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PCCN CCRN

CCRN/PCCN Review Course
Cardiovascular: Oxygenation

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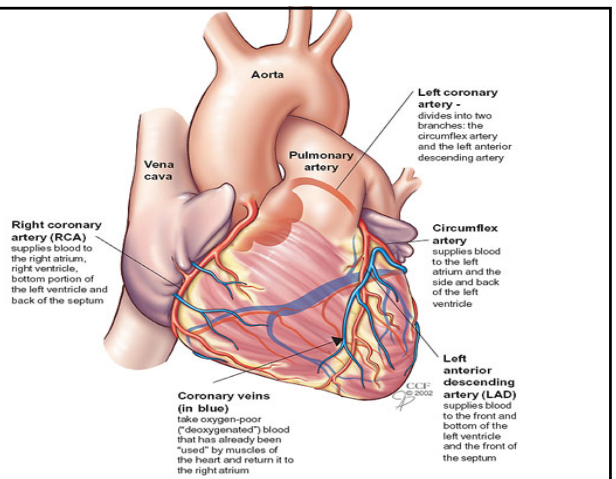
The author has no conflicts of interest to disclose

Determinants of Myocardial Oxygen Supply and Demand

Oxygen Supply	Oxygen Demand
• Coronary Artery Anatomy	• Heart Rate
• Diastolic Pressure	• Afterload
• Diastolic Time	• Preload
• Oxygen Extraction	• Contractility
– Hemoglobin	
– PaO_2	

Myocardial oxygen consumption



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Atherosclerosis

- Affects mainly large and medium-sized arteries
- Can begin in childhood and is progressive
- Stable plaque may narrow the lumen of the artery causing chronic ischemia (stable angina)
- Unstable plaque can rupture → thrombus that partially or completely occludes the artery → acute ischemia (unstable angina)

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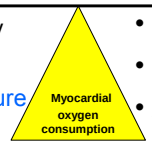


Diastolic Pressure

- The myocardium receives its blood supply during diastole
- Atherosclerosis (coronary artery narrowing) causes a significant drop in diastolic pressure as blood is forced through a narrow lumen

- Diastolic pressure
  - ↑ pressure, ↑ flow
  - ↓ pressure, ↓ flow

Diastolic Time

- Diastole determines the duration of coronary blood flow
- Fast heart rate means less time for coronary filling

- Diastolic Time
  - ↑ time, ↑ filling
  - ↓ time, ↓filling

Practice

Diastole comprises what percentage of the cardiac cycle?

- A. Half
- B. Two-thirds
- C. One-fourth
- D. On-third

Diastole comprises what percentage of the cardiac cycle?

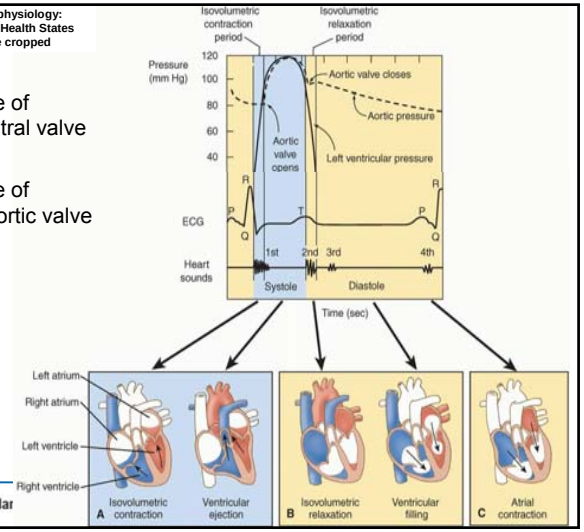
- **B. Two-thirds**

Diastole is a very important part of the cardiac cycle. It is the period during which 1) the heart chambers fill in preparation for systole and 2) the coronary arteries fill with freshly oxygenated blood to feed the heart muscle itself

Porth, Matfin, Pathophysiology: Concepts of Altered Health States 8th ed. 2009, picture cropped

S1 – closure of tricuspid, mitral valve

S2 – closure of pulmonic, aortic valve



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Oxygen Extraction

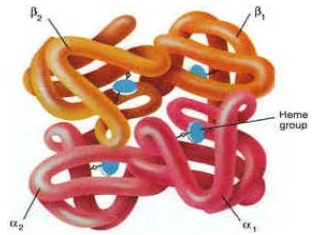
- Oxygen is transported through blood in two ways
  - –combined with hemoglobin
  - –dissolved in blood

Oxygen Extraction

- Oxygen is transported through blood in two ways
  - –combined with hemoglobin
  - –dissolved in blood
- 97% oxygen bound to hemoglobin (SaO_2)
- 3% oxygen dissolved in arterial blood (PaO_2)
  - – Tip: P = plasma to remember PaO_2 is oxygen dissolved in blood
- Only dissolved oxygen can pass through capillary walls for cellular use

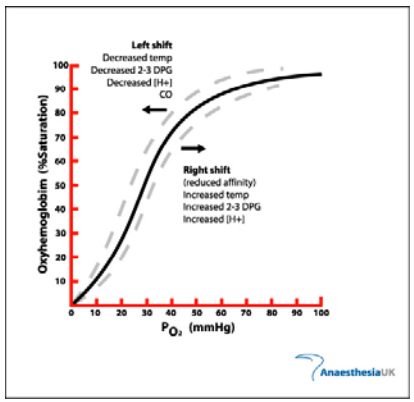
Oxyhemoglobin dissociation curve

- Hemoglobin with bound oxygen is called oxyhemoglobin



http://www.daviddarling.info/encyclopedia/H/hemoglobin.html

Oxyhemoglobin Dissociation Curve



http://www.anaesthesiauk.com/

Oxyhemoglobin Dissociation Curve

- Shift to the right

Hemoglobin releases oxygen more readily to tissues

- – ↓ pH (acidosis)
- – ↑ DPG
- – ↑ temp

- Shift to the left

Hemoglobin binds tightly to oxygen ↑ pH (alkalosis)

- – ↑ pH (alkalosis)
- – ↓ DPG
- – ↓ temp

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Myocardial oxygen consumption

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Heart Rate

- High heart rate
  - –More oxygen consumed at the tissue level
  - –Less oxygen rich blood delivered at the tissue level (less time for ventricular filling)

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Myocardial oxygen consumption

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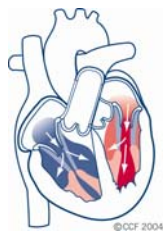
Preload



- Degree of ventricular stretch at end diastole
  - –*Determined by the volume of blood in the ventricle at end diastole*
- With increased preload…
  - –↑ in ventricular wall stress
  - –↑ in myocardial oxygen consumption

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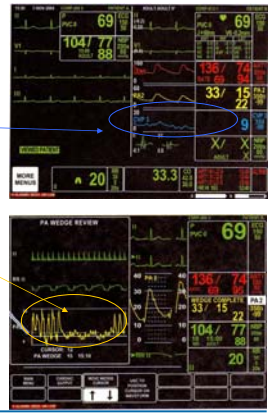
Principles of Oxygen Demand - Preload

- Preload numeric indicators:
  - *CVP (2-6 mmHg)*
  - *PAOP (2 – 12 mm Hg)*
- Preload drugs:
  - *Diuretics, vasodilators (nitroglycerin, morphine, nitroprusside, calcium channel blockers, ACE inhibitors)*



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Myocardial oxygen consumption

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Afterload

- The “load” against which the heart must contract to eject blood into the aorta
- When afterload is high…
  - –↑ in ventricular wall stress
  - –↑ in myocardial oxygen consumption

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Afterload

- Numeric indicators
  - *SVR (800-1200 dynes/sec/cm^5)*
  - *PVR (< 250 dynes/sec/cm^5)*
  - *Systolic blood pressure (indirect indicator)*
- Afterload reducing drugs: vasodilators
  - *SVR – systemic vasodilators (nitroprusside, hydralazine, ACE inhibitors, calcium channel blockers, alpha blockers)*
  - *PVR – systemic vasodilators (prostacyclin)*
- Intra-aortic balloon pump

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Practice

Increased afterload would be seen with

- A. Polycythemia
- B. Aortic insufficiency
- C. Hypovolemia
- D. Sepsis

Increased afterload would be seen with

- **A. Polycythemia**

Hypovolemia and sepsis decrease afterload, as does aortic insufficiency. Aortic stenosis increases afterload, as do peripheral vasoconstriction and hypertension**.**

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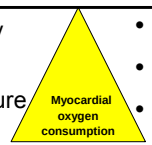

Contractility

- Strength of myocardial muscle fiber shortening during systole
  - – Influenced by preload
  - – Greater muscle fiber stretch → greater recoil
- When contractility is high
  - – ↑ in wall stress
  - – ↑ in myocardial oxygen consumption

Contractility

- Contractility indicators
  - Evidence of sympathetic nervous system stimulation
- Contractility numeric indicators:
  - Systolic B/P
  - Cardiac output
- Contractility enhancing drugs
  - Digoxin, dobutamine, dopamine, milrinone

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Signs of Decreased Cardiac Output

- Decreased level of consciousness
- Hypotension
- Weakness, fatigue, dizziness
- Nausea/vomiting
- Diaphoresis
- Shortness of breath, crackles
- Jugular venous distension
- Decreased peripheral pulses

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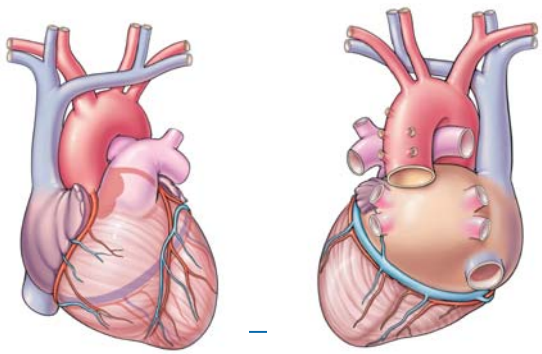
Acute Coronary Syndromes



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Acute Coronary Syndrome

- A reduction or absence of blood flow to a portion of the myocardium resulting in ischemia &/or injury
  - Unstable angina
  - Non-ST elevation MI (reduction in blood flow)
  - ST elevation MI (absence of blood flow)

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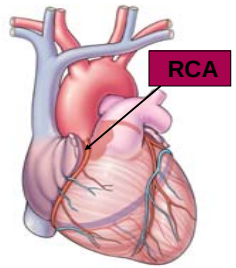
Six Primary **Risk Factors** for Atherosclerosis

- High blood cholesterol level
- Diabetes Mellitus - increases the rate of atherosclerotic progression
- Hypertension
- Tobacco use damages blood vessel walls → atherosclerosis
- Male gender - difference narrows with age (women catch up)
- Family history genetics & acquired health practices (e.g., smoking high-fat diet)

Bolooki & Bajzer, 2009

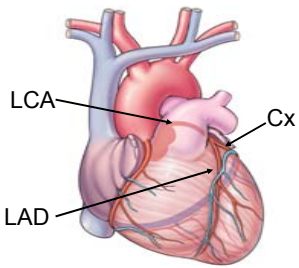
Coronary Artery Anatomy, Right Coronary Artery

- Supplies blood to:
  - Right atrium & ventricle
  - Bottom of both ventricles
  - Back of the septum
- Conduction System
  - SA node in 55% of people
  - AV node, 1st part of Bundle of His in 90% of people



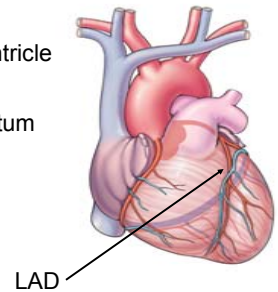
Left Coronary Artery

- Arises off of the ascending aorta and usually divides into two main branches:
  - Left Anterior Descending Artery (LAD)
  - Circumflex Artery (Cx)



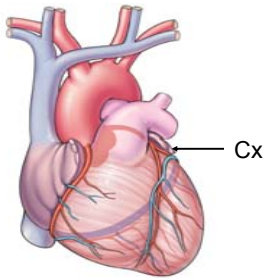
Left Anterior Descending (LAD)

- Supplies blood to:
  - Anterior wall of left ventricle
  - Apex
  - Anterior 2/3 of the septum
    - Bundle of His
    - RBB
    - LBB



Circumflux Artery (Cx)

- Supplies blood to
  - Left atrium
  - Lateral, posterior, inferior left ventricle
- Conduction System Blood Supply
  - SA node in 45% of people
  - AV node and 1st part of Bundle of His in 10% of people



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Coronary Atherosclerosis

- Slow, progressive disease
- Caused by accumulation of plaque within the arterial walls of the coronary arteries
- Plaque size enlarges over time
  - Soft on the inside
  - Hard fibrous cap covering the outside

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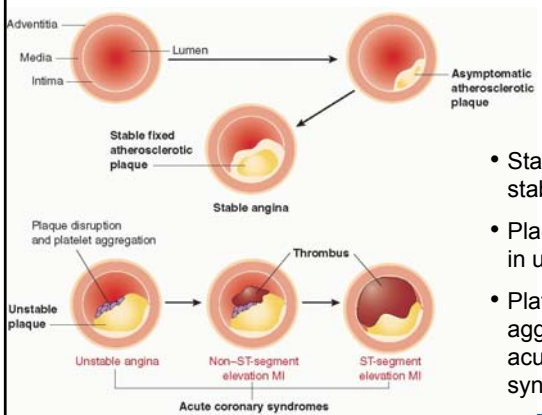
Atherosclerotic plaque



- Stable, fixed in stable angina
- Plaque disruption in unstable angina
- Platelet aggregation in acute coronary syndromes

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Porth & Matfin, 2009

Thrombus Formation

- Plaque ruptures - endothelial cover is torn away
  - Spontaneous
  - Sudden surge of sympathetic activity with ↑ in B/P, HR, force of contraction
- Platelets adhere, thrombus forms
- If thrombus enlarges enough →
  - complete occlusion of the coronary artery
  - loss of blood supply to myocardium dependant on that coronary artery
  - myocardial cell death after 20-40 minutes

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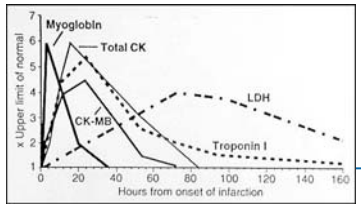
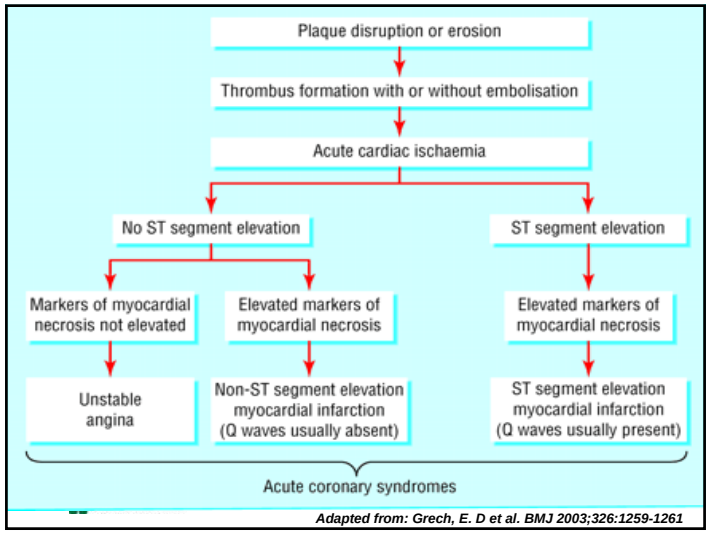
Serial Enzymes

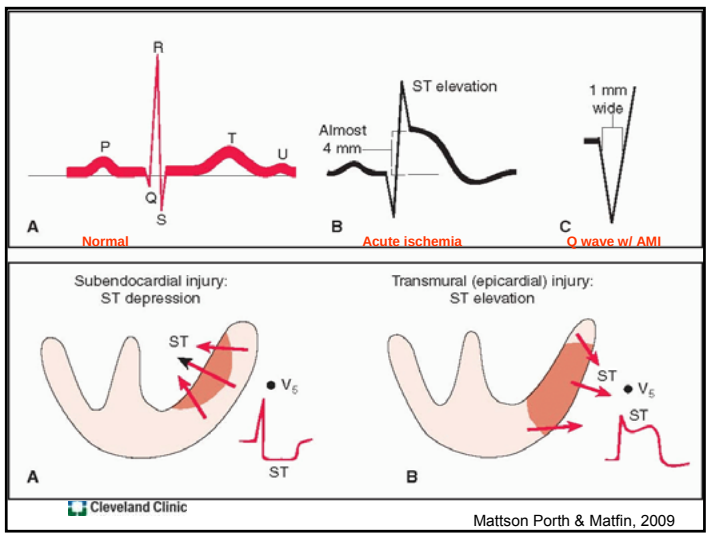
- Myocardial cells contain enzymes and proteins
- With ischemia and cell death, cell membrane loses integrity
  - –enzymes and proteins leak into the bloodstream



www.path.upmc.edu/cases/case178/dx.html



Adapted from: Grech, E. D et al. BMJ 2003;326:1259-1261



Mattson Porth & Matfin, 2009

Four Phases of Acute ST Segment Elevation Myocardial Infarction (STEMI)

Phase	Time After Onset	Pathophysiology
1	0 to 2 hours (first few hours)	▪ Extensive myocardial ischemia within seconds of coronary artery occlusion. ▪ About 30 min. after the interruption of blood flow, irreversible myocardial necrosis occurs in the subendocardium, and myocardial injury spreads toward the epicardium.
2	2 to 24 hours (1st day)	▪ By 3 hours, two-thirds of the myocardial cells within the affected myocardium become necrotic. ▪ By 6 hours, only a small percentage of the cells remain viable. ▪ The evolution of the transmural MI is complete.
3	24 to 72 hours (2nd to 3rd day)	▪ Minimal to no ischemic or injured myocardial cells remain because they have either died or recovered.
4	2 to 8 weeks	▪ Fibrous connective tissue replaces the necrotic tissue.

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Chest Pain

- P = Provocation
- Q = Quality
- R = Region/Radiation
- S = Severity
- T = Timing/Treatment

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Angina

- Temporary imbalance between myocardial oxygen supply and demand causing ischemia
- Ischemia → anaerobic metabolism causing an accumulation of lactic acid which causes chest pain
- No tissue injury
- Angina is associated with a coronary artery occlusion of 75% or more

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Treatment of angina

- Relieve chest pain
  - –Rest
  - –Oxygen
  - –Medication: nitroglycerin, calcium channel blocker or beta blocker, aspirin unless contraindicated
  - –Risk factor reduction/lifestyle modification

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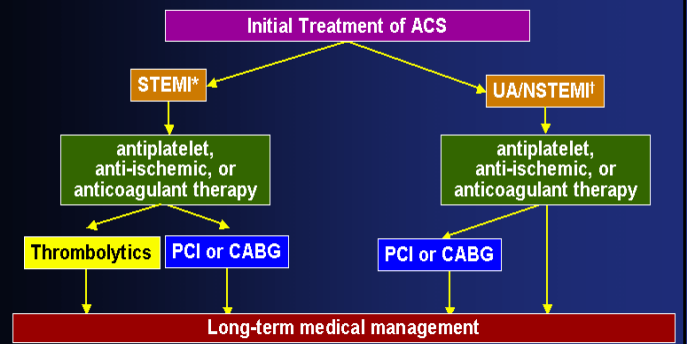
Treatment of Acute Coronary Syndrome



Braunwald E et al. Available at www.acc.org.
Bowen WE, Mckay RG. *N Engl J Med.* 2001;344:1939-1942.

*Also known as Q-wave MI.
†Also known as non–Q-wave MI.

Treatment of ACS

Goals for treatment include:

- Relief of chest pain
- Oxygen
- Prevent platelet aggregation
- Restore blood flow
- Salvage functional myocardium
  - Thrombolytics
  - Percutaneous Coronary Angioplasty (PCI)
  - Coronary Artery Bypass Graft Surgery (CABG)

Treatment of ACS - Immediate

Immediate treatment for chest pain suggestive of ischemia is “MONA”:

- Morphine Sulfate
- Oxygen
- Nitroglycerin
- Aspirin

Treatment of ACS - Nitroglycerin

- NTG SL or spray followed by intravenous drip
- Vasodilates systemic circulation (decreases preload) → decreasing myocardial oxygen demand
- Vasodilates coronary collateral circulation → increase in myocardial oxygen supply
- Avoid in SBP < 90
- Avoid if hypotension would result in serious hemodynamic decompensation such as in right ventricular infarction or severe aortic stenosis

Beta blockers

- Blocks catecholamine effects on beta receptors in the myocardium
- Decrease heart rate, contractility, and systolic blood pressure → decrease in myocardial oxygen consumption and increases duration of diastole
- Do not give to patients with active bronchospasm

Calcium Channel Blockers

- Reduce calcium influx thus inhibits myocardial and vascular smooth muscle contraction
- May slow AV conduction, depress SN impulse formation
- Used to
  1) control ongoing or recurrent ischemia-related symptoms in patients receiving adequate doses of nitrate and beta blocker and
  2) in patients unable to tolerate adequate doses of nitrates and/or beta blockers

Additional Therapies, Mechanical

- Intra-Aortic Balloon Pump (IABP)
  - With recurrent ischemia despite maximal medical management
  - With hemodynamic instability

Parenteral Anticoagulants

- Heparin inactivates thrombin, prevents conversion of fibrinogen to fibrin
- Glycoprotein IIb/IIIa receptor antagonists inhibit platelet aggregation by preventing fibrinogen binding:
  - Abciximab/Reopro
  - Eptifibatide/Integrilin
  - Tirofiban/Aggrastat

Fibrinolytics

- Fibrinolytics for STEMI when PCI not readily available. Dissolves thrombus
  - Alteplase (t-PA), Tenecteplase (TNK), Retaplase (r-PA)
- Goal: “door-to-needle” time 30 minutes or less
- Contraindications: previous hemorrhagic stroke, active internal bleeding, suspected aortic dissection

PCI

Percutaneous coronary intervention (PCI): Used in the management of STEMI

- Goal for STEMI patients: “door-to-balloon” time within 90 minutes
- Early PCI reduces mortality rates, and successfully restores coronary blood flow in 90-95% of AMI patients

Treatment AMI

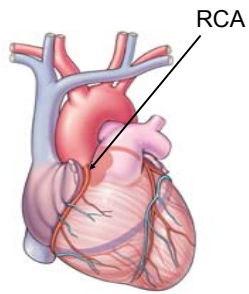
Emergent coronary artery bypass grafting (CABG): Used in failed PCI patients that are hemodynamically unstable

- Can limit myocardial injury and cell death if performed within 2-3 hours of symptom onset

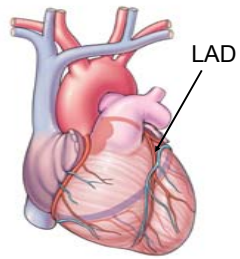
AMI and ECG

Inferior Wall MI (ECG leads II, III, aVF)

- Bradycardias
- SA node dysfunction
- AV blocks
- N/V
- RV Infarction
  - –V4R
  - –Hypotension



Anterior Wall MI (ECG leads V1-V4)



- AV blocks
- R & LBBB
- LV dysfunction
- LV failure
- Pulmonary edema
- Cardiogenic shock
- High mortality

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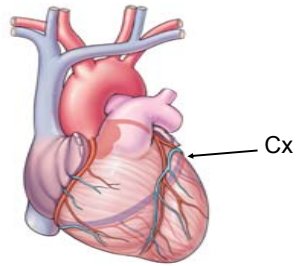
Lateral Wall MI (ECG leads V5, V6, I, aVL)

- Bradydysrhythmias
- AV Blocks
- LV dysfunction (HF symptoms)



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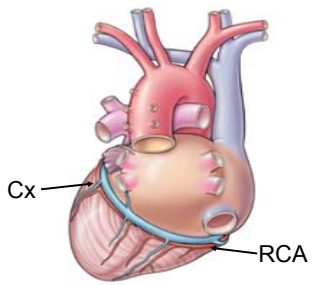
Posterior Wall MI
(reciprocal changes ECG leads V1 – V2 ST depression)



- Usually an extension of Inferior or Lateral MI (RCA or Cx occlusion)
- Bradydysrhythmias
- May see RV involvement and papillary muscle dysfunction

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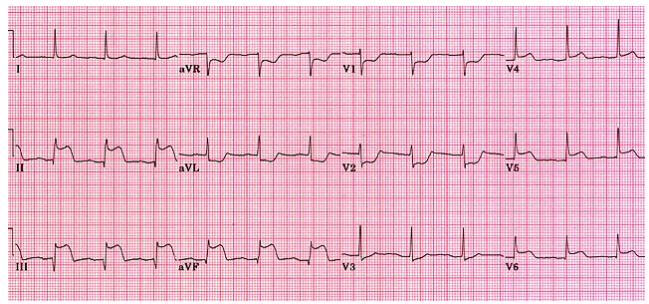
Interpretation of 12 Lead ECG



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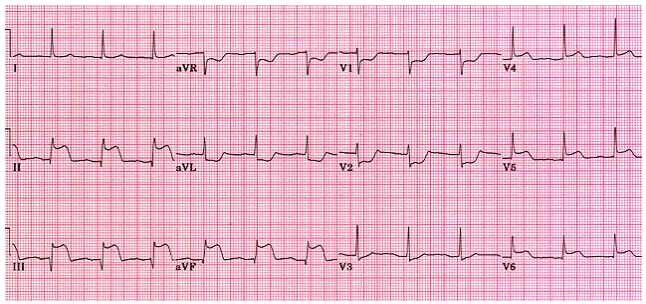
Interpretation of 12 Lead ECG

ST ↑ II, III, aVF = Inferior Wall MI, RCA

ST ↓ V1 and V2 = Posterior Wall MI, RCA or Circumflex



Practice

Your patient has had an anteroseptal MI. Where do you expect to see changes on the 12-lead ECG?

- A. V_1, V_2, V_3, V_4
- B. V_2, V_3, V_4, V_5, V_6
- C. V_1, V_2, II, III, AVF
- D. V_1, V_2, I, AVL

Your patient has had an anteroseptal MI. Where do you expect to see changes on the 12-lead ECG?

- **A. V_1, V_2, V_3, V_4**
- B. V_2, V_3, V_4, V_5, V_6
- C. V_1, V_2, II, III, AVF
- D. V_1, V_2, I, AVR
- V1 and V2 lie over the septum and V3 and V4 are over the anterior aspect of the LV

Practice

- In an inferior wall MI, the leads that will most directly reflect the injury are
- A. I, II, and aVF
- B. I, aVL,
- C. V1-V2
- D. V5-V6

Practice

- In an inferior wall MI, the leads that will most directly reflect the injury are
- **A. I, II, and aVF**
- B. I, aVL,
- C. V1-V2
- D. V5-V6
- Lead I and aVL will show damage to the higher areas of the lateral wall. Leads V1 and V2 will show septal wall damage. Leads V5 and V6 will show damage to the apical area.

Discharge Education

- Aspirin
- Beta Blocker
- ACE Inhibitor w/EF < 40%
- Lipid-lowering medication
- Risk factor modification
- Diet
- Smoking cessation
- Activity and exercise
- Cardiac rehabilitation

Practice

- Which of the following leads is best for monitoring for a RBBB?
- A. Lead II
- B. Lead I
- C. Lead V_1
- D. Lead V_6

- Which of the following leads is best for monitoring for a RBBB?
- **C. Lead V_1**

V1 is located at the 4th intercostal space right sternal boarder which lies directly over the right ventricle.

Practice

Which of the following may predispose an individual to ventricular fibrillation?

- A. Hypernatremia and hypomagnesemia
- B. Hypophosphatemia and hyperchloremia
- C. Hypermagnesemia and hyponatremia
- D. Hyperkalemia and hypocalcemia

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- C. Hypermagnesemia and hyponatremia
- **D. Hyperkalemia and hypocalcemia**

Practice

- A patient with an IWMI and sinus bradycardia suddenly develops atrial fibrillation. Which of the following should the nurse anticipate administering?
- A. Amiodarone
- B. Diltiazem
- C. Digoxin
- D. Synchronized cardioversion

- D. **Synchronized cardioversion**
- Inferior wall MI decreases perfusion to the right atrium and may result in SA node ischemia and atrial dysrhythmias. AF decreases CO and may further compromise coronary perfusion. All of the above meds are possible antiarrhythmic agents that could be used to treat AF, they take time to work.

Practice

- Occlusion of the LAD is associated with which of the following complications?
- A. Papillary muscle dysfunction
- B. LV aneurysm
- C. Bradycardia
- D. Pulmonary edema

- D. **Pulmonary edema**
- LAD supplies the LV and septum, including the bundle of His and bundle branches. LAD occlusion may cause LV failure → HF, pulmonary edema, and heart block.
- Bradycardia assoc w/occlusion of the RCA which supplies the SA and AV nodes.
- LV aneurysm is assoc w/ Cx occlusion and post MI.
- MR and papillary muscle dysfunction are associated with RCA and Cx occlusions

Practice

When attempting to auscultate the aortic area, the location of the stethoscope should be

- A. At the 2nd intercostal space, left sternal boarder
- B. Over the apical area
- C. At the 2nd intercostal space, right sternal border
- D. At the 5th intercostal space, left sternal boarder

Practice

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- A. At the 2nd intercostal space, left sternal boarder
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Practice

A patient in cardiogenic shock is in the ICU on vasopressor and IABP support. Which of the following assessment findings most reliably indicates that the current therapy is appropriate?

- A. HR 100 BPM, MAP 66 mm HG, SVR 1200 dynes/sec/cm^{-5}
- B. HR 117 BPM, MAP 53 mm HG, SVR 1900 dynes/sec/cm^{-5}
- C. HR 110 BPM, MAP 70 mm HG, SVR 2800 dynes/sec/cm^{-5}
- C. HR 117 BPM, MAP 53 mm HG, SVR 2400 dynes/sec/cm^{-5}

- **A. HR 100 BPM, MAP 66 mm HG, SVR 1200 dynes/sec/cm^{-5}**

- Theraputic goals for the pt. in cardiogenic shock includes achieving a MAP sufficient to ensure central and peripheral perfusion. MAP = 60 will provide cerebral perfusion.
- High SVR increases LV workload and can decrease end organ perfusion.
- HR nearing normal further indicates that myocardial work has decreased and oxygenation has potentially improved.

Practice

A patient with a recent MI suddenly develops a loud systolic murmur. The *most likely* cause is which of the following?

- A. Pulmonary embolism
- B. Congestive heart failure
- C. Ruptured papillary muscle
- D. Increased systemic vascular resistance

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- D. Increased systemic vascular resistance

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Heart Failure

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Clinical Nurse Specialist
Cardiac Medical Step-Down
The author have no conflicts of interest to disclose

Basics: Function of the heart

- Move deoxygenated blood
  - From the venous system
  - Through the right heart
  - To the lungs

Right side of heart

- Take freshly oxygenated blood
  - From the lungs
  - Through the left heart
  - To the arterial circulation

Left side of heart

Definition

- Heart Failure
  - Clinical presentation of impaired cardiac function in which one or both ventricles are unable to provide the cardiac output (CO) needed to meet the metabolic demands of the body

Key Concept

- Cardiac Output
  - Amount of blood ejected by a ventricle per minute
  - CO = HR x SV
    - HR x 1 minute
    - SV is the amount of blood ejected with each ventricular contraction
- Adequate CO → metabolic demands met

Sole, Klein, & Moseley (2009)

Cardiac Output

- Adjusts according to needs of the body
  - Sleep – decrease
  - Exercise – increase (cardiac reserve)
    - Normal young adult has a reserve of 300%-400%
    - Marathon runner has amazing cardiac reserve
      - CO may increase 5-6 times over resting level

Cardiac Output

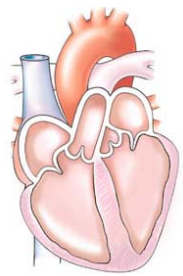
- Patients with HF often require their cardiac reserve at rest!
  - Climbing flight of stairs may exceed their reserve
    - Symptom: shortness of breath

Etiology of HF

- Caused by any cardiac condition that reduces the ability of the heart to pump blood
  - Right sided HF, left sided HF, or both
  - Right sided HF is usually caused by left sided HF
- Systolic dysfunction (poor contractility)
- Diastolic dysfunction (impaired relaxation)

Systolic Heart Failure

- HF with reduced LVEF
  - Systole is dysfunctional
  - ↓ contractility, SV, EF, CO
- EF < 40%
- Primary cause MI

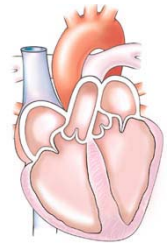


Ejection Fraction

- *Percentage* of blood pumped out of the ventricles with each contraction
- Normal range: 50% - 70%

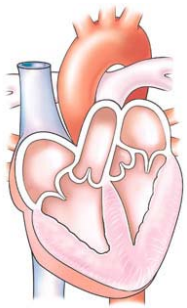
Clinical Presentation of Systolic Dysfunction

- Pulmonary crackles
- S3 gallop (vibration of aortic valve from noncompliant ventricle)
- Peripheral edema
- Cardiomegaly on chest x-ray
- Jugular vein distension (JVD)
- EF < 40%
- Hypotension
- Fatigue



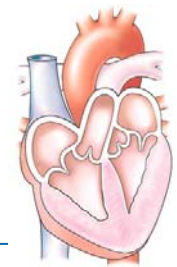
Diastolic Heart Failure

- HF with normal LVEF
  - –Diastole is dysfunctional
  - –Ventricles resist filling
  - –↑ contractility; may see ↓ cardiac output (CO) with exercise; pulmonary and systemic venous congestion
- EF is normal
- Causes include HTN, aortic stenosis

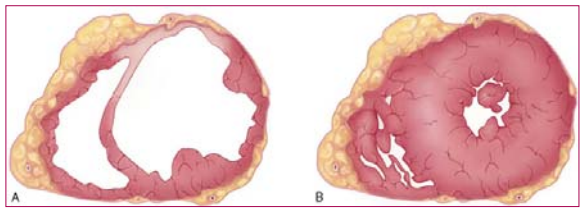


Clinical Presentation of Diastolic Dysfunction

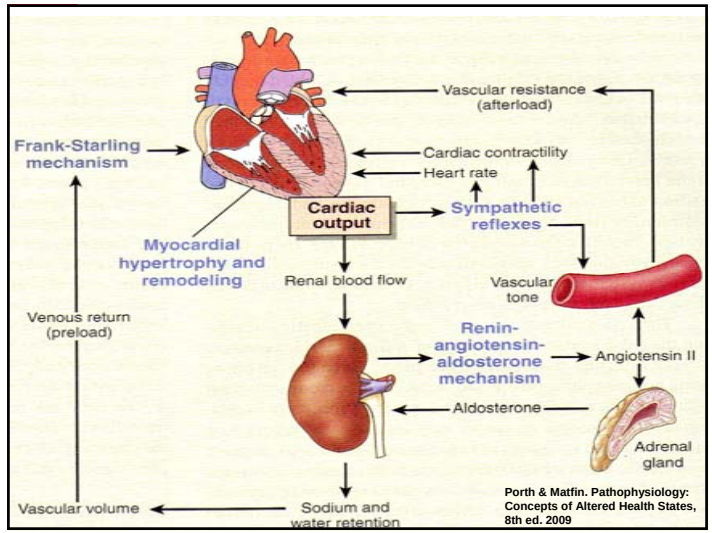
- Hypertension
- S4 (reflection of HTN, atrial contraction forcing blood into noncompliant ventricle)
- Normal jugular vein pressure (JVP)
- Pulmonary crackles
- Peripheral edema
- Normal heart size
- EF normal (>45%)



Heart Failure: Systolic and Diastolic



- A. Systolic HF: Weakened myocardial contraction
- B. Diastolic HF: Impaired myocardial relaxation



Porth & Matfin. Pathophysiology: Concepts of Altered Health States, 8th ed. 2009

Compensatory mechanisms

- ↓ CO →
  - –↓ Renal blood flow: renin-angiotensin-aldosterone system
    - – renin-angiotensin - ↑ vascular tone
    - – aldosterone → sodium and water retention
      - –↑ vascular volume
      - –↑ increased preload
      - –↑ increased myocardial wall stretch
      - –↑ force of contraction (inotropy)
- → Improved CO

Compensatory mechanisms

- ↓ CO →
  - –Sympathetic reflexes activated
    - –↑ HR
    - –↑ contractility } Epinephrine
    - –↑ vascular tone } Norepinephrine
- → Improved CO

Compensatory mechanisms

- Neurohumoral activation
  - –SNS reflexes
  - –Renin-angiotensin-aldosterone system (RAAS)
- Brain Naturetic Peptide (BNP) — Counter-balance the RAAS system
- Myocardial hypertrophy
- Myocardial remodeling — **In weeks to months**

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Compensatory mechanisms

- That's a good thing…for a while
- Over time it causes
  - –hypertrophy
  - –remodeling

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Signs of Decreased Cardiac Output

- Decreased level of consciousness
- Hypotension
- Weakness, fatigue, dizziness
- Nausea/vomiting
- Diaphoresis
- Shortness of breath, crackles
- Jugular venous distension
- Decreased peripheral pulses

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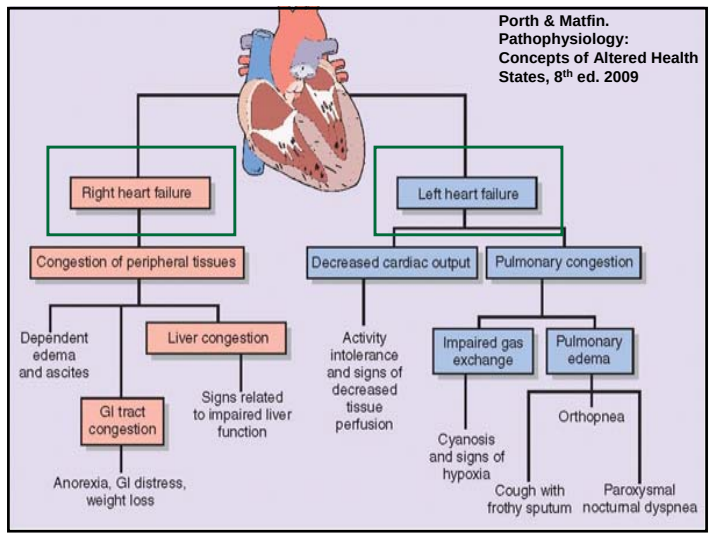


Patient Assessment

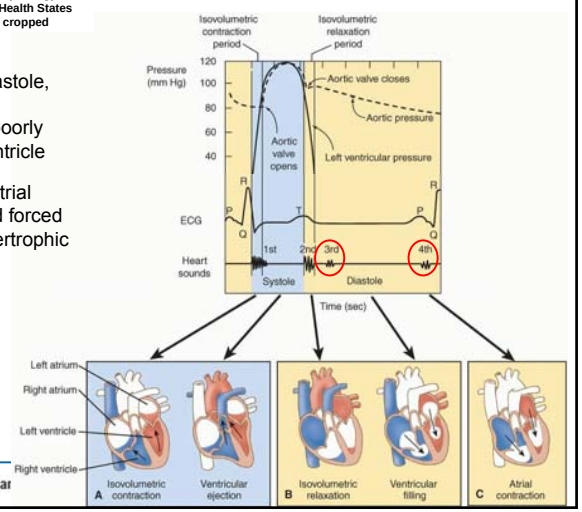
Right Ventricular Dysfunction	Left Ventricular Dysfunction
• Elevated jugular venous pressure	• Dyspnea/orthopnea (cough)
• Liver engorgement (hepatomegaly)	• Paroxysmal nocturnal dyspnea
• Peripheral Edema	• Fatigue/activity intolerance
• Elevated RAP (CVP)	• Diaphoresis
• Loss of appetite, nausea, vomiting	• Slow capillary refill/cyanosis
• Enlarged spleen	• S_3 and S_4
	• Increased heart rate

Sole, Klein, Moseley 2009

Porth, Matfin, Pathophysiology: Concepts of Altered Health States 8th ed. 2009, picture cropped

<u>S3</u> – early diastole, rapid filling of overfilled or poorly compliant ventricle

<u>S4</u> – during atrial systole, blood forced into stiff, hypertrophic ventricle



S_3 and S_4 Heart Sounds

- S_3 - Immediately after S_2
- Rapid filling in early diastole
- Heart already overfilled or poorly compliant
- Heard best with bell at 5th ICS, left midclavicular line

Increased volume

- S_4 - Just before S_1
- Atrial contraction more forceful than normal or blood being forced into stiff, hypertrophic ventricle
- Heard best with bell at 5th ICS, left midclavicular

Stiff ventricles

Causes of Right HF and Left HF

Right Heart Failure	Left Heart Failure
• # 1 - LV failure	• Hypertension
• Pulmonary hypertension	• Acute Myocardial Infarction
• Tricuspid or pulmonic stenosis or regurgitation	• Aortic or mitral valve stenosis or regurgitation
• RV infarction	
• Cardiomyopathy	
• Congenital heart defects	

Hemodynamic Profile

RV Failure

- ↑ CVP, RAP

LV Failure

- ↓ PaO_2, SaO_2
- ↑ PAP, PAOP
- ↓ CO and CI

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Practice

Which of the following parameters indicates successful management of RV failure?

- A. Decreased CVP
- B. Decreased PAOP
- C. Increased PAD
- D. Increased RV pressure

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Which of the following parameters indicates successful management of RV failure?

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- B. Decreased PAOP
- C. Increased PAD
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Practice

Which of the following findings is most consistent with high LV filling pressure?

- A. Sinus bradycardia
- B. Diastolic murmur
- C. Peripheral edema
- D. Bibasilar crackles

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- A. Sinus bradycardia
- B. Diastolic murmur
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- **D. Bibasilar crackles**

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Treatment Goals

Chronic

- Symptom relief
- Improve quality of life
- Reduce/eliminate risk factors such as HTN, diabetes, obesity
- Long-term goal slow, halt, or reverse cardiac dysfunction

Acute

- Stabilize hemodynamics
  - Diuretics
  - Vasodilators
  - Inotropics
- Correct cause

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Treatment Strategies

- Treat the cause/contributing factors
  - Reperfusion in AMI
  - Revascularization (CABG) for CAD
  - Beta blocker and ACE inhibitor to inhibit neurohormonal response
- Valve repair/replacement
- Treat dysrhythmias – including rate control and anticoagulants to prevent thrombi
- Improve oxygenation

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Pharmacologic Treatment Strategies

- Reduce preload:
  - Nitroglycerine, diuretics
- Reduce afterload:
  - Nitroprusside, hydralazine, ACE inhibitors
- Increase contractility:
  - Dobutamine - ↑ SV and CO, ↓ SVR (pro-arrhythmic++)
  - Milrinone - ↓ afterload and preload (pro-arrhythmic+)
  - Digoxin

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Pharmacologic Treatment Strategies

- Diuretics improve symptoms; ↓ JVD, edema
  - Monitor electrolytes (K^+, Mg^{++})
- Beta blocker inhibits excessive catecholamines
  - ↓ ventricular size and mass
  - ↑ exercise capacity
  - ↓ mortality
  - Once acute HF stabilized, start at low dose, increase gradually
  - Monitor for HF decompensation

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Treatment Strategies - Pharmacologic

- ACE inhibitors and ARBs – inhibits vasoconstriction & aldosterone release; inhibits ventricular remodeling
  - Angiotensin Recepter Blockers (ARB) - when patient has intolerance of ACE (cough, angioedema)
    - May be preferred in African American

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Treatment Strategies - Nonpharmacologic

- Biventricular Pacemaker
  - Resynchronizes contraction of the left and right ventricles
    - Improves ventricular function
    - B/P
    - Quality of life
    - Reduces risk of death

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Mechanical Support

- Ventricular assist device (VAD)
  - ↓ workload of heart
  - Maintain CO
  - Allows heart to rest and recover
  - Long-term or permanent support for end-stage heart failure
  - Used as bridge to transplant

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Treatment Strategies - Nonpharmacologic

- Heart transplant
  - End-stage HF, otherwise good life expectancy
  - In U.S., 5 year survival rates 66-71%

Patient Education, Nonpharmacologic

- Diet - restricted sodium, 2 grams
- Smoking cessation
- Limit alcohol intake
- Regular, moderate exercise
- Weight management
- Stress reduction
- Symptom recognition (when to call physician)
- Weigh self daily and report > 2 lb gain in one day or 5 lb in 1 week

Patient Education, Pharmacologic

- Medication adherence *
- Control HTN, hyperlipidemia, DM and thyroid disorders
- Avoid NSAIDs

Practice

A patient admitted with HF develops hypotension, tachycardia, decreased UO, cool clammy skin, decreased LOC, and tachypnea. Which of the following should be included in the patient's plan of care?

- A. positive inotropic agents, diuretics, vasodilators
- B. ACE-inhibitors, adenosine, beta-clockers
- C. Beta-blockers, diuretics, calcium-channel blockers
- D. Negative inotropic medications, digoxin, antiarrhythmics

1 of 10

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Practice

A normal value for an ejection fraction (EF) would be

- A. 65%
- B. 40%
- C. 30%
- D. 25%

2 of 10

A normal value for an ejection fraction (EF) would be

- **A. 65%**

  An EF should be > 50%. It represents the amount of blood ejected from the LV compared to the total amount available, expressed as a percentage.

Practice

Classic echocardiographic features of diastolic dysfunction in patients diagnosed with heart failure would include which of the following?

a. EF 55% and decreased early diastolic filling time

b. EF 50% and increased late diastolic filing time

c. EF 60% and prolonged left ventricular ejection time

d. EF 35% and shortened left ventricular ejection time

3 of 10

A. EF 55% and decreased early diastolic filling time

- The normal ejection fraction is 50-75%.
- Patients with diastolic dysfunction often have decreased early diastolic filling time and preserved ejection fraction.
- Beta-blocking agents prolong the diastolic filing time, resulting in improved left ventricular contraction and decreased diastolic pressure.

Practice

The resistance against which the right ventricle must work to ejects its volume is known as

- A. Resting heart pressure
- B. Systemic Vascular resistance
- C. Central venous pressure
- D. Pulmonary vascular resistance

4 of 10

The resistance against which the right ventricle must work to ejects its volume is known as

- A. Resting heart pressure
- B. Systemic Vascular resistance
- C. Central venous pressure
- D. **Pulmonary vascular resistance**

Practice

Actions of beta blockers include

- A. Increased myocardial oxygen demand
- B. Increased heart rate
- C. Increased diastolic filling time
- D. Increased afterload

5 of 10

Actions of beta blockers include

- A. Increased myocardial oxygen demand
- B. Increased heart rate
- C. **Increased diastolic filling time**
- D. Increased afterload

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Practice

The 4th heart sound (S4) is

- A. Heard as the mitral valve opens
- B. A low-pitched murmur
- C. Heard during atrial contraction
- D. Produced in congest6ive heart failure

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6 of 10

The 4th heart sound (S4) is

- A. Heard as the mitral valve opens
- B. A low-pitched murmur
- C. **Heard during atrial contraction**
- D. Produced in congest6ive heart failure

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Practice

During shift report, you are told that your patient has a 90% occlusion to the Cx. Which type of MI is this patient at greatest risk of developing?

- A. Lateral wall infarction
- B. Anterior wall infarction
- C. Posterior wall infarction
- D. Septal wall infarction

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Four days ago Gertrude, a 70 y.o. was admitted to your unit s/p laparotomy. History includes smoking for 55yrs, type 2 diabetes, PPM. She had a hypotensive episode 3 days ago; dobutamine was started. Today, weaning of dobutamine has been unsuccessful (became hypotensive). Which additional action could be taken to improve Gertrude's cardiac output and help wean her from dobutamine?

- A. Initiate a fluid challenge
- B. Start dopamine
- C. Place a pulmonary artery catheter
- D. Turn up the rate on the pacemaker

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Practice

Stroke volume is comprised of which of the following factors?

- A. Blood volume, viscosity, and impedance
- B. Cardiac output, heart rate and compliance
- C. Contractility, preload, and afterload
- D. Compliance, impedance, and heart rate

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When studying…

- For medications and clinical conditions, think in terms of how they impact preload, afterload, and contractility