Alterations of Cardiovascular Function

- Chapter 23
Simplified Path of Blood Flow
The Heart Layers
Cardiac Cycle—Diastole

- Ventricles relaxed
- Blood entering atria
- Blood flows through AV valves into ventricles
- Semilunar valves are closed

Cardiac Filling
Ventricles contract

Blood pushes against AV valves and they shut

Blood pushes through semilunar valves into aorta and pulmonary trunk
Cardiac Cycle
Arteriosclerosis:

- Normal consequence of aging
  - Hardening of the arteries, smooth muscle and collagen grow into lumen and stiffen the wall

Atherosclerosis:

- Type of arteriosclerosis
  - Fatty lesions in the intimal lining of large and medium sized arteries
Lipids get into the vascular endothelium

- Proportional to LDL level

- White blood cells try to clear them away → foam cells

- WBCs and vascular endothelium release growth factors that promote plaque formation

- Plaques block the arteries

- *** inflammation plays a BIG role as an initiator

Atherosclerosis in a Nutshell
1) Intimal injury allows LDL to penetrate vessel wall
2) LDL modifications trigger chemotaxis of monocytes
3) Monocytes enter vessel wall and phagocytize LDL
4) Engorged monocytes become ‘Foam’ cells & ‘Fatty Streak’ visible
Complicated plaques are the ones that rupture and this exposes collagen which activates platelets and thrombi form
Fatty Streak
Atherosclerosis

- Chronic inflammatory process ensues causing smooth muscle proliferation and fibrous tissue accumulation
- Now have fibrous plaque with isolated pools of lipid vulnerable to rupture
Atherosclerosis
The most important complication of atherosclerosis

- Vessel Occlusion
  - Ischemia: may be reversible
  - Infarction: permanent damage
Risk Factors for atherosclerosis and coronary artery disease

- Smoking
- Hypertension
- Gender
- Age
- Hyperlipidemia
- Sedentary
- Stress
- INFLAMMATION like in RA, obesity, periodontal disease
Sites of Atherosclerosis

- Internal carotid arteries
- Proximal coronary arteries
- Abdominal aorta and iliac arteries
- Thoracic aorta, femoral and popliteal arteries
- Vertebral, basilar, and middle cerebral arteries
Peripheral Arterial Disease

- Atherosclerotic changes in the peripheral arteries result in tissue ischemia
  - Lower extremities are most common
  - High risk group are men in their 60’s & 70’s
  - More common with atherosclerosis risk factors
Peripheral Artery Disease

- Gradual onset of intermittent claudication (uncomfortable, or tired feeling in the legs that occurs during walking and is relieved by rest)

- Pain at rest only if severe PAD
Peripheral Artery Disease

- Thromboangiitis obliterans (Buerger disease)
  - Occurs mainly in young men who smoke
  - Inflammatory disease of peripheral arteries resulting in the formation of nonatherosclerotic lesions
    - Digital, tibial, plantar, ulnar, and palmar arteries
  - Obliterates the small and medium-sized arteries
Atrophic Skin Changes
Peripheral Arterial Disease

- **Diagnostic Test Findings**
  - Inspection
  - Check pulses
  - Ankle Brachial Index

- **Treatment**
  - Walking program
  - Avoid injury
  - Reduce risk factors
  - Antiplatelet agents
  - Surgery
Raynaud phenomenon and Raynaud disease

- Episodic vasospasm in arteries and arterioles of the fingers, less commonly the toes
- Raynaud disease is a primary vasospastic disorder of unknown origin
Peripheral Artery Disease

- Raynaud phenomenon and Raynaud disease
  - Raynaud phenomenon is secondary to other systemic diseases or conditions
    - Collagen vascular disease (scleroderma), smoking, pulmonary hypertension, myxedema, and environmental factors (cold and prolonged exposure to vibrating machinery)
  - Seen in patients with RA, SLE, Sjogren’s
Raynaud’s Phenomenon

- Diagnosis
  - History
  - Example

- Treatment
  - Avoidance of cold
  - No Smoking
  - Medications (Ca++channel blockers dilate blood vessels)
Aneurysm

- Abnormal out pouching or localized dilation of a blood vessel or wall of the heart
- Various causes
  - Atherosclerotic plaque formation
  - Congenital abnormality of arterial wall
  - Trauma
  - Infection
  - "true" aneurysm is one that involves all three layers of the wall of an artery
  - Classified also by shape
Aneurysm

- Various locations (aorta most common)
  - Abdominal aorta (between renal arteries and iliac branches)
  - Thoracic aorta (ascending, transverse or descending)
  - Cerebral artery (at circle of Willis)
  - Femoral and popliteal arteries
Aneurysm

- Complications
  - Rupture – risk increases with increasing size
  - Pressure on adjacent structures
Berry Aneurysm
Aneurysms & Dissections

- Berry & saccular used interchangeably. Are often congenital
- Fusiform, either partially or fully, by thrombus, plaque
- Dissecting: tears
- associated with hypertension
Dissecting Aneurysm
Abdominal Aortic Aneurysms

- Develop after age 50
- Usually men
- Severe Atherosclerosis & Hypertension are major causes
- Most (>90%) occur below the renal artery
- Pulsating abdominal mass may be first sign
Hypertension

- **Normal**
  - Systolic <120
  - Diastolic <80

- **Pre-HTN**
  - Systolic 120 - 139
  - Diastolic 80 - 89

- **Stage 1**
  - Systolic 140 - 159
  - Diastolic 90 - 99

- **Stage 2**
  - Systolic ≥160
  - Diastolic ≥100
Hypertension

- Essential Hypertension
  - 90% of hypertensive patients
  - Unknown cause
  - Men > Women
  - Black > White
  - Poor > Rich
  - Older > Younger
  - Obese > Thin

- Secondary Hypertension
  - 10% of hypertensive patients
Hypertension

- Risk Factors
  - Constitutional Factors
    - Family History, Race, Sex
  - Lifestyle Factors
    - High sodium diet, obesity (increased vessel length increases resistance), Physical inactivity, Excessive alcohol consumption, Oral contraceptives, Sleep apnea
    - Not stress (acute changes only)
    - Not high fat diets (risk of CAD)
    - Smoking increases risk of CAD but ? risk in HTN
Hypertension

- Pathophysiologic Changes
  - Asymptomatic
  - Elevated blood pressure
  - Most are the result of chronic complications
Hypertension

- Complications
  - Target Organ Damage
    - Kidneys – Renal Failure
    - Brain – CVA, TIA
    - Heart – LVH, Angina, CAD, CHF
  - Eyes - Retinopathy
  - Blood Vessels - PVD
- Atherosclerosis
  - Cardiovascular disorders
Hypertension

- Diagnosis
  - > 2 BP readings on > 2 separate days

- Treatment
  - Lifestyle Modification
    - Reduce weight, Reduce sodium, Reduce alcohol, Increase exercise
  - Pharmacologic Treatment
    - Stepwise approach
Malignant hypertension: Rapidly progressive hypertension, diastolic pressure is usually >140 mm Hg with papilledema (optic disc swelling), biggest concern is encephalopathy

Up to 1% of patients with essential hypertension develop malignant hypertension, but the reason some patients develop malignant hypertension whereas others do not is unknown.
Orthostatic Hypotension

- Abnormal drop in blood pressure
  - Upon standing
  - Transient
- Symptoms
  - Dizziness and Syncope (fainting)
- Causes
  - Reduce blood volume, Drug induced, Aging, Immobility
Orthostatic Hypotension

- Diagnosis
  - Sitting and Standing BP measurements
  - Tilt table

- Treatment – alleviate the cause
  - Education
  - Adjusting medication
  - Support hose
Disorders of Venous Circulation

- Varicose veins
  - Dilated and tortuous veins
  - Venous valves are incompetent
- Thrombophlebitis
  - Clot formation with inflammation
  - Deep vein is most dangerous
Varicose Veins

- Dilated, tortuous veins, engorged with blood resulting from improper venous valve function
  - Weakening in valve allows backflow of blood
  - Increase pressure in the veins causing walls to stretch
- Primary
  - Originates in the superficial veins
- Secondary
  - Impaired flow in the deep venous channels
Varicose Veins
Varicose Veins

- Pathophysiologic Changes
  - Dilated, tortuous, purple veins most pronounced in the legs
  - Edema of the calves
  - Leg heaviness
  - Dull aching in the legs

- Complications
  - Blood clots secondary to venous stasis
  - Venous stasis ulcers
Varicose Veins

- Testing
  - Visual inspection
  - Doppler ultrasonographic flow analysis

- Treatment
  - Treat underlying cause
  - Anti-embolism stockings
  - Regular exercise
  - Surgical stripping
  - Weight loss if patient is obese
Diseases of the Veins

- Superior vena cava syndrome
  - Progressive occlusion of the superior vena cava that leads to venous distention of upper extremities and head
  - *usually the result of the direct obstruction of the superior vena cava* by malignancies
Chronic Venous Insufficiency

- Chronic venous insufficiency: inadequate venous return over a long period
- Consequence of valvular incompetence
  - Venous muscle pump is ineffective
  - Tissue congestion, edema, impaired tissue nutrition
  - Fat necrosis & skin atrophy
  - Brown pigmentation due to hemosiderin deposits
  - Stasis dermatitis and ulcers because poor O2 levels so cells die
Chronic Venous Insufficiency
Venous Thrombosis

- The acute development of a blood clot that may cause vessel occlusion or embolization
  - Thrombosis causes vessel irritation and resulting inflammation which contributes to occlusion or embolization
  - May occur in deep or superficial veins
Venous Thrombosis Triad

- Venous Stasis
  - Bed rest, immobility, shock, airplane rides, long car rides,

- Hypercoagulability of Blood
  - Pregnancy, childbirth, BCP’s, dehydration, cancer, SLE

- Vascular Trauma
  - Venous catheters, surgery, massive trauma, hip fracture, orthopedic surgery
Venous Thrombosis

- Pathophysiologic Changes
  - Tenderness, redness, and warmth over the affected area due to inflammation
  - Swelling of the affected leg from venous congestion
Venous Thrombosis

- Prevention
  - Ambulation
  - Support hose
  - Low dose anticoagulation

- Treatment
  - Anticoagulation
  - Surgical clot removal
Diseases of the Arteries and Veins

- Thrombus formation
  - Blood clot that remains attached to the vessel wall
  - Thromboembolus: blood clot that dislodges and travels to a new vessel
  - Arterial thromboembolus: source often the heart & obstructs lower extremities, coronary and cerebral vessels
  - Venous thromboembolus: source usually legs & obstructs pulmonary arteries
Embolism

- Bolus of matter that is circulation in the bloodstream
  - Dislodged thrombus, air bubble, amniotic fluid, aggregate of fat, bacteria, cancer cells, or a foreign substance
Pericardium
Pericarditis

- Inflammation of the pericardium as a result of systemic or cardiac diseases
  - Primary – usually viral or idiopathic
  - Secondary – infection, connective tissue disease or trauma
- Often associated with fluid collection within the pericardial space
Pericarditis

- Pathophysiologic Changes
  - Pericardial friction rub and sharp substernal pain
  - Shallow rapid respirations due to associated pleurisy
  - Mild fever resulting from inflammation
  - Dyspnea, orthopnea, tachycardia
  - Relief when sit up and slouch over
Pericardial Effusion

- Fluid in pericardial cavity restricts heart expansion
  - Left ventricle cannot accept enough blood: decreased cardiac output
    - Decreased blood pressure and shock
  - Right ventricle cannot accept enough blood: increased venous pressure; jugular distension
Pericarditis

- Testing
  - Echocardiogram shows pericardial fluid
- Treatment
  - Most cases resolve on their own
    - Pericardiocentesis
    - Pericardial window
    - Pericardiectomy
Cardiac Tamponade

- Rapid increase in intrapericardial pressure caused by fluid accumulation in the pericardial sac
  - Compression of the heart chambers
  - Causes impaired diastolic filling of the heart
Cardiac Tamponade

- Causes
  - Acute MI
  - Connective tissue disorders
  - Adverse drug reactions
  - Infective pericarditis
  - Hemorrhage from trauma
  - Post irradiation pericarditis
Cardiac Tamponade
Coronary Artery Disease

- Impaired coronary blood flow
  - Narrowing of coronary arteries due to atherosclerosis resulting in:
    - Heart disease secondary to myocardial ischemia
    - Strength of myocardial contraction is reduced
    - Wall motion is abnormal resulting in lower cardiac output
Coronary Artery Blood Flow.

- Coronary Arteries originate in the aortic sinuses
- Controlled by aortic pressure (aortic resistance)
- Perfuse during diastole
Imbalance in blood supply and the heart’s demands for oxygen

- Less blood
  - Atherosclerosis
  - Vasospasm
  - Thrombosis

- Higher oxygen demand
  - Stress
  - Exercise
  - Cold
Coronary Artery Disease

- Nontraditional risk factors
  - Markers of inflammation and thrombosis
    - C-reactive protein, fibrinogen, protein C, and plasminogen activator inhibitor
  - Hyperhomocysteinemia
  - Infection
Chronic Stable Angina
- Pain when heart’s oxygen demand increases
- coronary artery size is not changed

Variant Angina
- Pain when coronary arteries spasm
- Prinzmetal’s angina (vasospastic angina, occurs at rest), due to hyperactive sympathetic nerve, patient may have NO atherosclerosis or clots
- Intermittent coronary vasospasms

Silent myocardial ischemia
- Myocardial ischemia without pain
Angina
Coronary Heart Disease

- Coronary heart disease
  - Acute coronary syndrome
    - No ST-segment elevation
      - Unstable angina
    - ST-segment elevation
      - Non-ST-segment elevation AMI
      - Q-wave AMI

Subendocardial

Transmural
Acute Coronary Syndromes

- **ECG changes**
  - T-wave inversion
  - ST-segment depression or elevation
  - Abnormal Q wave

- **Serum cardiac markers**
  - Proteins released from necrotic heart cells
    - Myoglobin, creatine kinase, troponin I and T
Acute Coronary Syndromes

- **Unstable Angina**
  - Causes myocardial ischemia
  - Worse than stable angina but not quite a MI
  - Due to atherosclerotic plaque disruption → platelet aggregation → hemostasis
  - Vasospasm may play a role

- **Acute Myocardial Infarction**
  - Ischemic death of myocardial tissue
  - Sudden onset → Pain
Myocardial Infarction

- Reduced blood flow through one or more coronary arteries causing myocardial ischemia and necrosis
  - Caused by coronary artery occlusion
  - If ischemia lasts more than 45 minutes irreversible damage occurs
- All infarcts have a central hypoxic area
  - Transmural infarction involves the full thickness of the cardiac wall (myocardium)
  - Subendocardial infarction involves the inner half of the cardiac wall (myocardium)
- Injured tissue is potentially viable if circulation is restored
Acute Myocardial Infarction

- Ischemic death of myocardial tissue
Acute Myocardial Infarction
Myocardial Infarction

 Causes
  - Atherosclerosis
  - Thrombosis
  - Coronary artery stenosis or spasm
  - Platelet aggregation
Myocardial Infarction

- Pathophysiologic Changes
  - Persistent, crushing substernal chest pain
    - Caused by coronary artery occlusion
    - May radiate to left arm, jaw, neck or shoulder
  - Cool extremities, hypotension due to impaired myocardial function
  - Perspiration, anxiety, hypertension and feeling of impending doom resulting from pain or sympathetic stimulation
  - Nausea & vomiting due to pain or vagal stimulation
Myocardial Infarction

Complications

- Heart failure
  - AMI is the most common cause of left HF
- Cardiogenic shock
- Pericarditis
- Thromboembolism
- Rupture of the heart
- Ventricular aneurysms
- Cardiac Arrhythmias
  - Most common cause of sudden death with AMI
Ventricular Rupture of the Heart
Myocardial Infarction

- Structural and functional changes
  - Myocardial stunning: reversible reduction of function of heart contraction after reperfusion not accounted for by tissue damage or reduced blood flow. Last for hours to days
  - Hibernating myocardium: some segments of the myocardium exhibit abnormalities of contractile function. Until perfusion restored
  - Myocardial remodeling: refers to the changes in size, shape, and function of the heart after injury to the ventricles. Often necrosis followed by fibrous scarring which is not a contractile tissue therefore function declines
Myocardial Infarction

- Zone of ischemia
- Zone of infarction and necrosis
- Zone of hypoxic injury

<table>
<thead>
<tr>
<th>Normal</th>
<th>Ischemia</th>
<th>Injury</th>
<th>Infarction/necrosis</th>
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<tbody>
<tr>
<td><img src="image1" alt="Normal ECG" /></td>
<td><img src="image2" alt="Ischemia ECG" /></td>
<td><img src="image3" alt="Injury ECG" /></td>
<td><img src="image4" alt="Infarction/necrosis ECG" /></td>
</tr>
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</table>
Myocardial Infarction

- **Treatment**
  - Thrombolytic therapy within 3 hours of onset of symptoms
  - Percutaneous transluminal coronary angioplasty to open blocked or narrowed arteries
  - Oxygen
  - Bypass surgery
Dysrhythmias

- Disturbance of the heart rhythm
- Range from occasional “missed” or rapid beats to severe disturbances that affect the pumping ability of the heart
- Can be caused by an abnormal rate of impulse generation or abnormal impulse conduction

- *Respiratory sinus arrhythmia:* is a naturally occurring variation in heart rate. Inspiration triggers inhibitory signals to the vagus nerve so tonic attenuation of inherent SA node rate is transiently shut down thereby increasing heart rate
pulsus paradoxus (PP), also paradoxic pulse or paradoxical pulse, is defined as an exaggeration (more than 10 mmHg) of the normal variation during the inspiratory phase of respiration, in which the blood pressure declines as one inhales and increases as one exhales. It is a sign that is indicative of several conditions including cardiac tamponade, pericarditis, chronic sleep apnea, croup, and obstructive lung disease (e.g. asthma, COPD).[1]
Dysrhythmias

- **Examples:**
  - **Tachycardia:** >100 bpm
  - **Flutter:** *typical atrial flutter*, has an atrial rate of 250 to 300 bpm or atypical is typically faster, usually 340-350 bpm, *ventricular flutter* rate over 200 bpm
  - **Fibrillation:** >300 bpm irregular, and unsynchronized contraction of muscle fibers (atria or ventricles) *Ventricular fibrillation* is a cause of *cardiac arrest* and *sudden cardiac death*. The ventricular muscle quivers and is useless
  - **Bradycardia:** under 60 bpm, though it is seldom symptomatic until the rate drops below 50
- **Premature ventricular contractions PVC** the heartbeat is initiated by the heart ventricles rather than by the SA node

- **Junctional rhythm**: AV node instead of SA node sets the heart rate

- **Sick sinus syndrome**, also called **sinus node dysfunction**, is a group of abnormal heart rhythms (arrhythmias) presumably caused by a malfunction of the sinus node, the heart's primary pacemaker. **Bradycardia-tachycardia syndrome** is a variant of sick sinus syndrome in which slow arrhythmias and fast arrhythmias alternate
Following the PVC there is a pause and then the normal beat returns - only to be followed by another PVC. The continuation of this pairing of beats is an example of **bigeminy**.

PVCs that occur at intervals of 2 normal beats to 1 PVC is called **trigeminy**

**Quadrigeminy** is where one PVC occurs after every 3 normal beats of the heart
**Long QT Syndrome**

Heart condition in which delayed repolarization of the heart following a heartbeat, leading to re-entrant ventricular arrhythmias.

Symptoms may only manifest to patient during extreme exercise, but that 1st symptom may be sudden death like in young athletes.

May be the result of various gene mutations.

Patient may be put on beta blockers or may get a defibrillator implant.
Heart block: impeded neuroconduction any place along the conduction pathway. AV blocks have 3 degrees with 3rd degree causing complete nerve block between the atria and ventricles.
Cardiomyopathy

- Group of disorders affecting the myocardium
  - Primary ➡ Confined to the myocardium
  - Secondary ➡ Other disease conditions
Hypertrophic cardiomyopathy

- Autosomal dominant disorder or aortic stenosis or anything increasing the workload of the heart over a chronic period
- Hypertrophied ventricle becomes stiff and noncompliant and unable to relax causing a reduction in ventricular filling
- 1 out of 500 in the general population
- Common cause of sudden death in athletes
- Signs and symptoms vary greatly
Cardiomyopathy

- Dilated cardiomyopathy
  - Extensively damaged myocardium reduces contractility causing a decline in systolic function and cardiac output (heart failure)
  - Leading cause of heart transplant
  - Causes: viral, HTN, valve insufficiency

- Restrictive cardiomyopathy
  - Fibrosis and stiffening of the ventricle reducing the ability to relax and fill during diastole resulting in a decrease in cardiac output, amyloidosis
Cardiomyopathy

- Normal heart
- Hypertrophied heart (diastolic heart failure)
- Dilated heart (systolic heart failure)
Hypertrophic Cardiomyopathy
Myocarditis

- Focal or diffuse inflammation of the cardiac muscle which may be acute or chronic
  - Inflammatory cardiomyopathy
  - Inflammation may lead to hypertrophy, fibrosis or inflammatory changes of the conduction system
  - Usually resolves spontaneously
  - Heart muscle weakens and contractility is reduced
Myocarditis

- Causes
  - Infection: bacterial, viral, etc.
  - Radiation therapy
  - Hypersensitivity immune reaction, autoimmune SLE
  - Toxins such as lead, chemicals and cocaine
  - Chronic alcoholism
Myocarditis

- Complications
  - Arrhythmias and sudden death
  - Chronic valvulitis
  - Dilated cardiomyopathy
  - Heart failure
  - Pericarditis
  - Cardiac rupture
  - Thromboembolism
Endocarditis

- Infection of the endocardium, heart valves or cardiac prosthesis
  - Causing deformities and destruction of valvular tissue and chordae tendineae causing rupture and valvular insufficiency
  - Collection of microorganisms, fibrin and platelets form vegetations that can cover the valve surface
  - Vegetations may break off and form emboli
  - AKA: Bacterial Endocarditis
Endocarditis

- Causes
  - Bacterial or fungal invasion
  - Predisposing factors
    - Portal of entry
      - I.V. Drug abuse, surgery, teeth cleanings
    - Damaged endocardial surface
    - Presence of a prosthetic heart valve
    - Rheumatic heart disease
Endocarditis

- **Signs & Symptoms**
  - Fever and chills due to bacterial infection
  - Heart murmur due to turbulent blood flow
- **Diagnostic Test Findings**
  - Positive blood cultures
  - Elevated WBC count
Complications

- Acute renal failure
- Brain abscess
- Cardiac arrhythmia
- Cerebral emboli
- Heart failure
- Meningitis
- Death
Rheumatic Heart Disease

- Acute rheumatic fever is a systemic inflammatory disease of childhood that develops after infections with group-A streptococcus (pharyngeal not skin)
  - Inflammation mainly involves the heart, joints, CNS, skin and subcutaneous tissues.
- Rheumatic heart disease is the cardiac and valvular manifestations of rheumatic fever
  - Includes pericarditis, myocarditis & endocarditis during the early phase
  - Chronic valve disease in the later phase
Underlying Pathophysicsiology

- GAS infection leads to a autoimmune reaction in which antibodies produced to combat GAS affect connective tissues of the body
- Antibodies may attack healthy body cells by mistake because the bacterial antigens are similar to the body’s own cells
- Molecular mimicry
Rheumatic Heart Disease

- Pathophysiologic Changes
  - Polyarthritis caused by inflammation
  - Firm movable subcutaneous nodules
  - Erythema marginatum skin rash found on the trunk
  - Carditis with dyspnea and chest pain
  - Sydenham’s chorea is a CNS manifestation
Rheumatic Heart Disease

Erythema Marginatum
Rheumatic Heart Disease

- Complications
  - Destruction of the mitral and aortic valves
  - Heart failure
  - Infection
Rheumatic Heart Disease

- **Treatment**
  - Prompt treatment of GAS with antibiotics
  - Salicylates to relieve fever and pain
  - Corticosteroids for carditis
  - Corrective surgery
Valve Stenosis

- Occurs when there is narrowing of a heart valve opening
- Stenosis of the heart valve
  - Blood accumulates in chamber preceding stenosed valve
  - Results in increased chamber pressure due to resistance to flow through the valve
  - May cause hypertrophy of cardiac wall
Valve Insufficiency

- Occurs when heart valve leaflets don’t completely close
- Cardiac valve insufficiency
  - Incompetent heart valves allow blood to flow in both directions through valve
  - Volume of pumped blood increases
  - Involved heart chambers dilate to accommodate increased volume
Murmurs

- The blood going through the valve makes a noise.
- These are called **heart murmurs**.
- You can identify them by:
  - Where they are—which valve are they near?
  - How they sound—high or low pitched?
  - When they happen—systole or diastole?
Mitral valve prolapse is the most common cardiac valve disease in the US. More common in women and is believed to be inherited, causes regurgitation of blood into the left atrium. Some experience fatigue, anxiety, and palpitations. Whether the MVP causes these symptoms is in dispute.
Heart Failure

- Syndrome that occurs when the heart cannot pump enough blood to meet the body’s needs (pump failure)
  - Results in intravascular and interstitial volume overload and poor tissue perfusion
  - Classified as “left sided” or “right sided”
Heart Failure

- Left Sided Heart Failure
  - Caused by scars from MI
    - Pumping ability of the left ventricle fails
    - Cardiac output falls
    - Blood backs up into the left atrium...
    - Pulmonary edema
- Right Sided Heart Failure
  - Pumping ability of the right ventricle fails
  - Blood backs up into the right atrium and peripheral circulation
  - Peripheral edema develops
  - Caused by left heart failure and COPD
Heart Failure

Right heart failure
- Congestion of peripheral tissues
  - Dependent edema and ascites
  - GI tract congestion
  - Anorexia, GI distress, weight loss
  - Liver congestion
  - Signs related to impaired liver function

Left heart failure
- Decreased cardiac output
- Pulmonary congestion
  - Activity intolerance and signs of decreased tissue perfusion
  - Impaired gas exchange
    - Cyanosis and signs of hypoxia
    - Cough with frothy sputum
    - Orthopnea
    - Paroxysmal nocturnal dyspnea
  - Pulmonary edema
Pitting Edema
Ascites
Shock

- An acute failure of the circulatory system to supply the peripheral tissues and organs of the body with an adequate blood supply resulting in cellular hypoxia.
Shock

- Cardiogenic
- Hypovolemic
- Obstructive
- Neurogenic
  AKA
  vasogenic
- Distributive
  - Septic
Cardiogenic Shock

- Diminished cardiac output severely impairs tissue perfusion (pump failure)
  - The left ventricle initiates a series of compensatory mechanisms that attempt to increase cardiac output
  - The actions initially stabilize and later deteriorates as oxygen demands increase on an already compromised heart
Cardiogenic Shock
Cardiogenic Shock

- Causes
  - Acute myocardial infarction
  - End stage cardiomyopathy
  - Cardiac tamponade
  - Myocardial ischemia
  - Ventricular arrhythmias
Hypovolemic Shock

- Circulatory dysfunction and inadequate tissue perfusion caused by reduced intravascular blood volume
- Venous return is reduced and cardiac output fails
  - Acute loss of 15% to 20% of blood volume
Hypovolemic Shock

- Causes
  - Blood loss
  - Burns
  - GI fluid loss
- Pathophysiologic Changes
  - Tachycardia restlessness
  - Reduced urine output
  - Hypotension
  - Cyanosis
Hypovolemic Shock

- Diagnostic Test Findings
  - Hypotension
  - Many others...

- Treatment
  - Oxygen
  - Fluids
  - Blood replacement
Obstructive Shock

- Mechanical obstruction of blood flow
  - Great veins
  - Heart
  - Lungs

- Causes
  - Aortic aneurysm
  - Pulmonary embolism (most comm
Distributive Shock

- Inadequate tissue perfusion and circulatory collapse in response to infection (burns, anaphylaxis, neurogenic)
- Caused by loss of blood vessel tone and enlargement of the vascular compartment
- Capacity of the vascular compartment expands such that normal blood volume will not fill the circulatory system
Distributive Shock

- **Causes**
  - Decreased sympathetic activity increased parasympathetic activity: neurogenic
    - Brain or spine injury; anesthetics; insulin shock; emotion
  - Vasodilator substances in blood
    - Type I hypersensitivity (anaphylactic shock)
    - Inflammatory response to infection (sepsis and burns)
Distributive Shock

- Pathophysiologic Changes
  - Chills and fever due to proinflammatory cytokines
  - Tachycardia
  - Reduced urine output due to hypotension
  - Cyanosis
Distributive Shock

- Complications
  - Death
  - Heart failure
  - Renal failure

- Diagnostic Test Findings
  - Blood cultures are positive
  - And ruled out anaphylaxis
Distributive Shock

- Treatment
  - Antibiotics therapy
  - Medications to increase cardiac output
  - Oxygen
Multiple Organ Dysfunction Syndrome

- Progressive dysfunction of two or more organ systems resulting from an uncontrolled inflammatory response to severe illness or injury such as burns or other traumatic injury, infection, acute pancreatitis

- Mortality rate ~50-90%
Multiple Organ Dysfunction Syndrome
Left-to-right shunts

- Most common type of congenital heart defect
- Atrial septal defect – incomplete closure of the embryonic atrial septum
- Ventricular septal defect – incomplete closure of the embryonic ventricular septum; usually associated with other congenital heart defects; large defects require surgery
- Patent (persistent) ductus arteriosus – failure of the ductus arteriosus to close at birth; may close with medication or may require minor surgical intervention
Right-to-left shunts

- Less common - result when there is malrotation of the embryonic chambers
- Characterized by early cyanosis; require surgical intervention
- Tetralogy of Fallot – most common: 4 defects –
  - Ventricular septal defect
  - Pulmonary artery stenosis
  - Overriding aorta – misplaced and sits low on the ventricular septal defect
  - Right ventricular hypertrophy
Right-to-left shunts

- Transposition of great vessels – 2\textsuperscript{nd} most common
  - Aorta arises from the right ventricle and pulmonary artery from the left ventricle
  - Incompatible with life
  - Requires immediate surgical intervention