Case Study

JA is an 83 year old male who presents to his PCP complaining of a "strange rhythmic, throbbing sensation in the middle of his abdomen." He has sensed this feeling for the past three days. For the past several weeks he has also experienced deep pain in his lower back that "feels like it is boring into my spine." He describes the pain as persistent but may be relieved by changing position. "I think that I hurt my back lifting some firewood," he explains. The patient has never smoked.

What is probably causing this patient's lower back pain?

•**Family hx**: father with aortic aneurysm
•**PMH**: triple coronary artery bypass surgery at age 73, Cluster headache, PUD, OA, psoriasis, hypercholesterolemia
•**Medications**: Celecoxib 200 mg daily, Aspirin 81 mg daily, Clopidogrel 75 mg daily, Simvastatin 20 mg HS, Multivitamin 1 tab daily
•**Allergy:** NKDA
•**ROS**: denies nausea, vomiting, urinary problems, loss of appetite, heart failure
•**PE: BP** 150/95**, HR** 83**, RR** 14, **T** 98.8**, Wt** 158 lbs**, Ht** 5'9"
•**Abdomen:** bruit over the aorta, abnormal wide pulsation of the abd aorta with some tenderness

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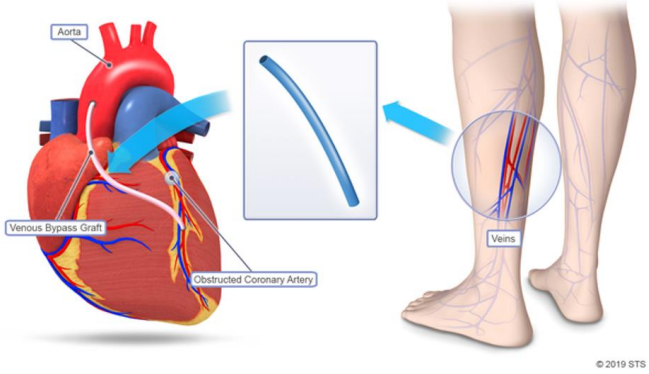

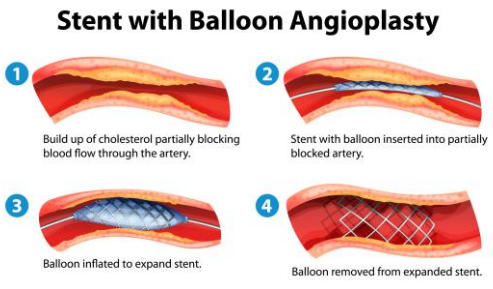

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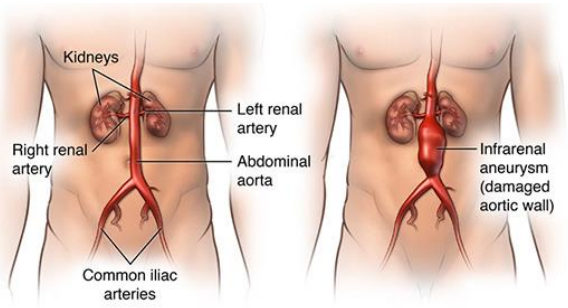


- **Clinical presentation of an aneurysm depends on its**:
  - Size
  - Location
  - integrity.
- **Aortic aneurysms** tend to develop gradually, with 75% undetected until they rupture.
- Rupture may be the first **sign** of an AAA.
- Before rupture, **symptoms** that should raise suspicion of an AAA include:
  - abdominal, flank, or back pain.
  - If the aneurysm is large, it can put pressure on adjacent organs.
  - Nausea, vomiting, bowel, or ureteral compression symptoms can occur.

3

Case Study

b. What is the mechanism of each medication? Do you anticipate more medication prescription by PCP?

- **Celecoxib** 200 mg daily,
  - nonsteroidal anti-inflammatory drug (NSAID).
  - works by reducing hormones that cause inflammation and pain in the body.
  - used to treat pain or inflammation caused by many conditions such as arthritis, etc.
- **Aspirin** 81 mg daily
  - Salicylate, NSAID. Works by stopping platelets from clumping together and forming blood clots that can cause heart attack or stroke.
- **Clopidogrel** 75 mg daily
  - Antiplatelet drug. Blocks platelets from sticking together and prevents them from forming harmful clots.
  - Taken with **aspirin** to prevent clots after a stent implant or other procedure
- **Simvastatin** 20 mg HS
  - HMG-CoA reductase inhibitor used to lower lipid levels
- **Multivitamin** 1 tab daily

4

Case Study

Hb	13.9 (12.5 to 17.5 g/dL)	Neutrophils	59% (40 – 60%)
HCT	43% (41 – 50%)	Lymphocytes	32% (20 – 40%)
WBC	5100 (4,500 – 11,000)	Monocytes	5% (4 – 8%)
RBC	6 (4,5 – 6)	Eosinophils	3% (1 – 3%)
Plt	315,000 (20,000 – 500,000)	Basophils	1% (0 – 1%)
ESR	6 mm/h (0 – 20 –vary)		

c. What important information can be gleaned from the pt's CBC?

5

Na	145 meq/L	**Glucose, fasting**	112 mg/dL	**AST** *(10-40)*	15 IU/L
K	4.9 meq/L	**Uric acid**	2.9 mg/dL	**ALT** *(7-55)*	37 IU/L
Cl	104 meq/L	**BUN** *(6-24)*	9 mg/dL	**Total bilirubin**	1.0 mg/dL
Ca	8.7 mg/dL	**Cr** *(0.7-1.2)*	0.7 mg/dL	**Cholesterol**	202 mg/dL
Mg	2.3 mg/dL	**Alk Phos** *(44-147)*	79 IU/L	**HDL**	50 mgdL
PO4	3.0 mg/dL	**PSA** *(1.0-1.5)*	11.6 ng/mL	**LDL**	103 mg/dL
HCO3	27 meq/L	**Alb** *(3.5-5.5)*	3.5g/dL	**Trig** *(<150)*	119 mg/dL

d. Which single lab value has to be of most concern?

6

High PSA causes:

7

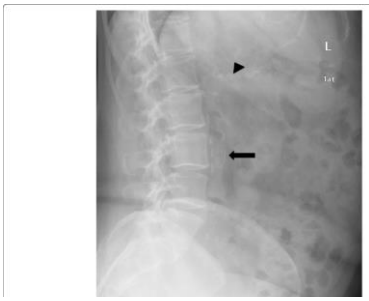
An abdominal x-ray was performed, a localized dilation of the abdominal aorta was visualized, and calcium deposits were seen within the aortic aneurysm.

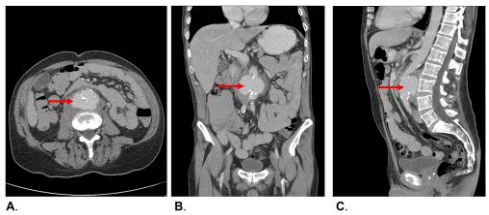
e. What has caused the calcium deposits in the aorta?

f. What type of imaging test is now most appropriate in this patient?

An abdominal aortic aneurysm of 6.5 cm in diameter was located at the level of the renal arteries and extended downward into the iliac arteries.

g. What treatment would you anticipate for this patient?





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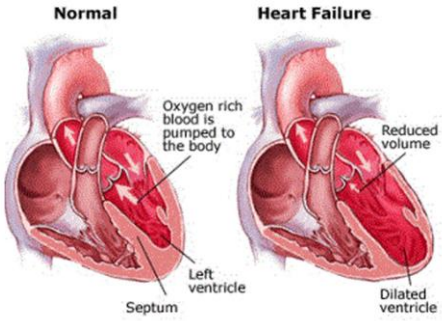
Congestive Heart Failure



Chapter 17

Heart Failure

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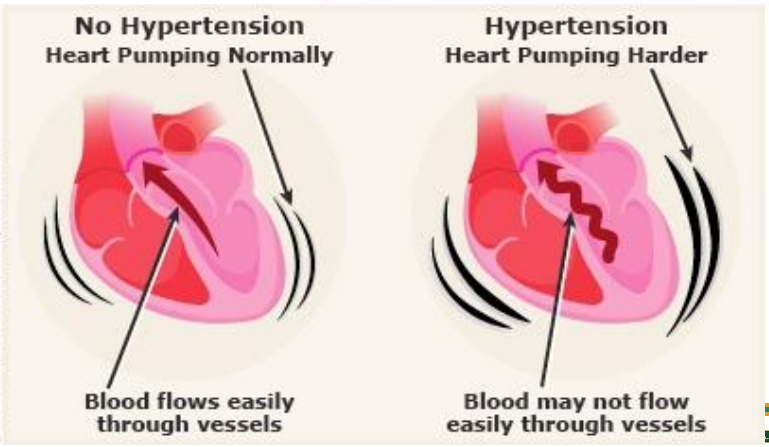
Hypertension



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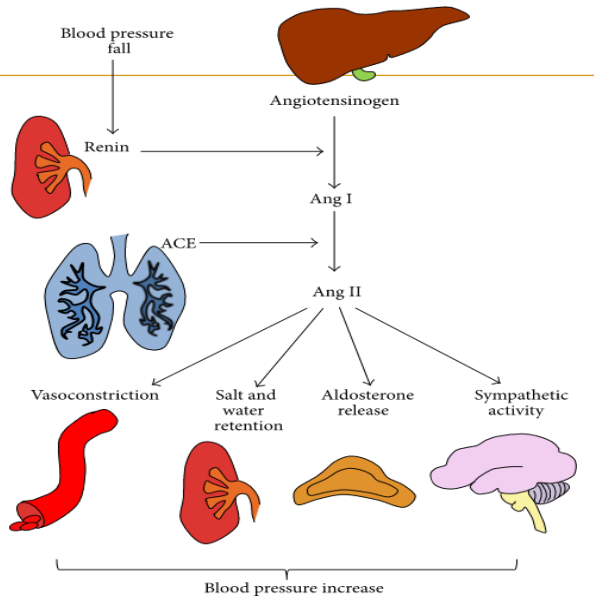
Renin-Angiotensin-Aldosterone (RAAS) pathway.



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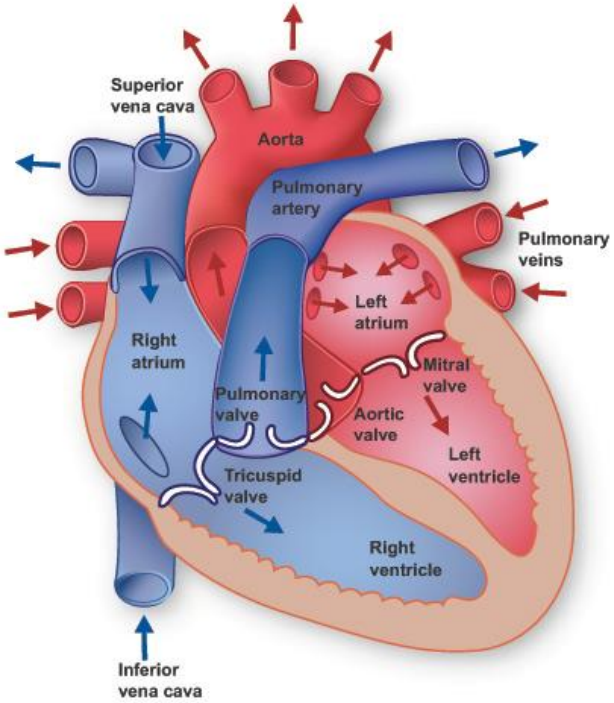

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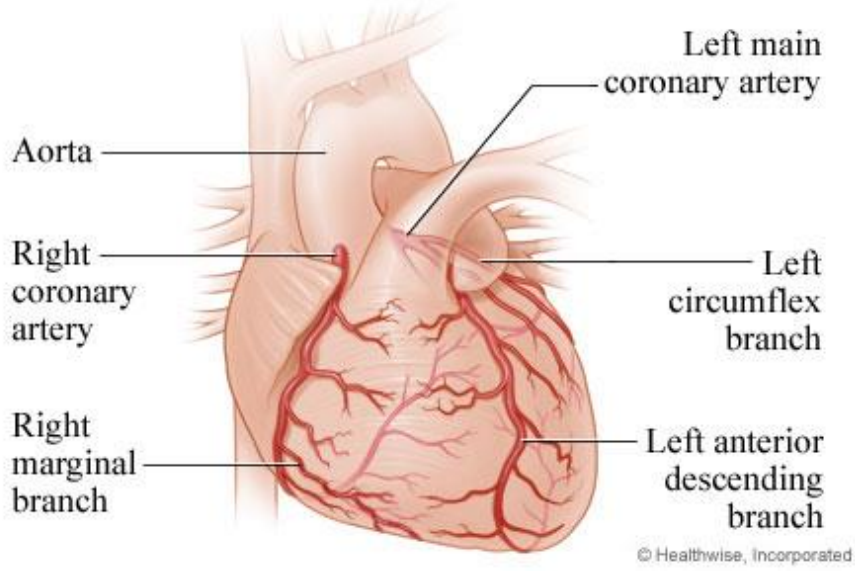

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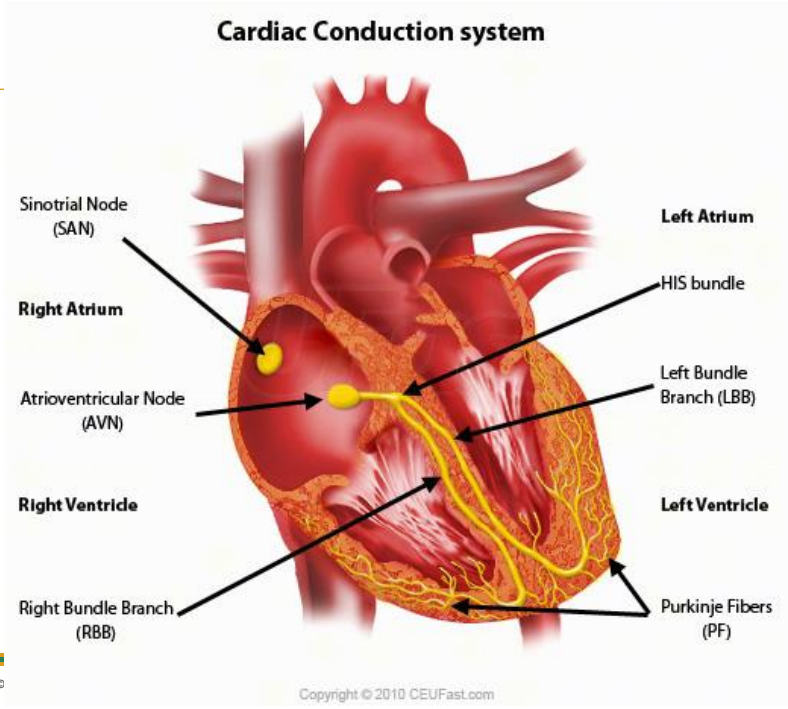

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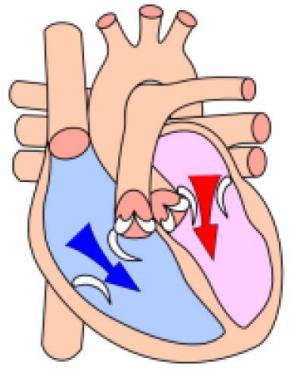
Cardiac preload

Preload can be defined as the volume of blood in the heart at the end of diastole.



The key difference between **preload** and **afterload** is that

- preload is the amount of stretch during diastole when the ventricles fill with blood,

while

- afterload is the pressure against which the heart must work to eject blood during systole.

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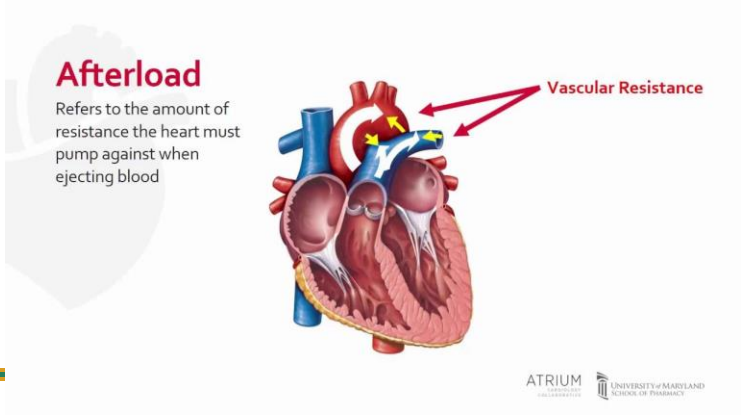
Cardiac afterload

- Cardiac afterload is the amount of resistance that the ventricle must overcome.
- The pressure against which the heart must work to eject blood during systole.
- The lower the afterload, the more blood the heart will eject with each contraction.



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Heart Failure

The following are common manifestations of left heart failure:

- Dyspnea
- Orthopnea
- Cough
- Lack of energy
- Swelling of feet/legs

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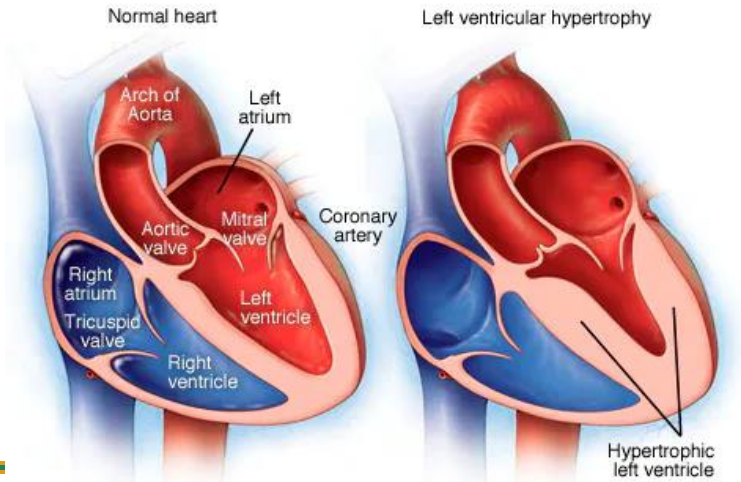
Heart failure

The increased workload on a heart with left heart failure results in change to the myocardial cells: hypertrophy



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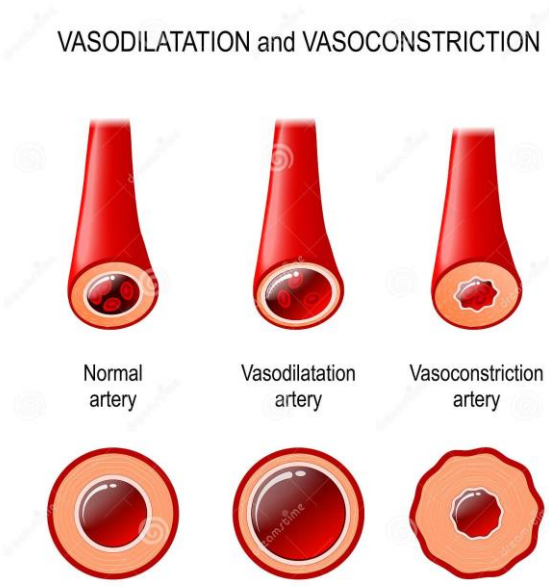
produced by vascular endothelial cells:

- Endothelin → potent vasoconstrictor
- Nitric oxide → dilates blood vessels



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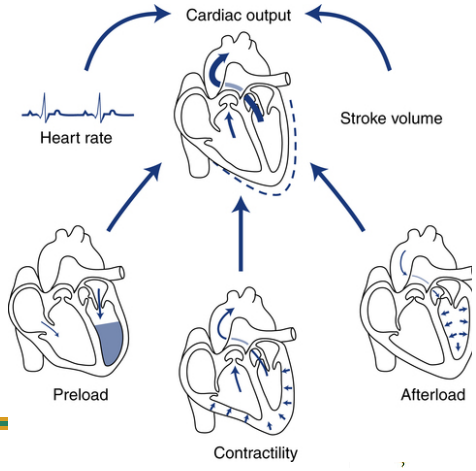
Cardiac performance

Cardiovascular performance is reflected in the arterial blood pressure and cardiac output (mean arterial blood flow), which in turn are dependent on four factors:

- Preload
- after load
- myocardial contractility
- and heart rate.



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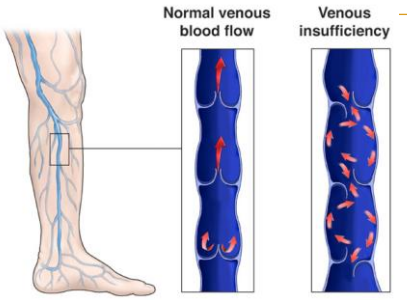
Chronic venous insufficiency.



Mainly 3 different types of venous disease:
1. venous insufficiency
2. DVT
3. varicose veins.

- more prevalent in women
- increases with age

- VENOUS INSUFFICIENCY Predisposing factors:
  - Gravity
  - valvular competence and skeletal
  - muscle contraction
  - Prolonged standing
  - any obstruction to upward blood flow from the lower extremities
  - Obesity
  - pregnancy

- Venous insufficiency = Retrograde blood flow → leads to stasis of venous blood and susceptibility to thrombus formation

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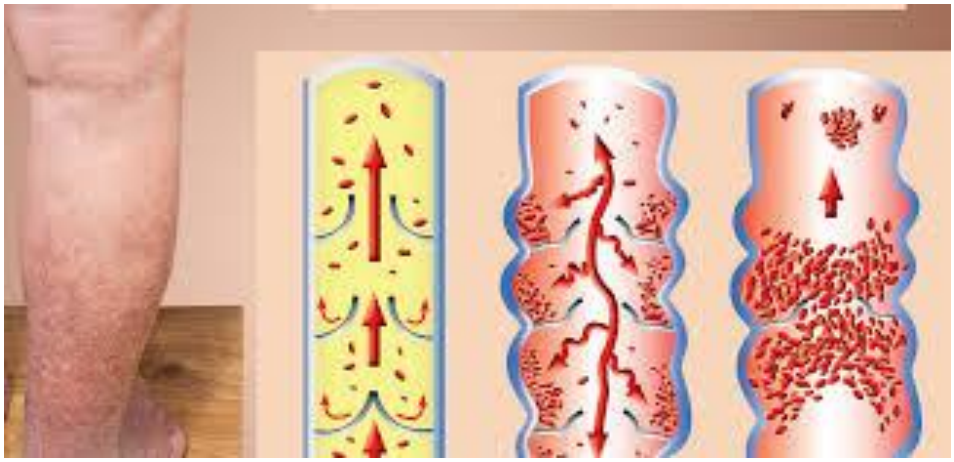
Vein insufficiency
From spider veins to Venous ulcers

22

Deep vein thrombosis (DVT)

→ is a medical condition that occurs when a blood clot forms in a deep vein



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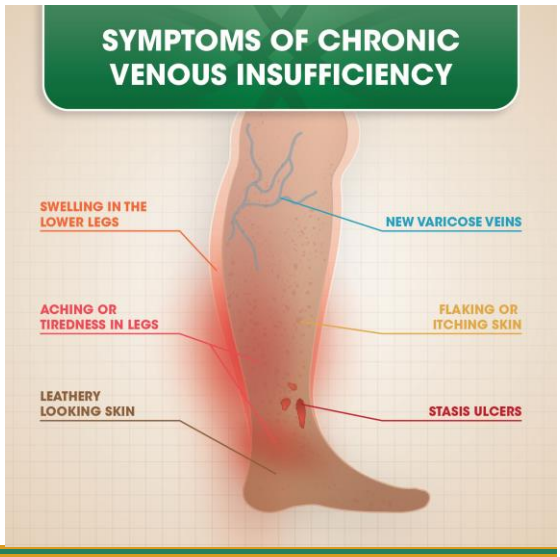
A nurse is caring for a client with chronic venous insufficiency. Which instruction is appropriate to include in the client's care plan?

- ❑ Try to reduce your walking throughout the day.
- ❑ Wear constriction garments.**
- ❑ Apply a cold compression.
- ❑ Elevate your legs periodically during the day.

**except for Thrombo-Embolus Deterrent (TED) Stockings (Compression Stockings)



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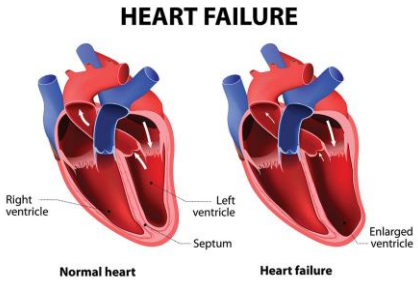
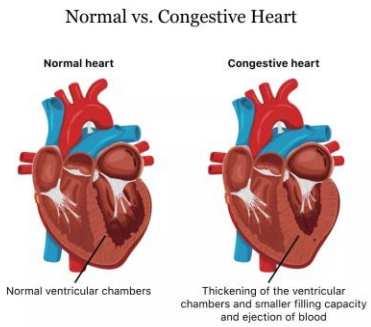
Heart Failure Basic Concepts

- HEART FAILURE: Inability of heart to pump well/to meet needs
  - LVEF: left ventricular ejection fraction
    - Normal: 60%–70%
    - Heart failure: less than 40%
- Should not be confused with cardiac arrest (cessation of all heart activity)
- Epidemic proportions in U.S.
  - As survivability of cardiac disorders (i.e., MI) has increased, incidence of heart failure has gone up

Overall Risks

- Hypertension
- Myocardial infarction
- CAD (Coronary Artery Disease)
- Metabolic syndrome
- Diabetes mellitus

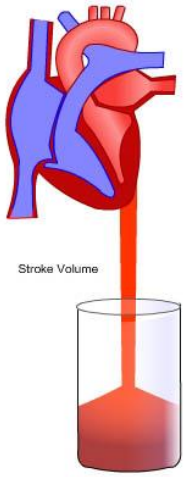




Cardiac Output

Amount of blood pumped by LV per minute

- Cardiac output = HR × SV (stroke volume)
  - Approximately 5 L/min at rest
- Cardiac index = cardiac output/body surface area



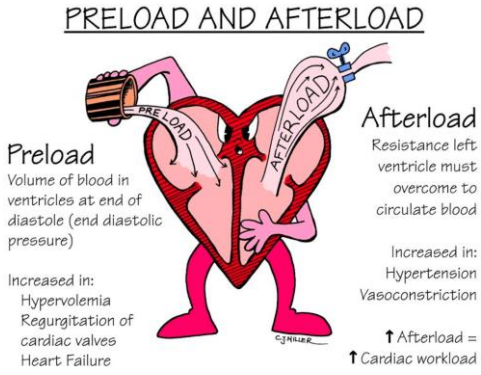
To increase cardiac output

Increase stroke volume
or

Increase heart rate
or
increase both

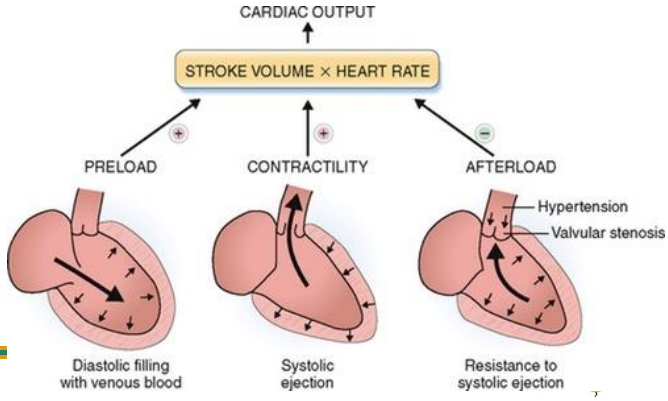
Factors Affecting Heart

- Preload
  - Amount heart is filled per beat (EDV: end-diastolic volume)
  - Increased EDV leads to increased SV (to a point)
- Afterload
  - Resistance that heart pumps against
  - Increased afterload reduces cardiac output



Factor Affecting Heart (continued)

- Contractility
  - Force of contraction
  - Increased preload increases contractility (to a point)
  - SNS activation increases contractility
  - High afterload reduces contractility



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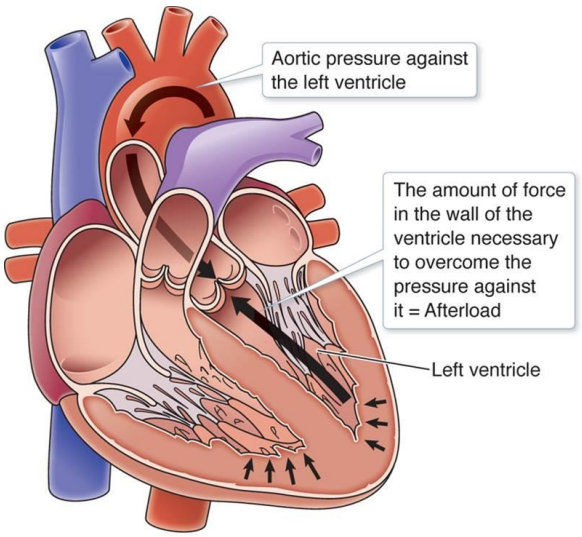
Reviewing: Cardiac Afterload



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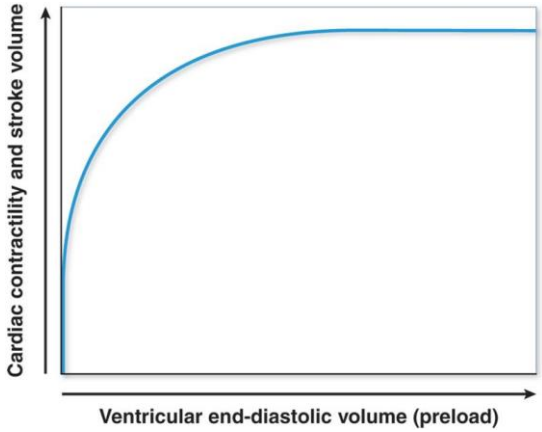
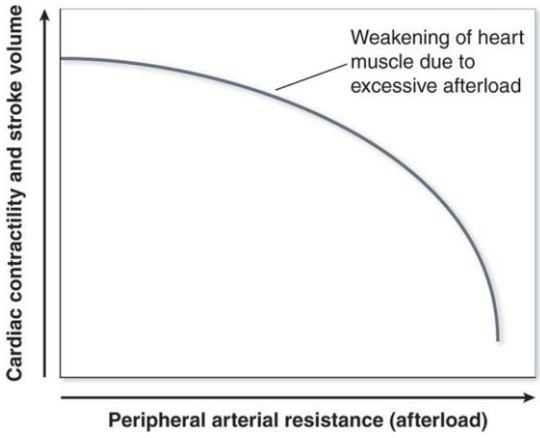
Failing Heart (heart working insufficiently)

- Increased preload fills the heart
- Weakened heart can not adequately pump excess fluid
- Excessive filling overtaxes ventricular fibers leading to decreased contractility
- SV (stroke volume) and cardiac output decrease

Preload, Contractility, and Stroke Volume in a Healthy Heart



Afterload, Contractility, and Stroke Volume



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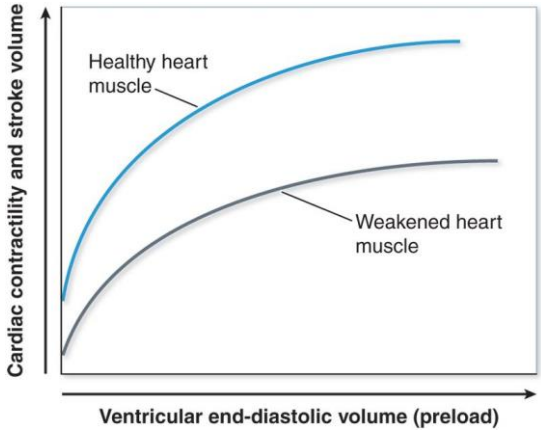
Preload, Contractility, and Stroke Volume in the Failing Heart



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Inotropic vs Chronotropic

Inotropic

- Force of contraction
- Ca++ and SNS activation
  - Positive inotropic agents
- Digitalis
  - Positive inotropic agent

Chronotropic

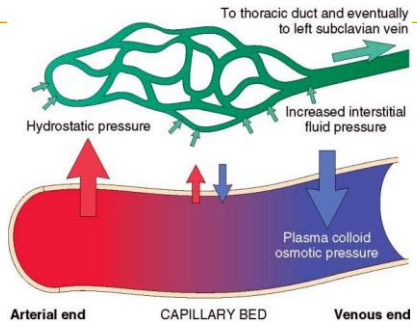
- Rate of heart
- Digitalis and beta blockers
  - Negative chronotropic effect
- Epinephrine and SNS activation
  - Positive chronotropic effect

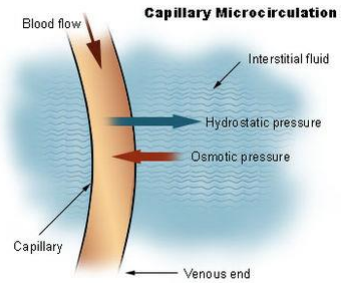
Digitalis Alert

- Evaluate pulse
  - Do not give digitalis if pulse lower than 60 bpm
- Evaluate potassium levels
  - Hypokalemia can cause digitalis toxicity

Heart Failure and Capillary Hydrostatic Pressure

- Starling capillary forces
  - Balance fluid movement at capillaries
    - Hydrostatic pressure: filtration force
    - Oncotic (osmotic) pressure: absorption force
- Fluid retention and "backup" in heart failure can increase hydrostatic pressure at capillaries
  - Increase risk for edema





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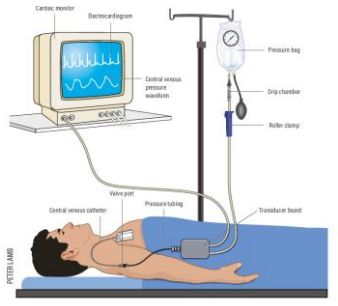
Hemodynamic Monitoring

Measurement of pressure in heart chambers and major vessels

- Specialized cardiac catheter device
- Central venous pressure (CVP)
  - Pressure in vena cava and right atrium
  - Elevated with right-side heart failure
- Pulmonary capillary wedge pressure (PCWP)
  - Pressure within pulmonary capillary bed
  - Elevate with left-side heart failure



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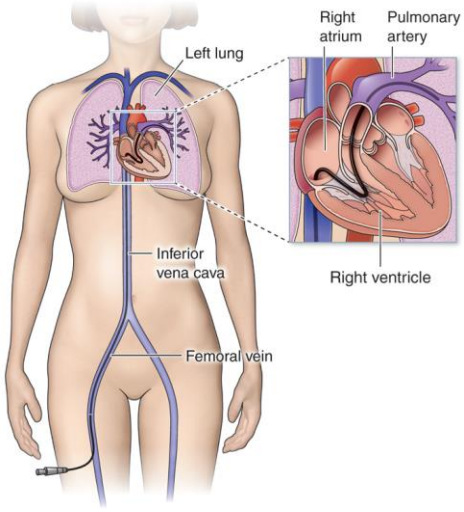
Hemodynamic Monitoring (continued)



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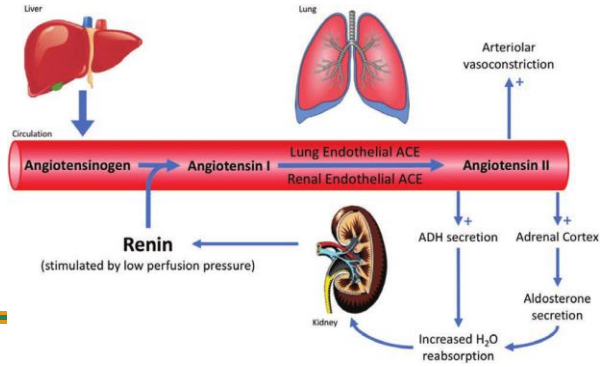
Cardiovascular Regulatory Mechanisms

Renin-Angiotensin-Aldosterone System (RAAS)

- Renin
  - From JG (juxtaglomerular) cells of kidneys in response to low pressure or perfusion
  - Converts angiotensinogen (from liver) to angiotensin I
- Angiotensin I converted to angiotensin II

→ACE (angiotensin-converting enzyme) <u>in the lung</u>

→ ACE inhibitors (<u>*anti-hypertensive medications*</u>) block



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Cardiovascular Regulatory Mechanisms (continued_1)

- Angiotensin II
  - Potent vasoconstrictor
  - Stimulates cardiac remodeling (hypertrophy, apoptosis, fibrosis)
  - Activates aldosterone from adrenal cortex
- Aldosterone stimulates sodium and water retention by kidneys to increase blood volume and pressure

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Cardiovascular Regulatory Mechanisms (continued_2)

- Compensatory mechanisms: detrimental effects over time, as failing heart is overloaded with fluid leading to edema
  - Although the RAAS is a vital compensatory mechanism and major regulator of blood pressure, it has detrimental effects in heart failure. The net effects of the RAAS are elevated blood pressure and blood volume, which increase workload for the left ventricle.

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Cardiovascular Regulatory Mechanisms (continued_3)

- Natriuretic peptides
  - Increase urine output (sodium and water excretion) in response to elevated blood volume
  - ANP: atrial natriuretic peptide
    - Increases urine output, blocks renin and aldosterone release, decreases vasoconstriction
  - BNP: brain or B-type natriuretic peptide
    - Released from ventricles when overstretched
    - High levels associated with heart failure

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Cardiovascular Regulatory Mechanisms (continued_4)

- Endothelin
  - Secreted by heart's endothelium and vasculature
  - Elevates in heart failure, especially post-MI
  - Increases vasoconstriction and ventricular remodeling
- Tumor necrosis factor-alpha (TNF-alpha)
  - Inflammatory mediator
  - Stimulates Hypertrophy, fibrotic changes, and apoptosis of myocardial cells
  - Also, has Negative impact on inotropic function

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Cardiovascular Regulatory Mechanisms (continued_5)

- Nitric oxide
  - Potent vasodilator
- Antidiuretic hormone (ADH, vasopressin)
  - Decreases urine output
  - Increases blood volume
- Autonomic Nervous System (ANS) regulation
  - SNS: beta-1 adrenergic receptors; increase HR and contractility; vasoconstriction
  - PNS: cholinergic receptors; decrease HR and contractility

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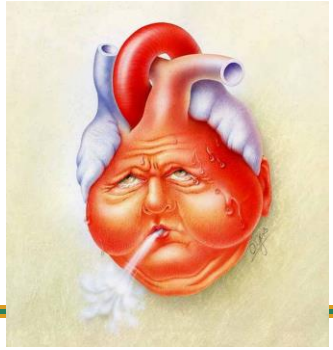
Pathological Changes Leading to Heart Failure

- Increased fluid volume or volume overload
- Impaired ventricular filling
- Degeneration of ventricular muscle
- Decreased ventricular contractile function



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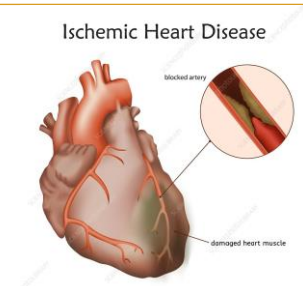
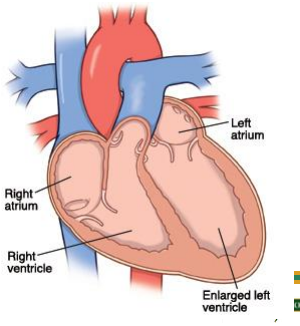
Heart Failure Causes

- Ischemic heart disease
  - Heart tissue compromised, unable to generate adequate pressure



- Hypertension leading to LVH (left ventricular failure)
  - Coronary circulation unable to meet demand
  - Restrictive cardiomyopathy: less filling space due to enlarged LV



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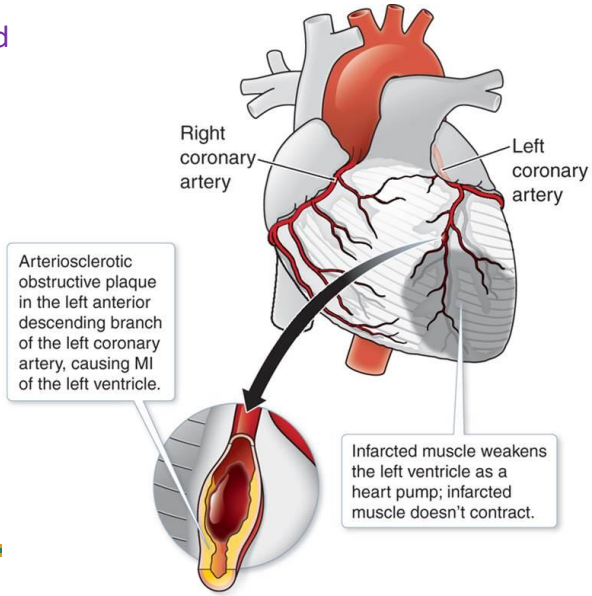
Heart Failure Causes (continued)

Repeated Ischemia and Heart Failure



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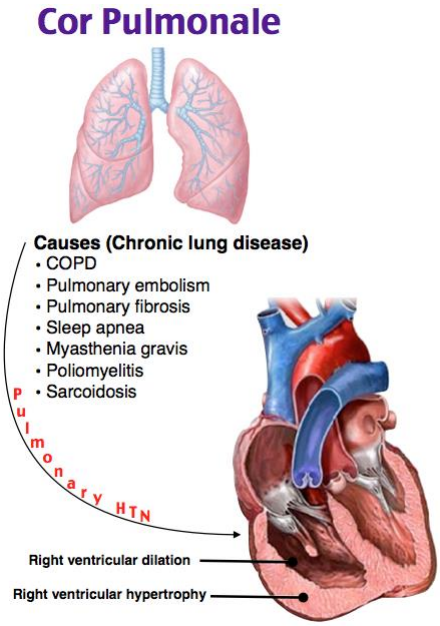
Heart Failure Causes (continued)

- Cor pulmonale
  - Right-side heart failure due to pulmonary issues
  - COPD-hypoxia-pulmonary vasoconstriction-increased workload on R-side of heart



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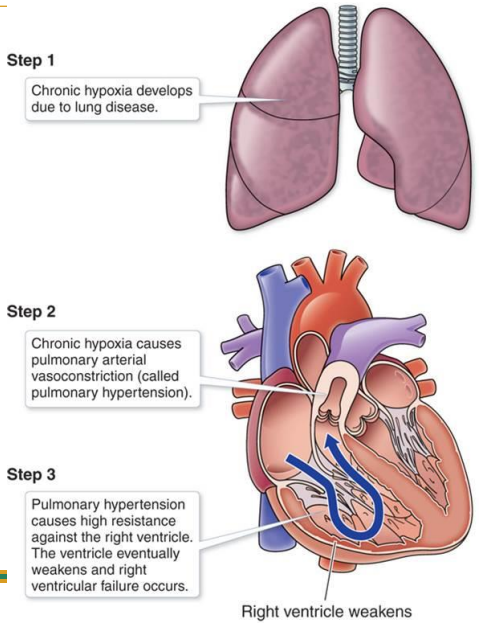
Heart Failure Causes (continued)

Cor Pulmonale: RVF Caused by Lung Disease



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Heart Failure Causes (continued_2)

- Cardiomyopathies
  - Ischemic
    - Myocardial fibrosis and scarring of heart
  - Dilated
    - Enlargement of ventricles
  - Restrictive
    - Can not fill
  - Hypertrophic
    - Usually LV muscle enlarged, encroaches on ejection of blood into aorta
    - Primary: genetic
    - Secondary: due to HTN

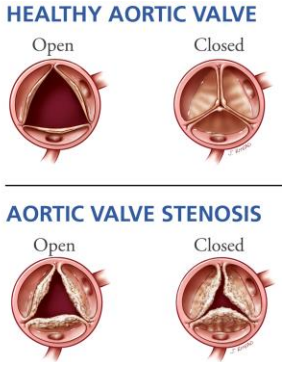
Heart Failure Causes (continued_3)

- Dysrhythmias
  - Irregular heart rhythm may precipitate failure
  - Both tachyarrhythmias and bradyarrhythmias
- Cardiac infection
  - Endocarditis, myocarditis
- Pulmonary embolism
  - Acute RV failure due to increased pulmonary artery pressure

Heart Failure Causes (continued_4)

Heart valve abnormalities:

- Mitral regurgitation (insufficiency)
  - May occur after transmural LV MI
  - Valve does not close properly, backward flow into right atrium (decreased blood ejected from LV)
- Aortic stenosis
  - LV must generate more pressure to eject blood
  - LVH develops



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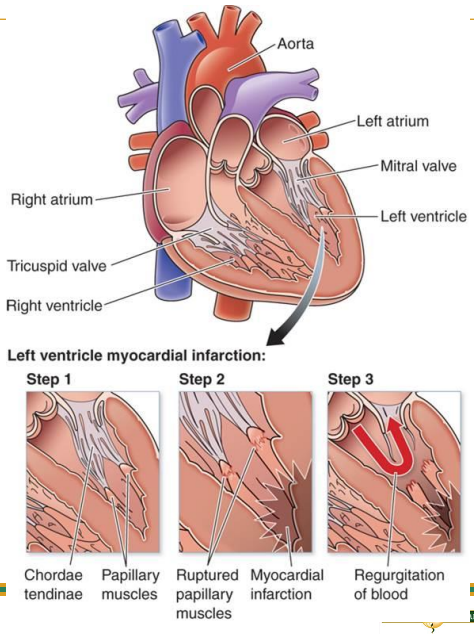
Papillary Muscle Rupture, Valve Disorder and Heart Failure



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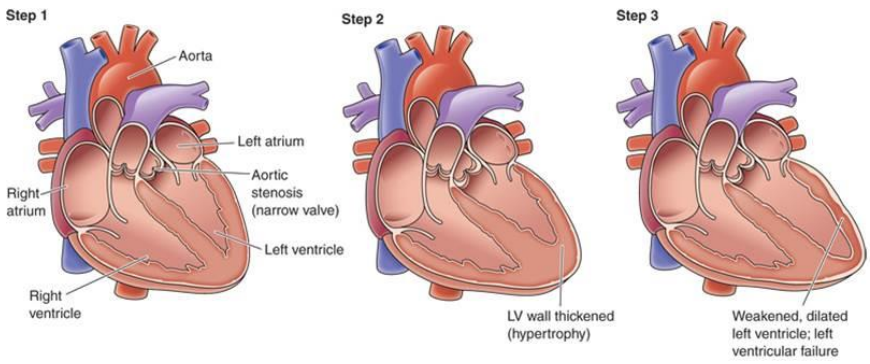
Aortic Stenosis and Heart Failure



Heart Failure Risk Factors

- Age
- Ethnicity
- Family history
- Diabetes
- Obesity
- Sleep apnea
- Congenital heart defects
- Lifestyle
- Smoking
- Sedentary
- Mediations
- Anabolic steroids
- Viral myocarditis
- Alcohol abuse
- Kidney conditions

Heart Failure Classifications

- Acute vs chronic
- Systolic or diastolic
- High-output or low-output
- Right-side or left-side
  - Failure of one side leads to failure of other side
  - Most common way to classify heart failure
  - Forward effects: decreased pressure and perfusion
  - Backward effects: backup of hydrostatic pressure due to heart's failure to eject blood

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Systolic vs Diastolic Failure

Systolic

- Difficulty ejecting blood (HFrEF)
- Decreased SV and cardiac output
- Activation of RAAS, SNS, vasoconstriction place more stress on heart
- Backup of fluid from failed ventricle

Diastolic

- Difficulty relaxing/filling (HFpEF)
- Stiff or enlarged ventricles can not easily fill
- EF may be normal (due to reduced EDV)

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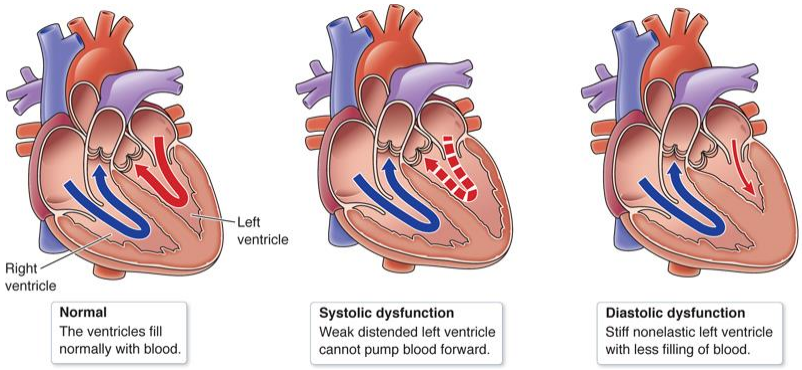
Systolic vs Diastolic Dysfunction



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High-Output vs Low-Output Failure

High-output

- Heart output can not meet high circulatory needs
- Relatively uncommon
- Thyrotoxicosis, severe anemia

Low-output

- Impaired venous return
- *Example:* severe leg trauma preventing blood return

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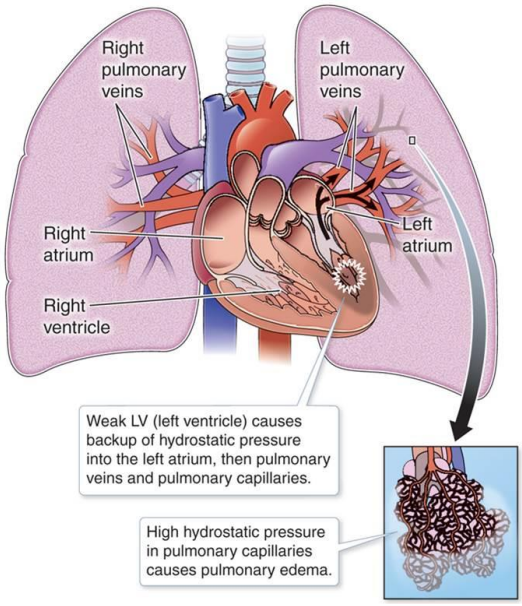
Left Ventricular Failure (LVF)

- Can occur with both diastolic (can't fill) and systolic (can't eject) forms
  - Diastolic
    - LVH due to HTN
  - Systolic
    - Damage to heart (i.e., MI) prevents adequate pressure generation

Left Ventricular Failure (LVF) (continued)

- Forward effects
  - Decrease perfusion
  - Activate SNS, RAAS
- Backward effects
  - Hydrostatic pressure backup into pulmonary circulation
    - Crackles, orthopnea, paroxysmal nocturnal dyspnea (PND)

Pulmonary Edema Formation From LVF



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Pathophysiological Processes LVF

- Activation and cycling of RAAS due to poor renal perfusion
- Results in:
  - Increased vasoconstriction (angiotensin II)
  - Increased blood volume (aldosterone)
  - Increased resistance against LV
  - Ventricular remodeling
- LV fails, further working against these responses

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Pathophysiological Processes LVF (continued_1)

- Adrenergic stimulation
  - Decreased BP stimulates baroreceptors
  - Activate SNS: increased HR and vasoconstriction
- Ventricular remodeling
  - Angiotensin II, TNF-alpha
  - Progressive weakening of myocardium leading to further failure
- Pulmonary edema
  - Oxygen diffusion is hindered

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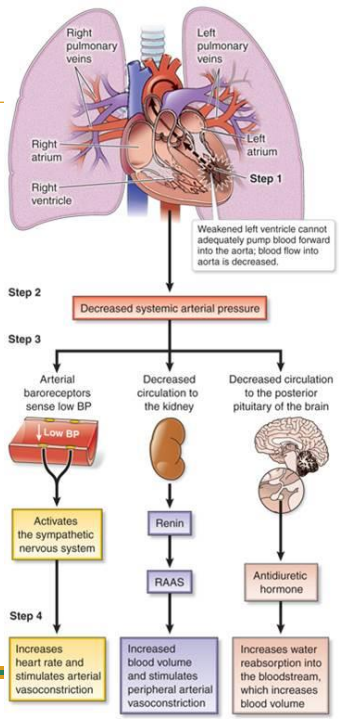
Response to Forward Effects of LVF



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Pathophysiological Processes LVF (continued_2)

- Orthopnea
  - Difficulty breathing while lying down
  - Classified based on number of pillows patient needs to elevate head to breathe comfortably
- Paroxysmal nocturnal dyspnea (PND)
  - Fluid accumulates in lungs while supine
  - Patient may report night terrors that awaken

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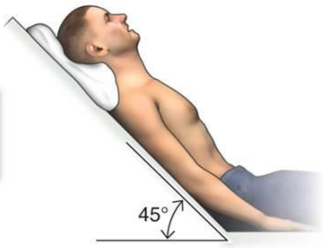
Orthopnea

Lying flat, the patient has the most difficulty breathing because fluid traverses throughout the lung tissue.

Semi-Fowler's position causes fluid in the lungs to move down toward the bases.



Seated position (also called high Fowler's position) gives greatest relief in orthopnea.

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Pathophysiological Processes LVF (continued_3)

- Cerebral symptoms
  - Confusion, memory loss, anxiety
- Constitutional symptoms
  - Decrease blood flow to GI tract
  - Muscle weakness
  - Poor urinary output
  - Cold and pale extremities

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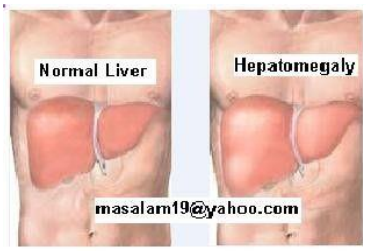
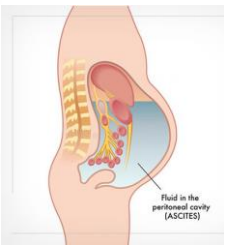
Right Ventricular Failure

- Backward effects most significant
  - JVD: jugular vein distention
  - Increased central venous pressure (CVP)
  - Hepatomegaly, splenomegaly, ascites
  - Hepatojugular reflex may be present
  - Venous congestion of GI tract causing anorexia, nausea
  - Hypoxia and cyanosis may develop
  - Peripheral edema




Normal spleen


Splenomegaly



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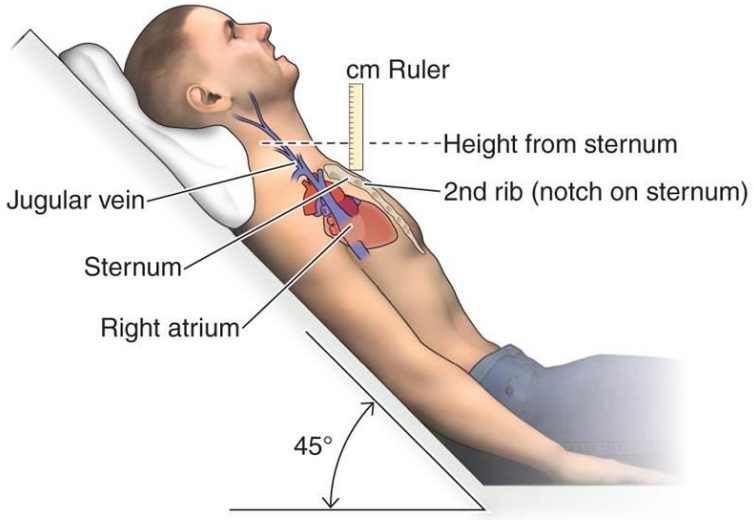
Jugular Venous Distension



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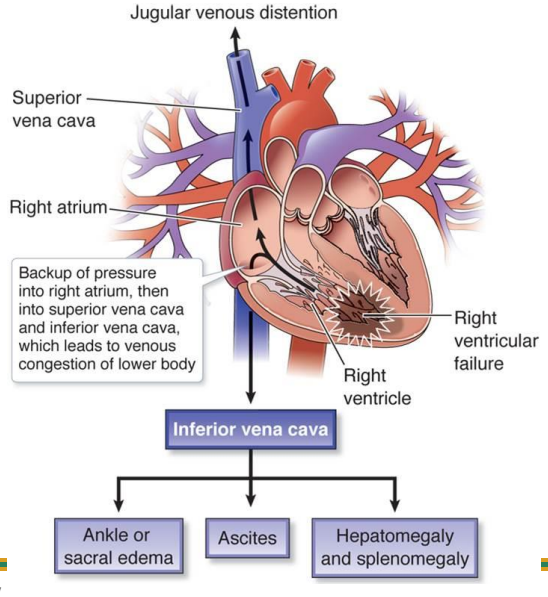
Right Ventricular Failure (RVF)



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Biventricular Failure

→ Dysfunction in 1 heart chamber affects other chambers

- Patients most often present with failure of both sides of heart
- Signs and symptoms of both right- and left-side failure are normally present

General Clinical Presentation

- If mild to moderate failure:
  - Minimal sign and symptoms
- Pulmonary crackles, JVD, cardiomegaly, shifted point of maximal impact (PMI), edema of heart may be present
- S_3 heart sound
  - High ventricular volume and pressure
- S_4 heart sound
  - Atrium contracting against noncompliant ventricle

Heart Failure Diagnosis

- Framingham Criteria for Diagnosis of Congestive Heart Failure

Major Criteria	Minor Criteria	Major or Minor Criteria
• Paroxysmal nocturnal dyspnea • Jugular vein distension Pulmonary crackles • Cardiomegaly • Auscultation of S_3 heart sound • Increased CVP (greater than 16 cm H_2O) • Positive hepatojugular reflux	• Bilateral extremity edema • Nighttime cough • Dyspnea on exertion • Hepatomegaly • Pleural effusion • Reduced pulmonary vital capacity by one-third from normal • Tachycardia (120 beats/min or greater)	• Weight loss of 4.5 kg or more over 5 days of treatment for heart failure
To establish a diagnosis of heart failure, at least one of the major criteria and two of the minor criteria should be present from the Framingham Criteria for Diagnosis of Heart Failure.		

Adapted from Ho, K. K., Pinsky, J. L., Kannel, W. B., & Levy, D. (1993).The epidemiology of heart failure: The Framingham Study. *Journal of the American College of Cardiology*, *22* (4 suppl A), 6A–13A.

Heart Failure: Laboratory and Diagnostic Studies

- Elevated B-NP
- Diluted electrolytes due to fluid retention
- Chest x-ray
  - Cardiomegaly
  - Vascular congestion in pulmonary fields
- ECG
  - No specific sign
  - Some changes such as enlarged QRS, ST segment elevation may be apparent

Heart Failure: Laboratory and Diagnostic Studies (continued)

- Echocardiogram
  - Estimate LVEF, size of heart chambers, valve disorders
- Multiple-gate acquisition scan
  - Radionuclide ventriculography
    - Radioactive dye enables view of heart contraction
- Cardiac catheterization
  - Hemodynamic monitoring
- Angiography
  - Use of dye to view vessels

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Measurements in Heart Failure

Hemodynamic Measure	Normal Value Range
Central venous pressure (CVP)	1 to 5 mm Hg
Pulmonary artery pressure	17 to 32/4 to 13 mm Hg
Cardiac output	4 to 8 liters/min
Systemic arterial blood pressure	90 to 140/60 to 80 mm Hg
Pulmonary capillary wedge pressure (PCWP)	12 to 15 mm Hg
Left ventricular ejection fraction (LVEF)	50% to 70% of blood in ventricle Lower than 40% indicative of LVF

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Heart Failure Classifications (continued)

- The NYHA Classification of Heart Failure
  - Class I
    - Mild heart failure
  - Class II
    - Mild heart failure
  - Class III
    - Moderate heart failure
  - Class IV
    - Severe heart failure
- ACC/AHA Classification of Heart Failure
  - Stage A
  - Stage B
  - Stage C
  - Stage D

With permission from American Heart Association. NYHA classification system. Retrieved from https://www.heart.org/en/health-topics/heart-failure/what-is-heart-failure/classes-ofheart-failure

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Heart Failure Treatment

- Lifestyle modifications
  - Exercise, low sodium diet, smoking cessation
- Medications
  - Diuretics
    - Reduce fluid volume (some can lead to hypokalemia)
  - Aldosterone antagonist
  - ACE inhibitors
    - Block angiotensin-converting enzyme
    - Cornerstone of heart failure treatment

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Heart Failure Treatment (continued_1)

- Medications
  - Angiotensin II receptor blockers (ARBs)
    - Often used in those who can't tolerate ACE inhibitors
  - Beta 1-adrenergic blockers
    - Slow HR, lessen vasoconstriction
  - Positive inotropic and negative chronotropic agents
    - Digitalis
  - Positive inotropic and chronotropic agents
    - Dopamine and dobutamine

Heart Failure Treatment (continued_2)

- Synthetic natriuretics
  - Nesiritide
    - Induces diuresis
- Neprilysin inhibitors
  - Neprilysin breaks down B-NP and ANP
  - Neprilysin inhibitors reduce this breakdown
  - Neprilysin also prevents breakdown of angiotensin II, thus often prescribed with ARBs

Heart Failure Treatment (continued_3)

- Nitrates
  - Arterial and venous vasodilators
  - Reduce heart's workload
  - Coronary artery dilation
- Arterial vasodilators
  - Reduce afterload
  - May be used with nitrates (isosorbide dinitrate/hydralazine)

Devices and Cardiac Transplantations

- Cardiac resynchronization therapy (CRT)
  - Biventricular pacing to coordinate heart rhythm
  - Specialized pacemaker
  - May be used in conjunction with implantable cardiac defibrillator (ICD)
- Intra-aortic balloon pump
  - Inserted into thoracic aorta
  - Helps facilitate adequate coronary flow

Devices and Cardiac Transplantations (continued)

- Left ventricular assist device (LVAD)
  - Pump that enhance LV ejection
  - Helps pump blood into aorta
- Cardiac transplantation
  - Based on age and presence or absence of comorbidities
  - Compatibility of donor and recipient