CARDIAC PATHOLOGY
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CARDIAC DYSRHYTHMIAS

Mechanisms of dysrhythmias

- Alterations in normal rhythm of cardiac cycle
- Abnormality of formation of conduction of electrical impulse resulting in heart rate or regularity
- Occurs when disruption of normal action potential of heart
  Abnormal automaticity - enhanced or depressed
  Re-entry phenomenon: re-excite tissue previously depolarized through anatomic or functional circuits
  Circus movement or re-entrant excitation - continuous excitement by normal or abnormal paths
- Etiology
  Underlying cardiac disease (CAD, cardiomyopathies, valvular lesions, congenital defects)
  Acute MI:
  Systemic-metabolic diseases (DM, HTN, pulmonary disorders, hyperthyroidism, anemia)
  Electrolyte imbalance
  Anesthesia
  Drug intoxication
  CNS disorders
  Psycho-neurogenic disorders
  Exercise

Common cardiac dysrhythmias

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CORONARY ARTERY DISEASE/ ISCHEMIC HEART DISEASE

OVERVIEW

Terminology: - interchangeable terms

- **Coronary heart disease (CHD)**
- **Coronary artery disease (CAD)**
- **Ischemic heart disease (IDH)**

**Etiology:** virtually always from **atherosclerosis**

**Elevated cholesterol** correlates with atherosclerotic changes

- Serum cholesterol above 260 mg/dl has twice risk MI as levels < 160 mg/dl
- **Lipid profile** (components of cholesterol) has bearing on CAD risk

**Dietary fat** - composed of fatty acids

- Fatty acids: chains of carbon, hydrogen, oxygen atoms
- Classified based on degree to which hydrogen saturates molecule

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**ARTHROSCLEROTIC CORONARY ARTERY DISEASE**

CAD results from atherosclerosis of coronary arteries

Disease starts **early in life;** 2-4 decades ensue between onset and disease symptoms

Starts as **fatty streak** which later develop into **fibrous plaque** or **atheroma**

**Atherosclerotic plaques**

- Plaque is white, becomes elevated and partially occludes lumen of artery
- Core of plaque becomes **necrotic** and hemorrhage-calcifications result
- Thrombosis on or around plaque develops - occludes lumen of vessel

Lesions normally asymptomatic until well advanced

**Ischemia** occurs with occlusion. **Collateral circulation** may avoid ischemia

**Plaques** common in bifurcations, curvatures, tapering of arteries

- Right and left coronary artery branch sharply off aorta thus main target
- Coronary arteries bifurcate and taper rapidly thus very vulnerable

When blood flow decreases - anoxia results causing **myocardial ischemia**

**Angina pectoris** - results from ischemia; symptomatic when 75% obstructed

**Myocardial infarction** may result from hemorrhage into plaque (may be sudden)
CLASSIFICATION OF FATTY ACIDS

**Saturated Fatty Acids** - solidify at room temperature
- Plant or animal origin - no double bonds
- Coconut oil, palm oil, animal fats
- Most atherogenic - decreasing intake directly lowers cholesterol levels
- Goal is to limit consumption to less than 10% of total energy

**Monounsaturated Fatty Acids** - single double bond
- Olive oil, peanut oil, canola (rapeseed) oil
- Can lower serum cholesterol but mechanism is unknown

**Polyunsaturated** - long chain fatty acids with 2 or more double bonds
- Liquid at room temp; least atherogenic
- Vegetable oils, cold-water fish
- Includes essential fatty acids, linoleic and alpha-linolenic
- Structural component of cell wall component, precursors to prostaglandins, thromboxane, prostacyclins (immune function, inflammatory response, BP regulation, onset labor, coagulation)

**Trans fatty acids**: formed by partial hydrogenation of polyunsaturated vegetable oil
- Hydrogenation solidifies oil; increases shelf-life of food
- Believed to increase serum cholesterol and triglycerides
- Atherogenic via increasing serum cholesterol and triglycerides

**CLASSIFICATION OF FATS**

**Triglycerides** - fat molecules having 3 fatty acid molecules attached to a glycerol molecule
- Compose most of lipids in food
- Probable independent risk factor of **atherosclerotic heart disease**
- Stored in adipose tissue to be catabolized for energy source

**Cholesterol**
- Sterol form of lipid found only in animal products
- Humans can synthesize thus no dietary requirements
- Concentrated dietary sources: egg yolk, dairy products, beef, pork
- Precursor: sex hormones, cortisol, aldosterone, vit D
- Structural component of cell membrane
- **Excess** cholesterol promotes **atherosclerotic heart disease**

**Phospholipids** - 2 fatty acids molecules and phosphate group bound to glycerol molecule
- Cellular structural components; also found in liver and nervous system
- Found in most foods; not a factor in atherosclerosis
Lipoproteins
Molecules containing protein and various types of lipids
Synthesized in liver; transport insoluble lipids in blood
Classified based on density

Chylomicrons
Very low density lipoproteins (VLDL)
Low-density lipoproteins (LDL)
High-density lipoproteins (HDL)

Risk factors
Alterable risk factors
a. Diet
   High fat diet; liver uses saturated fats to make cholesterol; cholesterol and LDL levels correlate with MI - high fat, high CHO elevates TC

b. Cigarette smoking
   Alters HDL/LDL, myocardial O2 levels; increases risk of dysrhythmia and sudden cardiac death

c. Hypertension
   Enhances atherogenesis and increases myocardial O2 consumption; mechanism not clear

d. Stress
   Type A may increase risk of CAD; stress correlates with disease

e. Sedentary lifestyle
   Altered HDL/LDL relationship increases plaque; exercise improves collateral circulation

f. Diabetes mellitus
   Increases all atherosclerotic disease processes due to altered CHO/fat metabolism - mechanism is not clear
Nonalterable risk factors

a. Advancing age - increases all atherosclerotic diseases
b. Sex - men higher to age 65 then nearly equal incidence
c. Genetic predisposition: early CAD is familial; genetic hyperlipidemia

PATHOPHYSIOLOGY OF CORONARY ARTERY DISEASE

Atherosclerotic Coronary Artery Disease

Initial change occurs early in life as **fatty streak**
Streak subsequently develops into **fibrous plaque** or **atheroma**
Plaque is white, elevated and partially occludes arterial lumen
Core becomes necrotic, hemorrhagic and calcified
Thrombosis around plaque - partially or completely occludes lumen
Lesions normally asymptomatic until 75% vascular supply is occluded

Ischemia results from decreased O2 secondary to occlusion
- Decreased blood supply: **atherosclerosis, spasm, emboli**
  - **Cold temperature** esp with snow shoveling causes peripheral vascular constriction -> angina (ERs with many patients)
- Decreased O2 in blood: **anemia, CO, alkalemia, cyanide**
- Increased demand:
  - **Hypertension**
  - **Cardiomyopathy** - cardiac hypertrophy or dilation
  - **Valvular disease**: stenosis or insufficiency (regurgitation)
  - **Hyperthyroidism**, hyperthermia
  - **Stress** producing increased **catecholamines**
Collateral circulation may delay ischemic symptoms (angina)

Plaques most common at vessel bifurcations, curvatures or branching

Right and left main coronary arteries branch sharply
Circumflex artery common site

Angina results from myocardial ischemia
Myocardial infarction (MI) - cell death or necrosis secondary to total occlusion
- Hemorrhage into plaque, thrombosis on established plaque
- Coronary artery spasm

Nonatherosclerotic coronary artery disease

Cocaine abuse - very common; other substance abuse
Emboli: vegetative endocarditis, tumor, thrombosis
Bacterial endocarditis very common with IVDA
Aortic or coronary artery dissection; trauma, spasm
Increased demand: hypertension,
Arteritis, intimal proliferation (transplantation, angioplasty, radiation)
Nonatherosclerotic thrombosis: polycythemia, hypercoagulability

Polycythemia from athlete's illicit injection of erythropoietin in an effort to improve performance can lead to dehydration and death

ANGINA PECTORIS

Pain caused by ischemia
- Intermittent pain
- Typically substernal but often radiates (arm, jaw, neck, etc)
- Referred pain from esophagus (GERD-esophageal spasms) often mistaken angina
- May be perceived as pressure; may not be present with neuropathic DM
- Severe ischemia results in substernal pain which is crushing
- Source of pain is not entirely understood
- Episodes subside within minutes if imbalance is corrected
- Normal metabolic, functional, hemodynamic balance returns if corrected
- Portends risk of future MI

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Pathophysiology of myocardial ischemia

**Imbalance** between myocardial oxygen **supply and demand**
- Virtually always secondary to atherosclerotic narrowing of coronary arteries
- Pain is intermittent and relieved by rest or taking vasodilator (e.g. NTG)
- Ischemia implies that the changes are reversible (MI when they are not reversible)

**Anaerobic metabolism** (O2 insufficiency) results in accumulation of lactic acid
- Ischemia of cardiac muscles causes intense pain (mechanism not clear)
- Lactic acid may stimulate muscle nerve endings
- Heart is not otherwise sensitive to pain from manipulation or tissue cutting
- Referred pain (left or right arm/neck) likely mediated via sympathetic NS

**Clinical manifestations**
- Substernal feeling of tightness, fullness, pain or oppression
- Pain commonly radiates down one or both arms or into neck/jaw
- Person becomes immobile with pallor, diaphoretic and dyspneic
- EKG changes during angina:
  - T wave inversion, ST-segment depression
  - ST segment elevation when pain is severe and progressive
- Majority (50-70%) patients have no resting EKG changes (in absence of previous MI)
- Stress testing (treadmill with monitoring) will produce diagnostic changes
- Cardiac enzymes during angina are normal (differentiates from MI)

**TYPES OF ANGINA**

**Stable angina** symptoms predictable; onset results from certain level activity/stress

**Variant or Prinzmetal’s angina**
- Not precipitated by activity or stress; often occurs at night
- Demonstrated due to **coronary vasospasm** vs atherosclerosis
  - Usually occurs in vessels that have some degree of arteriosclerosis
  - Some have no CAD
- More common in women
- Characteristic ST elevation during pain

**Silent Ischemia**
- Asymptomatic in many persons with significant CAD
- May be first noted during stress testing (ST-segment depression; T-wave inv)
- Increased incidence of dysrhythmias and sudden death

**Unstable or Pre-infarctional Angina**
- Pain more severe and more frequent; ECG changes common
- Platelet emboli, non-occlusive thrombi over fissured plaque
- Angina changes in character, duration and intensity
- Usually in persons with long history of stable angina
- Pain may occur at night or at rest or at less and less strenuous activity
- Increasing severity or frequency may herald impending MI
Ischemic Myocardial Dysfunction (Stunned Myocardium)
- Mechanical dysfunction due to ischemia
- Ischemia insufficient for MI but produces hypokinesis
- Commonly associated conditions
  - Angina, post-thrombolytic therapy, post-thrombolytic therapy
  - After percutaneous coronary angioplasty
  - Post coronary artery bypass procedure
- Pathophysiology is unknown; possibly due to depression of ATP
- Possibly due to oxygen free radicals which are products of reperfusion
- “Stunned heart” may recover after rest and treatment
  - Intraaortic balloon pump
  - Inotropic drug (increases contractility)

MYOCARDIAL INFARCTION

Oxygen deprivation resulting in cell death
Angina is reversible
MI is non-reversible and results in necrosis/death
Single most common cause of death in US
Most due to atherosclerotic plaque which occlude coronary artery
Can be secondary to cocaine abuse typically in younger men
  - Sympathetic effects (tachycardia, HTN, vascular constriction)
  - Increased thrombogenicity due to platelet aggregation
  - Coronary artery constriction; direct myocardial muscle damage
  - Premature atherosclerosis
ASA (81-325 mg/day) is therapeutic in prevention and also limits infarct with actual MI
Pathophysiology of Myocardial Infarction

- **Ischemic necrosis** of myocardium: irreversible cell damage and muscle death
- Onset within 20-40 minutes with extent of necrosis complete within 3-6 hrs
- Factors affecting infarction size
  - Extent, severity and duration of ischemic episode
  - Amount of collateral circulation
  - Metabolic needs of myocardium at time of event
  - Reduced contractility with abnormal wall motion
  - Altered left ventricular compliance
  - Reduced stroke volume
  - Reduced ejection fraction
  - Elevated LV end-diastolic pressure

Classifying Infarct

**VESSEL LOCATION** - Dysfunction affected by location of infarct

**Left anterior descending coronary artery (LAD)**
- Anterior left ventricular infarct
- Apex, interventricular septum

**Right coronary artery**:
- Posterior left ventricular infarcts
  - Inferior/posterior wall of left ventricle
  - Posterior interventricular septum,
  - Right ventricle

**Circumflex artery**:
- Lateral wall infarcts

**MYOCARDIAL SURFACE** affected - Infarct described in terms of location on myocardial surface

**Transmural infarction**:
- Endocardium to epicardium

**Subendocardial infarction**:
- Endocardial surface extending into myocardium

**Intramural infarction**:
- Patchy areas of myocardium

**Q-WAVE STATUS**:
- Reflects EKG appearance of Q waves with MI
- **Q-wave infarction**:
  - Usually results from sustained occlusion, necrosis
- **Non-Q wave infarction**:
  - Early spontaneous or thrombolytic therapy-induced reperfusion

**REGIONS OR PATTERNS** of necrosis or ischemia (each area emits characteristic ECG patterns)

- Central area of necrosis
- Surrounding area of injury
- Ring of ischemia

**Right ventricular infarction**
- Right coronary artery occlusion; resultant inferior wall infarction
- Central venous pressure may be elevated
- Low right ventricular output may cause cardiogenic shock
- Diagnosis is difficult: right-sided EKG and ECHO
Diagnosing Infarct

23% of MIs go unrecognized - denial is also a big element with this disease
Atypical symptoms either ignored or attributed to another cause
Respiratory difficulties, epigastric pain, vomiting
Diagnosed both with physical exam, history and diagnostics

EKG changes - characteristic - will vary with type of infarct
ST segment elevation during the acute phase - varies with leads
Q-waves - develop subsequently (“Old” MI) - varies with lead
Leads: I, II, III, aVF, aVR, aVL, V1, V2, V3, V4, V5, V6

Cardiac isoenzymes elevation
Myocardial cells liberate intramyocardial cellular enzymes on cell death
Used to date infarct and estimate severity
CPK-MB, Troponin, Myoglobin - serially measured

Other diagnostic blood work: elevated ESR, leukocytosis (with fever elevation)
Myocardial imaging: echocardiogram and nuclear imaging

Clinical Manifestations

- Manifestations from sudden death to asymptomatic
- Most common symptomatology
  - Acute substernal, radiating chest pain with radiation
  - Diaphoresis; Dyspnea
  - Nausea and vomiting
  - Extreme anxiety, fever
  - Any type of dysrhythmia

Sequelae of Infarct

Cardiac scar formation
- Myocardial muscle does not regenerate
- Healing involves formation of scar tissue to replace necrosis
- Scar tissue inhibits contractility - significance depends on amount
- Heart failure may ensue with deceased contractility
- Ventricular dilation is common

Cardiogenic shock may ensue with large ventricular myocardium loss

Complications of MI

Dysrhythmias - 90% of MIs
Predisposing causes:
  - Tissue ischemia, hypoxemia, sympathetic and parasympathetic nervous system
  - Lactic acidosis, hemodynamic abnormalities, drug toxicity, electrolyte imbalance
Mechanisms: abnormal automaticity and/or reentry phenomenon
Cause decline in cardiac output and increase in cardiac irritability
Ventricular fibrillation s/p MI is most common cause of death
Congestive heart failure and cardiogenic shock

See discussion on CHF
Circulatory congestion produced by myocardial dysfunction
Reduced contractility resulting in abnormal wall motion
Compromised ventricular function -> decreased stroke volume and output
Increased residue volume results in pulmonary congestion
Cardiogenic shock - profound ventricular failure (massive MI)
  Occurs s/p MI 10-15% of cases
  Mortality is 80%

Thromboembolism
Mural thrombi common on autopsy
Thrombi associated with large infarct - possibly more common in non-survivors
Fragments produce systemic embolization (brain, kidney, spleen, mesentery)
Pulmonary emboli secondary to DVT from bedrest is common complication

Pericarditis aka Dressler’s syndrome
Common after transmural infarction
Pericardial friction rub
Usually transient - appears after first week of infarction

Myocardial rupture
Rupture of free wall of LV - causes immediate tamponade and death
Tamponade restricts filling of heart -> decreased BP
Rupture of septum less common
Rupture of papillary muscle uncommon - more common with RCA occlusion
  Immediate onset mitral regurgitation
  Symptoms of heart failure

Ventricular aneurysm
Late complication
Thinning, ballooning and hypokinesis of LV after transmural infarct
Aneurysm creates paroxysmal motion of LV
Ballooning of aneurysmal segment with ventricular contraction
Dysfunctional area often fills with necrotic debris and clot - calcium rimming
Debris/clot may embolize into systemic arterial circulation
Occasionally rupture -> tamponade/death (less common than embolization or CHF)

HEART FAILURE
- Inability of pump to meet body’s metabolic demands
- Pump is impaired (heart failure)

- Hypoperfusion resulting from extracardiac conditions
- Hypovolemia, peripheral vasodilation
- Inadequate Hgb oxygenation
- Circulatory congestion: cardiac or noncardiac causes
- Cardiac causes: congestive heart failure
- Noncardiac causes
  - Increased blood volume e.g. salt retention
  - Decreased peripheral resistance
Classification concepts

**Right-sided vs left-sided heart failure**

**Forward vs backward effects**
- Forward (systolic dysfunctions): low output syndrome
- Backward (diastolic dysfunctions): congestive symptoms

**Systolic vs diastolic effects**

Pathophysiology

Onset may be acute or insidious
Systolic or diastolic overload with myocardial weakness
Contractility increases via Starling’s Law to a point (beyond which there is decompensation)
Stress reaches critical level then decompensates - cardiac output declines
Venous input to ventricles remains same resulting in congestion

**COMPENSATION MECHANISM INVOLVED IN HEART FAILURE**

- Systemic response to decreasing cardiac output - R-A-S activation *
  - Reflex increase in sympathetic activity * Renin-angiotensin system
  - Release of renin from juxtaglomerular cells of kidneys
  - Anaerobic metabolism of affected cells
  - Increased extraction of O2 by peripheral cells

- Heart’s response to increased blood volume (short and long-term mechanisms)
  - Dilation of heart’s chambers esp in acute CHF
  - Hypertrophy of myocardium esp in chronic heart failure

- Acute or short-term mechanisms of compensations
  - Dilation and increased force of contraction as end-diastolic fiber length increases *
  - Starling Law of the Heart

- Long term mechanism: ventricular hypertrophy
  - Increases ability of heart to contract and push blood in circulation
  - May exist for long periods before reaching point of decompensation
Cardiac output declines (↓EF) due to ventricular dysfunction → **INCREASED PRELOAD**

**Cardiac dilatation (LVH):** chambers enlarge to accommodate excessive blood volume

Compensatory (neurohumoral) mechanism are activated (to maintain normal circulation)
- **Sympathetic NS** (immediate response - minutes)
- **Renin-angiotensin system** (delayed response: days to weeks)

**Compensatory mechanisms ultimately worsen HF over time**
- Create a "vicious cycle" effect
- Decreased LV ejection fraction → progressive decline systolic function

Additional **neurohormones** are activated

- **Endothelin:** contributes to systemic vasoconstriction
  Produced locally at tissue level (vascular endothelial cells)
- **Arginine vasopressin:** contributes to systemic vasoconstriction (posterior pituitary)
- Increased amounts of counter-regulatory vasodilatory substances present in HF
  - **Atrial natriuretic hormone** (cardiac atria)
  - **Endothelium-derived relaxing factor**

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**Right** - CXR CHF with Kerley B lines
**Left:** Cor Pulmonale
**Left lower:** Dilated cardiomyopathy

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SYMPATHETIC NERVOUS SYSTEM ("Fight or flight" response)
- Occurs immediately
- Attempts to maintain circulatory stability in face of ↓ CO
- HR controlled by baroreceptors in aortic and carotid artery which detect pressure
- High circulating levels of norepinephrine -> down-regulation of cardiac beta-adrenergic receptors
  - Decrease heart’s ability to respond to sympathetic stimulation.
  - May contribute to decreased exercise tolerance
  - Sympathetic NS possibly activated in asymptomatic LV dysfunction
  - Increased activation when symptoms of CHF develop
Normal individual:
- Baroreceptors respond to ↓ or ↓ BP -> signal vasomotor center (brain stem)
- Inhibitory signals (from baroreceptors) -> ↓ sympathetic outflow -> ↓ renin secretion
Heart failure
- Baroreceptors become dysfunctional -> fail to inhibit vasomotor center (brain stem)
- Sympathetic + parasympathetic control is blunted -> lack of control SVR and HR

MYOCARDIAL HYPERTROPHY
- Takes much longer than dilation (preload) or NS mechanisms
- Results from chronic elevation of myocardial wall tension
- Chamber enlarges -> ↓ pressure -> ↓ tension -> hypertrophy of myocardial contractile elements
- Hypertrophy boosts pumping force: compensatory mechanism
  - Works for extended period of time
  - Eventually ability to compensate is exceeded and DECOMPENSATION ensues

Renin-Angiotensin System (R-A-S) - occurs more slowly
- Activated in response to ↓ GFR from decreased cardiac output
- Activation of plasma renin, angiotensin II and aldosterone
  - Results in vasoconstriction and Na+ retention
    - Occurs in both peripheral vascular and cardiac-tissue- based renin-angiotensin systems
- Elevated blood volume (Na-H2O retention) -> ↓ force contraction (Starling’s Law of the Heart)
- Increased force of contraction compensates only up to a point then heart decompensates
- Local production of angiotensin II (tissues)
  - Increases arterial tone in PVS
  - Facilitates release of norepinephrine from cardiac sympathetic NS
  - Contributes to ventricular remodeling
  - May increase incidence of ventricular arrhythmias.
- Activation of renin-angiotensin system (later stages of heart failure except with diuretics)
COMPENSATION VS DECOMPENSATION

- Point of compensation vs decompression is poorly understood
- No morphological difference in examining heart
- Degree of hypertrophy-dilation is not enlightening
- Evidence primarily via hypoxic and congestive dysfunction of other organs vs heart itself
- **Compensatory mechanism**: ↑CO but also ↑myocardial workload + O2 requirements

**Decompensation**: inability to generate adequate contractile force
- Compensation + primary disease overwhelm ability to generate adequate contractile force
- **Early stages**: enlargement is an adaptive process (heart attempts to augment output)
- **Late stages**: Past a certain point dilation is a pathologic process (heart overcompensates for declining systolic performance)

**Goal of therapy to maintain state of compensation via minimizing cardiac work while optimizing cardiac output (CO)** (difficult to achieve)

- Pathologic features of myocardial dysfunction (end stage) is similar regardless of cause
- Degree of cardiac enlargement is important prognostic factor in pt w systolic dysfunction

- **CHF often occurs s/p MI** -> myocyte death and segmental scarring w fibrosis
  - **Chronic ischemia** -> fibrosis in both R and L ventricles
    - Increased degradation of the normal collagen matrix occurs
    - Fibrosis and increase in myocardial interstitium follows
  - **Neurohormones aldosterone and angiotensin II** exacerbate this process

LEFT VS RIGHT FAILURE

- Describes which side has decreased pumping ability
- Etiology, clinical manifestations, and treatment differ
- **Dysfunction of one side will eventually affect the other**
  - **Right side**: blood from systemic venous circulation - pumps to pulmonary system
  - **Left side**: blood from pulmonary circulation and pumps to systemic arterial circulation

FORWARD FAILURE: refers to insufficient cardiac pumping i.e. poor cardiac output
BACKWARD FAILURE: congestion behind pump

Forward and backward failure manifest differently according to side of heart involved.
LEFT HEART FAILURE

CAUSES
  - Infarction and cardiomyopathy: impaired contractility
  - Aortic/mitral valvular disease and HTN -> excessive workload

FORWARD EFFECTS:
  Due to insufficient CO with diminished O2-nutrients to tissue

  - CV: narrow pulse pressure, faint pulses, ↑ HR
  - Brain: restlessness, mental fatigue, confusion, anxiety, impaired memory
  - Generalized: fatigue, activity intolerance, lethargy
  - Kidney: Activation of renin-angiotensin-aldosterone cascade from ↓ GFR -> fluid retention
    - Decreased urine output via conserving Na-/H20 -> ↑ BP
    + ↓ blood volume
    - Vascular congestion -> ↑ afterload against which damaged LV must pump
    - Kidney failure may follow severely reduced perfusion

Sympathetic NS activation
  1. Vasoconstriction (to maintain BP in face of reduced output) -> ↑ LV afterload
  2. Increases HR -> ↑ CO but also raises myocardial energy consumption

BACKWARD EFFECTS

  - RV hypertrophy plus pulmonary congestion
  - Dramatic clinical s/s due to pulmonary dysfunction
  - LV failure -> pulmonary blood congestion-backup
    - ↓ capillary-vein pressure -> fluid forced into interstitial and alveolar spaces
    - Results in pulmonary edema

Pulmonary Effects
  - Dyspnea: breathlessness - cardinal symptom
  - Dyspnea on exertion (DOE): exacerbated w exertion
  - Orthopnea: redistribution from periphery to heart w recumbency
    Ask re: number of pillows used to attempt to quantify during history
  - Paroxysmal nocturnal dyspnea (PND): intermittent attacks of severe dyspnea
    - Suffocation and panic as feeling of inability to overcome dyspnea
    - One of most distressing forms orthopnea
CLINICAL SIGNS

- Cough
- Respiratory crackles (rales)
- Hypoxemia
- High L atrial pressure
- Cardiomegaly on CXR

- Frothy blood tinged sputa if severe
  - Breakage of fragile capillaries
  - Frothy from fluid buildup in alveoli

- Crackles indicated severity of CHF
  - Caused by air movement thru partially fluid-filled alveoli
  - Edema collects in dependent lung fields due to gravity
  - Progressively moves upward with increasing fluid
  - Hypoxemia may result from fluid in lungs: measured via ABG- pulse oximetry

- Cyanosis: bluish coloration skin - late sign - large amt Hgb (5 g/dl) is deoxygenated
- L atrium hypertrophy: compensate for pressure - measure pressure w Swan-Ganz
- Pulmonary edema w 16-18 mg LAP

- Radiographic findings
  - Enlarged heart
  - Engorged pulmonary capillaries
  - Engorged lymphatics (Kerley B lines)

Acute cardiogenic pulmonary edema

- Life threatening - severely impaired gas exchange
- Dramatic S/S
  - Severe dyspnea and anxiety
  - Bolt upright posture
  - Bubbly rales from bases to apices
  - Pink frothy sputa
  - Anxiety/hypoxemia -> tachycardia - worsens pumping efficiency
  - Needs immediate treatment to reduce fluid volume/ support O2
RIGHT-SIDED FAILURE

- LHF increases burden on right -> right heart failure (RHF)
- Pure right heart failure is rare
  - Right ventricular infarction (only 3% of MI) - difficult to treat
  - Pulmonary disorders w increased pulmonary vascular resistance thus high right afterload

- Cor pulmonale: right ventricular hypertrophy -> RHF
- Causes of RHF - disorders which increase pulmonary vascular resistance
  - Hypoxemia -> pulmonary art constriction -> pulmonary resistance
  - Destruction or blockage of vascular bed -> pulmonary resistance
    - Emphysema
    - Pulmonary embolism

- Right Ventricle (RV) - accommodates gradual changes w ↓ preload/ hypertrophy
- RV: thin musculature - little ability to compensate acute changes
  - Right ventricular infarct
  - Large pulmonary embolus

FORWARD EFFECTS: similar to LHF - due to resultant decreased output to LV

- Decreased right CO -> decreased left CO -> systemic effects even if LV functional
- Activation of renin-angiotensin system: ↓ GFR -> volume expansion-activity intolerance
- Activation of sympathetic NS
- No direct pulmonary function impairment but peripheral hypoxemia from ↓ perfusion

CV: narrow pulse pressure, faint pulses, ↑ HR
Brain: restlessness, mental fatigue, confusion, anxiety, impaired memory
Generalized: fatigue, activity intolerance, lethargy

BACKWARD EFFECTS: systemic venous congestion from blood backed up behind failing RV

- Systemic venous congestion
- Impaired function of liver, portal system, spleen
- Impaired kidney function

- Impaired perfusion to sub-Q tissue and brain
- Hepatomegaly w atrophy and necrosis

- Ascites from increased pressure thru portal system -> edema in peritoneal cavity
- Splenomegaly
- GI s/s: anorexia, abdominal discomfort

- Renal congestion -> ↓ GFR/fluid retention
- Congested liver > ↓ aldosterone metabolism -> worsens problem

- Systemic edema esp dependent edema
- JVD: superior vena cava drainage impeded
- Impaired mentation
- Hepatojugular reflex: tests severity of RHF
- When liver is manually compressed -> sudden increase in venous return to RH
- Compression causes jugular vein distention (from increased flow)
- Normally no JVD: increased blood flow (from liver) would enter unimpeded into heart

**BIVENTRICULAR FAILURE:** Failure not localized to one side of heart

- Most frequently result of primary LHF which has progressed to right side of heart
- S/S of both
  - LHF: pulmonary congestion
  - RHF: systemic venous congestion
**VALVULAR HEART DISEASE**

**Valvular Stenosis** (failure to fully open)
- Valve orifice narrows; valve leaflets (cusps) fuse
- Result is that valve cannot open freely
- Narrowing of opening obstructs flow
- Rheumatic fever main etiology
- Other causes: endocarditis, congenital
- Obstructed flow results in increased resistance
- Myocardial hypertrophies in response to resistance

**Valvular Regurgitation** (failure to close completely)
- Incomplete closure results from scarring and retraction of valve leaflets
- Blood flows backward (retrograde) through opening
- Heart chamber which receives backward flow forced to pump added volume
- Muscle fibers stretch and lengthen from added volume
- Hypertrophy and dilation are compensatory mechanisms

**Mixed Lesion** - simultaneous stenosis and regurgitation
- Usually present with advanced disease
- Clinically classified via predominant mechanical load on heart and valvular defects
- Complex clinical picture

**Rheumatic Fever** - rheumatic heart disease
- Inflammatory disease in susceptible patients
- Complication of 1% to 3% untreated streptococcal pharyngitis
- Occurs after untreated pharyngeal infection with Group A Beta-hemolytic streptococcus
- Streptococcal antigenic response activates cytotoxic T cells
  - Cross reaction with cardiac structures causing acute cardiac inflammation
  - Inflammation of joints, heart, skin and nervous system
- Frequently results in valvular disease (rheumatic heart disease)

**MITRAL STENOSIS**

**Pathophysiology:** mitral valve fails to open completely
- Flow of blood from left atrium (LA) to left ventricle impaired
- Normally when mitral valve is open during diastole -> pressures in atrium and ventricle nearly equal
- Stenosis results in atrial pressure which are higher then ventricular pressure during diastole.
- Stenosis worsen results in pressure gradient increases
- Increased atrial pressure -> atrial chamber enlargement and hypertrophy -> pulmonary hypertension -> Right CHF
**Etiology:** most rheumatic in origin

**Auscultatory characteristics:**

- **Diastolic murmur at apex**
  - Varying length depending on extent of valve disease
  - Blood rushes thru narrowed mitral valve during ventricular diastole

- Characteristic **localized low-pitched middiastolic murmur** whose duration varies with severity of stenosis and HR

- **Opening snap** may be heard
  - Widely distributed over chest and occurs early after A2 in severe and later in milder varieties of MS
  - Severe MS murmur may be soft but opening snap usually heard

- Note: if both MS and MR: dominant features may be systolic murmur of mitral regurgitation with or without short diastolic murmur and delayed opening snap.

**Signs of symptoms of mitral stenosis**

- First symptom = dyspnea on exertion (DOE)
- Worsened by strenuous activity, fever, emotion and AF, tachycardia and tachy-dysthymias
  - All of which will increase blood flow and/or decrease diastolic filling time
  - Symptoms due to backup pressure of LA and pulmonary circulation and decreased stroke vol LV due to deficient filling

- Atrial dysrhythmias eg atrial fibrillation common due to excessive atrial volume (40-50%)
- Atrial enlargement and fibrillation predispose to clots which may embolize
  - Systemic embolization in 10-20%

- Pulmonary congestion and right sided-HF end result
  - Orthopnea
  - Cough
  - Dyspnea on exertion
  - Paroxysmal nocturnal dyspnea
  - Abnormal breath sounds
  - Poor arterial oxygenation

- Reduced LV stroke volume
  - Fatigue
  - Poor activity tolerance
  - Poor arterial oxygenation

**Clinical course**

Symptom free 10 years, DOE next 10 years, dyspnea increases with other symptoms over next 10 years, prognosis of 5 years once symptoms occur at rest.

<table>
<thead>
<tr>
<th>TREATMENT MITRAL STENOSIS</th>
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<tbody>
<tr>
<td>- Convert atrial fibrillation if short duration</td>
</tr>
<tr>
<td>- Anticoagulation if a-fib long duration</td>
</tr>
<tr>
<td>- Treat HF</td>
</tr>
<tr>
<td>- Surgical options if disease is severe</td>
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MITRAL REGURGITATION

Auscultatory characteristics

- **High pitched, usually holosystolic**
- Heard best at **apex** with diaphragm, usually with patient in left lateral recumbent position after exercise
- **Unchanged or decreases with inspiration**

Note: Aortic stenosis is also heard at apex. To differentiate, No systolic murmur at RICS in presence of a high pitched systolic murmur at the apex is usually mitral regurgitation. If the same quality murmur is heard at both areas it is probably aortic stenosis

Etiology:

- Rheumatic heart disease
- Non-rheumatic causes
  - Mitral valve prolapse
  - Papillary muscle dysfunction (failure of muscles around mitral ring to contract)
  - Calcified mitral valve annulus -> dilation of mitral ring
  - Weakening of papillary muscle by infarction or fibrosis
  - Improper closure of MV due to absence of atrial contraction (afib or AV dissociation)

Pathophysiology: (valve fails to close completely)

- Backflow of blood from LV to LA during ventricular systole
- Regurgitant flow elevates LA volume and pressure
- Severity of mitral insufficiency (f) amt of LV stroke volume which is regurgitant
- LV must pump greater stroke volume to maintain effective stroke volume

- Both LA and LV generally dilate and hypertrophy to compensate for extra volume they need to pump -> eventual heart failure

  Underlying hemodynamic insult ins the volume overload imposed upon LV by the regurgitant leak. Regurgitant blood plus normal forward flow into ventricles -> increase LV end diastolic volume -> increased wall tension. Chronic overload and increasing LV size -> decrease in systolic function and heart failure as LV is no longer able to eject large LV stroke volume -> decompensation

- May lead to left CHF if severe and uncorrected

Note: acute MR is different: LA is non-compliant and small increases in LA volume -> marked rises in intra-atrial pressure. LA HTN produced by regurgitant volume is reflected back to pulmonary venous system -> acute pulmonary edema

Signs and Symptoms

- Similar to mitral stenosis
- Result from **pulmonary congestion** and poor cardiac output
- Progressive deterioration of LV function may occur in absence of symptoms
- LV function may be irreversibly impaired when becomes symptomatic
Medical Management

1. Improve LV systolic function
2. Decongest circulation
3. Improve forward cardiac output

- Digitalis and diuretics are standard
- Afterload reduction using vasodilator agents popular

Surgical intervention

Indicated if LV function good and not severely impaired below EF 40% - EF will deteriorate post op (close open hole and increase resistance)

Note: treatment of acute MR is basically same but requires quick thinking and action to preserve pt hemodynamically. Atrial lines, nitroprusside, IABP and stat surgical consult.

MITRAL VALVE PROLAPSE

- Mitral valve that balloon up into LA during ventricular systole
- **Myxomatous mitral valve** ("floppy or billowing") valve

Incidence

- Women between ages of 20-40 years most often affected
- Common valvular disease (4-7% in US)
- First ID in 1965 - now most common valvular disorder in industrialized countries
- Often diagnosed only incidentally to P/E

- Sometimes prolapse is sufficient to cause mitral regurgitation but MVP does not usually produce hemodynamically significant regurgitation. Amt of regurgitation does NOT increase over time
- Cause commonly associated w other connective tissue disorders
  - Marfan's syndrome
  - Scoliosis

- AHA suggests endocarditis prophylaxis only for MVP with regurgitation or evidence of mitral insufficiency

Pathophysiology

ANATOMY

- Normal mitral valve has two leaflets
  - Longer anterior - broader posterior leaflet
  - Ratio of anterior to posterior surface area 2:1
- Leaflets attached to chordae tendineae which insert into papillary muscles.
- Ventricular myocardium and mitral annulus below papillary muscle
PHYSIOLOGY

Ventricular contraction -> papillary muscles contract
-> leaflets together and coapt as rising pressure in L ventricle pushes them against one another. Mitral annulus contracts -> reduces size of orifice

Loss of any part of sequence -> Mitral regurgitation

PROLAPSE MECHANISM

- Most persons leaflets COAPT toward ventricular side of mitral annulus
- Minority of persons: leaflets displaced superior and posterior to normal location during systole -> bellies of leaflets move into left atrium rather than coapting

- MVP occurs when leaflets, annulus and chordae tendineae are not in normal proportion to muscular supports (papillary muscles and left ventricular myocardium) i.e. structural changes
  Increased mitral valve surface area, increased size of mitral annulus, chordal abnormalities, plus characteristic histologic hallmark (myxomatous degeneration)

REGURGITATION (does not always occur with MVP)

- More serious consequence of prolapse
- Can result from any one or more of the structural anomalies

Auscultatory characteristics

1. **Mid to late systolic clicks** - single or multiple
   - Will shift in time with respect to first and second heart sounds in response to provocative maneuvers

2. **Mid or late systolic murmur (high pitched)** (in pts w no other cause of mitral regurgitation)
   - May or may not e preceded by a click
   - May crescendo into second heart sound
   - May decrescendo at some point before S2

   - Best heard at apex but may radiate along LSB to base of heart

Postural auscultatory changes: very specific to diagnosis

- Standing:
  - Click occurs earlier in systole
  - Murmur occurs earlier, closer to S1 thus longer

- Squatting (rapidly from standing) - clicks and murmur move later systole (closer to S2)
- Changes are due to sudden increase in venous return from standing to squat -> left ventricle enlarges
- Classic changes are not always present and may vary from exam to exam
Signs and symptoms: (if symptomatic)

- Palpitations, rhythm abnormalities, dizziness, fatigue, dyspnea, chest pain, psychiatric manifestations (anxiety or depression esp panic attacks) - see Syndrome

- Association with panic attacks is controversial
- Majority are unaware and have no untoward effects
- Four serious through rare complications
  - Infective endocarditis more frequent and may warrant prophylactic antibiotics for invasive conditions
  - Slow insidious onset of mitral regurgitation - may necessitate treat
  - Dysrhythmias may develop
  - Sudden death may occur in association w fatal ventricular dysrhythmia (remote risk)
- Surgical repair is rarely indicated

Diagnosis

- Often based on classic auscultatory findings
- Echo can confirm when auscultatory findings non-definitive

MVP Syndrome

- Symptoms not explained via valvular abnormalities alone
- Palpitations, chest pain, fatigue, exercise intolerance, dyspnea, syncope or pre-syncope and neuropsychiatric symptoms eg Panic attack.

- Controversy re: whether syndrome exists (data is sparse)
- Autonomic dysfunction theory (one of many theories)
  - Hyperadrenergic state creates symptoms
  - Increased catecholamines present in MVP patients
  - Isoproterenol infusion reproduces symptoms

Treatment of MVP

- Beta blocker therapy may help subset with hyperadrenergic state
- Exercise may benefit -> increases parasympathetic tone
- Anxiolytic agents
- Meditation, biofeedback
- Echo yearly for significant regurgitation and q other year if mild regurgitation
- Surgery if LVH or decline in EF over several studies
- Treatment of HTN reduces risk of chordal rupture
- ACE to reduce afterload if significant regurgitation

- Assess for arrhythmias (as cause of palpitations)
  - A-fibrillation PVCs and short runs V-tach result from LVH
  - Halter may be useful w palpitations

- Exercise - most can exercise
  - Some patients to avoid high intensity competitive sports

  Hx syncope associated with arrhythmia, family hx sudden death w MVP, significant SVT or ventricular arrhythmias, moderate to sever mitral regurgitation or embolic event
AHA UPDATES ON DENTAL PROPHYLAXIS FOR PREVENTING BACTERIAL ENDOCARDITIS

- Pt w mitral valve prolapse usually don’t need antibiotics unless they have a heart murmur.
- A single 2 m (4x500 caps) amoxicillin before procedure
  - previous dose was 3 gms before, 1.5 grams after.
- Erythromycin is no longer recommended for allergic penicillin pts due to GI upset
- Penicillin allergic pts to receive
  - Azithromycin 2 x 250 mg
  - Clarithromycin 500 mg
  - Clindamycin 4 X 150 mg
  - Cephalexin 2 gms is also an option

per 9/23/97 the new AHA prophylaxis recommendations were published in June 11, 1997 of JAMA.
Also found on American Heart Association (AHA) world wide web site at

DISORDERS OF THE AORTIC VALVE

- Primary disorders are stenosis and regurgitation
- Decline in rheumatic fever: primary cause of AS is age-related calcification
- Hallmark: calcium deposits piled up on aortic cusps
- Calcifications build up over several decades and become clinically significant p age 70-90 yrs
  - Rheumatic disease:
    - Occurs primarily in children and young adults
    - Accounts for only 10% of cases of acquired AS
- Causes of aortic regurgitation similar to those of mitral regurgitation

AORTIC STENOSIS

- Most common acquired valvular disease among adults
  - Predominantly affects older men
  - Incidence affected by
    - Use of penicillin for syphilis and rheumatic heart disease
    - Lengthening life span
  - Secondary to congenital bicuspid valve (vs tricuspid) - past adolescence but before 80 yrs
  - Sclerotic degeneration of normal tricuspid in elderly
  - Need to distinguish from innocent aortic flow murmurs (common adolescents and young adults)

Pathophysiology

- Stenotic process reduces aortic orifice by half (normal is 2-4 cm) -> ventricle has progressively increasing pressure burden
  - Results in ventricular hypertrophy
    - Systolic stress wi normal range
    - Resting cardiac output is normal
  - Significant ventricular dilation not present in uncomplicated cases
  - Dilation when present signifies failure of compensatory mechanism

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- Decline of ejection fraction - secondary to systolic wall stress
- Most of these anatomical and functional changes recede after surgery

- Obstruction of aortic outflow from LV during systole
  Characterized LV/Ao pressure gradient during ventricular ejection
- LV develops high systolic pressure to overcome resistance of stenotic aortic valve
- Slow development of AS allows heart to maintain stroke volume by compensatory LV hypertrophy
- Combination of high LV pressure and hypertrophy
  - Ischemia
  - Attacks of anginal pain

- Continued high LV afterload may lead to L HF
- Symptoms due to decreased cardiac output
- Pulmonary complications occur later as LV fails
- Heart rate generally slow: allow for necessarily long ejection phase

Auscultatory characteristics:

- Characteristic crescendo-decrescendo murmur during ventricular systole
- Harsh in quality
- Crescendo-decrescendo
- Best heard middle to lower left sternal border

Diagnostic studies: (ECHO)

- Mandatory with ejection murmurs of III/VI or greater esp when accompanied by additional signs of significant stenosis

- Refer adolescents or adults w less prominent murmurs if early systolic sound compatible with aortic ejection click heard

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<thead>
<tr>
<th>MANAGEMENT</th>
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<tbody>
<tr>
<td>- Cardiac catheterization</td>
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<tr>
<td>- Successful surgery has largely supplanted medical therapy for AS</td>
</tr>
<tr>
<td>- My need to treatment for CHF before cath or surgery</td>
</tr>
<tr>
<td>- 2/3 of pt w congenital bicuspid valve develop some AS or insufficiency beyond age 40</td>
</tr>
<tr>
<td>- Pathological AS less than 1/3</td>
</tr>
<tr>
<td>- Lower still is percentage of whom require SX</td>
</tr>
<tr>
<td>- Antibiotic prophylaxis a consideration</td>
</tr>
<tr>
<td>- Justifies ECHO in young people for dx purposes</td>
</tr>
<tr>
<td>- Surgical correction indicated for symptomatic AS</td>
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History and physical exam

- May be symptom free for extended periods
- Average age of onset 60 yrs
- Classic s/s: dyspnea, syncope and angina
  - Dyspnea from abnormal compliance of in hypertrophied ventricle
- Resting diastolic pressures elevated esp after exercise
- Exertional angina may occur even w normal cor arteries
- With advent of symptoms -> sharp downward turn in life expectancy with average survival of 3 years
- Suspect/refer to aortic stenosis with presentation of angina, syncope, CHF where there is reasonable suspicion

Functional vs pathologic murmur

- Distinction can not always be made
- Aortic murmurs radiate to R base
- Innocent pulmonic flow murmur
  - Radiates to upper L parasternal region
  - May be prominent along lower sternal margin
- Murmur characteristically associated w high-output states
  - Pregnancy fever, anemia
  - These conditions can induce aortic murmurs
  - Generally II/VI or less
  - Peak amplitude during early systole
  - Relatively brief
  - Diminish/disappear during strain of Valsalva maneuver
- What is not ominous in an adolescent may be consequential in middle-aged adult
  - Especially concern if adult is 5th and 6th decade
  - May progress rapidly over 2 years to severe

- Peripheral manifestations of critical aortic stenosis

  - Reduced pulse volume
  - Delayed upstroke - best appreciated on carotid artery palpation
  - Systolic thrill often palpable at base
  - Normal first sound followed usually after brief gap by late-peaking harsh murmur which radiates as noted above.
  - Second heart sound is often single in older adults - due to rigid aortic cusps
  - Paradoxical splitting of S2 may reflect delayed ventricular emptying in adolescents and young adults
AORTIC REGURGITATION

- Incompetent aortic valve - blood leaks back from aorta to LV during diastole
- Results in LV volume overload
  - LV has usual preload volume from atrium plus regurgitant blood from aorta
- LV compensates w hypertrophy and dilation
- Larger than normal stroke volume achieved -> high system pressure
- Diastolic pressure gen lower than normal from rapid runoff of blood into ventricle

- **Bounding peripheral pulse** from
  - Large stroke volume and
  - Rapid decline in diastolic BP

- High-pitched blowing murmur during ventricular diastole
- C/O palpitations and throbbing/pounding heart - due to large ventricular stroke volume
- Left heart failure is major complication - due to high ventricular workload

- 2 categories: acute and chronic
- Distinction is (f) response rate at which valvular incompetence develops

**Chronic**

- Most likely etiology is congenital bicuspid aortic valve
- In past due to rheumatic fever but now isolated AR unusual
- Other causes
  - Connective tissue disorders (Marfan, Ehlers-Danlos) aortic aneurysm, myxomatous valvular degeneration, syphilis, aortic involvement by rheumatoid arthritis.

**H/P**

- Most often asymptomatic except for s/s predisposing disorder
- Essential features
  - Bounding, collapsing peripheral pulses
  - Downwards laterally displaced hyperdynamic apex impulse characteristic murmur

- High pitched blowing diastolic murmur - best heard along left sternal margin
- Audibility to right of sternum suggests syphilitic basis or aortic aneurysm
- **Hill sign:** difference in systolic BP between simultaneous supine measurements in arm and leg
  - Normally no difference
  - Difference 20 mm Hg or less suggests mild incompetence
  - difference 20-40 suggests moderate disease
  - Greater than 60 mm severe regurgitation
  - Rough index only in asymptomatic patient
  - Prone to underestimate severity in CHF
INFECTIVE ENDOCARDITIS

- Invasion and colonization of endocardial structures by microorganisms
- Variety of organisms have affinity for heart esp valves
- Valvular lesions: growths of microorganisms enmeshed in fibrin deposits aka vegetations
  - Can become large and interfere with valvular function
- Predispose to emboli formation (septic emboli)
- Common organisms
  - Streptococci
  - S. aureus

- Requisite is invasion of blood stream by infective organisms
- Obvious portals of entry
  - Overt infection
  - IVDA
  - Invasive sx or dental procedures
- Less obvious portals of entry
  - GI tract
  - Oral cavity

Acute infective endocarditis

- May develop where
  - Host resistance is low
  - Organism is highly virulent
  - Bacterial invasion is sufficiently large
- Usually affects persons w previously normal valves
- Mortality 50-60%
- IVDA particularly susceptible

Subacute infective endocarditis

- More insidious onset
- Affects persons w some preexisting propensity for valvular colonization
- Organisms less virulent
- Predisposing conditions
  - Rheumatic heart disease
  - Congenital heart abnormalities
  - Mitral valve prolapse
  - Calcified valves
  - Artificial valves
  - Immunosuppression
  - IVDA (persistent inoculation)
- Skin colonizers common offenders in IVDA
  - S. aureus
  - S. epidermidis
  - Candida
- Right heart valves usually affected in IVDA
- Organisms of SBE not virulent enough for healthy endocardium
- Gain foothold only where underlying predisposition
- Heart disease may permit platelet-fibrin deposits on valves - stagnant flow blood patterns
- Deposits become site of organism attachment
- Antibodies further assist attachment via clumping of organisms
BACTERIAL ENDOCARDITIS

DIAGNOSTICS (same for BE and SBE)
Large bulky bacteria-laden vegetations
Hang from heart valves and adjacent endocardial surfaces

PATHOLOGICAL PROCESSES (from vegetation)
- Risk of embolization
- Erosion or perforation of underlying valve leaflets
- Erosion/abscess of adjacent myocardium (acute forms)
- Valvular vegetations eventually become calcified

CLINICAL PRESENTATION very nonspecific
- Fatigue, wt-loss, flu-like symptoms
- Positive blood cultures may confirm
- Acute has more obvious onset
- Acute: fever, chills, malaise and heart murmur

TREATMENT
- Antibiotic therapy
- Surgical valve replacement
- Prophylactic antibiotic prevention for those at risk

CARDIOMYOPATHIES

Congestive or Dilated Cardiomyopathy (idiopathic cardiomyopathy)

Usually unknown etiology but has other associations
Ischemic cardiac disease, beriberi, thyrotoxicosis, alcoholism,
childbirth/postpartum, DM, drug toxicity, cobalt therapy, neuromuscular disorders
Cardiomegaly with activation of compensatory mechanisms
Dilation and hypertrophy resulting in hypokinesis and ensuing CHF
Ischemic cardiomyopathy has poorest prognosis

Restrictive Cardiomyopathy

Etiology unknown; biventricular failure is common
Idiopathic form: extensive fibrosis but no pathologic substance
Ventricular filling is impeded
End-diastolic ventricular pressures are high
Poor prognosis for survival - death from CHF

Hypertrophic Cardiomyopathy

- Asymmetrical increase in ventricular mass
- Etiology unknown - familial occurrence
- Diastolic dysfunction, myocardial ischemia, dysrhythmias, outflow obstruction
- Symptoms of left heart failure
- Sudden death during or after vigorous activity esp young athletes
- Prognosis variable
**MYOCARDITIS - PERICARDITIS**

**Myocarditis** - inflammation of the myocardium

**Etiologies**
Infectious processes, chemical agents (chemotherapy), hypersensitivity, viruses esp coxsackievirus, bacteria, protozoa, fungal infections

**Often self-limiting**

**Symptoms:** heart failure, tachycardia, gallop, flu-like symptoms

**Bed rest, fluid restriction, limited drug therapy**

**Infrequently progresses to dilated cardiomyopathy**

**Pericarditis** – inflammation of pericardium

**Secondary to a variety of conditions**
- Open heart surgery (leading cause), MI, viral, bacterial infection, anticoagulants, trauma
- Other causes: uremia, SLE, RA, malignancies-lymphomas affecting pericardium

**Acute pericarditis:** serous, serofibrous or purulent exudates on epicardial- pericardial surfaces

**Can result from blunt trauma:** MVA steering wheel, CPR

**Clinical presentation - often misdiagnosed as angina**

- **Pericardial friction rub.** ECG changes, pericardial effusion with tamponade, paradoxic pulse (pulsus paradoxus), pain, characteristic ECG changes, faint heart sounds, absent apical pulse; enlarged cardiac silhouette on CXR, echo shows fluid

- **Effusion:** collection of noninflammatory fluid which collects in pericardium
  - Serous, sanguinous, purulent, serosanguinous depending on etiology

- **Tamponade:** rapid accumulation of fluid sufficient to impair heart function
  - Must remove fluid on an emergent basis with tamponade

- **Pulsus paradoxus:**
  - Large inspiratory reduction in arterial pressure - SBP drops more than 10 mm Hg during inspiration

**Chronic Constrictive Pericarditis**

**Healing of acute pericarditis with formation of granular tissue**

**Gradually contracts to form firm scar surrounding heart**

**Scar constricts heart thus interferes with filling**

**Similar to pericardial effusion except it develops gradually over weeks to months**

**Clinical presentation**
- Weakness, fatigue, weight loss, anorexia, edema
- Abdominal discomfort (hepatic congestion and swelling)

- **Jugular neck vein distention** is characteristic sign

**Echocardiogram**
- Pericardial thickening and paradoxic septal motion
- Left ventricular wall moves outward in early diastole then little movement

**CXR:** calcifications on pericardium
CONGENITAL CARDIOVASCULAR DISEASE

Types: Shunts, Congestive Heart Failure

COMMON CONGENITAL CARDIOVASCULAR DEFECTS

**Patent Ductus Arteriosus**
- Embryonic ductus fails to close after birth
- Persistence results in shunt between pulmonary artery and aorta
- Second most common defect - may be isolated
- Systolic then machine-like murmur
- Increased volume and pressure in pulmonary system short circuiting V output
- Other symptoms: pulmonary congestion, heart failure

**Atrial Septal Defects**
- Very common - failure of atrial septum to close
- Various forms
  - Ostium primum
  - Persistent atrioventricular communis
  - Ostium secundum
- Clinical manifestations - (f) size of defect - most asymptomatic
  - Right V hypertrophy, respiratory infections, feeding difficulties, dyspnea, fatigability, growth retardation

**Ventricular Septal Defects**
- Most common heart lesion - 8-20% of congenital heart lesions
- Ventricular septum grows in cephalad fashion and fuses with endocardium
- Left to right shunt from high pressure in left ventricle to low pressure in right
- Shunt produces high-grade holosystolic murmur
- Palpable thrill
- Pulmonary hypertension and right ventricular failure
- Eisenmenger’s syndrome: pulmonary hypertension resulting in right-to-left shunt

**Tetralogy of Fallot**
- Primary causes of cyanotic heart disease - more common in males
- Combination pulmonary stenosis, VSD, dextroposition of aortic root, hypertrophy of right ventricle
- Clubbing, hypoxia, polycythemia, susceptibility to infection

**Transportation of the Great Vessels**
- Aorta arises anteriorly from right ventricle
- Pulmonary artery arises posteriorly from left ventricle
- These two closed circuits are incompatible with life

**Coarctation of the Aorta**
- High blood pressure and decreased circulation to lower extremities
- Headaches, dizziness, epistaxis, intermittent claudication, coolness or pallor to lower extremities
- Cyanosis of lower extremities - CHF after age 5 yrs
OTHER CONGENITAL HEART DEFECTS

- Total anomalous pulmonary venous connection
  Pulmonary veins are connected to right atrium
- Truncus arteriosus
  Embryonic truncus fails to divided into aorta and pulmonary arteries
- Endocardial cushion defect
  Valves and septi fail to form adequately
  May result in one chambered heart
- Ebstein’s malformation
  Downward displacement of tricuspid valve
  Results in part of right ventricle becoming part of right atrium
  Decreases output to lungs - increases right atrial pressure
  Possible associated ASD: right to left shunt causes cyanosis
- Tricuspid atresia:
  Tricuspid valve does not form or no right ventricle
  Hypoplastic pulmonary artery may be present
  PDA must be present to sustain life
  Right atrium enlarged - left ventricle hypertrophied
- Isolated pulmonic stenosis
  Relatively common
  Symptomatic when severe
- Bicuspid aortic valve
  Aortic valve with only two cusps
  Common and usually asymptomatic
- Mitral valve prolapse