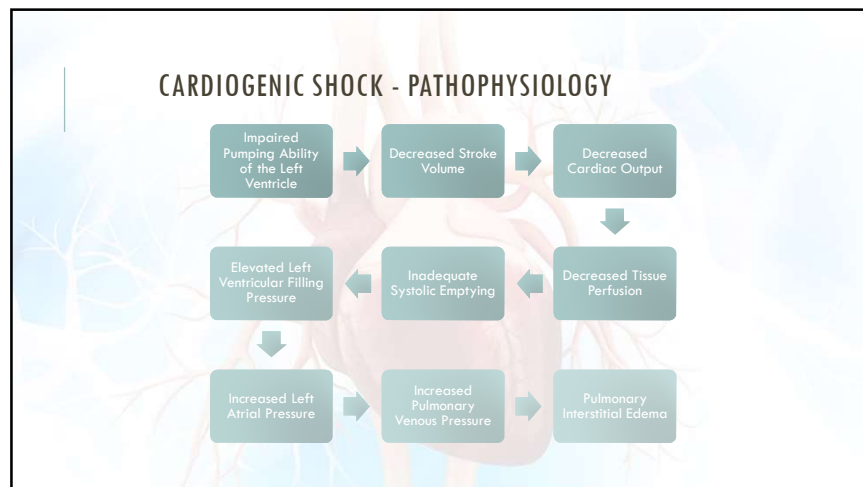
Cardiogenic shock is the inability of the heart to maintain cardiac output necessary to meet body needs. Extra strain on the heart causes decreased tissue perfusion.



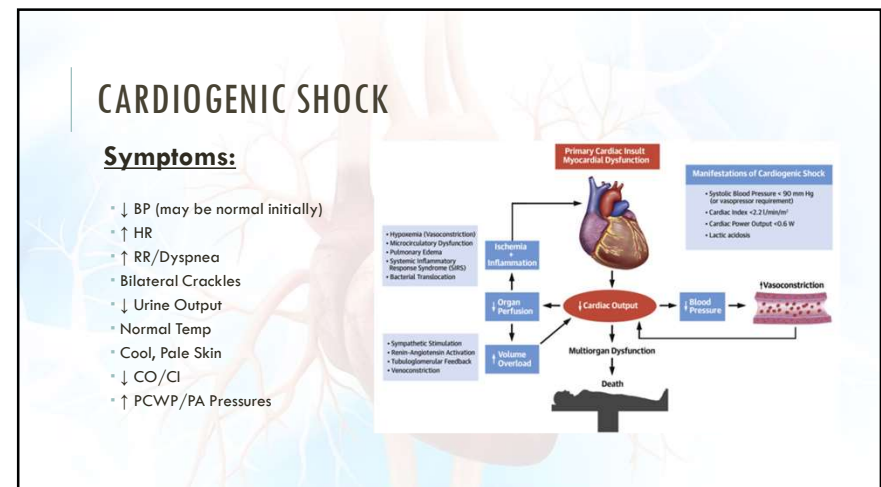
Causes:

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

34



35



36

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

37

CARDIOGENIC SHOCK HEMODYNAMICS

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



38

OBSTRUCTIVE SHOCK

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

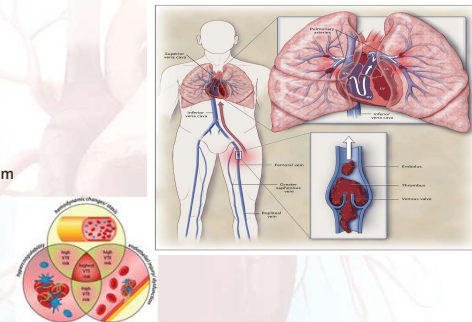
Cause	Treatment
Pregnancy	Roll patient to her side
Tension Pneumothorax	Chest Tube/Needle Decompression
Cardiac Tamponade	Pericardiocentesis
Pulmonary Embolism	Thrombolytics
Aortic Aneurysm	Surgical Intervention
Aortic Stenosis	Surgical Intervention
Excessive Positive End Expiratory Pressure	Readjust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

39

OBSTRUCTIVE SHOCK - PE

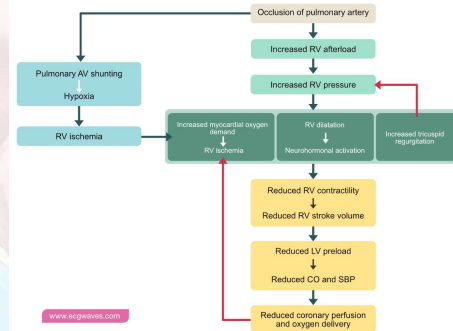
Source

- DVT
- Air embolism
- Fat embolism
- Amniotic fluid embolism
- Septic embolism
- Tumor embolism



40

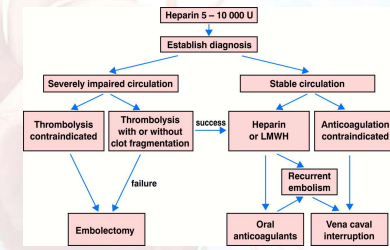
PE PATHOPHYSIOLOGY



41

OBSTRUCTIVE SHOCK TREATMENT - PE

ABCs
Thrombolytics
Embolectomy

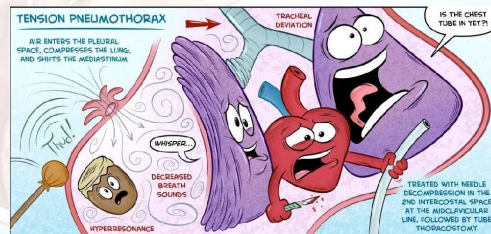


42

OBSTRUCTIVE SHOCK — TENSION PNEUMOTHORAX

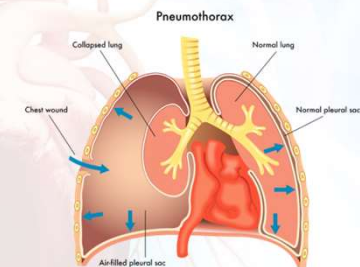
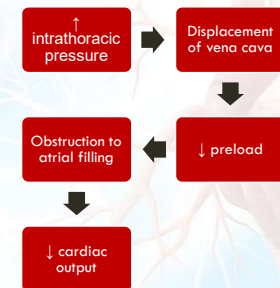
Signs/Symptoms:

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



43

OBSTRUCTIVE SHOCK — TENSION PNEUMOTHORAX



44

OBSTRUCTIVE SHOCK TREATMENT — TENSION PNEUMOTHORAX

Needle Decompression

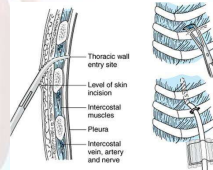
Insert 14 g x 3.25 in angiocath into chest wall

- 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



45

OBSTRUCTIVE SHOCK — CARDIAC TAMPONADE



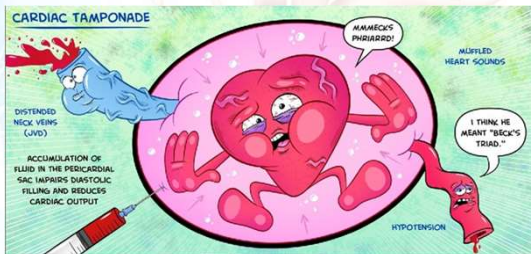
Accumulation of fluid in the (inflexible) cardiac sac

Impediment of diastolic expansion and filling of ventricle

Low preload, SV, CO, and End-Organ Perfusion

46

OBSTRUCTIVE SHOCK — CARDIAC TAMPONADE

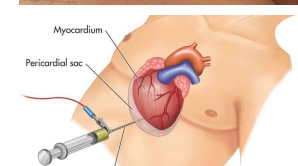


47

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%



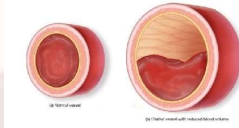
48

DISTRIBUTIVE SHOCK

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

3 Types:

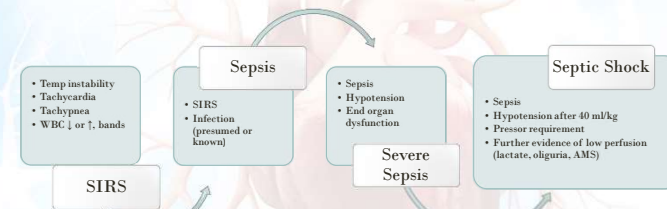
- Septic
- Neurogenic
- Anaphylactic



Occurs when blood vessels dilate without subsequent increase in volume

49

SEPTIC SHOCK (DISTRIBUTIVE)



50

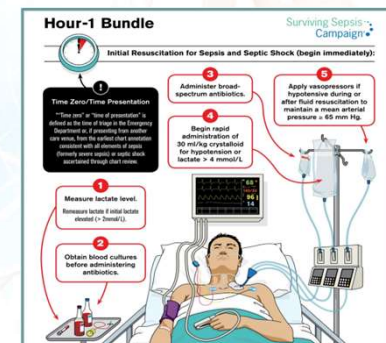
SEPSIS GUIDELINES (DISTRIBUTIVE)

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

51

SEPTIC SHOCK (DISTRIBUTIVE) – INITIAL RESUSCITATION



52

SEPTIC SHOCK (DISTRIBUTIVE) – INITIAL RESUSCITATION

Intervention	Goal	Notes
Fluid	30 ml/kg crystalloid within 1 st 3 hours	- Additional fluids guided by frequent reassessment - Albumin may be considered
Hemodynamic Assessment	Maintain MAP $\geq$ 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	

53

ANAPHYLACTIC SHOCK (DISTRIBUTIVE)

Immune system overreaction that results in a host of vasoactive reactions

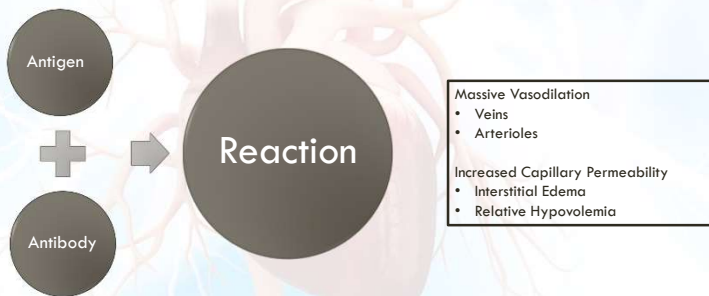


Causes:

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

54

ANAPHYLACTIC SHOCK - PATHOPHYSIOLOGY



55

ANAPHYLACTIC SHOCK

Symptoms

- Urticaria/Pruritis
- "Sense of Impending Doom"
- Bronchoconstriction
- Increased Capillary Permeability
- Decreased CO
- $\uparrow$ HR/RR
- $\downarrow$ PAP/PCWP



56

ANAPHYLACTIC SHOCK – TREATMENT GOALS

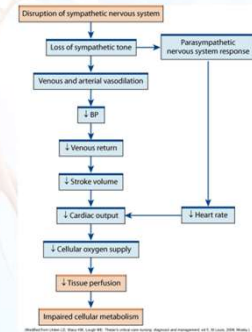
- ID and remove causative agent
- Fluids
- Epinephrine
- Antihistamines
- Corticosteroids
- Bronchodilators
- Patient Education

57

NEUROGENIC SHOCK (DISTRIBUTIVE)

Causes:

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

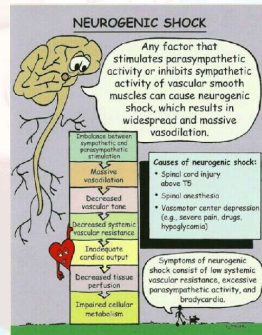


58

NEUROGENIC SHOCK (DISTRIBUTIVE)

Symptoms:

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



59

NEUROGENIC SHOCK (DISTRIBUTIVE)

Treatment

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

60

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



61

AGE-RELATED SHOCK CONSIDERATIONS

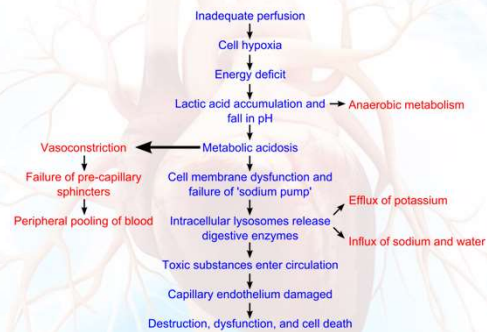
Geriatric Shock Patient

- 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

62

IN SUMMARY:



63

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

64



QUESTIONS?

Contact Kelly Urban @
klurban@uams.edu



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

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

Objectives

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

2



Cardiac Risk Factors



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

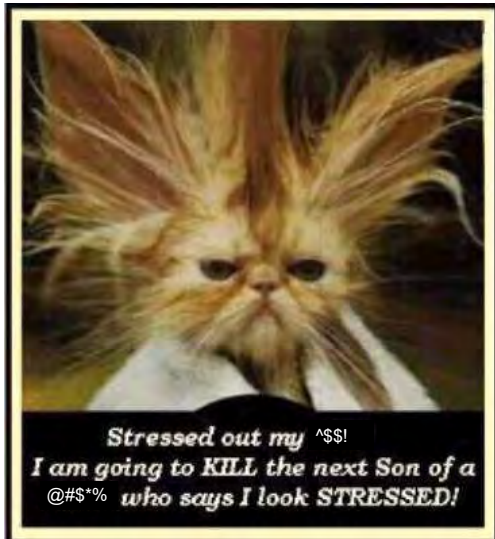


TESTING

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

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

6

GI Complaints...



Gall Stones
Acid Reflux
Indigestion
Hiatal Hernia
Esophageal Spasm

7

Apex
of
Right
lower
lobe



8

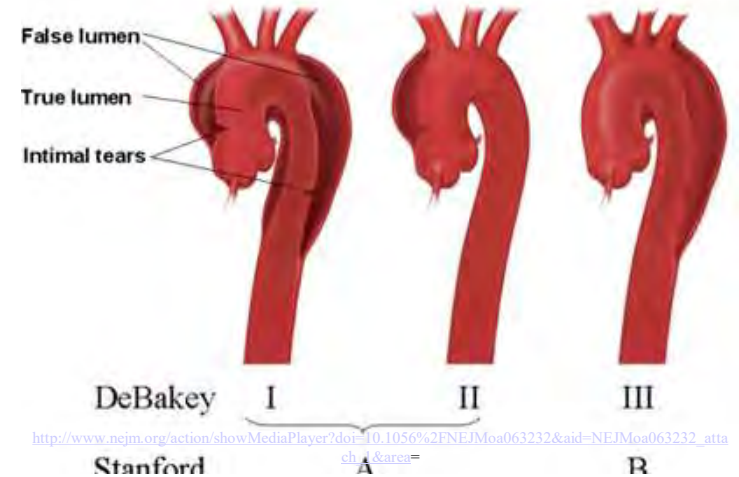
8

DISSECTED AORTA



9

Anatomy and Classification of Aortic Dissection



10

Cardiac – non CAD ischemia



Non CAD
ischemia.....

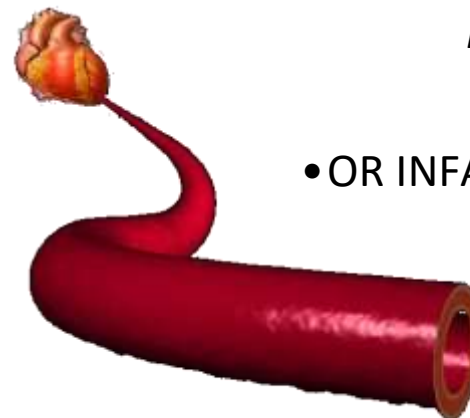
Anemia
Dehydration
HTN crisis
tachycardia
spasm

11

11

Cardiac

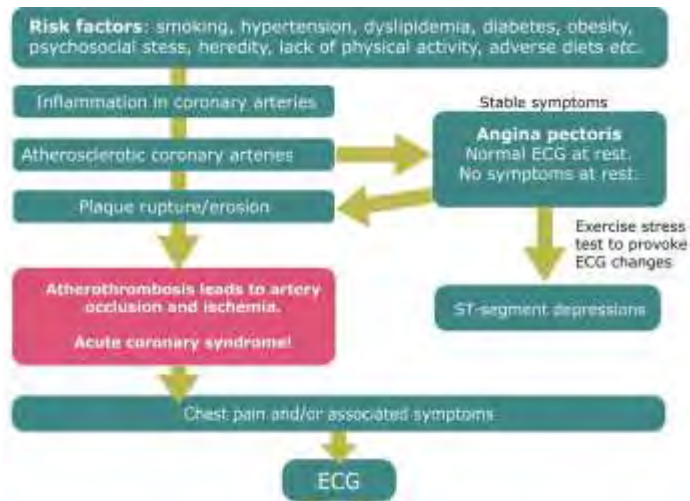
• MAY BE ANGINA
Ischemia



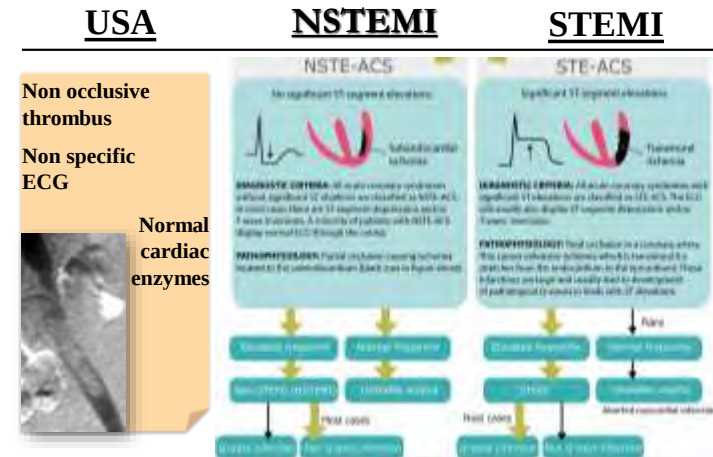
• OR INFARCTION
Injury

12

12



13



14

Diagnosis of Acute MI STEMI / NSTEMI

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



15

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

16

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

17

Evaluation

- Efficient & direct history
 - Initiate stabilization interventions
- } Occurs simultaneously

Plan for moving rapidly to indicated cardiac care



19

Acute Management



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

18

Description



Pain

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

20

20

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

21

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

23

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



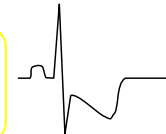
22

ECG assessment

ST Elevation or new LBBB
STEMI



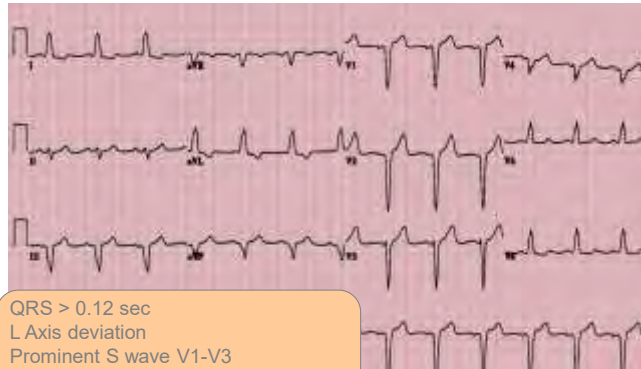
ST Depression or dynamic T wave inversions
NSTEMI



Non-specific ECG
Unstable Angina

24

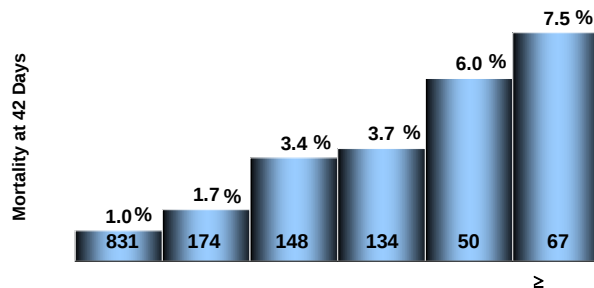
New LBBB



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

25

Prognosis with Troponin



27

Cardiac markers

Troponin (T, I)

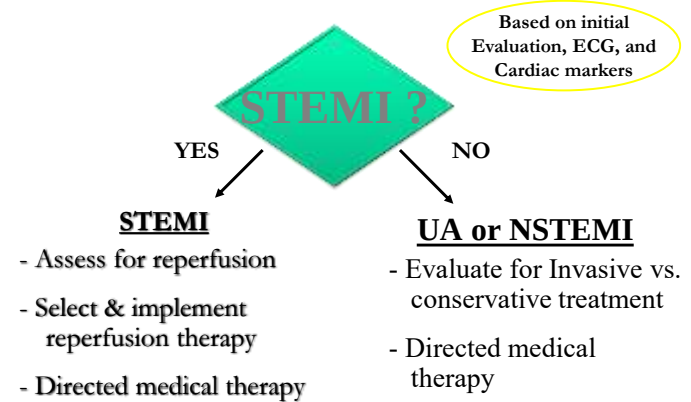
- Very specific and more sensitive than CK
- Rises 4-8° after injury
- May remain elevated for up to two weeks
- Can provide prognostic information
- Troponin T may be elevated with renal dz, poly/dermatomyositis

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

26

Risk Stratification



28

Cardiac Care Goals

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



29

Killip Class

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

<https://acc.org/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%

31

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



30

TIMI Score for USA/NSTEMI

Antman et al JAMA 2000; 284:0.35.042

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)

32

32

Fibrinolysis indications

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



33

ACS/STEMI Pharmacotherapy Medical Therapy MONA + BAH

38

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

34

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



42

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 O₂ demand
 - To reduce extension of ischemia by reducing oxygen 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**



43

43



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



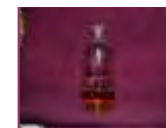
Careful with active PUD, hypersensitivity, bleeding disorders

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

45

45

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

44

44

β-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 β₁ receptors
- Reduces HR, BP, myocardial contractility
- Decreases AV nodal conduction
- Decreases incidence of primary VF

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



46



β-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 ¹
Atenolol	5-10 mg IV × 1, then 100 mg/day PO	ISIS-1 ²
Carvedilol	6.25 mg bid titrated to 25 mg BID	CAPRICORN ³



47



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



49

- **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% ¹
Ramipril	1.25 mg bid titrated to 5 mg bid	AIRE: 3-10 days post-MI with symptoms of heart failure ²
Captopril	6.25 mg bid titrated to 50 mg bid	ISIS-4: started within 24 hr of MI ³
Lisinopril	5 mg/day titrated to 10 mg/day	GISSI-3: started within 24 hr of MI ⁴

48

Unfractionated Heparin Dosing

Loading Dose

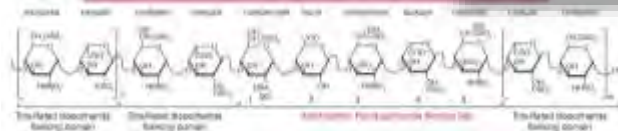
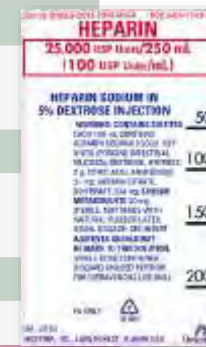
- 60 U/kg IV bolus
- Max 5000 U if >65 kg or 4000 U if <65 kg

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

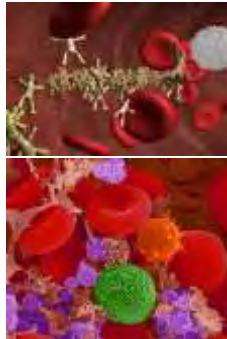


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Antiplatelet Agents

- **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 (<i>reopro</i>)	Ticagrelor
– Eptifibatide (<i>integrilin</i>)	Vorapaxar
– Tirofiban (<i>Aggrastat</i>)	Cangrelor
– Prasugrel	



51

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



53

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

52

52

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**



54

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
Pacitaxel 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.

55

Cases to Ponder...

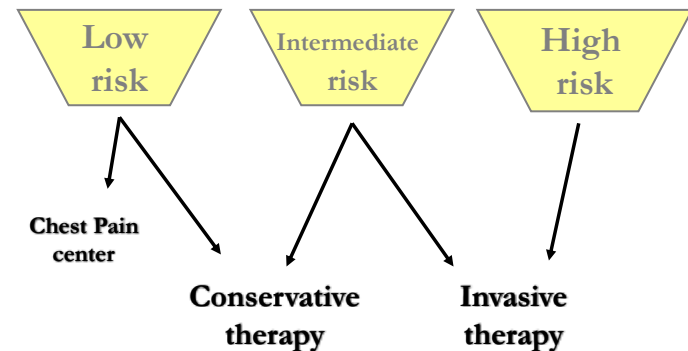
- 64 y/o female collapses in the cafeteria with c/o new onset chest pain. She has no cardiac history, her troponin is negative and her ECG has non-diagnostic, fixed changes.
- A 75 y/o male visitor is sagging against the wall in the hallway, he is diaphoretic, with weak rapid pulses. You notice the top 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

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

56



64

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



65

Secondary Prevention

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



67

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



66

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%



68

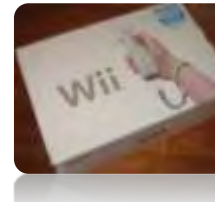
Secondary prevention behavioral intervention

- **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



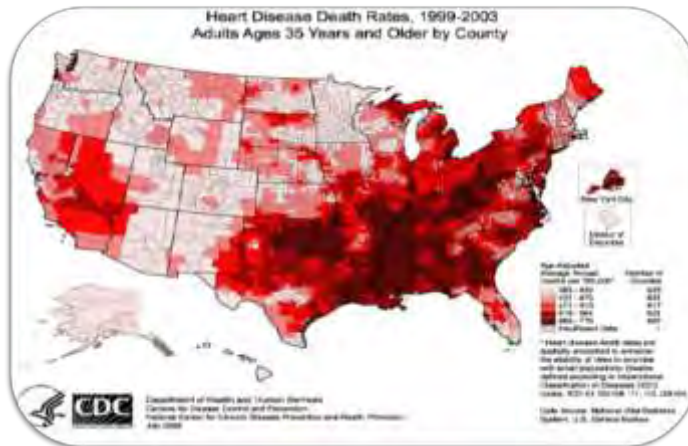
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Thinking outside the box...



70

Or maybe just move....



71

Secondary prevention cognitive

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



72

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)



73

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

76



75

- *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.*

77

77

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78

78



79

Unraveling the web of 12-Lead Interpretation



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

80

80

81

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
- fascicular blocks
 - LAFB
 - LPFB
- Bifascicular
- Trifascicular

Hypertrophies

- LVH
- RVH
- LAE
- RAE

Other conditions

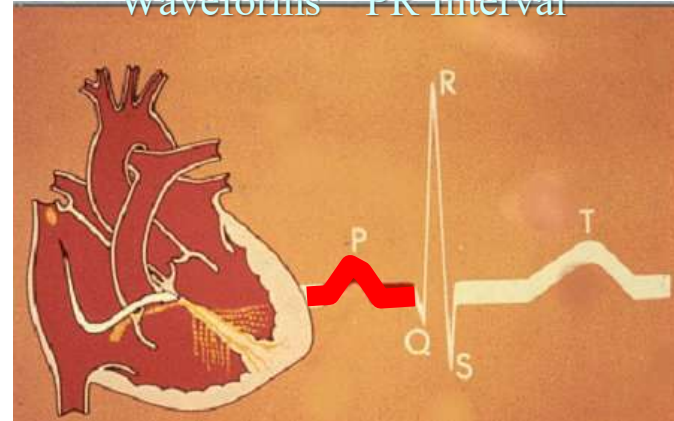
- Pulmonary Emb.
- Cerebral Events
- Brugada Syndrome
- Long QT syndrome
- Wolfe-Parkinson White syndrome

Aberrant conduction

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

82

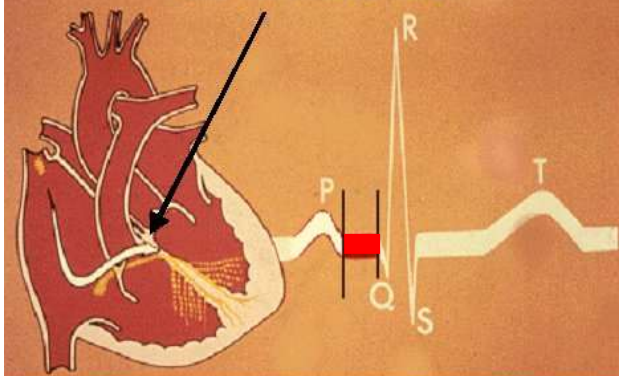
Waveforms PR Interval



83

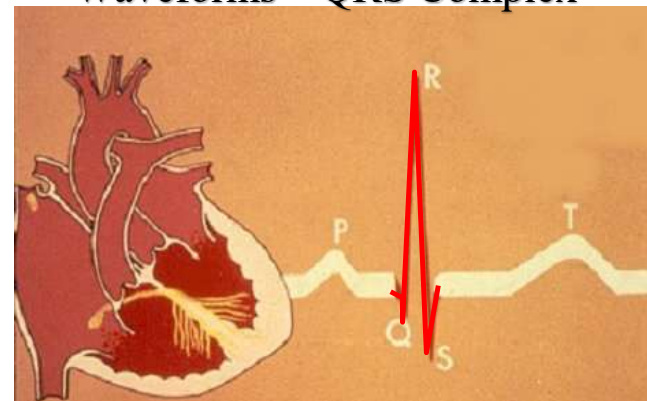
Waveforms PQ segment

Atrio-Ventricular Node



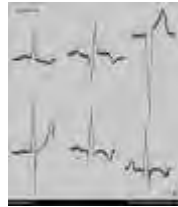
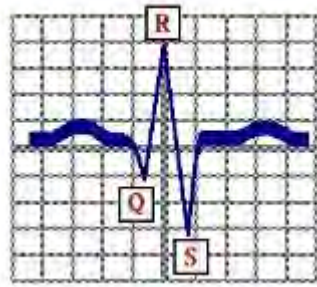
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Waveforms QRS Complex

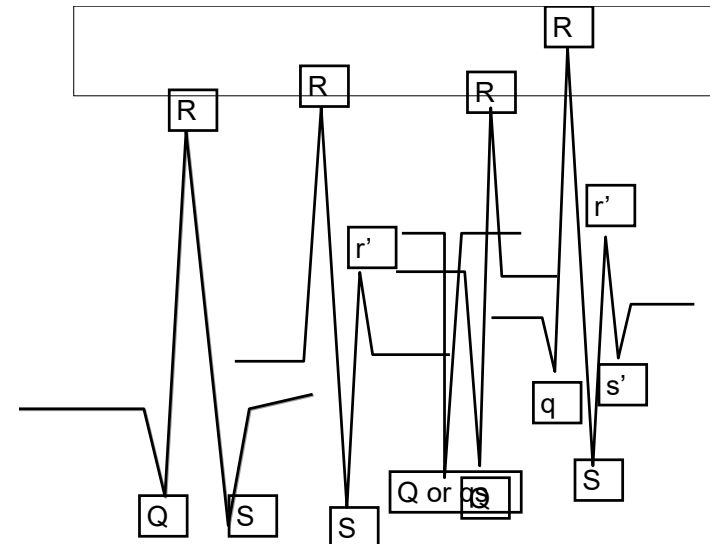


85

QR&S Waves

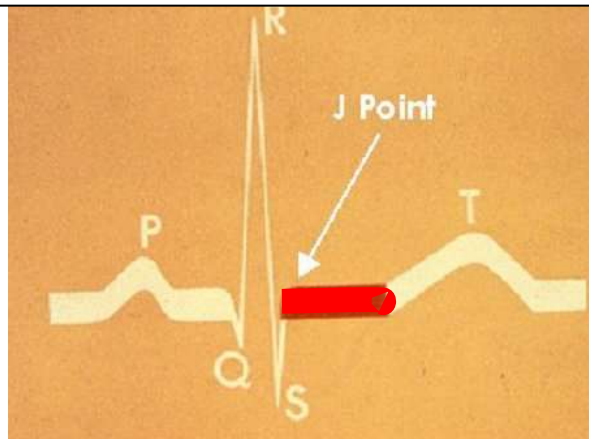


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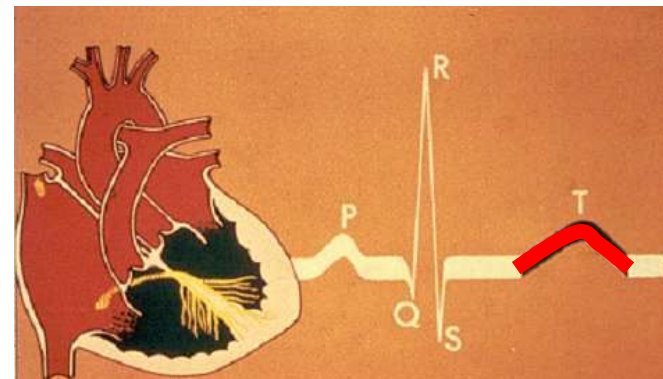
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Waveforms ST Segment



88

Waveforms T-Wave

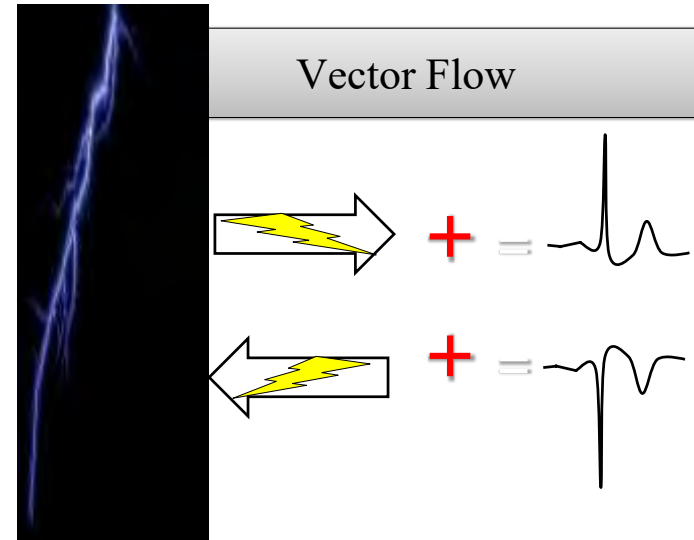


89

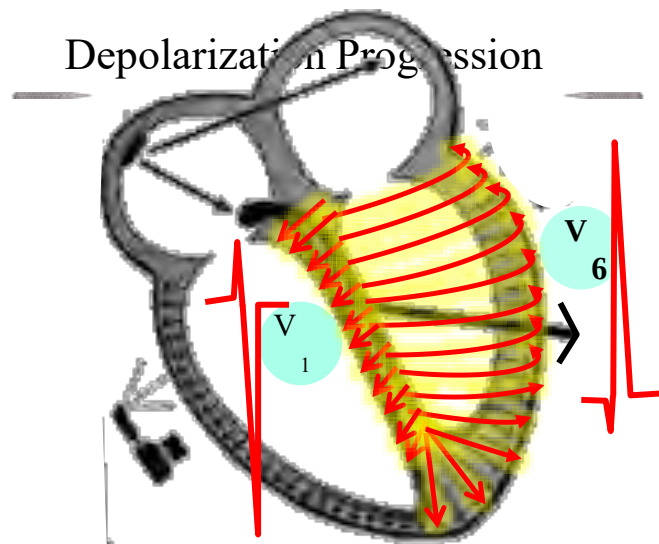
Standard Lay-out

Lead I	aVR	V1	V4
Lead II	aVL	V2	V5
Lead III	aVF	V3	V6
Rhythm Strip one			
Rhythm Strip two			
Rhythm Strip three			

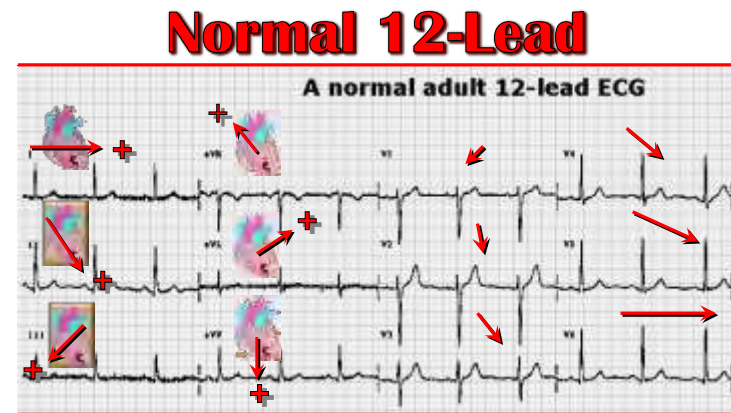
90



91



92



93

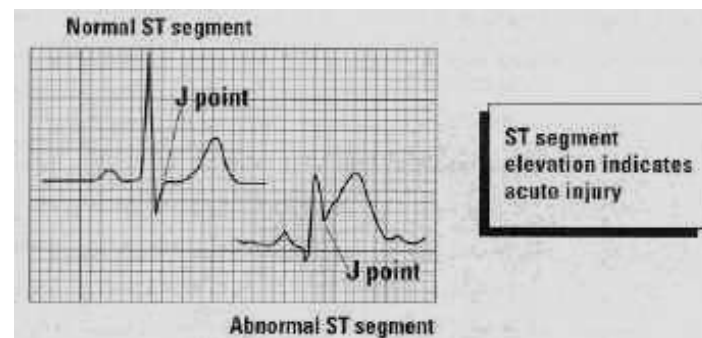


Triad of Anoxic Changes

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

94

Triad of Anoxic Changes ST Elevation

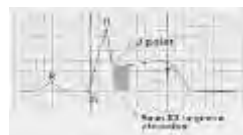
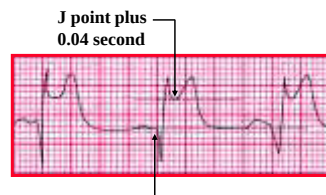


95

12-Lead ECG

- ST elevation
- = Injury
- = Acute MI

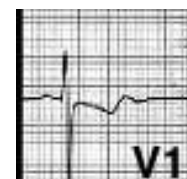
- STE >1mm indicates MI
- Must be present in at least 2 contiguous leads



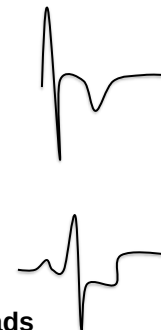
96

12-Lead ECG

- T-Wave Inversion
- = Ischemia
- = Angina

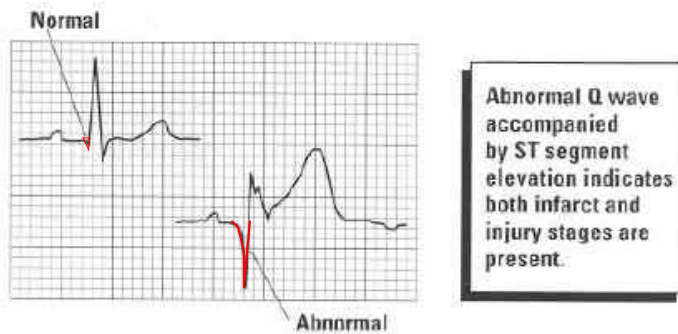


- Resolves when perfusion restored
- Global may be pericarditis
- Must be present in 2 contiguous leads



97

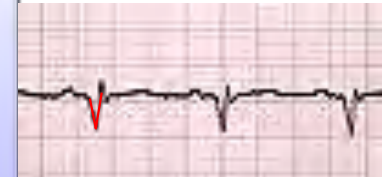
Abnormal Qs



98

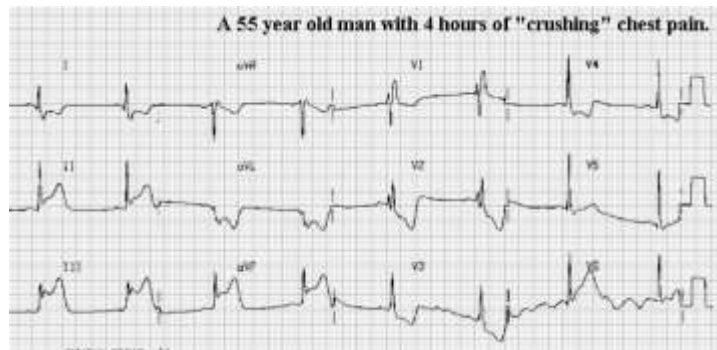
12-Lead ECG

-  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



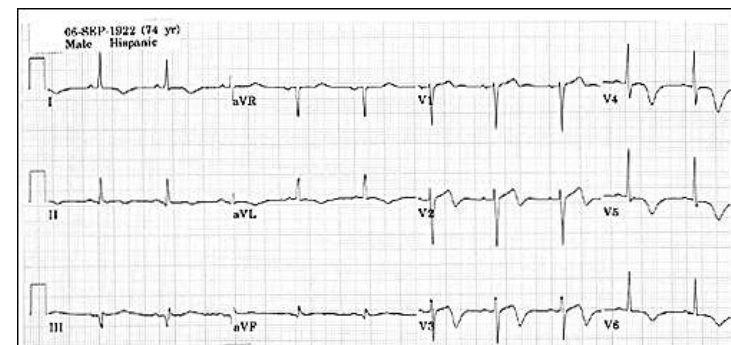
99

Intro #1



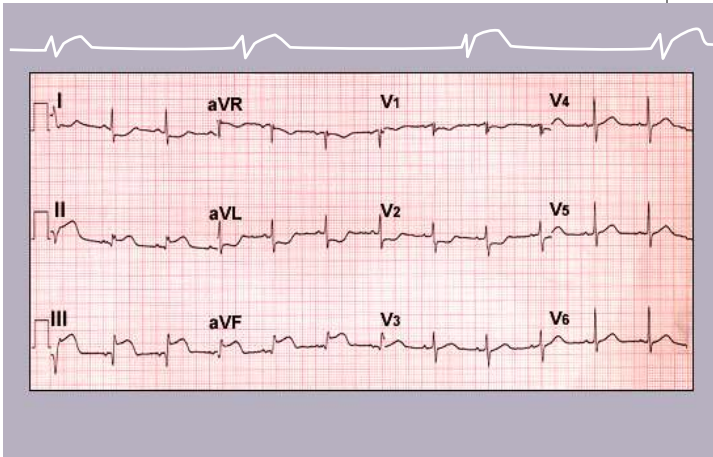
100

Intro #2



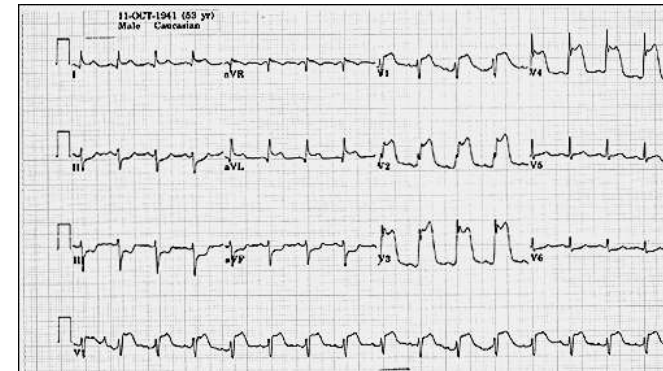
101

Intro #3



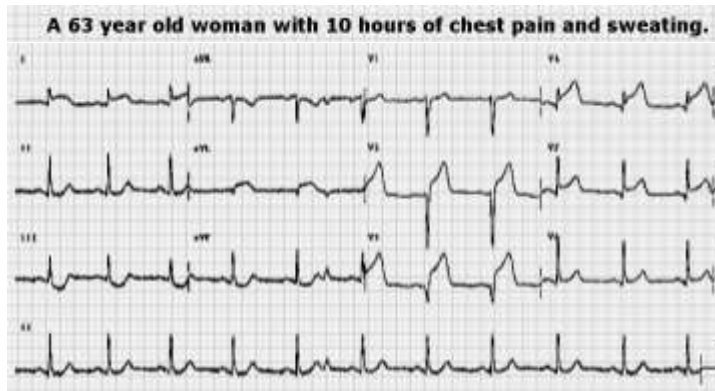
102

Intro #4

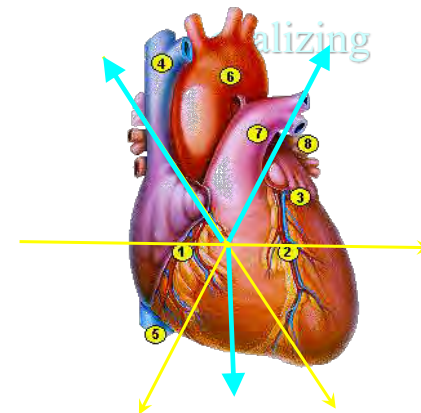


103

Intro #5



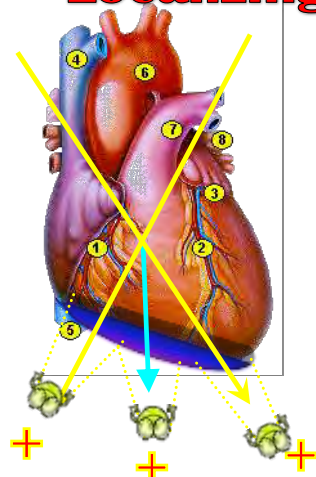
104



105



Localizing



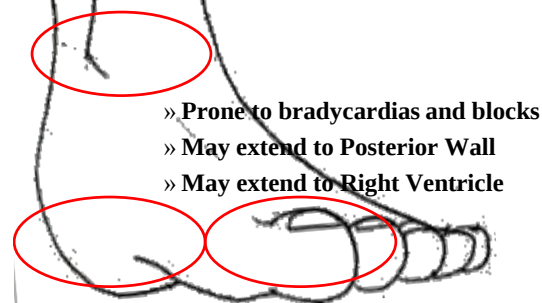
106



Localizing

Changes

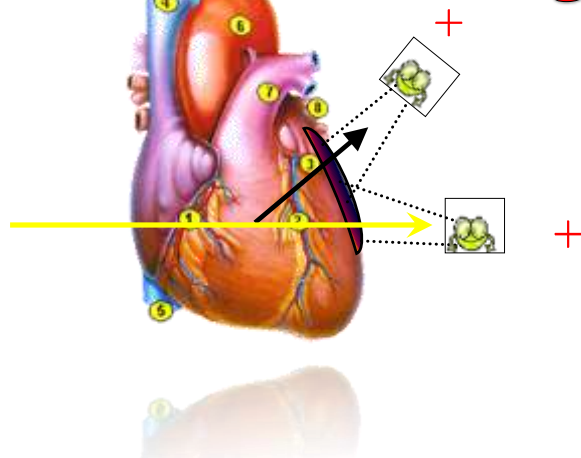
- **Inferior Wall**
- II, III, avF
- Fed by Right Coronary Artery



107



Localizing



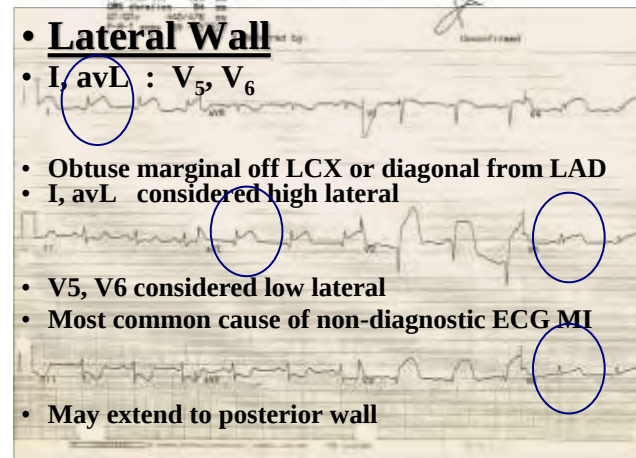
108



Localizing

Changes

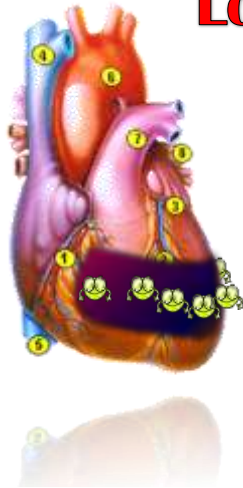
- **Lateral Wall**
- I, avL : V₅, V₆
- Obtuse marginal off LCX or diagonal from LAD
- I, avL considered high lateral
- V₅, V₆ considered low lateral
- Most common cause of non-diagnostic ECG MI
- May extend to posterior wall



109



Localizing



110

Localizing

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

Changes

111



Localizing

More Specific V Lead:

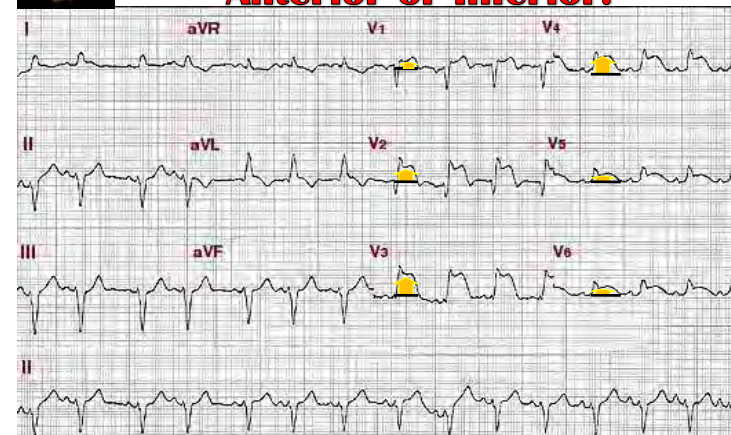
- ♥ V_1-V_3 = Antero-Septal
 - Highest mortality MI
 - Prone to bundle branch, and fascicular blocks
 - High-risk for septal rupture and PEA
- ♥ V_5-V_6 = Antero-Lateral
 - Tied to Inferior, Lateral or Anterior MI
 - Indicate LAD dominance

Changes

112

ECG #1

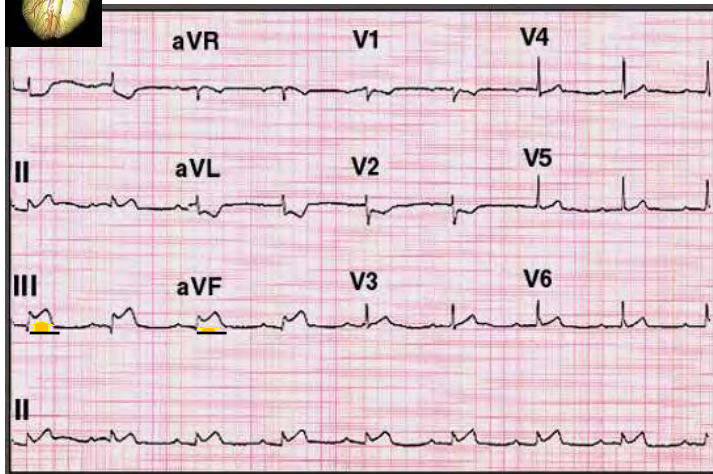
Anterior or Inferior?



113



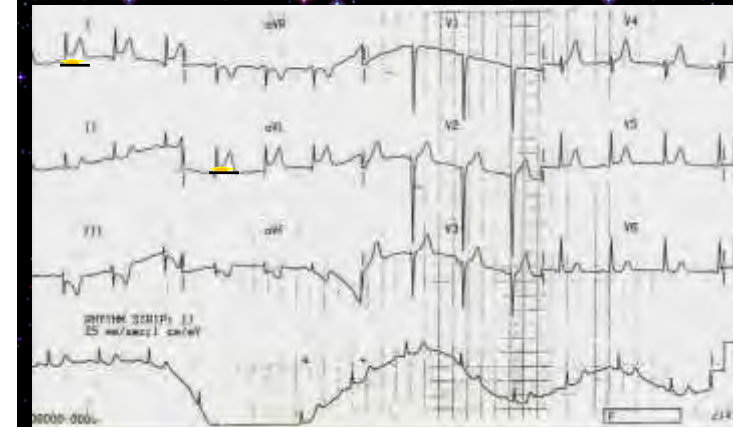
ECG #2



114



Jose Cuervo



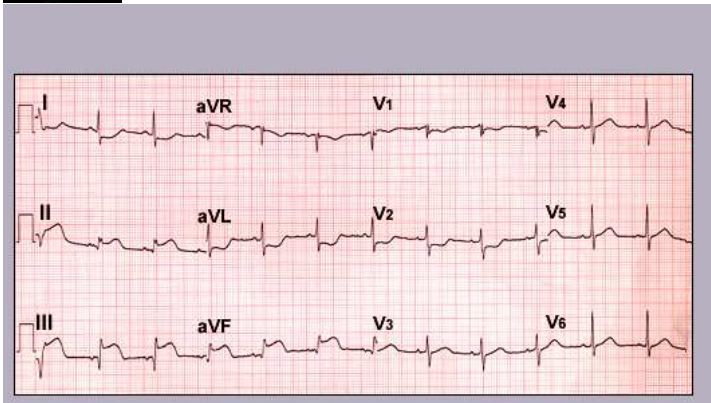
115



ECG #3

Anterior, Inferior?

30 minutes old or 3 hours old?



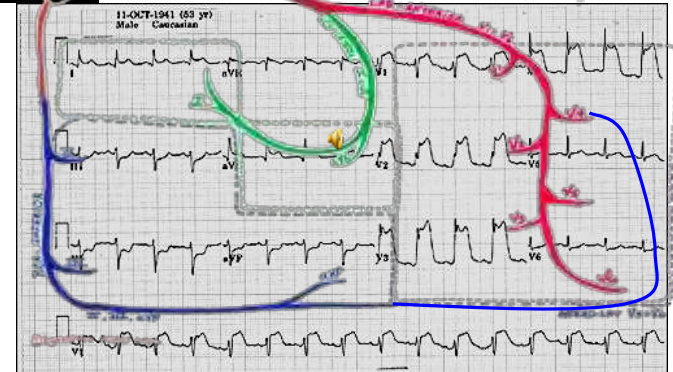
116



ECG #5

Anterior, Inferior?

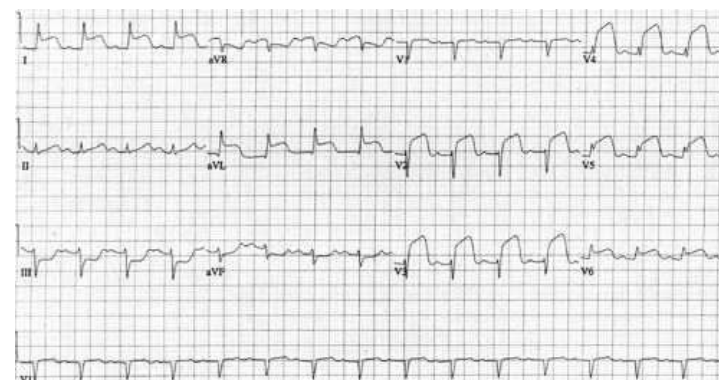
30 minutes old or 3 hours old?



117



118



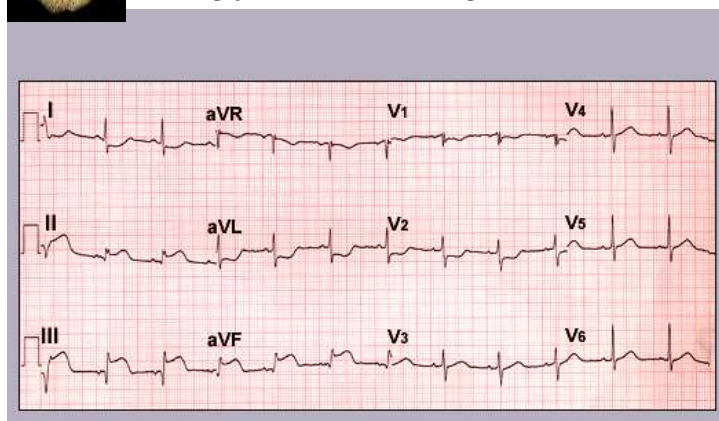
119



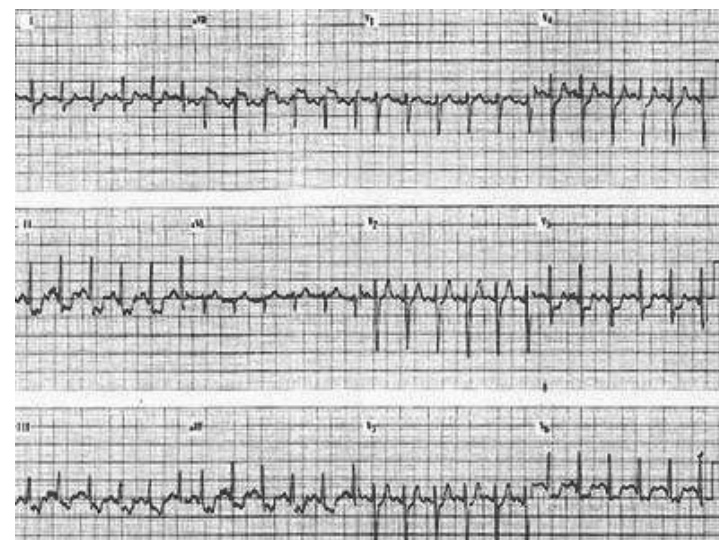
ECG #3

Anterior, Inferior?

30 minutes old or 3 hours old?



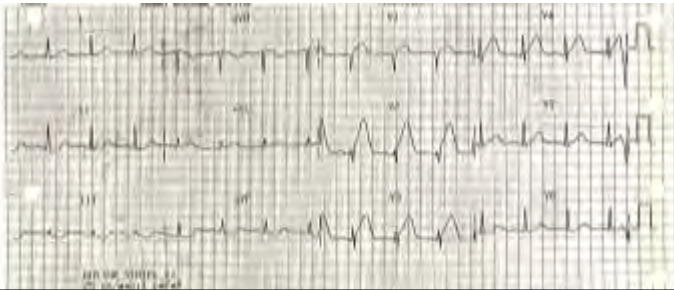
120



122

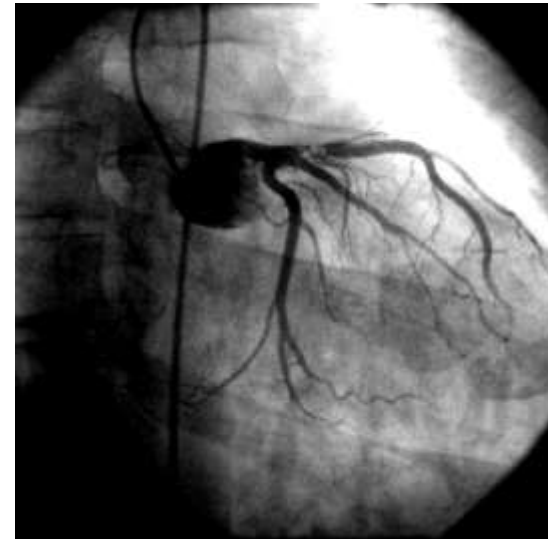


Patient being evaluated for USA



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

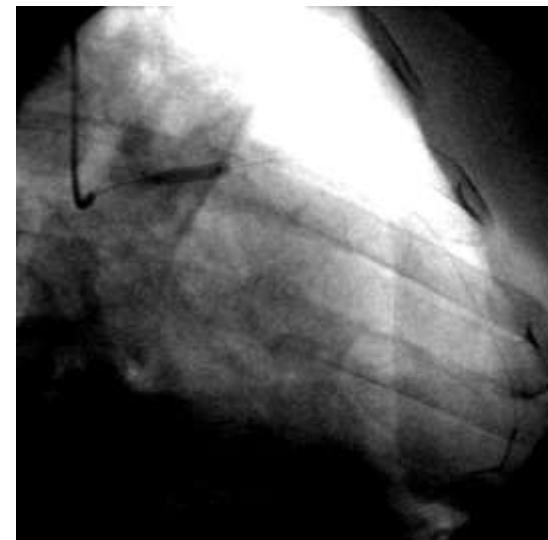
123



124



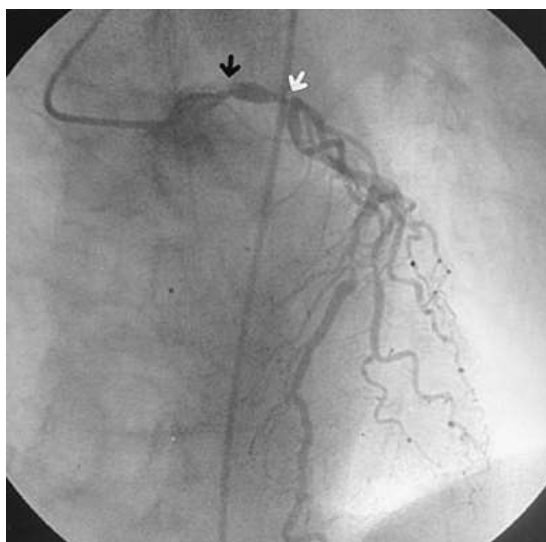
125



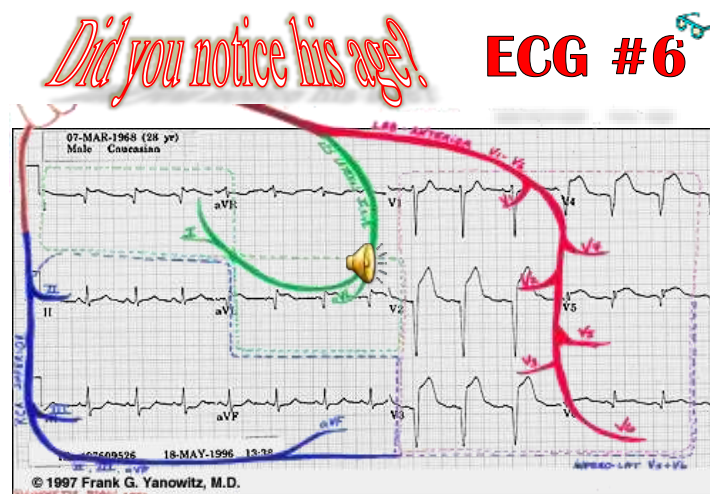
126



127



129



128

Worksheets

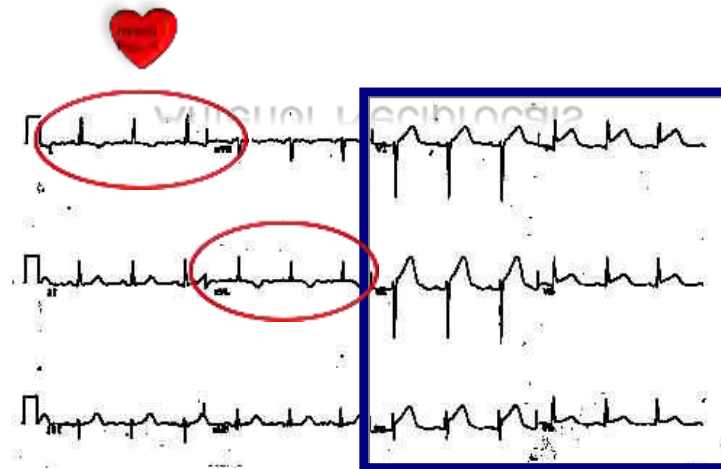
- Review 1 and 3
- Find all STElevation
- Find any pathological Q waves
- Localize all changes to Anterior, Inferior or Lateral

130

Reciprocals



131



133



Reciprocal MI

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

Inferior leads

II III aVF

Mirror leads

I aVL

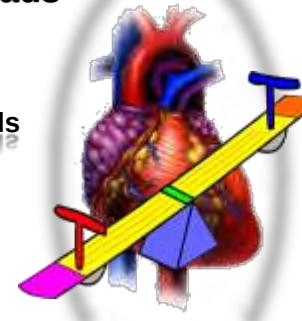
Anterior Leads

V1-V6

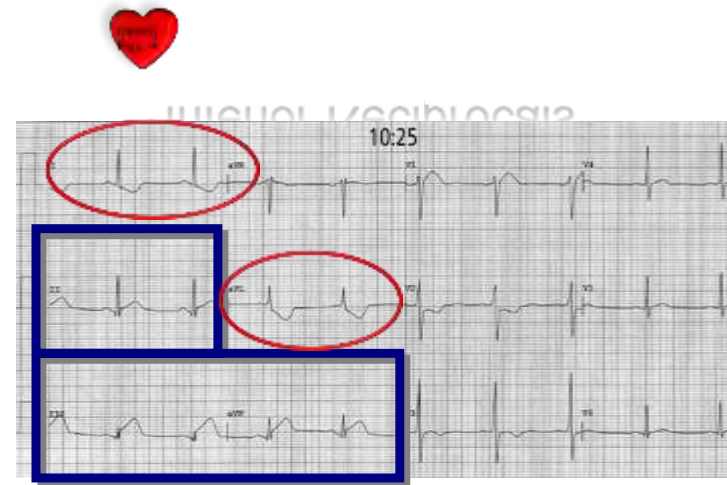
Limb leads

Posterior Wall

V1-V3



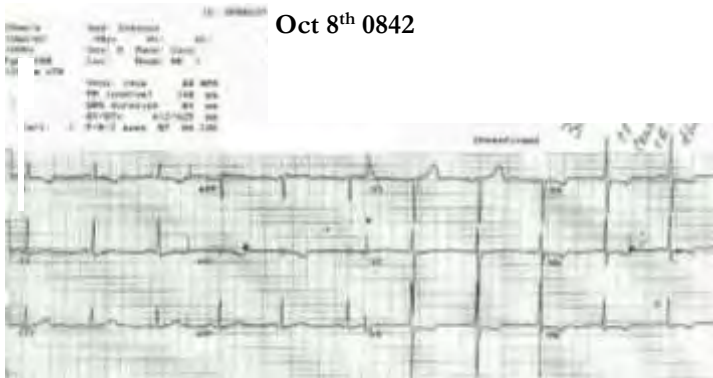
132



134

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

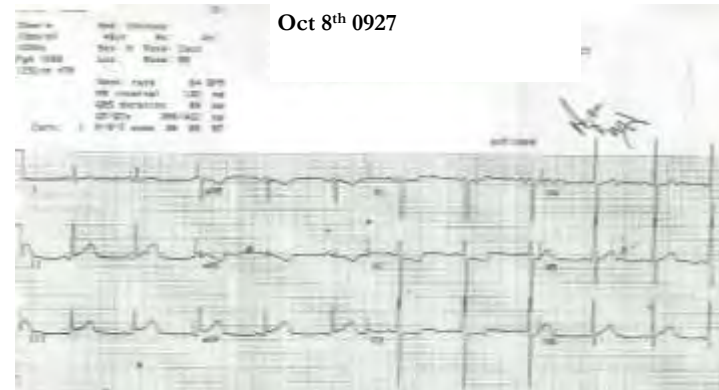
Oct 8th 0842



135

TPA started @ 0927

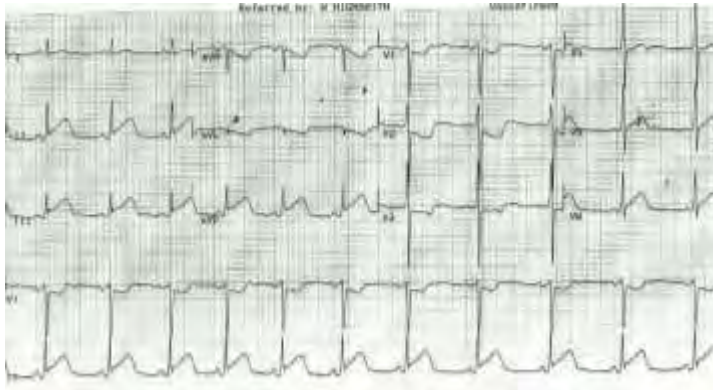
Oct 8th 0927



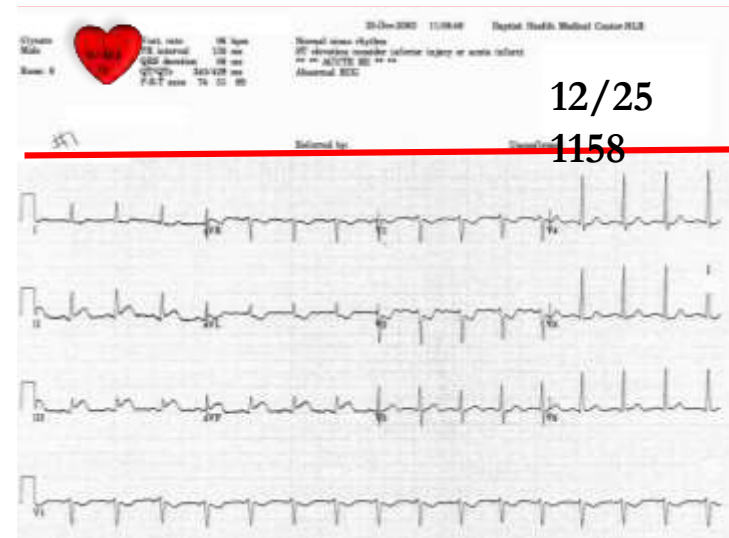
136

**October 8th
1002**

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



137



138

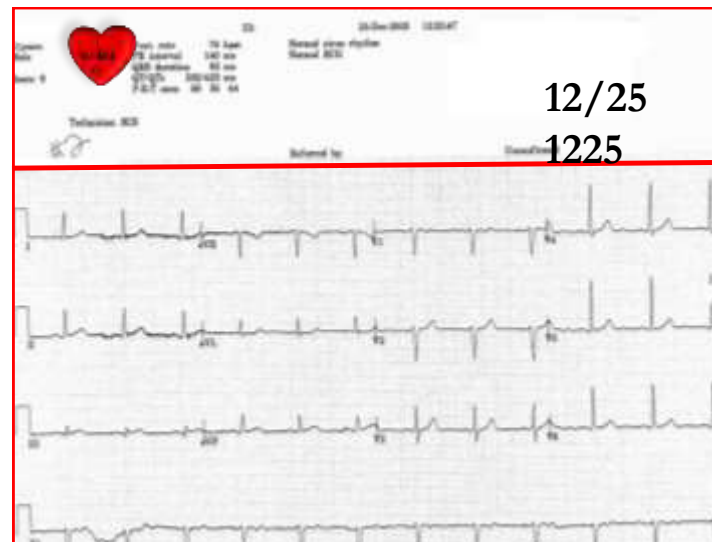


139

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



141

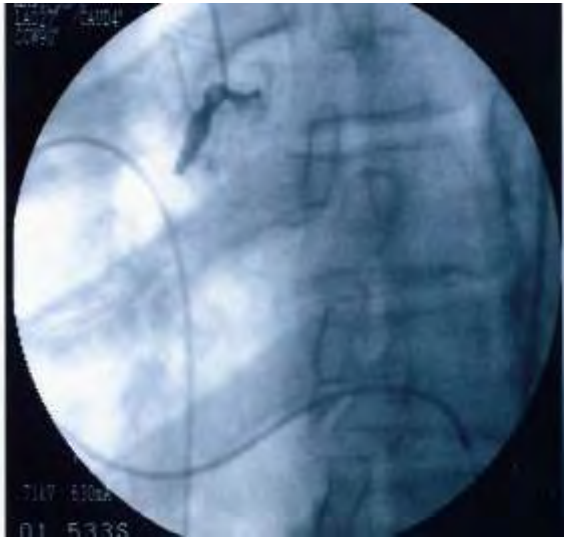


140

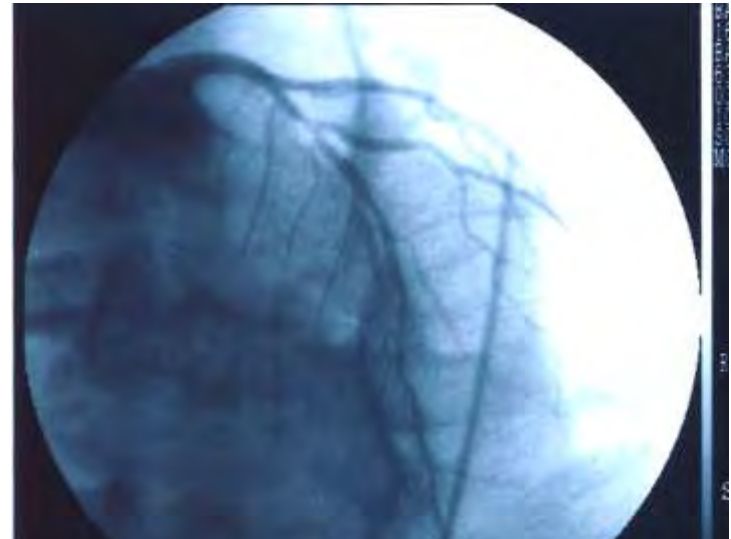
20 minutes later. After ASA O2, Heparin, and MSO4 patient is
asleep and felt to be pain free..... 9/25 @ 23:44



142



143



144

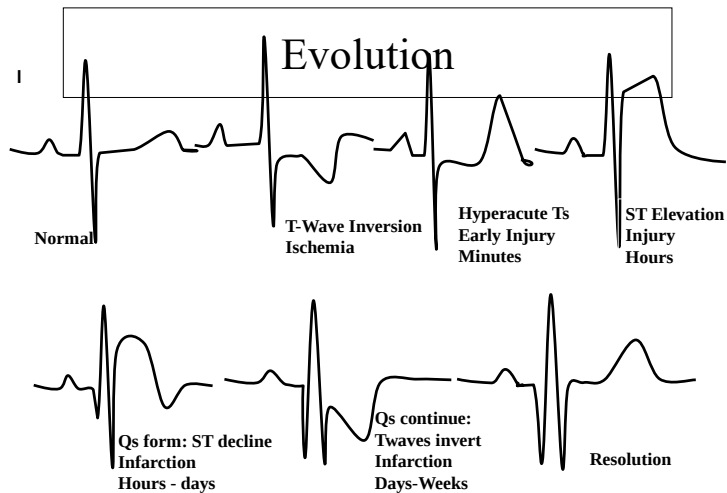
Worksheets

- Review 1
- Review 3
- Find all reciprocal changes

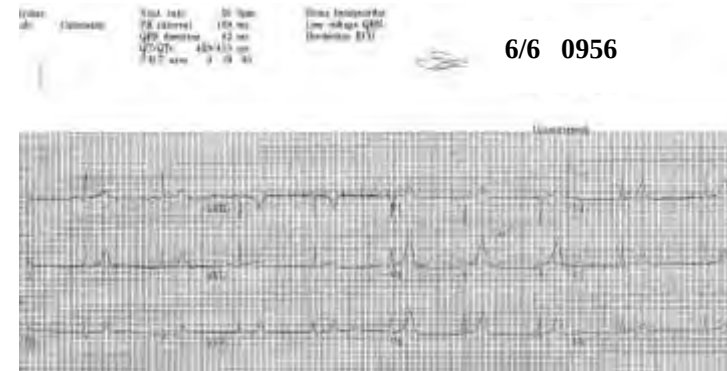
145

Evolution

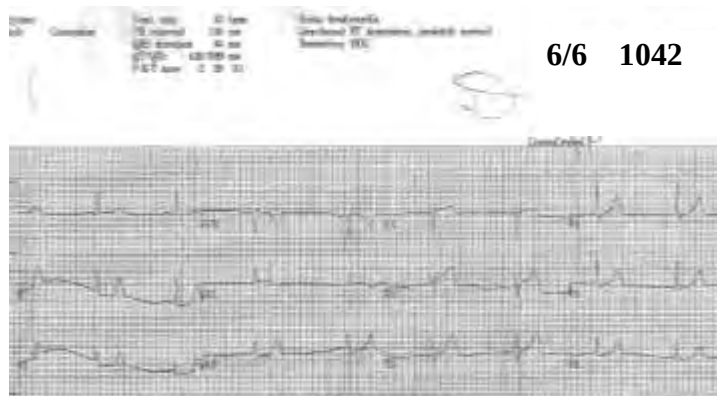
146



147



148



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.

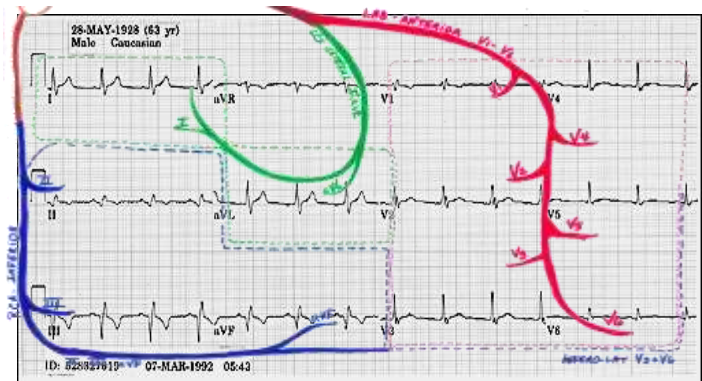
On questioning he admitted that about 5 days ago he had a weak spell while out in the garden and had to lie down for several hours before regaining the strength to walk into the house.

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

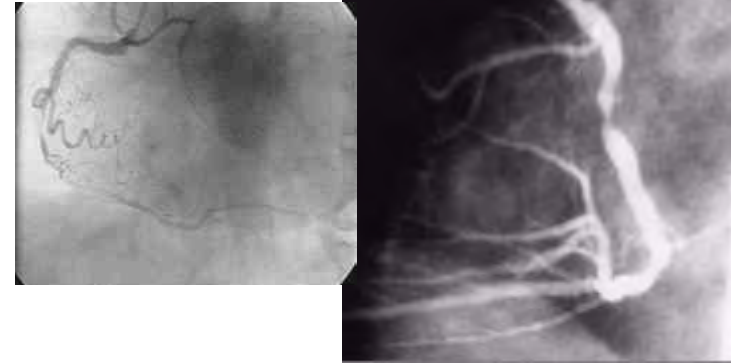
150



Mid RCA



151



152

Worksheets

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

- Compare 1 and 2
- Compare 3 and 4

153

154



155

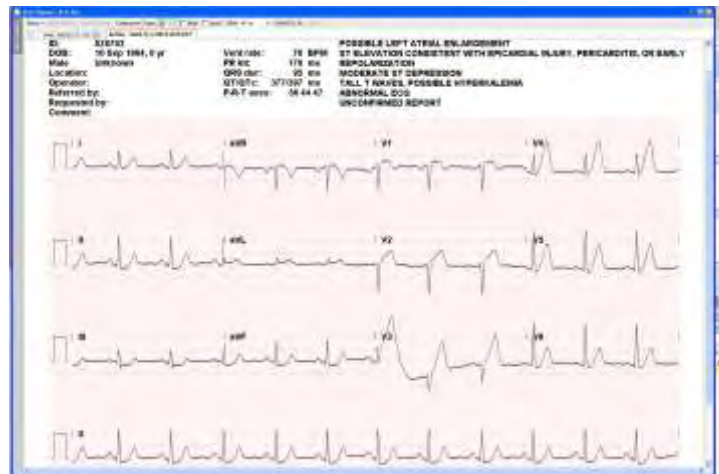
CHIEF COMPLAINT:
Interscapular aching.

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

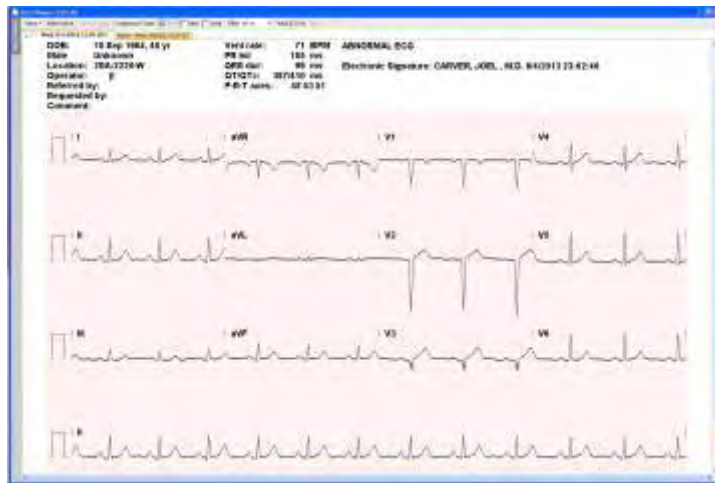


157

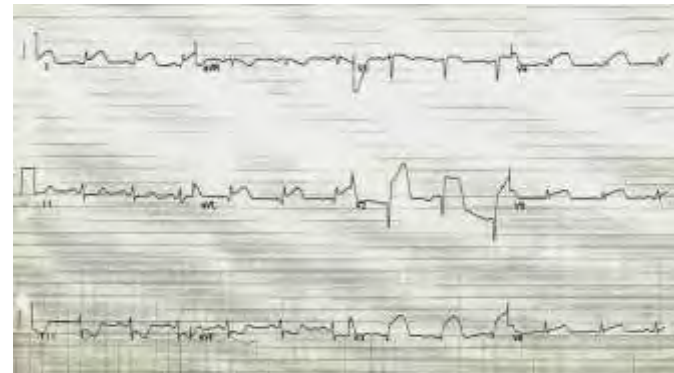
- SELECTIVE CORONARY ANGIOGRAPHY
- A. LEFT MAIN CORONARY ARTERY: The left main is free of disease.
- B. LEFT ANTERIOR DESCENDING CORONARY ARTERY: The left anterior descending coronary artery is a large wrap around vessel. It was totally occluded in its mid segment. This is reduced to no appreciable narrowing 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

Study Strips #1

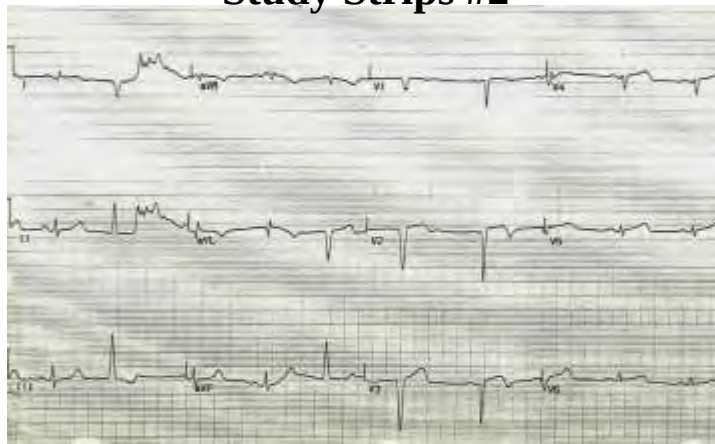


159



160

Study Strips #2



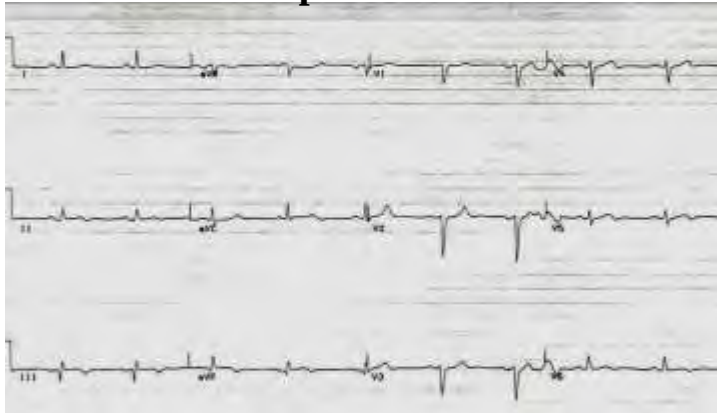
161

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

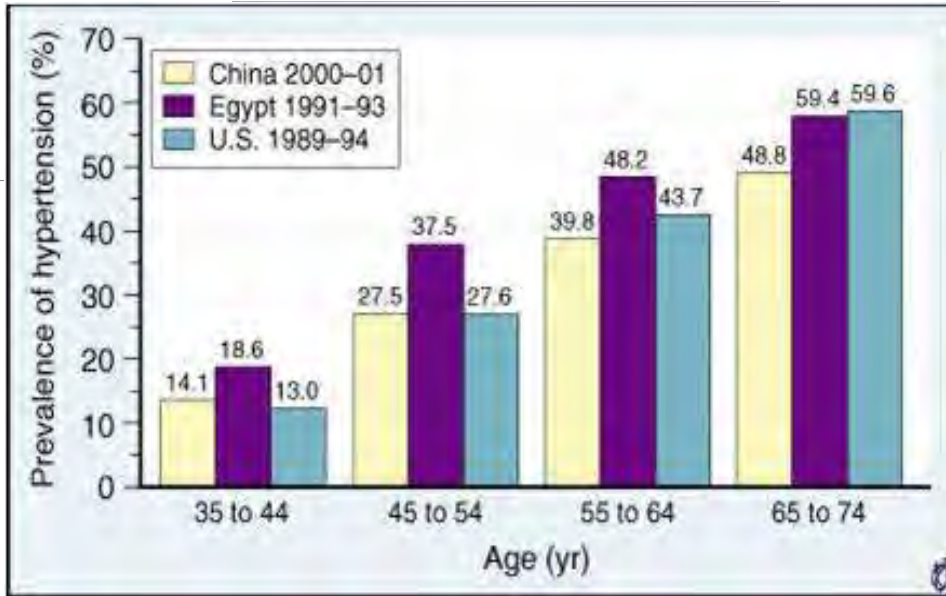
Key Concept



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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AR

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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

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Heart Effects



Heart

Tachycardia

↑ CO

↓ perf. → angina → MI

CAD

LV hypertrophy

LV failure

Angina



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Brain Effects

Brain

Loss of autoregulatory mechanisms

Arterial spasm → ischemia → TIA

Weakened vessels → aneurysms →
hemorrhage → CVA

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Kidney Effects

Kidney

- ↓ Renal perf
- ↓ Ability to concentrate
- ↑ BUN, CR
- ↑ Proteinuria
- Kidney failure
- Uremia

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Diagnostics

Laboratory

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

ECG

Radiologic

- CXR
- Echocardiogram
- MRI or CT
- Renal ultrasound



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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

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Pharmacology

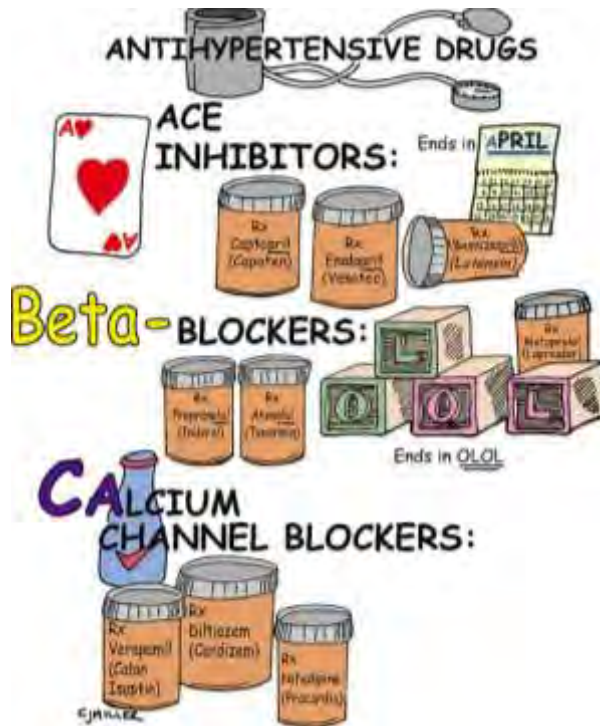
Nipride: gold standard for malignant HTN therapy.

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

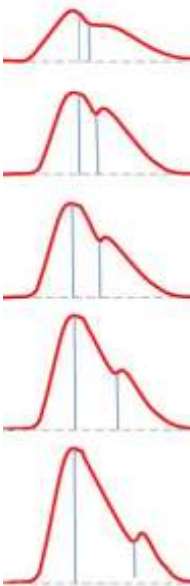
- 0.25 to 0.5 mcg/kg/min IV Titrate every 5 min
 - max 10 mcg/kg/min.
 - titrate to lowest dose.
 - Run at Max no more than 10 min
- Acts in seconds, reversed by stopping (1-5 min)
- Protect bag and lines from light
- Watch for cyanide toxicity after 48 hours or with RI.
 - Blurred vision, tinnitus,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



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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 α & β 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/kg/min IV

IV NTG for hypotension due to cardiac (AMI, failure)

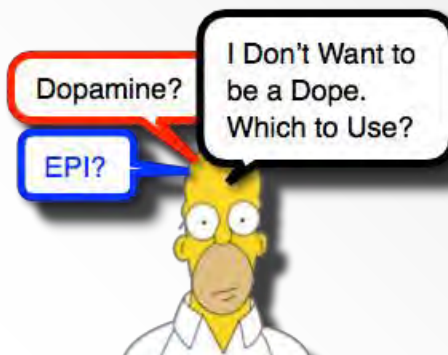
Loop diuretics (torsemide, furosemide, ethacrynic acid) for LV failure, pulmonary edema.

- Watch for volume depletion.

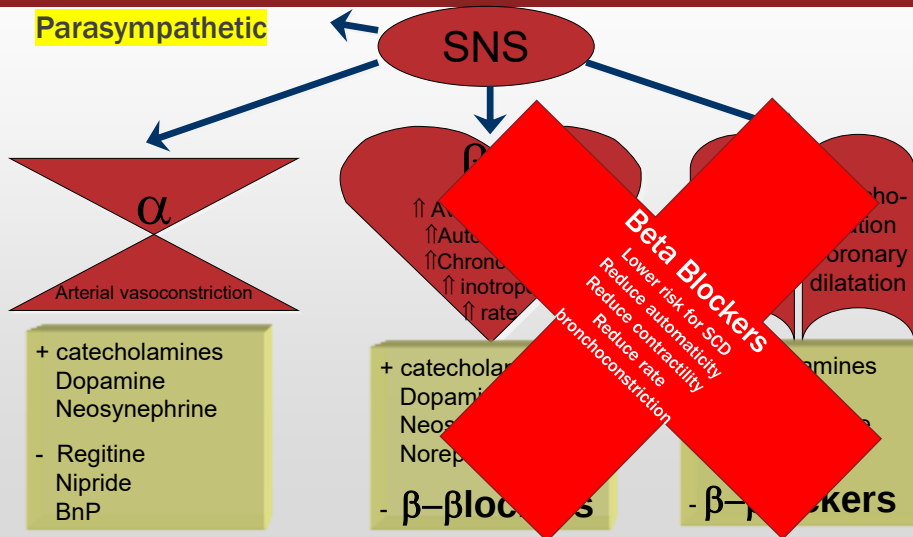
ARKANSAS HEART HOSPITAL[®]

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

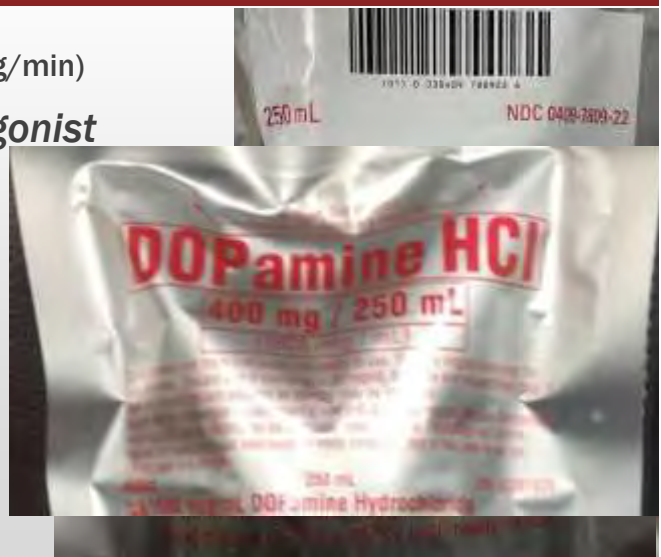
- **Receptors: Strong α -1**
- Increased SVR, significantly
- No effect on PVR
- Increased BP
- No effect on HR
- No effect on systolic or diastolic function
- No effect on myocardial oxygen demand
- Decreased CO and renal perfusion



Effects of Various Vasopressors

Dopamine, low dose (1-5 μ g/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 $\mu\text{g/kg/min}$)

▪ ***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 $\mu\text{g/kg/min}$)

▪ ***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	• Yes	• No
Cardiac failure	• No	• Yes
Ischemic LVF	• No	• Yes
Renal failure	• 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. www.heartfailurematters.com		

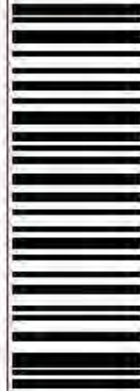
Effects of Various Vasopressors

NDC 0517-0410-10
VASOPRESSIN
 INJECTION, USP
Synthetic
20 Units/mL (200 Units/10 mL)

10 mL
 MULTIPLE DOSE VIAL
 FOR IM OR SC USE ONLY

Rx Only
 AMERICAN REGENT, INC.
 SHIRLEY, NY 11967

Each mL contains:
 Vasopressin 20 units,
 Sodium Chloride 9 mg,
 Chlorobutanol 0.5% (as a
 preservative), Water for
 Injection q.s. pH adjusted
 with Acetic Acid.
 Store below 23°C (73°F).
 Do not freeze.
 Usual Dosage: See
 Package Insert.
 Rev. 5/11



Lot / Exp.

PRESSOR	RECEPTOR	MAIN EFFECT	MAIN SHOCK USE	OTHER
EPINEPHRINE	$\alpha 1$ $\alpha 2$ $\beta 1$ $\beta 2$	VASOCONSTRICTION IONOTROPY DROMOTROPY CHRONOTROPY	AAA - Anaphylaxis, Asthma, Arrest (Cardiovascular)	DIRTY - Nonselective α and β receptors FLOGs the Heart
NOREPINEPHRINE	$\alpha 1$ $\beta 1$	VASOCONSTRICTION IONOTROPY	SEPTIC SHOCK Most commonly used in US for most kinds of shock	First line for most kinds of shock
PHENYLEPHRINE	$\alpha 1$	VASOCONSTRICTION	Hypotension (often used as "push dose" pressor in anesthesia and ED)	BRADYCARDIA - may cause reflex bradycardia
DOBUTAMINE	$\beta 1$ $\beta 2$	IONOTROPY MILD VASODILATION	CARDIOGENIC SHOCK	Minimal changes in heart rate FLOGs the Heart
DOPAMINE	$\alpha 1$ $\alpha 2$ $\beta 1$ $\beta 2$ 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

- B/P 80/60
- HR 130
- SVR 1800
- Sat 78%
- Lungs Wet
- CVP 35
- PCWP 40
- CO 1.8

Patient B

BP 80/p
HR 135
SVR 600
Sat 78%
Lungs Dry
CVP 4
PCWP 2
CO 7

Patient C

BP 80/60
HR 135
SVR 1800
Sat 78%
Lungs Dry
CVP 4
PCWP 2
CO 3.2

- www.Pie.med.utoronto.ca

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



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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

SIZE OF TREATMENT EFFECT												
CLASS I <i>Benefit >>> Risk</i> Procedure/Treatment SHOULD be performed/ administered	CLASS IIa <i>Benefit >> Risk</i> Additional studies with focused objectives needed IT IS REASONABLE to per- form procedure/administer treatment	CLASS IIb <i>Benefit ≥ Risk</i> Additional studies with broad objectives needed; additional registry data would be helpful Procedure/Treatment MAY BE CONSIDERED	CLASS III No Benefit or CLASS III Harm <table><tr><th></th><th>Procedure/ Test</th><th>Treatment</th></tr><tr><td>COR II: No Benefit</td><td>No Harmful</td><td>No Proven Benefit</td></tr><tr><td>COR III: Harm</td><td>Excess Harm w/o Benefit or Harmful</td><td>Harmful to Patients</td></tr></table>		Procedure/ Test	Treatment	COR II: No Benefit	No Harmful	No Proven Benefit	COR III: Harm	Excess Harm w/o Benefit or Harmful	Harmful to Patients
	Procedure/ Test	Treatment										
COR II: No Benefit	No Harmful	No Proven Benefit										
COR III: Harm	Excess Harm w/o Benefit or Harmful	Harmful to Patients										

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

ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT	LEVEL A
	Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses
	LEVEL B
	Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies
	LEVEL C
	Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care

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 of the disease and is an independent predictor of mortality.

AHA/ACCF HEART FAILURE STAGES A-D

5 yr survival A 97% Stage A	B-96% Stage B	C-75% Stage C	D-20% Stage D
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

Heart Failure *reduced* ejection fraction (HFrEF)
Heart Failure *preserved* ejection fraction (HFpEF)

Classification	EF (%)
I. Heart failure with reduced ejection fraction (HFrEF)	≤40
II. Heart failure with preserved ejection fraction (HFpEF)	≥50
a. HFpEF, borderline	41 to 49
b. HFpEF, improved	>40

HFrEF

Definition: the clinical diagnosis of HF and an EF of $\leq 40\%$.

Prevalence: 50% of all HF cases with $\frac{1}{2}$ having variable degrees of LV enlargement.

- Commonly have elements of diastolic dysfunction



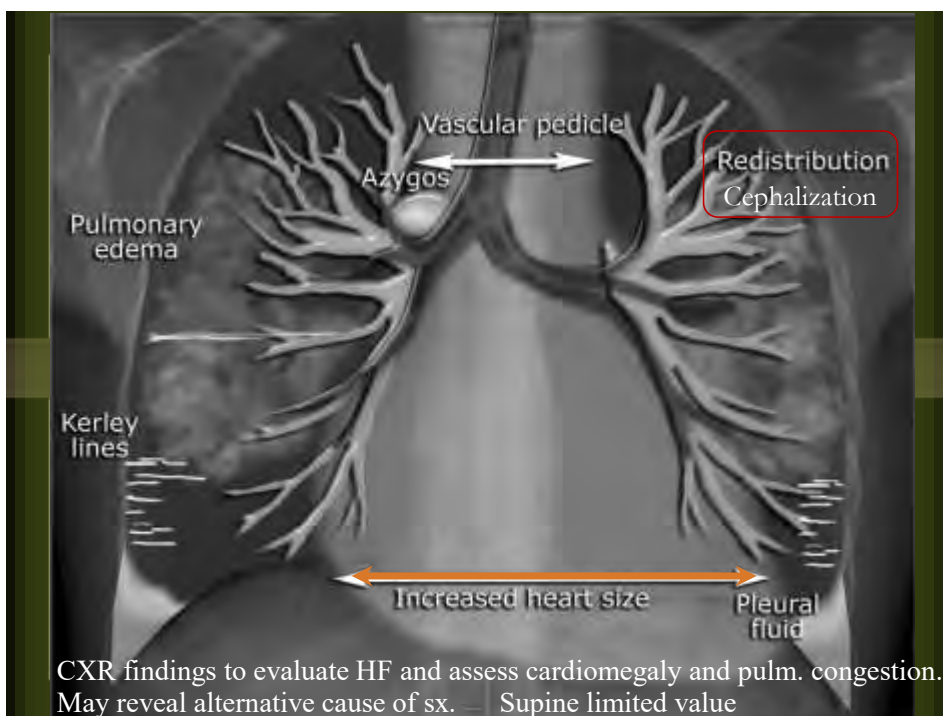
HFrEF

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

Keys to Diagnosing Broken Heart Syndrome (Takotsubo Cardiomyopathy)

Mark P. Brady, PA-C
February 17, 2015

02 of 17






17

Interstitial Edema



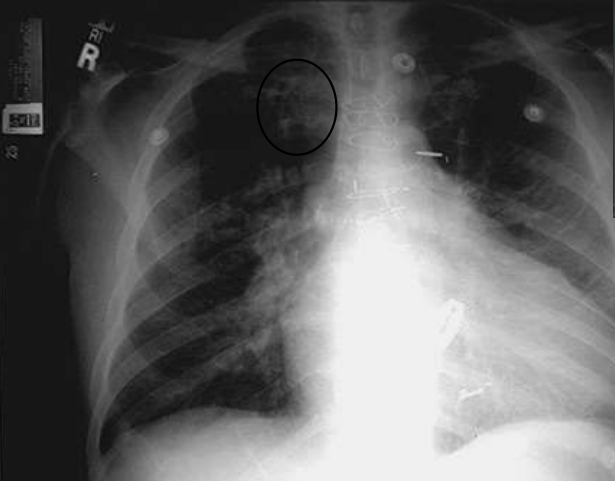

25-30

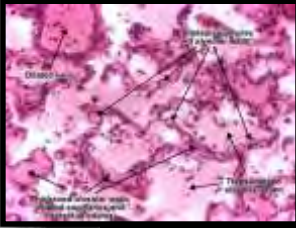
- Kerley's A lines from hila toward periphery - lymphs
- Kerley's B lines in periphery extending to pleura - interlobular septal thickening
- Peribronchial cuffing - thick bronchial walls

58

58

Alveolar Edema



- 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%)

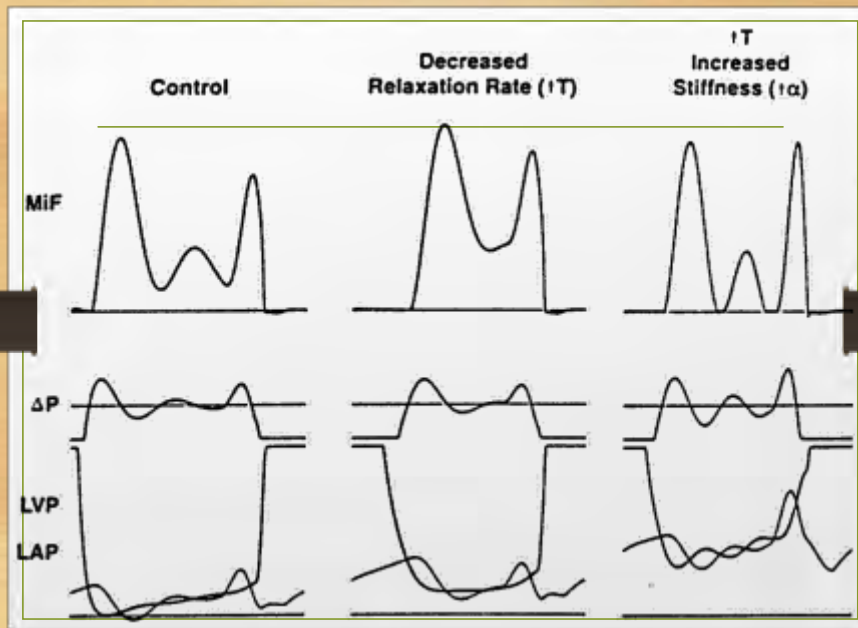
Concentrically remodeled left ventricle 2° arterial hypertension, obesity, and diabetes, with no evidence of CAD ([Barry A. Borlaug, 2011](#))

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



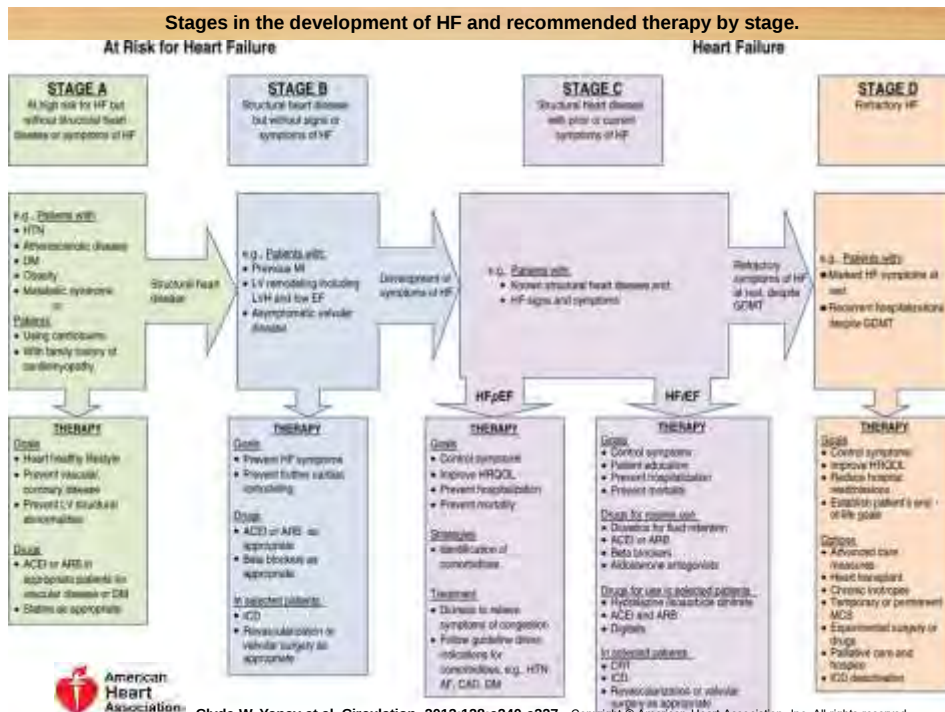
Patient Characteristics

Older women with a hx of:

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



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



Clyde W. Yancy et al. Circulation. 2013;128:e240-e327 Copyright © American Heart Association, Inc. All rights reserved.

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 (<i>trig</i> < 150, <i>tot chol</i> < 200, <i>LDL</i> < 100)



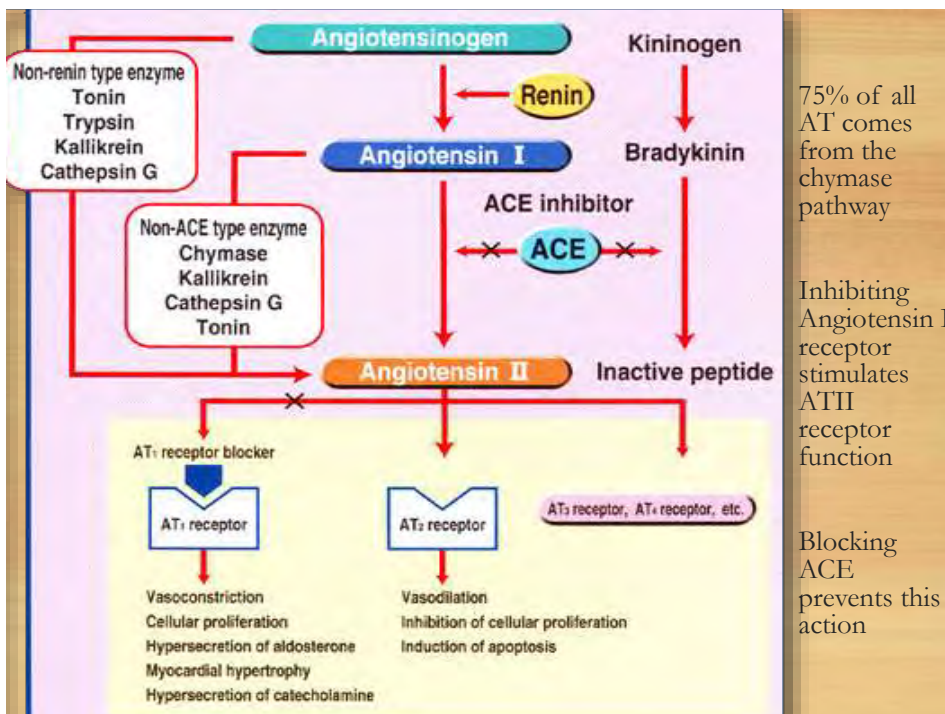
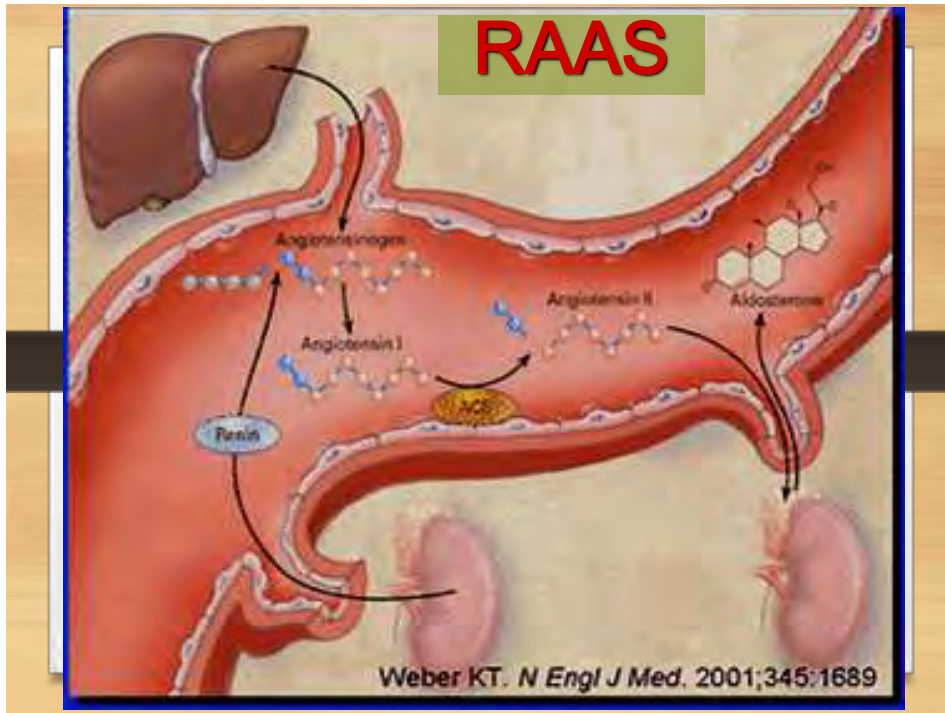
Left Ventricular Remodeling

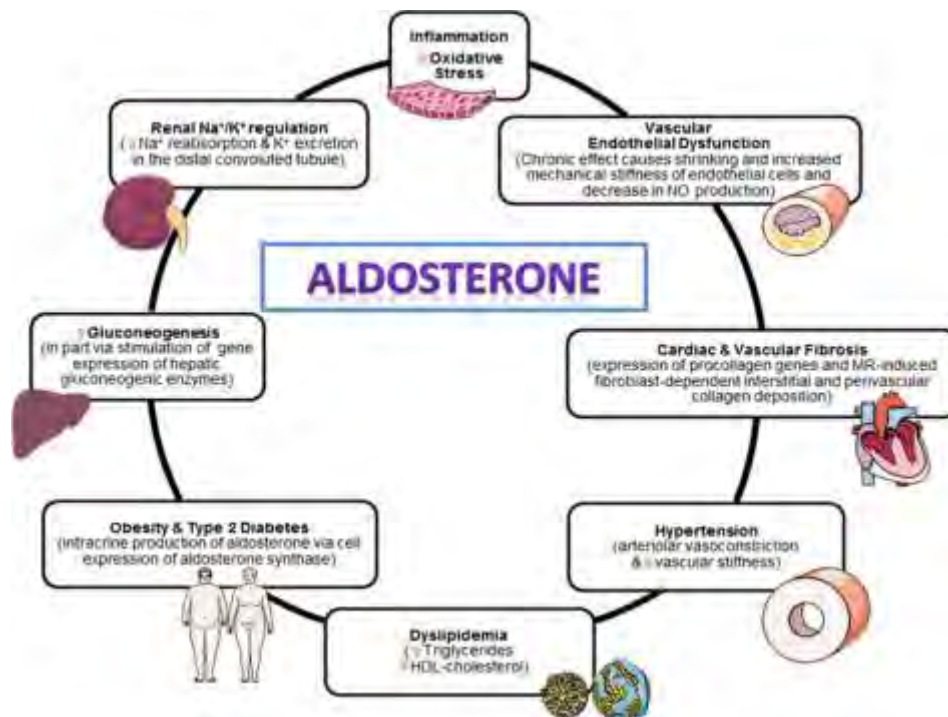
Relatively normal chamber size & geometry

Remodeled ventricle

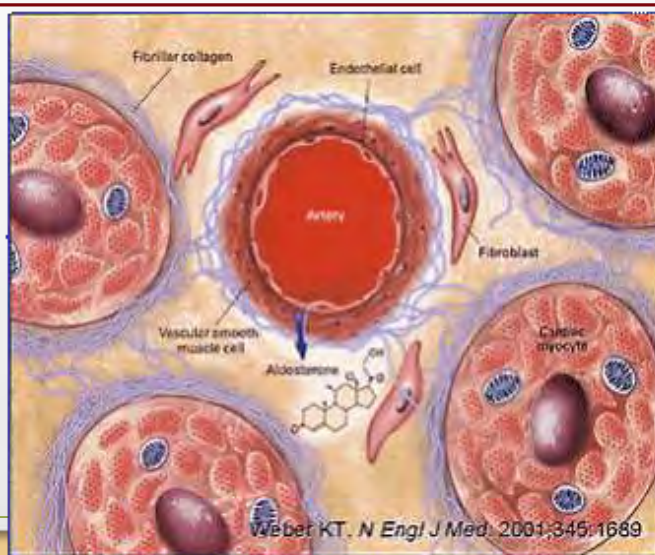
Angio II synthesis of fibroblasts
 Aldosterone collagen synthesis
 Norepinephrine directly toxic to myocytes

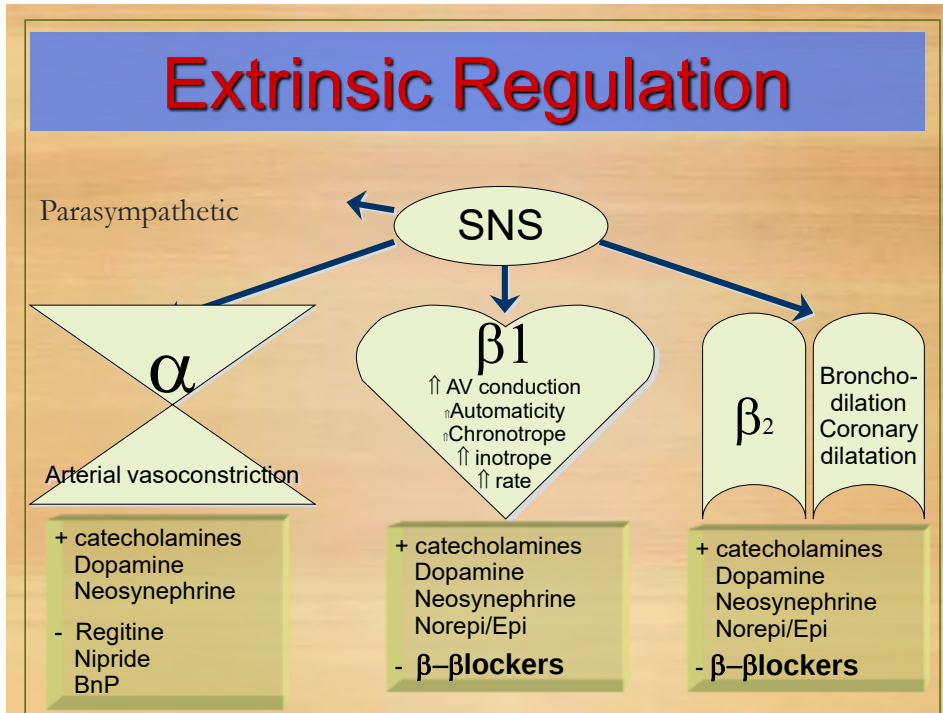
Cohn JN, et al. *J Am Coll Cardiol.* 2000;35:569-582.





Extra-Adrenal Production of Aldosterone





Neurohormones and Heart Failure

Neurohormones that are Beneficial in Heart Failure

- ANP** – Atrial natriuretic peptide
- BNP** – B-type natriuretic peptide
- CNP** – C-type natriuretic peptide




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 BBIs 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 BBIs Aldosterone antagonists Selected patients: Hydralazine/isosorbide Digitalis CRT - ICD Revascularization/valve OR

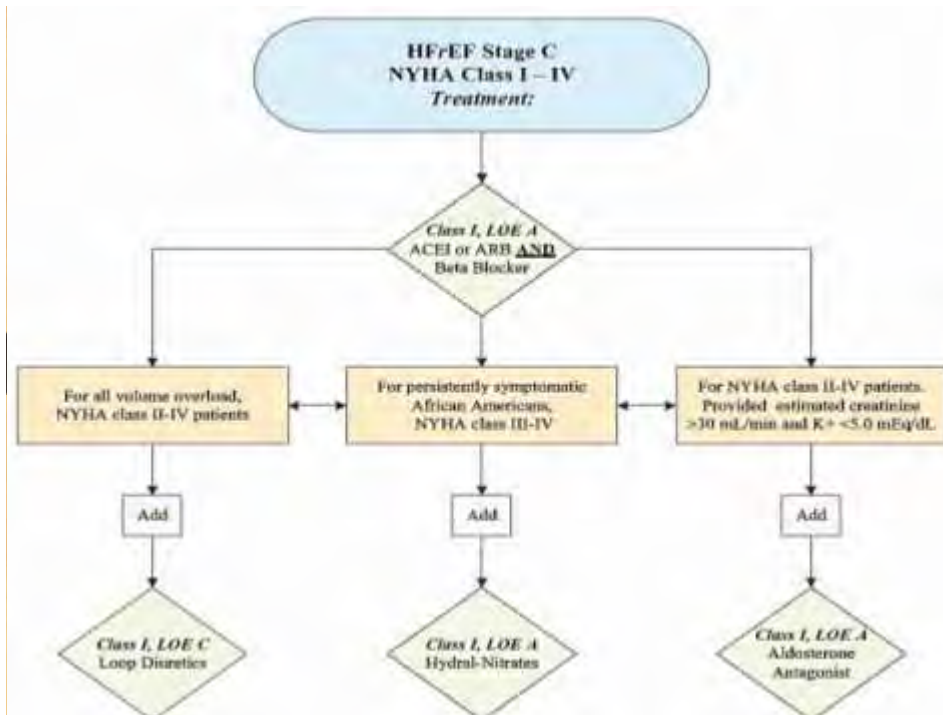


Pharmacologic Tx. Of HFrEF

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 <i>Avoid ACEi, ARB and Ald blkr 2^{to} ↑K+</i>
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 H ₂ O retention and blunt effects of diuretics

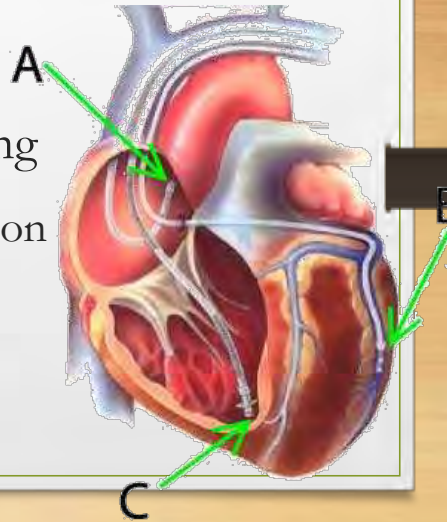
Pharmacologic tx: HFpEF

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



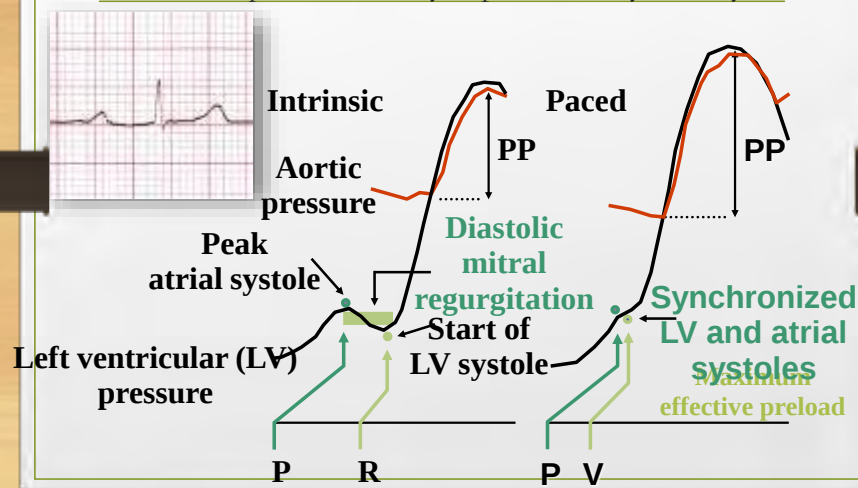
Non-Pharmacologic Therapies

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

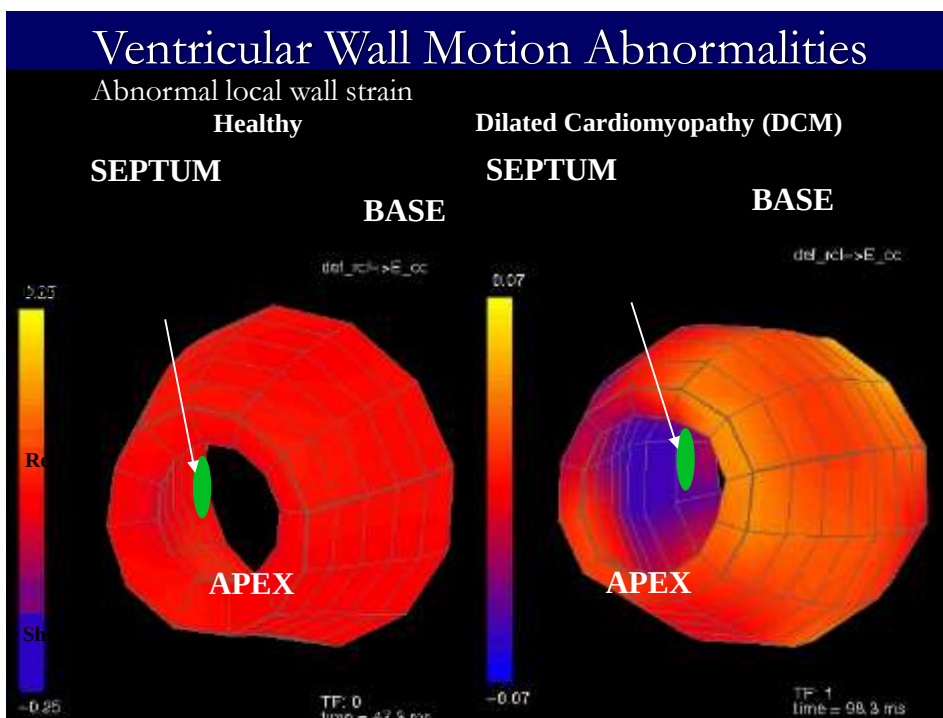


AV Dysynchrony

Mechanism I—optimal AV delay improves AV synchrony

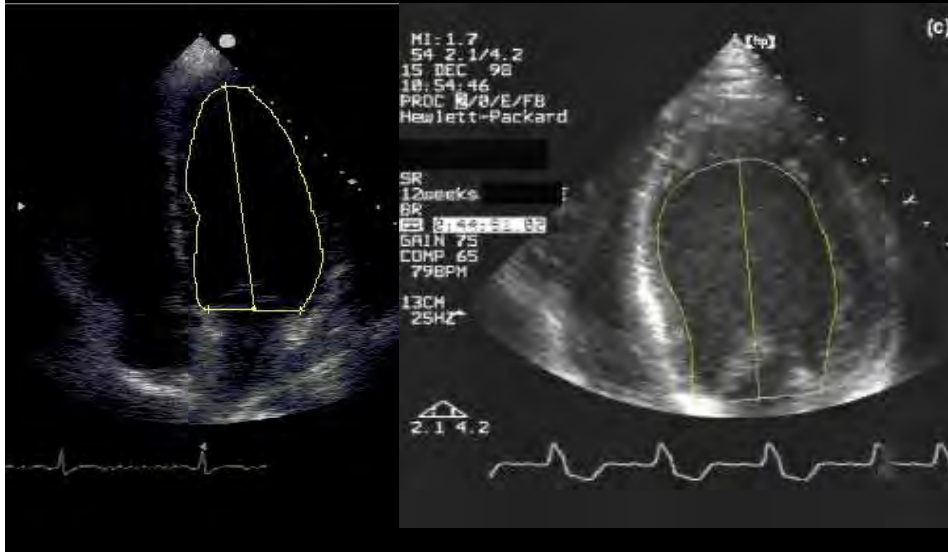


Resynchronization



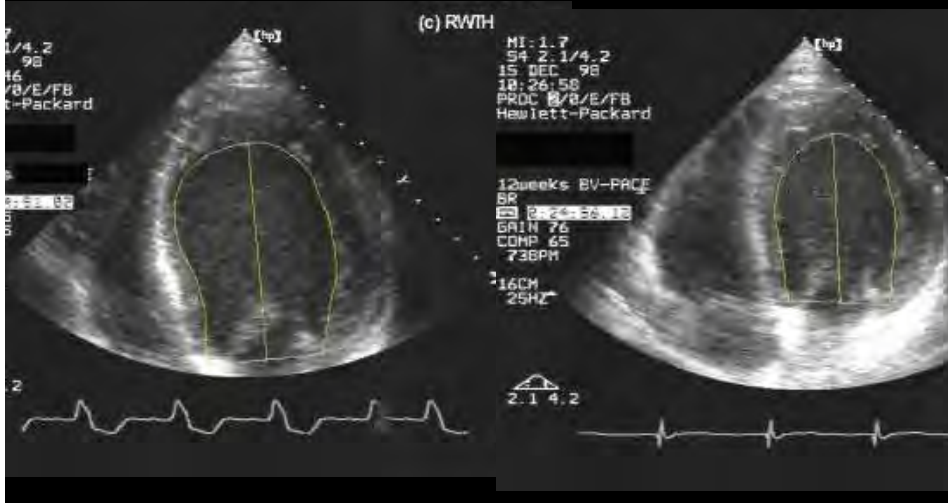
Ventricular Dysynchrony by Echo

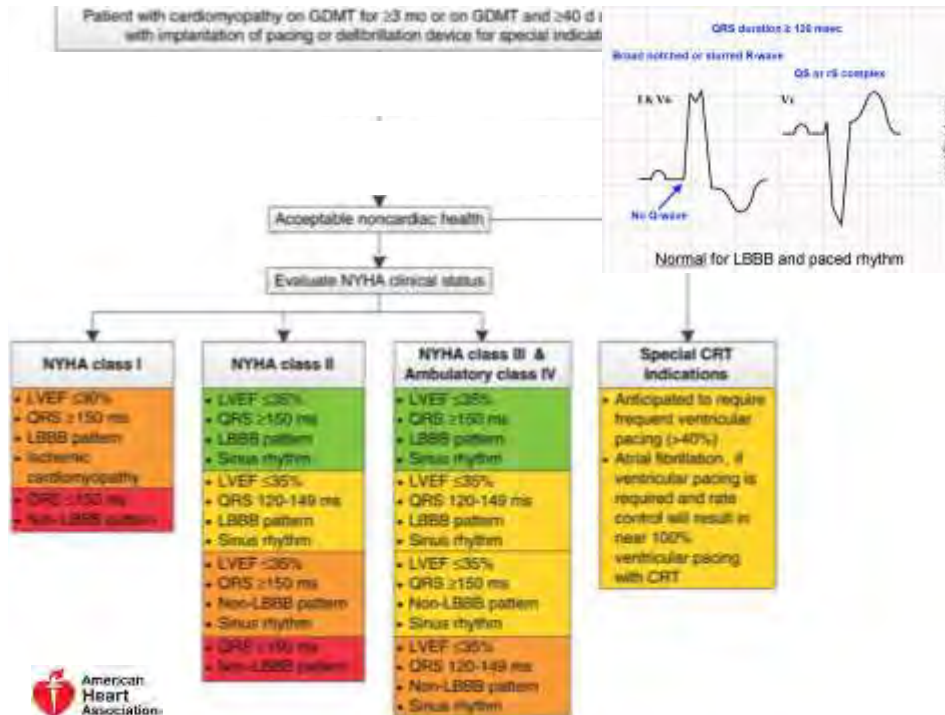
Abnormal wall motion



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

		CONGESTION AT REST		S&S OF CONGESTION
		No	Yes	
LOW PERFUSION AT REST	No	Warm & Dry PCWP normal CI normal	Warm & Wet PCWP elevated CI normal	
	Yes	Cold & Dry PCWP low/nml CI decreased	Cold & Wet PCWP elevated CI decreased	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	IIIb
Routine intravenous use, either continuous or intermittent, is potentially harmful in stage D HF	III Harm

Recent Literature



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.

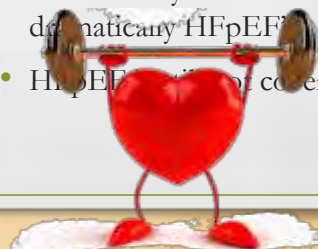
Lithium: Increased risk of lithium toxicity.



Cardiac Rehab

Aug 20, 2015 Louisville Kentucky School of Medicine – Dr. Greg Fonarow

- **2005-2014 Rehab was self-pay**
 - 36 sessions over 3 months at 65-120/session (\$2340-4320)
 - 10% of HF patients were referred for rehab
- **Feb 2014 CMS extended coverage to cardiac rehab**
 - Patients with symptomatic HF and reduced EF should receive cardiac rehab to increase functional status
- “There is substantial evidence that Cardiac Rehab decreases symptoms, hospitalizations and mortality in HFrEF and similarly but less dramatically HFpEF
- HFpEF patients not covered



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

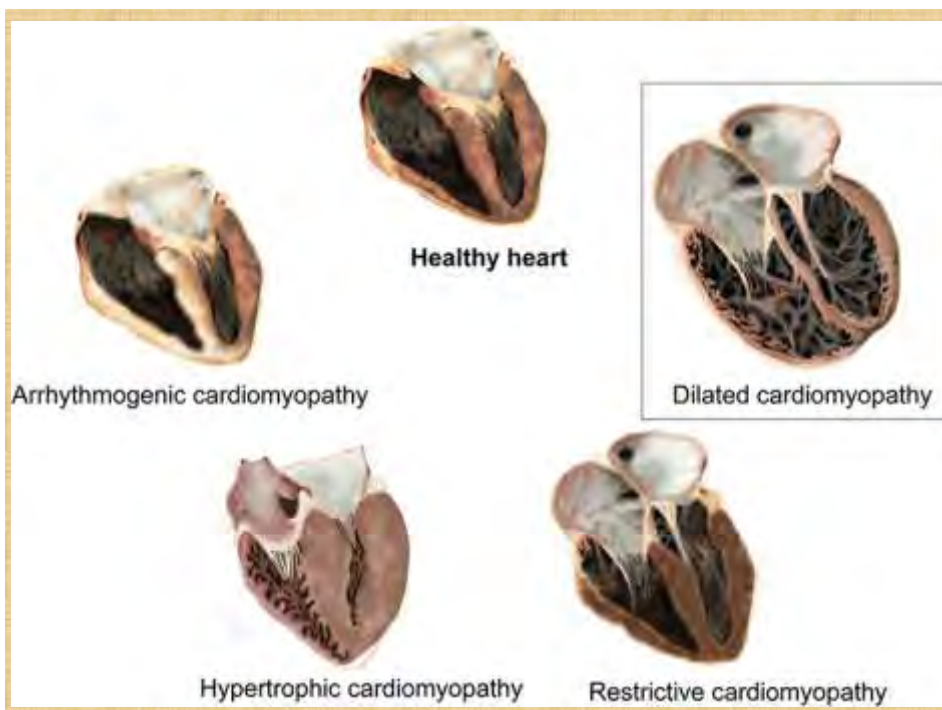
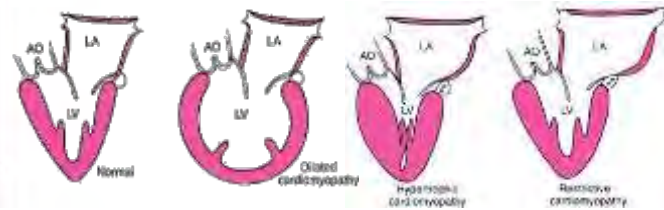


Types

Definition:

A Heterogeneous 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
- Löffler'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 stroke 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



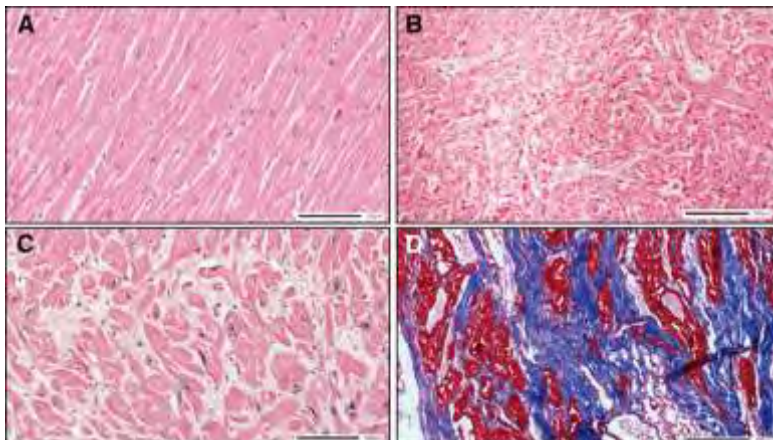
- 1 of every 500
- Primary genetic cardiomyopathy Effects men and women equally
- Hypertrophy of myocardial muscle mass in the absence of increased ventricular afterload
- Associated with decreased ventricular filling (diastolic dysf.) and decreased CO
- Most common cause of sudden death in young adults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

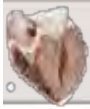


Hypertrophic Cardiomyopathy



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



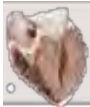
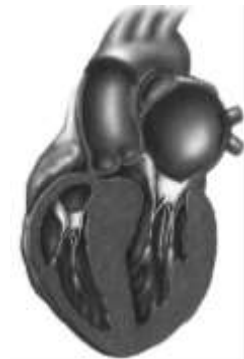


Hypertrophic Cardiomyopathy



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

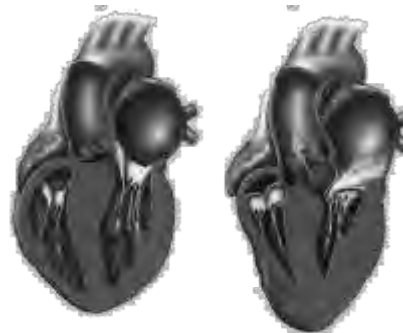
May involve entire septum or only a portion of the septum



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



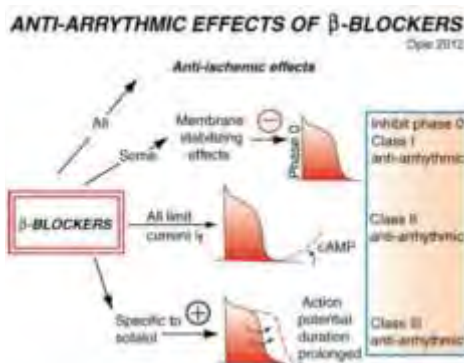
Hypertrophic Cardiomyopathy Presentation

- 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

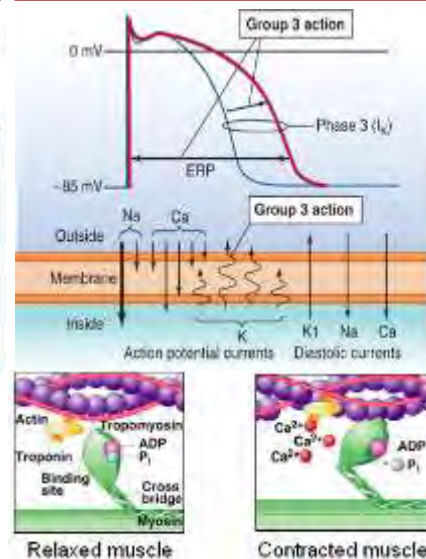


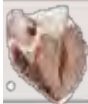
Hypertrophic Cardiomyopathy Treatment

Beta Blockers



Calcium Channel Blockers

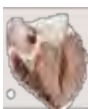




Treatment



- **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

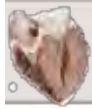



Surgical Myectomy



- 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%)

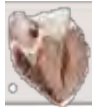




Percutaneous Alcohol Septal Ablation



- 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

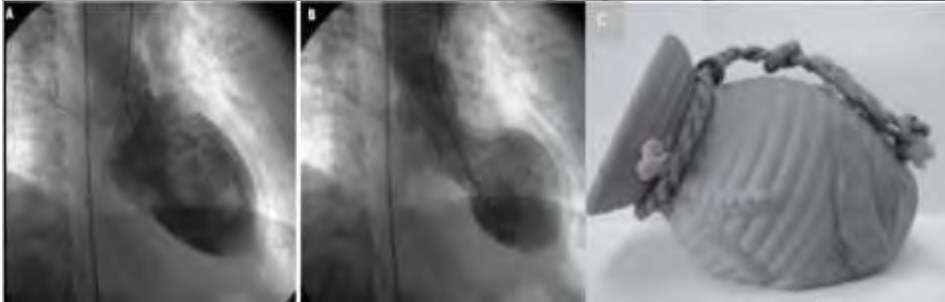
Routine f/u every 12-18 mos.

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

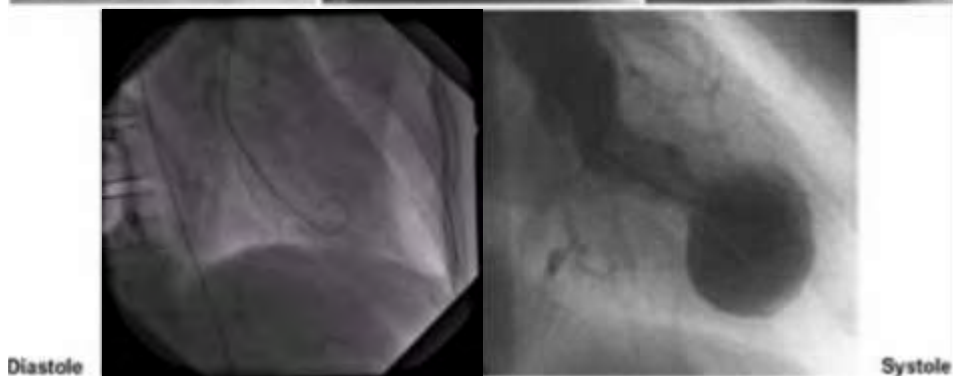
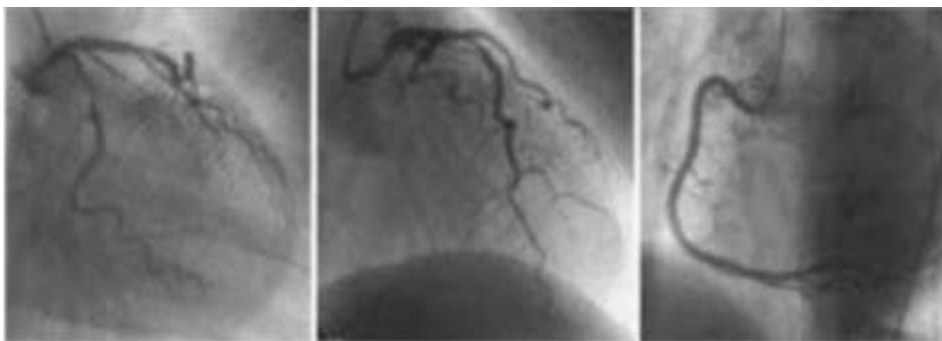
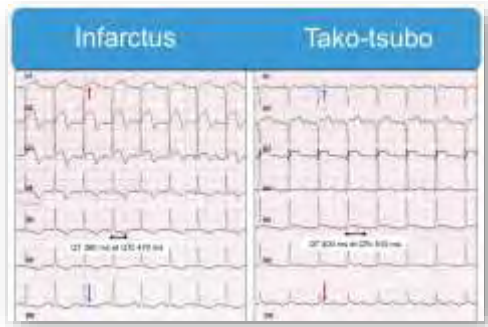
- **Cardiac Cath**

- 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



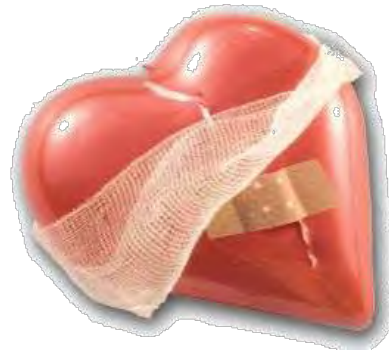
Diastole

Systole

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

