MEDAVIE HealthEd ÉduSanté

# Cardiovascular Pathophysiology

DND Primary Care Paramedicine

Module: 04

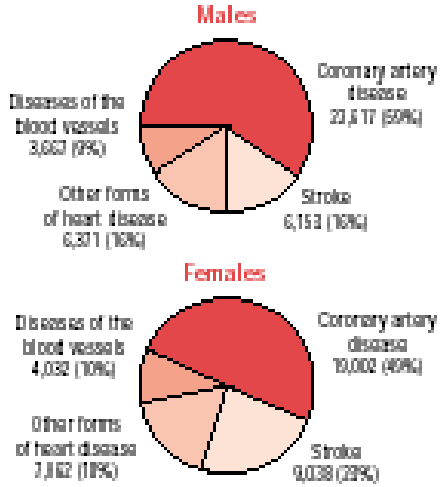
Section: 04

# Cardiovascular Disease

Leading Causes of Death

| | Canada | Ontario |
|---|---|---|
| Heart disease and stroke | 78,942 (36%) | 29,851 (37%) |
| Cancer | 62,768 (29%) | 23,189 (28%) |
| Respiratory disease | 22,026 (10%) | 7,745 (10%) |
| Accidents, suicide, violence | 13,956 (6%) | 4,372 (5%) |
| All other causes | 41,797 (19%) | 16,236 (20%) |
| Totals: | 219,530 (100%) | 81,393 (100%) |

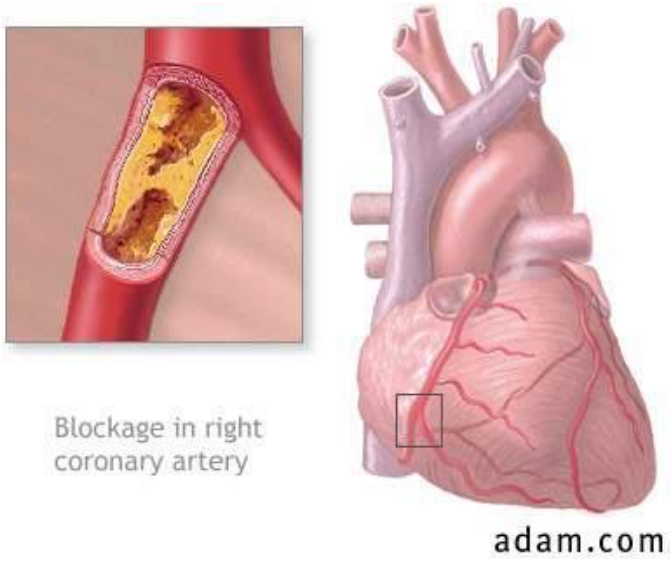
CVD Deaths in Canada by Gender




Stats: Canada 1999 (most recent data available)

- Accounts for the death of more Canadians than any other disease.
  - In 1999 (the latest year for which Statistics Canada has data), cardiovascular disease accounted for 78,942 Canadian deaths.
    - Male 35%
    - Female 37%
  - CAD 54%
  - Stroke 20%
  - Other problems 16%
    - Electrical system, viral heart infections, and heart muscle disease
  - Vascular problems 10%
    - HTN
- Cost of cardiovascular diseases to the Canadian economy is over $18 billion a year according to a 1994 study by the Heart and Stroke Foundation.

# Atherosclerosis




- Narrowing of the lumen of the medium and large arteries
- Narrowing caused by collection of plaque (atheromas) which decrease blood flow to the tissues
- Usually found in areas of turbulence (bifurcations of the vessels)
- Turbulence may rip atheromas free causing emboli and produces a lesion of the tunica intima
- Lesion provides access to the tunica media and platelets will adhere to the collagen producing further narrowing as the clot forms
- Lesions allow for Ca to enter the tunica media which calcifies the layer decreasing or destroying the elasticity of the vessel at that point (Arteriosclerosis)

# Atherosclerosis

- Risk Factors
  - Age and Sex
    - Increased age is a dominant risk factor
    - Predominant in Men at early ages, appears to be a 10 year lag for women though the gap closes after menopause
  - Family History
  - Smoking
    - Causes endothelial damage and therefore promotes plaque thrombosis

# Atherosclerosis

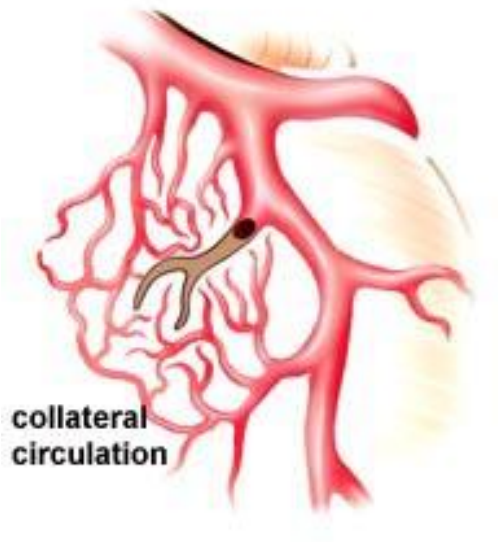
- Risk Factors
  - Obesity, Lack of Exercise
  - Hypercholesterolemia
    - Risk is proportional to serum level of LDL cholesterol.
    - Hyperlipidemia may be familial, and thus may account for the fact that a strong family history of premature CAD is a significant risk factor.
    - HDL cholesterol is protective.
  - Alcohol use
  - Stress
  - Sedentary Life Style
  - Type ‘A’ personality

# Atherosclerosis

- Risk Factors
  - Obesity, Lack of Exercise
  - Predisposing illness
    - Hypertension
      - Although definitely a risk factor, HTN alone probably does not cause plaques.
      - It may act synergistically with hypercholesterolemia by first causing mechanical wall stress and damage.
    - Diabetes Mellitus
      - Strong independent risk factor.
      - Believed to be a result of sugar level changes cause release of growth factors that stimulate smooth muscle proliferation
  - Race
    - Variances in statistics world wide but seems to equal out when lifestyles mimic national norm

# Atherosclerosis

- Effects
- The severity is dependent on:
  - Time of onset
  - Degree of obstruction
    - Most people with clinical disease have extensive CAD, usually with at least one major vessel showing severe narrowing of at least 75% reduction in the cross-sectional area of its lumen.
  - Pt's ability to produce collateral circulation



# Atherosclerosis

- Can manifest as any of four clinical entities
  - Angina Pectoris
  - Acute Myocardial Infarction
  - Sudden Cardiac Death
  - Chronic Ischemic Heart Disease

# Angina Pectoris

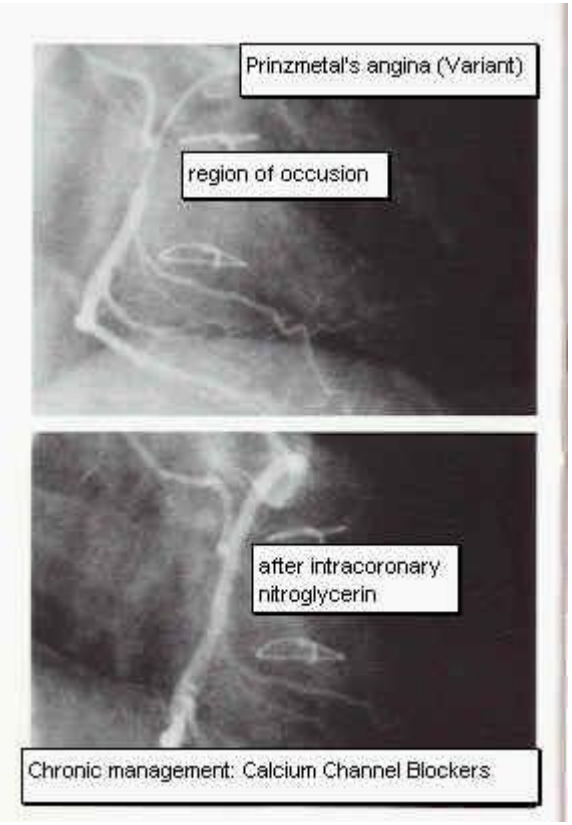
- Etiology
- When the circulatory system is unable to provide enough oxygen to meet the demands of the heart muscle
- Angina Pectoris means “Choking of the Heart”
- The lack of $O_2$ to the coronary vessels creates a build up of $CO_2$ resulting in anaerobic metabolism (without $O_2$). This metabolism’s major byproduct is Lactic Acid
- The lactic acid irritates the nerve endings in the heart muscle and produces a pain response
- Types of angina that will be discussed
  - Prinzmetal’s Angina
  - Stable Angina
  - Unstable Angina
  - Angina Decubitus

# Angina Pectoris

- Precipitating Factors
- Atherosclerosis tends to be major cause
- Emotional stress
- Increased activity

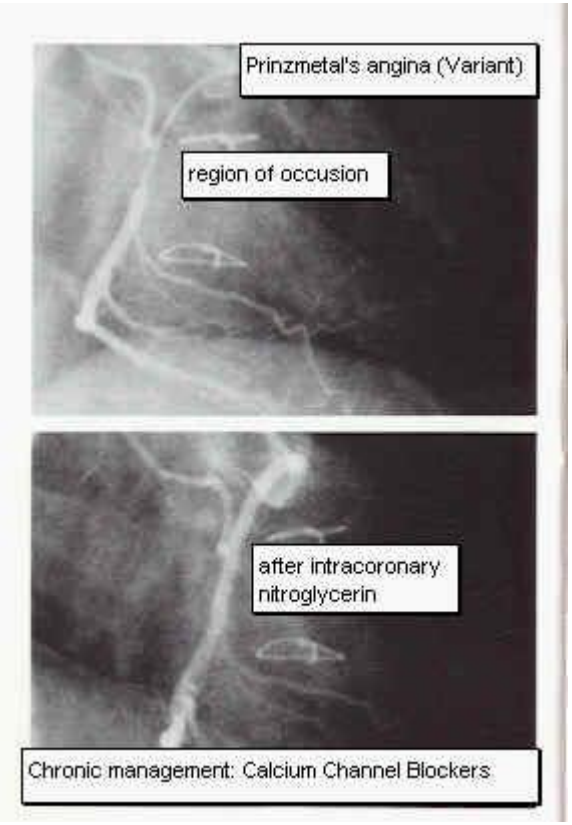
- Note:
  - If increased $O_2$ demands are not met, may lead to ischemia of the muscle
  - This ischemia may result in arrhythmias or infarction

# Prinzmetal's Angina




- Also known as Variant Angina Pectoris
- Caused by temporary spasm of coronary vessels and may or may not include atherosclerosis
  - About 2/3 have severe coronary atherosclerosis in at least one major vessel.
  - The spasm usually occurs very close to the blockage.
  - Can occur in people with valvular heart disease, hypertrophy or uncontrolled HTN
- Appears to be substantially less common than typical forms
- Unlike typical angina, it nearly always occurs when a person is at rest.
  - It doesn't follow physical exertion or emotional stress, either. Attacks can be very painful and usually occur between midnight and 8 a.m.

# Prinzmetal's Angina




- Signs and Symptoms
  - typically complains of a pressure-like, squeezing retrosternal chest discomfort of several minutes duration
  - Transient ST elevation in association with chest pain
    - both of which resolve spontaneously or with nitroglycerin
- Usually treated with calcium-channel blockers with or without long-acting nitrates

# Angina Decubitus

- Occurs when a person is lying down (not necessarily during sleep)
- Due to fluids being redistributed in this position due to gravity, and the heart has to work harder
- Usually a complication of cardiac failure due to the strain on the heart resulting from the increased intravascular volume
- Patients usually have severe coronary artery disease.

# Stable Angina

- AKA “Exertional Angina”
- Usually precipitated by exertion or stress
- Pain lasts up to 10 minutes (may last up to 15 minutes)
- Usually relieved with rest, $O_2$ administration and NTG
- Attacks tend to be similar to each other and are typically relieved in the same manor (though we do allow for variances)

# Unstable Angina

- AKA “Pre-infarction Angina”
- Usually a change from the stable angina’s normal presentation (in either precipitating factor, frequency, duration, intensity and quality of the pain)
- Includes new-onsets of angina pain
- May occur with light exertion and may even come on at rest
- Pain usually lasts 10 minutes or more
- Responds to rest, $O_2$ administration and NTG though may take increasing amounts compared to stable
- May mimic MI S/S and should be treated as such

# Angina

- Signs and Symptoms
- Chest Pain
  - Described as burning, heavy, pressure, tightness, squeezing
  - May radiate to shoulders, jaw, arm, neck and back
- Anxious
- Dyspnea
- Cool to the touch
- Diaphoresis
- N/V
- Syncope

# Angina

- Management
- Primary Survey (ABC's, RBS)
- Place pt at rest (Sitting or lying)
- Emotional Support
- Administer $O_2$ (100% via NRB)
- Baseline Vitals
- Administer Aspirin (160 mg PO)
- Administer NTG (0.4 mg SL, q 3-5 min with BP > 100/50)
- Initiate IV (Lock preferred)
- Monitor (3 lead, 12 lead)
- Transport

# MI

- Etiology
- Results from partial or total occlusion of $O_2$ rich blood to the cardiac tissue
- This results in ischemia, injury and death (necrosis) of the tissue distally to the occlusion
- May be caused by:
  - atherosclerosis
  - rupture of the vessels
  - angina
  - severe hypoxia
  - shock

# MI

- Size of infarct is determined by:
  - the metabolic needs of the tissue
  - presence of collateral circulation
  - duration of time before reperfusion is established
- Most AMI's occur in the L Ventricle or the interventricular septum (40 – 50%)
- R ventricle MI is usually a result of occlusion to the RCA (30 – 40%)
- Lateral wall is usually L circumflex (15 – 20%)
- Are typically classed into 3 categories
  - Unstable angina
  - Non-Q-Wave MI
  - Q-Wave MI

# MI

- Unstable angina
  - Not a complete obstruction that may progress
- Non-Q-Wave MI
  - Evident only with ST elevation or T wave abnormalities
- Q-Wave MI
  - Abnormal Q wave in 2 or more leads
    - > 5 mm in depth or > 0.04 s in duration

# MI

- As with angina the lack of $O_2$ causes the cells to switch to anaerobic metabolism and produces a build up of lactic acid an $CO_2$
- Cells begin to lose ability to maintain electrical charge and remain depolarized (reversible at this point)
- After a period of time, tissue distal to occlusion will be necrotic and is replaced with scar tissue over a period of weeks
- These scar tissue areas may result in weaker tissue and develop into aneurysms on the ventricular walls or ventricular rupture

# MI

- Death Secondary to MI
- Lethal arrhythmias
  - VT
  - VFib
  - Cardiac Standstill
- Pump Failure
  - Cardiogenic Shock
  - CHF
- Rupture of Myocardium
  - Ventricle, Septum or papillary muscle

# MI

- Signs and Symptoms
- Chest Pain
  - Onset most likely will occur at rest and is not alleviated by rest or NTG
  - May be absent (with Diabetics and some elderly); known as “Silent MI”
  - May only have symptoms of dyspnea, syncope or confusion
- Dyspnea
- Cyanosis
- Agitation
- N/V
- Diaphoresis
- Palpitations
- Sense of impending doom
- Syncope

# MI

- Vitals
- Variant based on size of infarct
- Inferior
  - Parasympathetic response
- Anterior
  - Sympathetic response

# MI

- Management
- Primary Survey (ABC's, RBS)
- Place pt at rest (Sitting or lying)
- Administer $O_2$ (100% via NRB)
- Baseline Vitals
- Administer Aspirin (160 mg PO)
- Administer NTG (0.4 mg SL, q 3 - 5 min with BP > 100/50)
- Initiate IV (Lock preferred)
- Morphine (2.5 mg IV, q 3 – 5 min)
  - If pain is not relieved after 3 sprays of NTG
- Monitor (3 and 12 leads)
- Treat dysrhythmias as required
- Transport

# MI

- Management Keys
- Return patency of coronary circulation
  - Fibrinolytics, angioplasty, CABG
- Decrease area of ischemia/infarct
- Other pharmaceutical adjuncts
  - Heparin
  - Magnesium Sulphate
  - ACE Inhibitors
  - B-Blockers
  - Ca Channel Blockers

# Anticoagulant Therapy

- Heparin
  - Inhibits growth of thrombus
  - Inhibits formation of new thrombus
- Warfarin (Coumadin)
  - Inhibit Vitamin K use thus inhibiting coagulation
- Novasen (ECASA)
  - Inhibit the Thromboxane A-2 thus inhibiting aggregation
- Integrilin and Clopidogrel
  - Antithrombotic agent that reversibly inhibits platelet aggregation by preventing binding of fibrinogen to the GP IIb-IIIa receptor.

- Fragmin (LMWH)
  - Only small chains of polysaccharides
  - Works the same as heparin
- Fondaparinux (Arixtra)
  - a synthetic pentasaccharide
  - Inhibits thrombin formation

# Fibrinolytic Therapy

- Pharmaceutical support to dissolve a thrombus thus restoring blood flow to ischemic tissue
- Common forms
  - Streptokinase
    - acts with plasminogen to form a “activator complex” that converts residual plasminogen into plasmin
  - Tissue Plasminogen Activator (t-Pa)
    - converts the proenzyme plasminogen to plasmin See each individual drug for specifics:
    - Alteplase (Natural form), Reteplase and Urokinase (rTPA)
  - Tenecteplase (TNK)
    - rTPA
- Time restraints
- Eligibility restraints

# Fibrinolytic Therapy

- ISIS 2 Study (Second International Study of Infarct Survival) – 1988
  - Was a randomized trial of intravenous streptokinase, oral aspirin, both, or neither among 17,187 cases of suspected AMI
  - Showed ASA alone improved risk of CV death by 20%
  - When added to management with Streptokinase showed a 40% reduction
- West Study?

# Fibrinolytic Therapy

- Eligibility
  - AMI clinical presentation
  - ECG Criteria
  - Absence of contraindications
  - Absence of cardiogenic shock

# Fibrinolytic Therapy

- Inclusions
  - Pain of ischemic origin
  - Time > 20 minutes but < 12 hours
  - > 30 y/o (some suggest < 75 y/o)
  - A/O X 3
  - BP (Systolic < 180 mmHg, Diastolic < 110 mmHg)
  - STE > 1 mm (0.1 mV) in 2 or more contiguous leads
- Exclusions
  - CVA, IC bleed or CNS dysfunction in last 6 months (some suggest 1 year)
  - Active bleeds or bleeding disorders (menses excluded)
  - Suspected Aortic Dissection

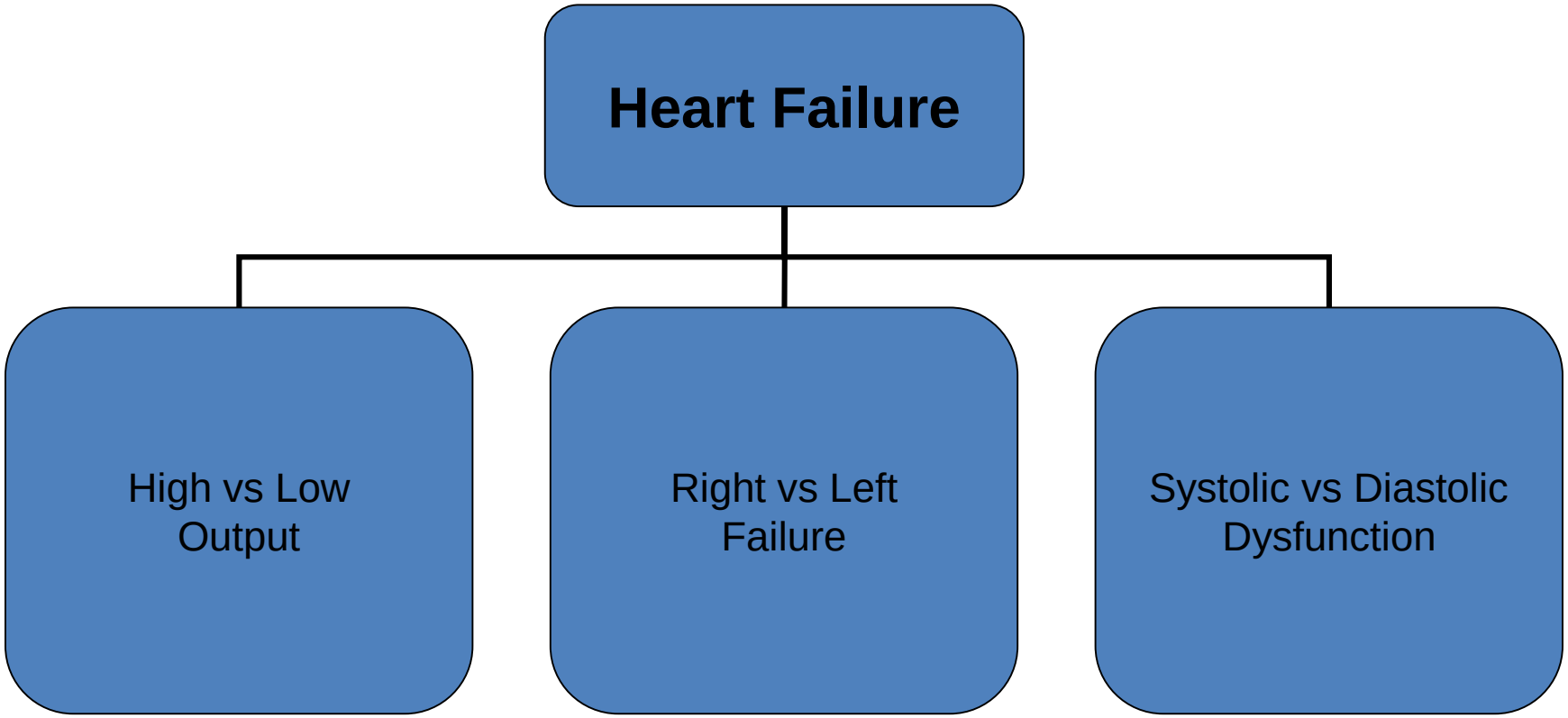
# Fibrinolytic Therapy

- Relative Contraindication
  - Pregnancy or post partum state
  - Uncontrolled HTN
  - Major surgery within last 3 weeks
  - Intercranial tumor
  - AAA
  - CPR
  - Trauma in last 2 to 4 weeks
  - Use of anticoagulants
  - Terminal illness
  - Other possible criteria based on hospital/EMS protocols

# Heart Failure

- The inability for the heart to function at its normal capacity
- May be caused by
  - Myocardial ischemia (Acute or chronic)
  - Valvular dysfunction (Aortic, mitral and prosthetic)
  - LV outflow obstruction (Aortic stenosis)
  - Idiopathic myopathy (Hypertrophy)
  - Acquired myopathy (toxic, metabolic)
  - Myocarditis (radiation, infection)
  - Pericarditis
  - Pericardial tamponade
  - Systemic HTN
  - Dysrhythmias
  - Anemia

# Heart Failure



# High vs Low Output

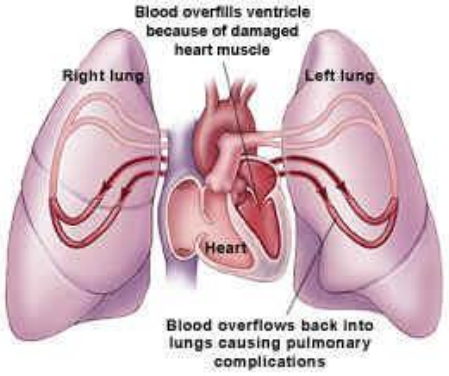
- Low output
  - Inherent problem in myocardial contraction
- High output
  - Inability to meet excess functional demands
  - Anemia, thyrotoxicosis (Grave's Disease) and even large AV shunts

# Systolic vs Diastolic

- Systolic
  - Impairment of myocardial contraction
    - Increased afterload
    - Damaged myocytes (necrosis)
- Diastolic
  - Impairment of myocardial relaxation
    - Myocardial ischemia
    - Hypertrophy

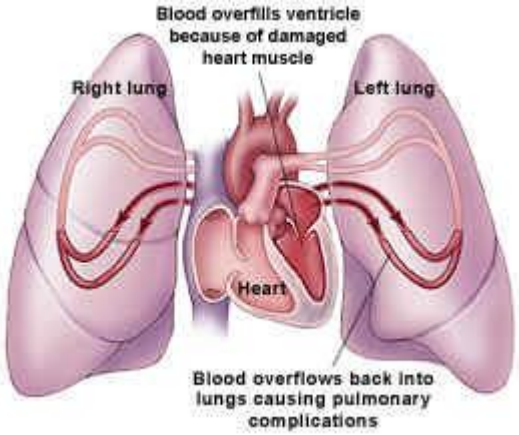
# Left Ventricular Failure

- Failure is as a result of damage to the function of the left side of the heart
- With ineffective pumping of the ventricle an increase in LVEDV and LVEDP.
- Results in a back-up of blood into the pulmonary circulation as the pressure is transferred into the atrium and pulmonary veins.
- This increase in pressure increases hydrostatic pressure and forces plasma into the alveoli (Pulmonary Edema)
- May be as a result of:
  - AMI or Ischemia
  - Valve damage
  - Hypertension



# Left Ventricular Failure

- The resulting failure decreases SV and the body tries to maintain homeostasis
  - Tachycardia, vasoconstriction activation of renin-angiotensin-aldosterone system (increasing BP)
- This increases oxygen demand of the heart and results in further complications



# Left Ventricular Failure

- Signs and Symptoms
- Dyspnea
  - Orthopnea
  - Spasmodic cough with foamy pink sputum
  - Paroxysmal nocturnal dyspnea
- Apprehension, agitation or confusion
- Cyanosis
- Diaphoretic
- Adventitious lung sounds (crackles, rhonchi, wheezes – “cardiac asthma”)
- JVD (if pressure backs up into the R ventricle)
- Hypertension
- Tachycardia
- Tachypnea, laboured
- Chest Pain
- Heart sounds
  - S3 gallop (early diastolic due to abnormal filing of the dilated ventricle)
  - S4 (forceful atrial contraction due to stiff ventricle)

# CHF – with Pulmonary Edema

- Management
- Keyed on
  - decreasing venous return to the heart
  - Decreasing $MvO_2$
  - Improving oxygenation
- Primary Survey (ABC's, RBS)
- Place pt at rest (Sitting with legs down)
- Baseline Vitals (Include auscultation)
- Administer $O_2$ (100% via NRB) assist if required
  - May require CPAP or BiPAP, consider PEEP of 5 – 10 $cmH_2O$
- IV Lock
- Pharmacological support
- Monitor (3 and 12 lead)
- Transport

# CHF – with Pulmonary Edema

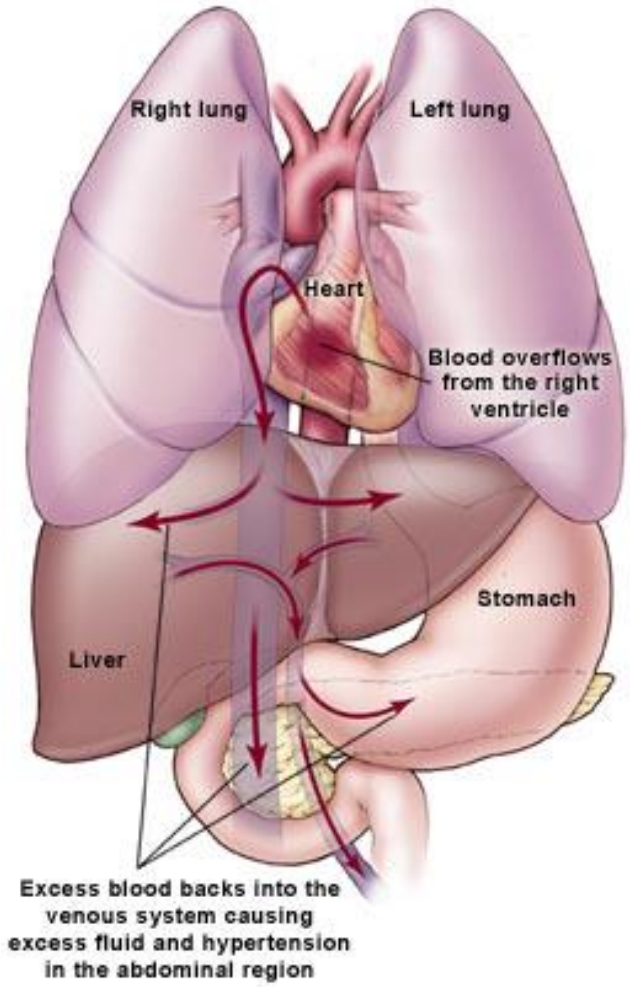
- Pharmacology
- NTG (0.4 mg SL, q 3 – 5 minutes)
  - decrease preload and afterload thus decreasing $MvO_2$
- Ventolin (5 mg Aerosol - if wheezing present and decompensating)
- Furosemide (0.5 – 1.0 mg/kg or double home dose)
  - Vasodilatation and decrease in intravascular volume
- Morphine (2. 5 – 5.0 mg IV)
  - Decrease $MvO_2$ with dilation of venous system and decrease respiratory effort
- Consider nitrate drip
  - NTG (5 µg/min and increase until desired SBP is achieved)
  - Nitroprusside (0.1 – 5.0 µg/kg/min)
- ACE inhibitors (Captopril, Monopril, enalapril)
- Cardiogenic shock or hypotensive
  - Dopamine (5 to 15 µg/kg/min)
  - Dobutamine (2 to 20 µg/kg/min)
  - Levophed (0.5 – 30 µg/min)

# Right Ventricular Failure

- Etiology
- Results from failure of the right side of the heart
- May be caused by LVF as the increased pressure is returned to the RV through the pulmonary circulation
- Right atrium is unable to keep up with venous return and produces a back-up in the systemic system
- This back-up creates an increase in venous pressure, increasing hydrostatic pressure on the venous end of the capillary.
- This increase in turn inhibits the return of plasma to the system and fluid accumulates in the tissue (edema)

# Right Ventricular Failure

- May be as a result of:
  - LVF
  - Hypertension
  - COPD
  - PE
  - Value disease
  - Right MI



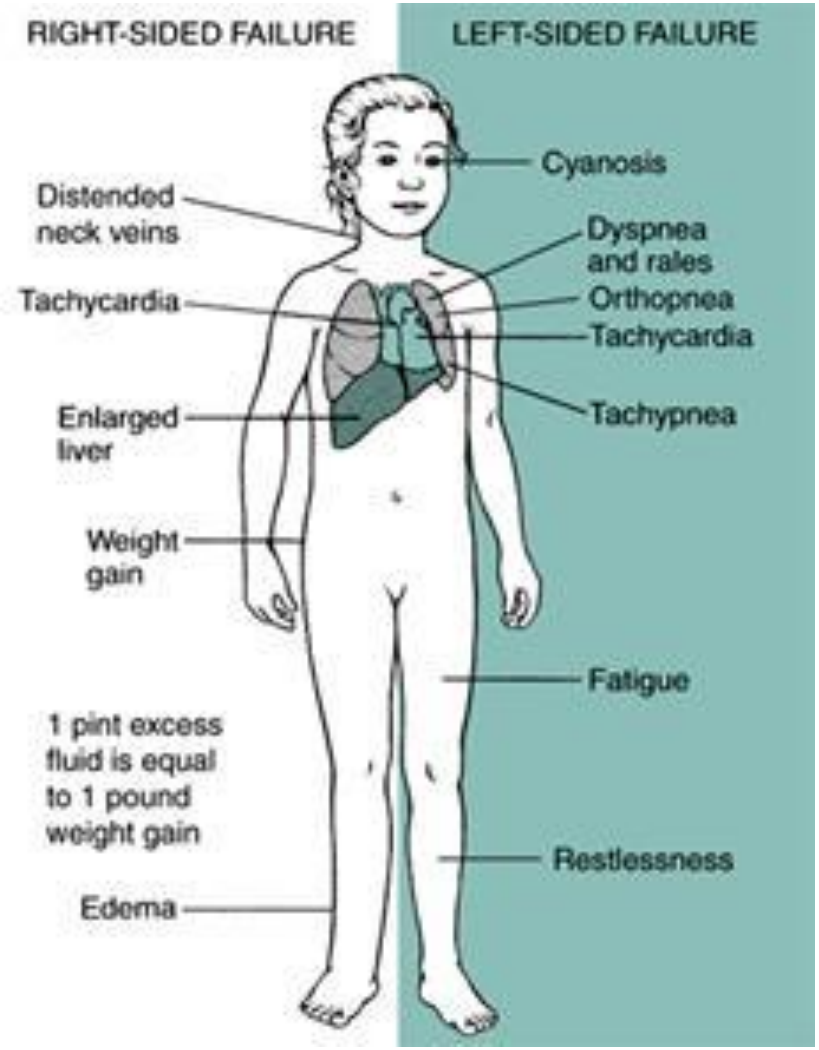
# Right Ventricular Failure

- Signs and Symptoms
- Chest pain, may have RUQ pain
- Dyspnea
- Hypotension
- Tachycardia
- Venous congestion
  - Engorged liver and/or spleen (hepatomegaly, splenomegaly)
    - May see hepatojugular reflux
  - Distend Neck Veins
- Peripheral Edema (may be pitting)
  - feet and hands, entire body
  - sacral area for the bedridden
- Edema in serous cavities
  - Peritoneum (Abdomen) - causes ascites
  - Pericardium – pericardial effusion (may be able to tolerate large quantities if develops over an extended period of time)

# Right Ventricular Failure

- Management
- Primary Survey (ABC's, RBS)
- Place pt at rest (Sitting with legs down or semi-Fowler)
- Baseline Vitals
- Administer $O_2$ (100% via NRB)
- Initiate IV (Fluid challenge for hypotension to relieve LV filling)
- Monitor
- Treat LVF if present as well (watch fluids)
- Transport

# CHF - Comparison



# Cardiogenic Shock

- Etiology
- Occurs as a result of complete failure of the pump to meet the metabolic needs of the body
- Usually resulting from massive MI (> 40%) or by large areas (diffuse) ischemia
- The decrease in function decreases SV, which also decreases CO and eventually the BP
- Results in inadequate perfusion of organs
- High mortality rate (> 70%) even with aggressive therapy

# Cardiogenic Shock

- Signs and Symptoms
- An accumulation of S/S of shock and AMI
- Altered LOC
- Hypotension (SBP usually < 80 mmHg)
- Dyspnea or tachypnea
  - Pulmonary edema with crackles
- Hypoxemia
- Acidosis
- Tachycardia
- Cool, clammy skin maybe pale, cyanotic or ashen in color
- Chest pain
- Sense of impending doom

# Cardiogenic Shock

- Management
- Primary Survey (ABC's, RBS)
- Place pt at rest (Supine or semi-Fowler if dyspneic)
- Baseline Vitals
- Administer $O_2$ (100% via NRB) assist if necessary
  - Auscultation for adventitious sounds
  - May require aggressive airway management
- Initiate IV (TKVO)
- Monitor
- Transport (Emergent situation – do not delay on scene time)
- Pharmacological support
  - Inotropic agents (Dopamine or dobutamine)

# Hypertension

- Etiology
- A chronic condition where BP is consistently greater than 140/90 mmHg
- The increase creates an increase PVR and forces the heart to work harder to overcome it by increasing rate and SV
- This excess workload, as with any muscle, causes it to enlarge (hypertrophy) which will eventually lead to failure

# Hypertension

- Categories
  - Transient
  - Mild (uncomplicated)
  - Urgencies
  - Emergencies

# Hypertension

- Transient
  - May be found in
    - Anxiety
    - Pancreatitis
    - Early dehydration
    - Alcohol withdrawal
    - Drug OD
    - Catecholamine induced HTN emergencies
      - MAOI OD, phenochromocytoma, Tyramine (an enzyme contained in many foods, especially aged cheese) with MAOI
  - Treatment is based on correcting the cause versus treating the HTN

# Hypertension

- Mild (uncomplicated)
  - Defined as a diastolic pressure < 115 mmHg without S/S of end organ damage
  - Acute management not required
  - May require medication support

# Hypertension

- Urgencies
  - Elevation of DBP > 115 mmHg without S/S of end organ damage
  - Reduce pressure over 24 – 48 hours
  - Commonly seen with non-compliance of HTN medications

# Hypertension

- Emergencies
  - Increase in BP with end organ damage or dysfunction
  - Is not determined by the BP but the degree of end organ dysfunction
    - Eclampsia may result in BP of 160/90 mmHg
  - S/S can progress over hours to days
  - Treatment must be initiated ASAP to prevent further damage

# Hypertension

- Emergencies
  - Management
    - Lowering of BP to a level consistent with the patient's normal in a controlled graded manner
    - Recommend 30% reduction in 30 minutes
    - End point is resolution of S/S
  - May use
    - Nitrates (Nitroprusside, NTG)
    - Beta-blockers (Propranolol, Esmolol, Metoprolol)
    - Ca channel blockers (Verapamil, Nefedipine, Diltiazem)
    - Diuretics (Furosemide)
    - ACE inhibitors (Captopril)
    - Clonidine (Catapres – decreases renin production)

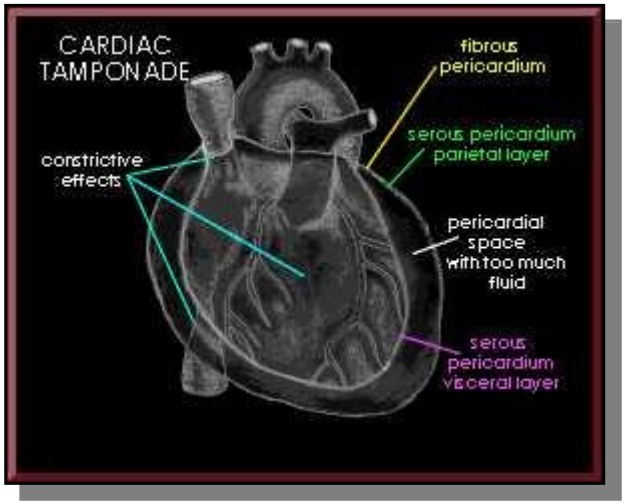
# Hypertension Emergencies

- Where the hypertension leads to irreversible end-organ damage such as the heart, brain or kidneys
- Include
  - MI with HTN
  - Aortic Aneurysm with HTN
  - Pulmonary Edema with HTN
  - Intracranial hemorrhage with HTN
  - Toxemia with HTN (pregnancy, sepsis)
  - Encephalopathy with HTN

# Hypertension Emergencies

- Signs and Symptoms
- Paroxysmal nocturnal dyspnea
- SOB
- ALOC
- Vertigo
- Headache
- Epistaxis
- Tinnitus (ringing in the ear)
- Changes in visual acuity
- N/V
- Seizures
- ECG changes

# Pericardial Tamponade




- Defined as impaired diastolic filling of the heart by increased intrapericardial pressure and volume
- Will alter the ability of the atria and ventricles to fill
- Thus decreasing SV
- Maybe
  - Gradual in onset (neoplasm, infection)
  - Acute (trauma, CPR)

# Pericardial Tamponade

- Signs and Symptoms
- Chest pain
- Tachycardia
- Ectopics
- Other ECG changes
  - Low-voltage QRS and T waves
  - ST elevation or non-specific T wave changes
- JVD (elevated venous pressures)
- Decreased SBP (late)
- Pulsus paradoxus
- Faint or muffled heart sounds

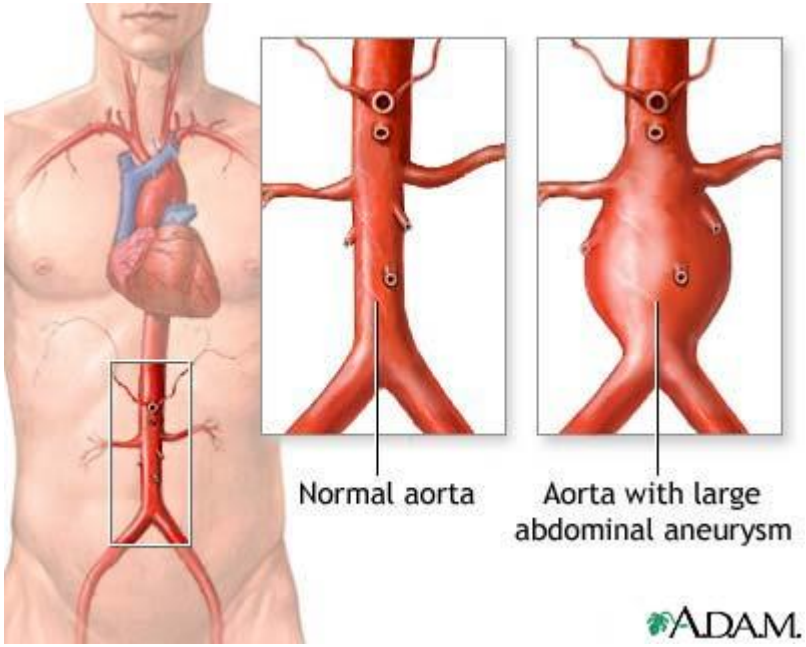
# Pericardial Tamponade

- Management
- History
- Ensure adequate oxygenation
- Transport
- If hypotensive consider fluid challenge
- Definitive treat would include pericardiocentesis

# Aortic Aneurysm

- Etiology
- Dilatation of a vessel through the weak lining of the lumen
- Usually develop in areas where the tunica media is weak and allows for expansion
- This increases turbulence and pressure and may eventually rupture
- May be caused by
  - Atherosclerosis
  - Infectious diseases
  - Trauma
  - Genetic disorders

# Abdominal Aortic Aneurysm




- Usually occurring below the renal arteries before the aorta divides into the common iliac
- 10 X more common in men
- Most prevalent between 60 – 70 y/o
- May remain asymptomatic if stable
- If unstable, leaking and rupture may occur as more blood fills the cavity
- Upon rupture the retroperitoneal tissues may tamponade the leak and the pt will present asymptomatic
- If tamponade does not occur than massive hemorrhaging will begin
- Either will develop S/S of shock

# Abdominal Aortic Aneurysm

- Signs and Symptoms
  - May present with syncope followed by hypotension and bradycardia (vagal response)
  - Unexplained hypotension
  - Unexplained Syncope
  - Sudden onset of abdominal pain or tearing/ripping back pain
  - Low back pain or flank pain radiating to the groin, thigh or perineum that is not relieved with rest or position
  - Peritoneal irritation
  - Urge to defecate
  - Pulsatile mass above the umbilicus (left of midline) – usually greater then 5 cm
  - Weak or absent distal pulse
  - Hardening and distention of abdominal cavity

# Abdominal Aortic Aneurysm

- Management
  - Primary Survey (ABC's, RBS)
  - Gentle handling of pt
  - Place pt at rest
  - Baseline Vitals
  - Administer $O_2$ (100% via NRB) assist if necessary
  - Initiate IV (TKVO but if ruptured fluid challenge)
  - Attach monitor
  - Transport

# Dissecting Aortic Aneurysm

- Usually an acute occurrence that may be caused by
  - Systemic hypertension
  - Atherosclerosis
  - Congenital abnormalities
  - Degenerative changes in the aortic tunica media
  - Trauma
  - Pregnancy
- Results from a small tear of the tunica intima and rapid filling of the medial layer causing rupture of the adventitia
- May happen in any section of the aorta, but is more common in the ascending aorta
- Affects twice as many men as women

# Dissecting Aortic Aneurysm

- As the aneurysm expands it may impede blood flow to areas by blocking their respective arteries as they branch off the aorta
- For these reasons DAA may result in
  - Syncope
  - Stroke
  - Absent or reduced pulses
  - LVF (as a result of aortic semilunar valve regurgitation)
  - Pericardial tamponade
  - AMI

# Dissecting Aortic Aneurysm

- Signs and Symptoms
  - Altered LOC
  - Sudden onset of pain (> 70%)
    - Tearing, ripping or cutting
    - Located in the back, epigastrium, abdomen or extremities depending on location of AA
    - Pain may radiate to from the interscapular region downward
    - Pain is maximal from onset
  - Pale, cool and clammy
  - Peripheral cyanosis
  - Significant differences in BP from R to L
  - Peripheral pulses are unequal

# Dissecting Aortic Aneurysm

- Management
  - Primary Survey (ABC's, RBS)
  - Gentle handling of pt
  - Place pt at rest and reduce stress
  - Baseline Vitals
  - Administer $O_2$ (100% via NRB) assist if necessary
  - Initiate IV (TKVO but if ruptured fluid challenge)
  - Attach monitor
  - Transport
  - Prepare for aggressive management if rupture occurs

# Arterial Occlusion

- A sudden blockage of an artery by trauma, embolus or thrombosis
- Severity depends on location and size as well as collateral circulation
- Ischemia begins distal to occlusion and may eventually lead to necrosis of tissue
- Common types are
  - CVA/TIA
  - MI
  - PE

# Arterial Occlusion

- Signs and Symptoms
  - Pain in extremity distal to occlusion
  - Pallor distal to site (mottled or cyanotic)
  - Lower temperature distal to site
  - Changes in sensation and function
  - Weak or absent pulses
  - Bruit over site (auscultated turbulence)
  - Decrease capillary refill
  - May see S/S of shock

# Arterial Occlusion

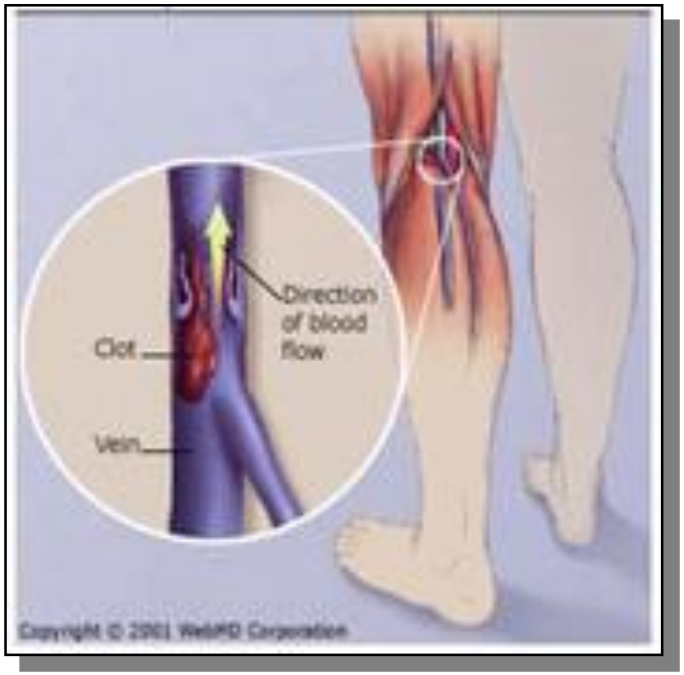
- Management
  - Immobilize the affected limb
  - Manage shock if present
  - Consider analgesic (with consultation with OLMC)

# Peripheral Vascular Disease

- Includes
  - Varicose veins
    - Permanent dilation of the veins, may result from weak valves (typically in the legs)
    - May be seen in pt's whose occupation requires long periods of standing, pregnancy
  - Superficial Thrombophlebitis
    - Inflammation of a vein associated with the formation of a thrombus
  - Deep Vein Thrombosis
    - May produce life threatening PE

# Deep Vein Thrombosis

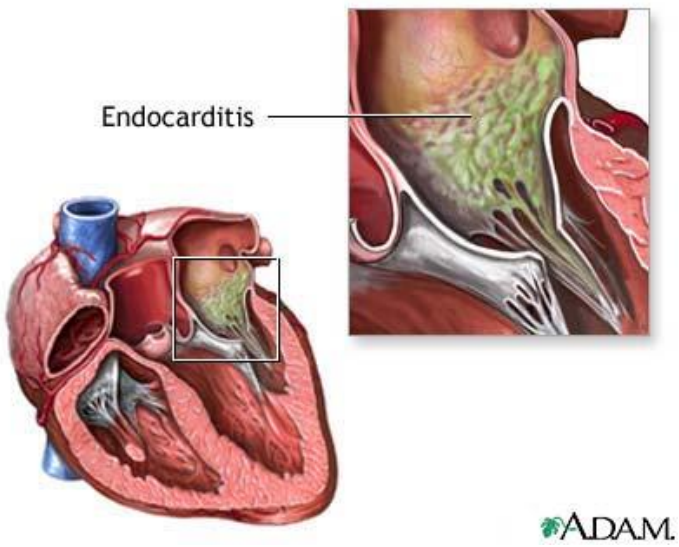
- Occlusion of the deep veins
- Most common in the lower extremities
- Risk factors
  - Recent trauma
  - Advanced age
  - Recent MI
  - Inactivity
  - CHF
  - Cancer
  - Oral contraceptives
  - History of thrombus
  - Obesity
  - Smoking



# Deep Vein Thrombosis

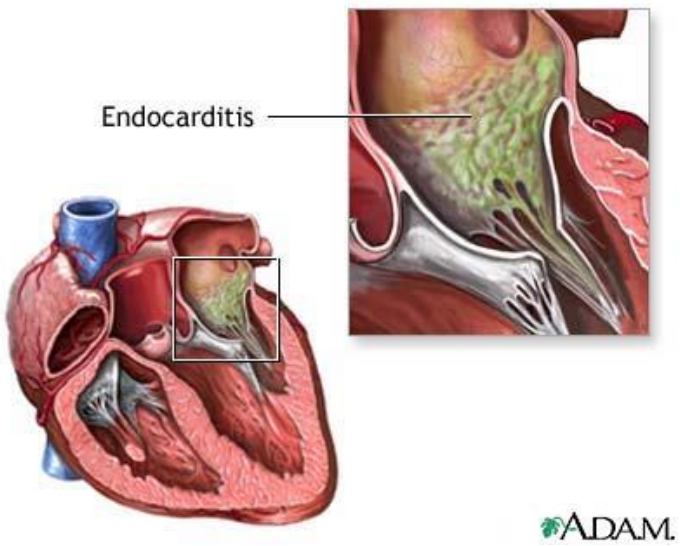
- Signs and Symptoms
  - Pain
  - Edema
  - Warmth
  - Erythema (redness of the skin) or cyanosis
  - Tenderness
- Management
  - Immobilization and elevation of the extremity

# Endocarditis



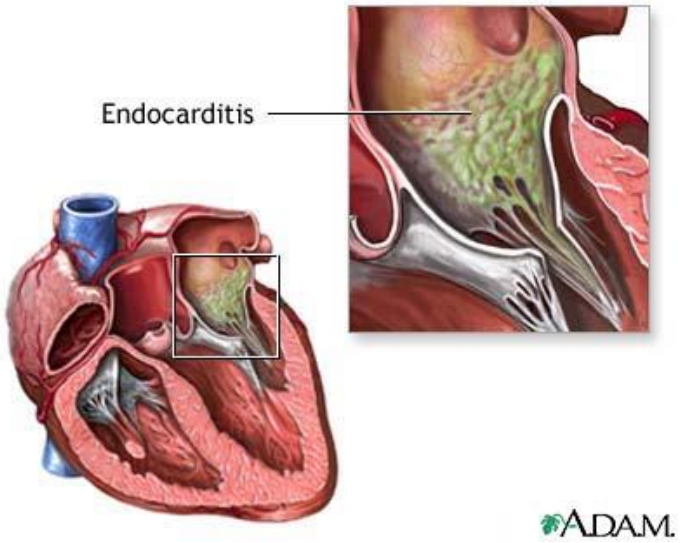

- Infection and inflammatory process that affects the lining of the heart and valves
- Detected by performing blood cultures and an echocardiogram
- Usual cause is a bacteria (staph or strep) or by a fungal infection
  - The bacteria or fungus can enter the bloodstream from infections elsewhere in the body (urinary tract, GI tract, or the skin), or as a result of any surgical or dental procedure.

# Endocarditis




- Signs and symptoms
  - Chest pain
  - Fever
  - Fatigue, weakness
  - Chills and night sweats
  - Muscle and joint pain
  - Heart murmur
  - Late signs may include swelling of the feet and legs, and shortness of breath with an irregular heartbeat.

# Endocarditis



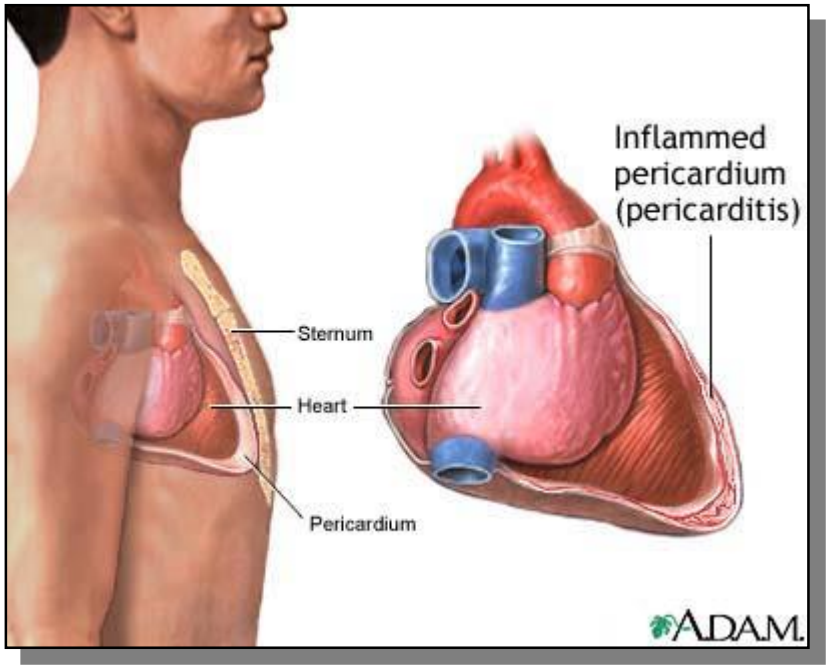

- Treatment
  - Antibiotics
  - Non-aspirin medications such as Tylenol can be used for fever and minor pain
  - A regular diet can be followed as tolerated.
  - Fluid intake should be increased while fever is present.
  - Good dental hygiene is needed to prevent infection.

# Myocarditis

- Inflammation of the myocardial layer by an inflammatory response to an injury or infection.
- May be caused by radiation or side effects of some medications.
  - Most commonly it is caused by a virus

- Signs and Symptoms
  - Chest pain (from fluid collection)
  - Dyspnea
  - Fever or chills
  - Fatigue
  - Some patients have a rash or joint pain (arthritis) related to rheumatic fever (from previous streptococcal infection)
  - Arrhythmias
- Treatment
  - Symptoms generally clear up with rest and time.
  - Avoid strenuous exercise until the condition has completely cleared.
  - Management of chest pain and arrhythmias is most important.
  - if heart failure occurs, treat as needed.
  - NSAIDs
    - In more severe instances, steroid-containing medications or immunosuppressive drugs are used.
  - Antibiotics are given for acute rheumatic fever or other infections.

# Pericarditis



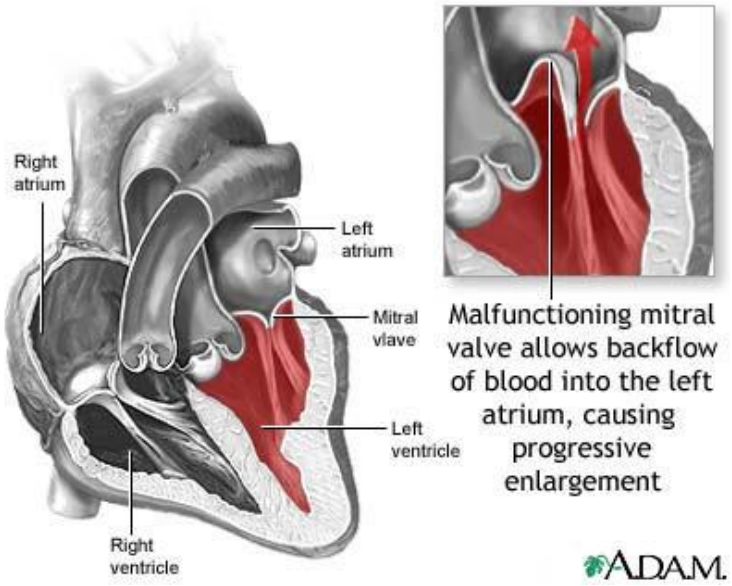

- Inflammation of the pericardium
- Possible causes
  - Young, otherwise healthy persons who develop pericarditis often have had a recent viral infection
  - MI
  - Kidney failure caused by the buildup of certain toxins
  - Tumors (Cells from tumor may metastasize to the pericardium)
  - Radiation therapy
  - Tuberculosis
  - Overactive immune system (rheumatoid arthritis and lupus)

# Pericarditis

- Signs and Symptoms
  - Chest pain
    - predominantly felt below the sternum and/or below the ribs on the left side of the chest and, occasionally, in the upper back or neck.
    - Increased on inspiration, lying supine
  - Pericardial effusion
  - Low grade Fever
  - Dyspnea
    - of concern because it may indicate that the amount of fluid is reaching a critical point and requires urgent medical treatment
  - Friction rub
  - PR segment depression with diffuse STE without Q wave changes
  - Need to rule out other causes of CP
    - MI, DAA, PE, Pneumothorax, Digestive tract perforation
- Treatment
    - Anti-inflammatory agents (Aspirin)
    - NSAIDs (ibuprofen, Motrin, Aleve)
    - If pericardial effusion is significant treat as tamponade

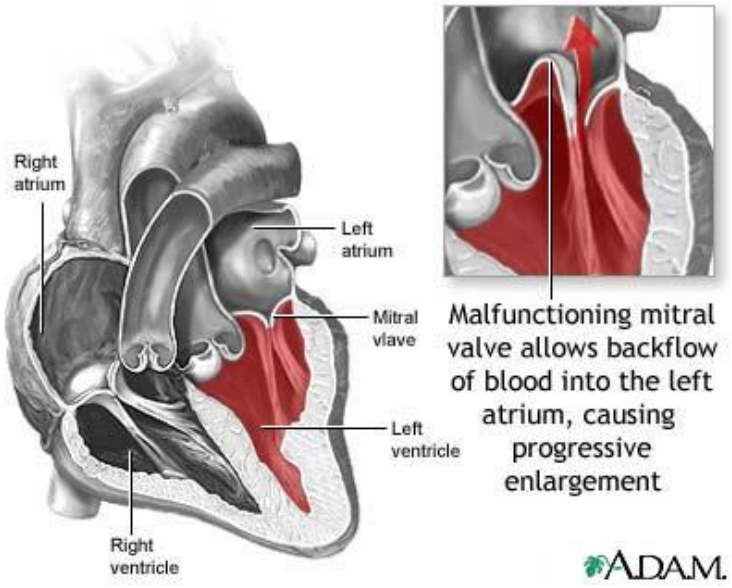
# Mitral Valve prolapse

- Valve billow upward during atrial systole
- Mitral regurgitation may occur
- Seen in young women and may be inherited
- May be asymptomatic
- If symptomatic may see
  - Palpitations (tachycardia)
  - Syncope, lightheadedness
  - Fatigue, lethargic, weakness
  - Dyspnea, hyperventilation
  - Chest tightness
  - Anxiety
  - Atypical chest pain



# Mitral Valve prolapse

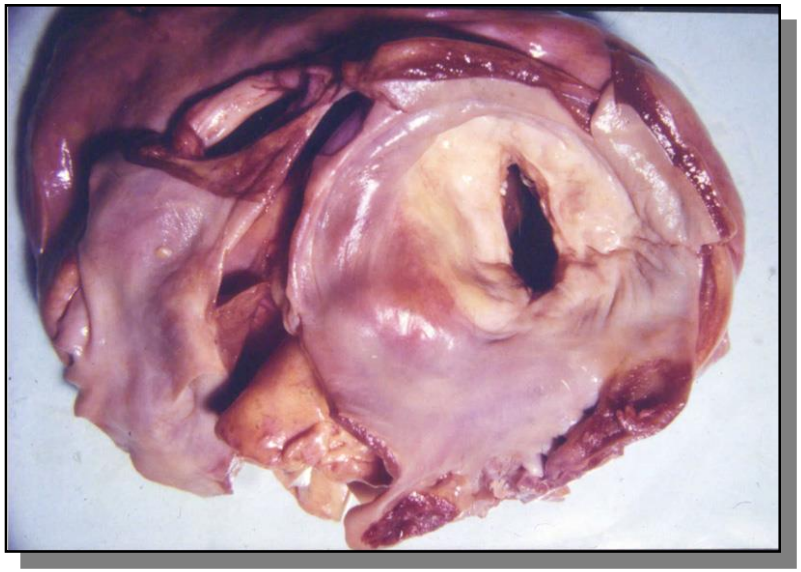
- Treatments include
  - Antibiotics for endocarditis
  - Beta-blockers
  - Surgical repair



# Valve Problems

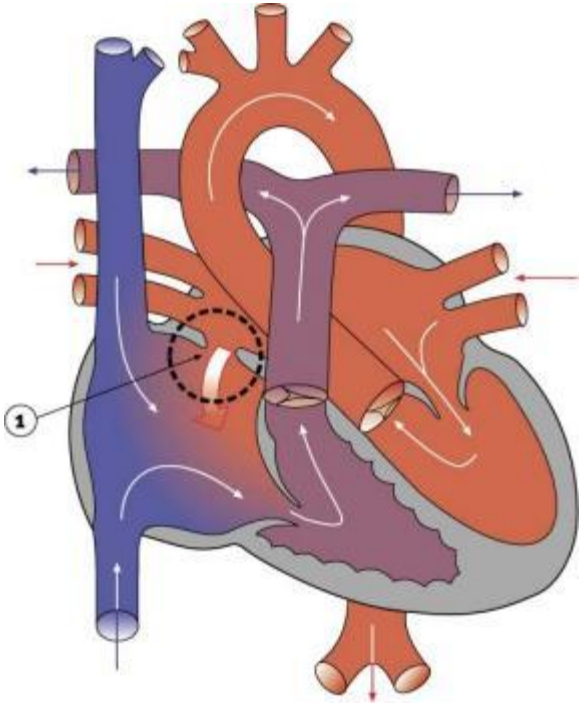
- Aortic stenosis
- Pulmonary stenosis
- Mitral valve stenosis

# Mitral Stenosis



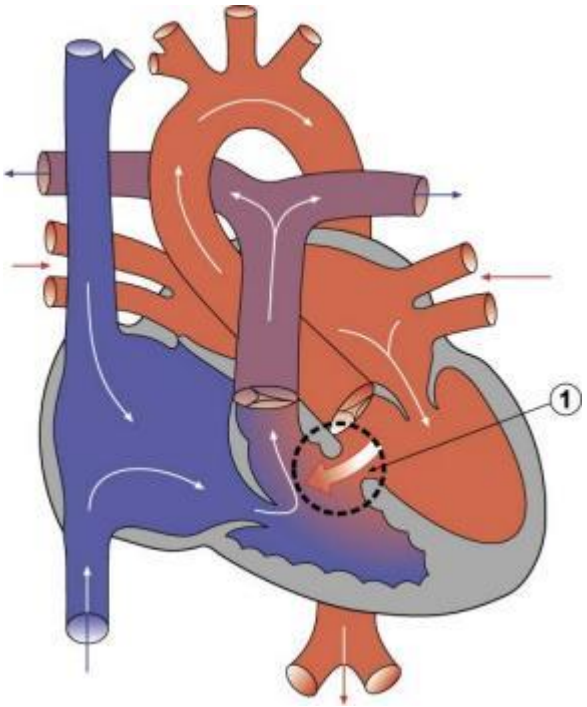
- Impairs flow of blood
- Commonly caused by
  - Acute rheumatic fever
  - Bacterial endocarditis
  - May be congenital
- Causes increase in LVEDV and may result in hypertrophy
- Development of arrhythmias (AFib)

# Atrial Septal Defect (ASD)




- May be
  - Ostium primum (low)
  - Ostium secundum (middle)
  - Sinus venosus defect (high)
- Usually asymptomatic
  - May develop CHF
  - Experience SOB with exertion

# Ventricular Septal Defect (VSD)

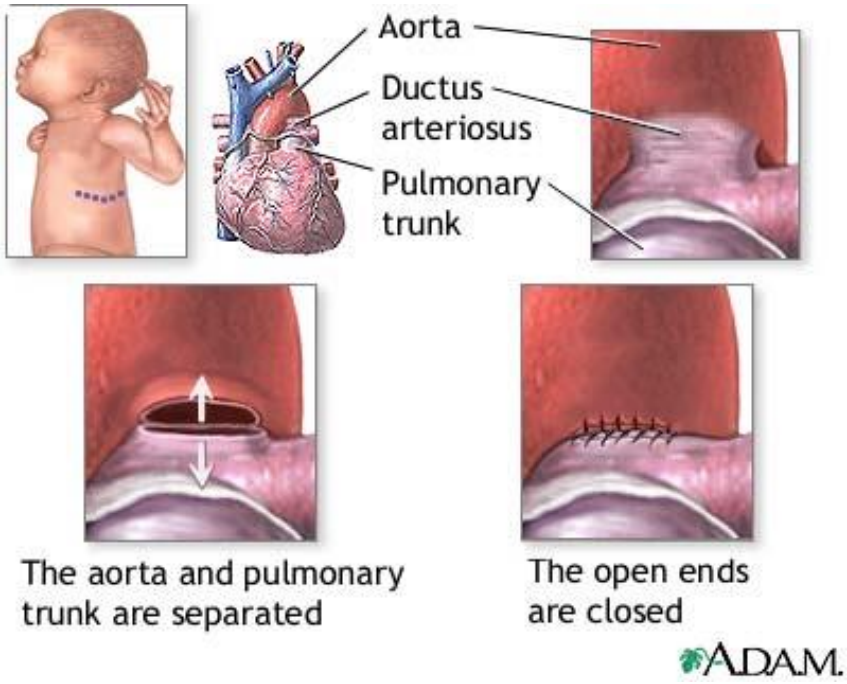



- Classed
  - Pre-membranous (high)
  - Muscular (low)
- May spontaneously close (before 2 y/o)
- May show signs of CHF
- Pulmonary hypertension

# Patent ductus arteriosus

- Ductus arteriosus remains open
- some of the oxygenated blood that should be going to the body may flow back and reenter the blood circulating in the lungs
- the baby may not receive enough oxygen and there is an increase in carbon dioxide in the baby's blood
- Results in
  - Dyspnea
  - May have a heart murmur
  - Pulses may feel full and the baby's blood pressure may go up
  - May not feed well and may not gain weight
- The signs and symptoms just described help to make the diagnosis. Often a PDA is then confirmed by an echocardiogram
- Treatment
  - If opening is small it may not cause any problems for the baby, treatment is not necessary and the ductus will close by itself as the baby matures
  - Decrease the amount of fluid the baby is getting (through IV and feeds)
    - Lasix may be given to help remove fluid through the urine
  - Large ductus opening who are having difficulty will need repair

# Patent ductus arteriosus



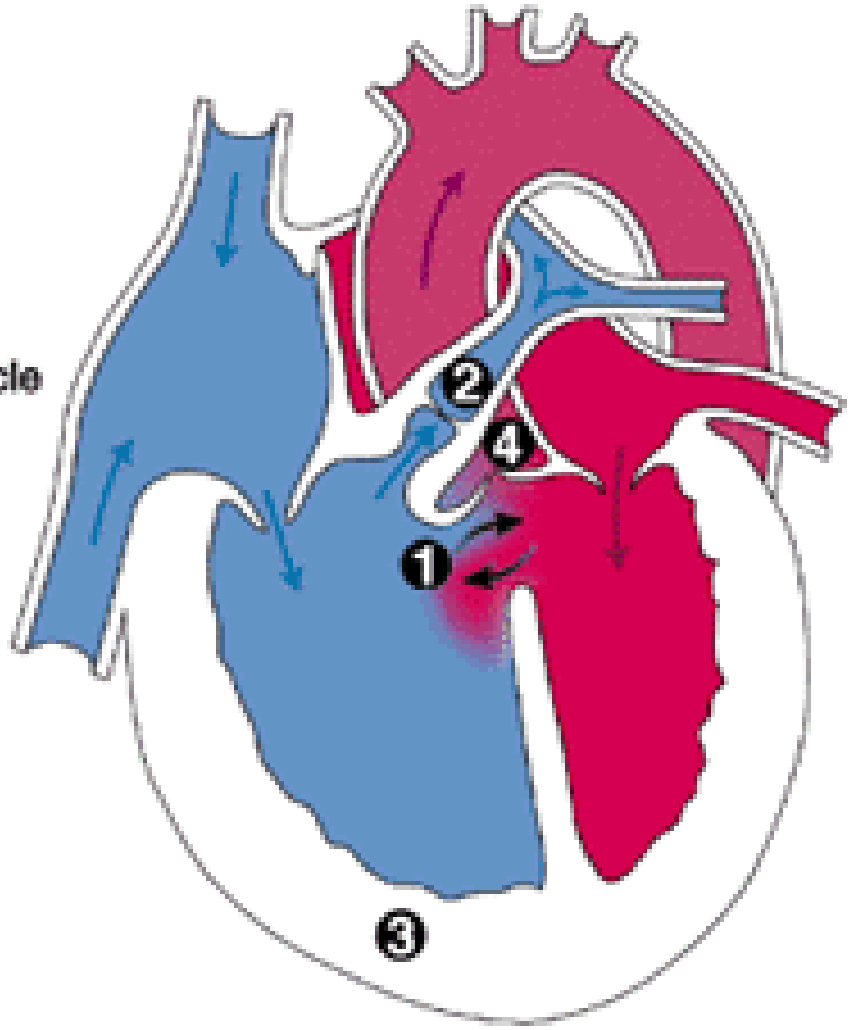

- Indomethacin (Indocin)
  - medication causes constriction of the ductus
  - given IV and several doses may be needed
- Surgery
  - PDA Ligation

# Tetralogy of Fallot

- Also known as
  - Blue Baby
  - Fallot's Tetralogy Pulmonic Stenosis-Ventricular Septal Defect
- Most common form of cyanotic congenital heart disease
- Consists of a combination of 4 different heart defects
  - ventricular septal defect
  - pulmonary stenosis
  - Aorta overrides the ventricular septal defect
  - right ventricular hypertrophy
- The severity of the symptoms is related to the degree of blood flow obstruction from the right ventricle.
- If not treated, the symptoms usually become progressively more severe.
- Blood flow to the lungs may be further decreased and severe cyanosis may cause life-threatening complications.
- The exact cause of Tetralogy of Fallot is not known.
- Symptoms
  - Cyanosis at birth or may be seen with increased demands (feeding, crying)
  - Prolonged cyanosis may produce clubbing, poor growth
  - Are at risk for embolotic diseases, CV disease, brain abscess, seizures or sudden death

# Tetralogy of Fallot

1. Ventricular Septal Defect
2. Pulmonary Stenosis
3. Hypertrophy of Rt. Ventricle
4. Overriding Aorta



# Transposition