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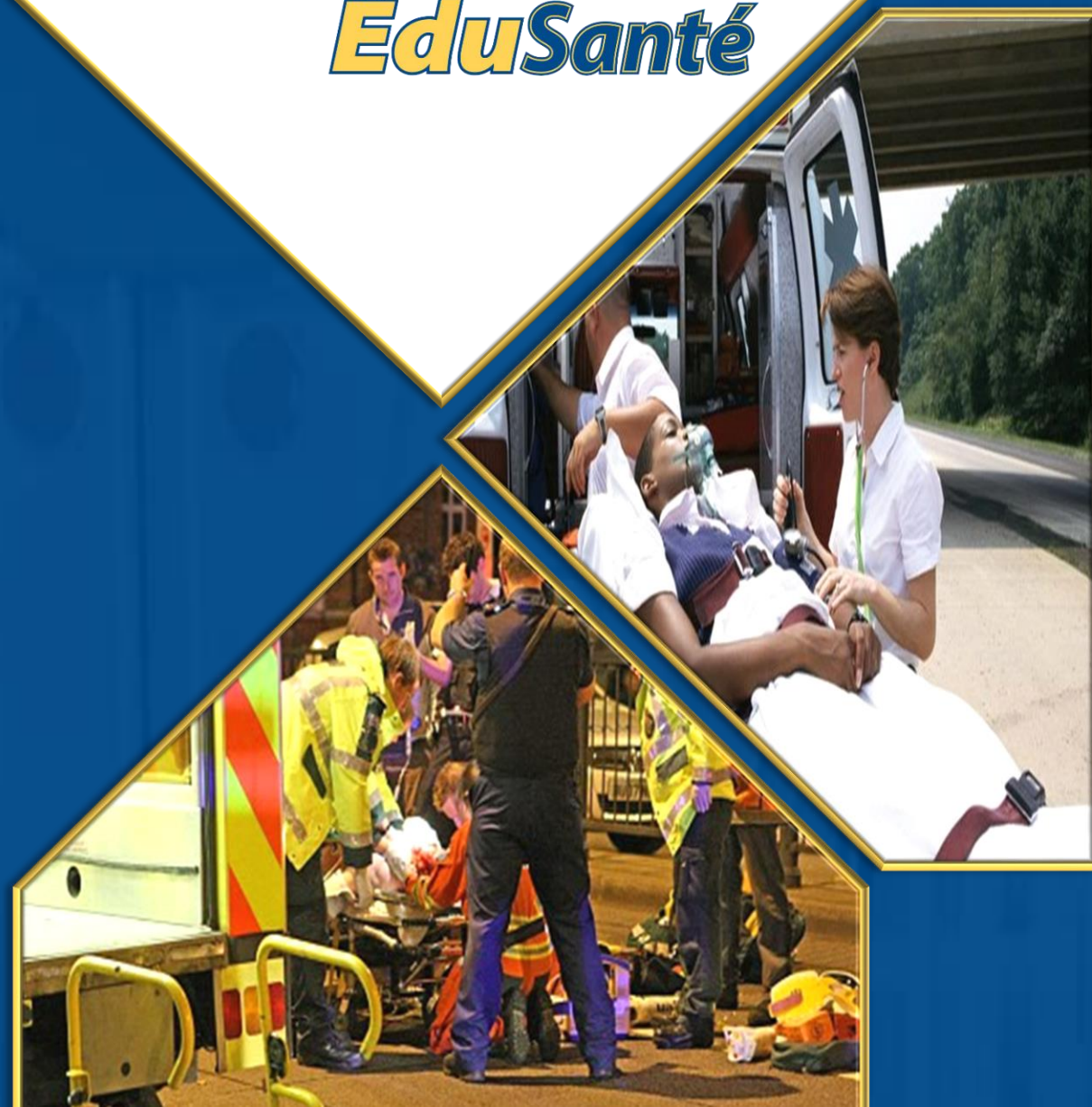
Cardiovascular Pathophysiology

Primary Care Paramedicine



Module: 12

Section: 03

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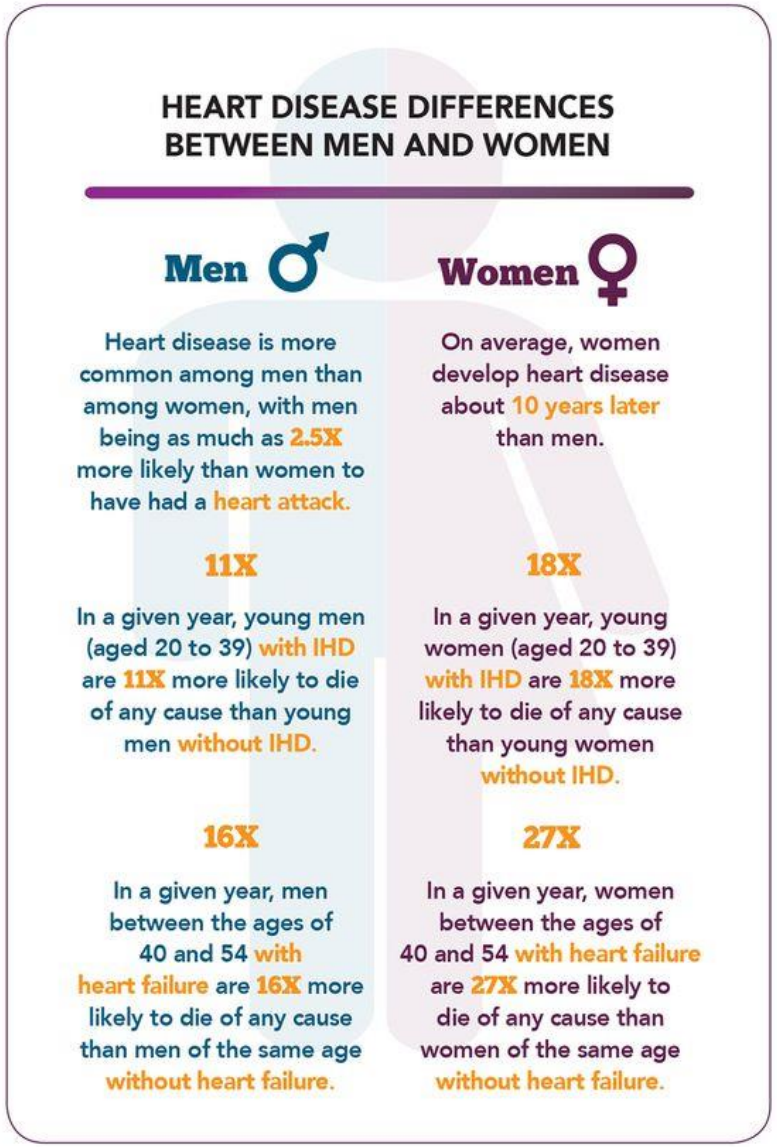
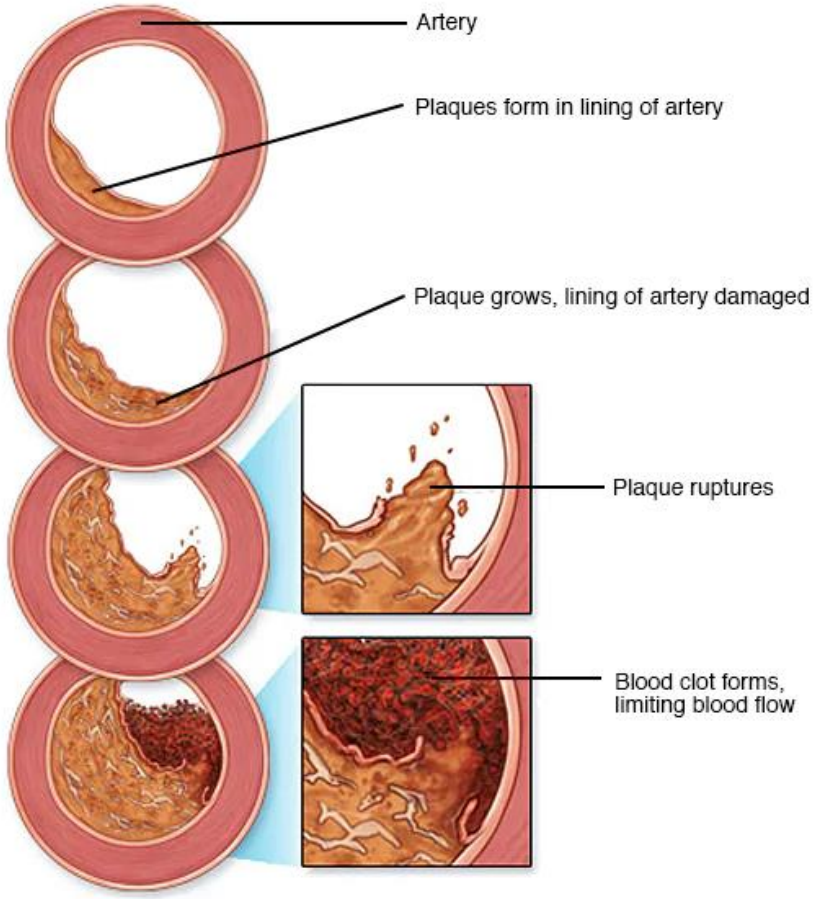
Cardiovascular Disease



- Heart disease is the 2nd leading cause of death in Canada (after Cancer)
- Providing early, definitive treatment for patients with acute myocardial infarction (AMI) was the goal of creating the position of paramedics.
- Each year, about 159,000 Canadians are newly diagnosed with heart disease and about 33,600 Canadians die of heart disease.

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

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Risk Factors

Unpreventable Risk Factors

- Age
- Gender
- Family history of heart disease or stroke
- Race

Preventable Risk Factors

- Diabetes
- Excessive alcohol consumption
- High blood pressure
- A diet high in saturated fat, trans fat, and cholesterol
- Obesity
- Lack of physical activity
- Cigarette smoking
- Stress

Coronary Heart Disease and Angina

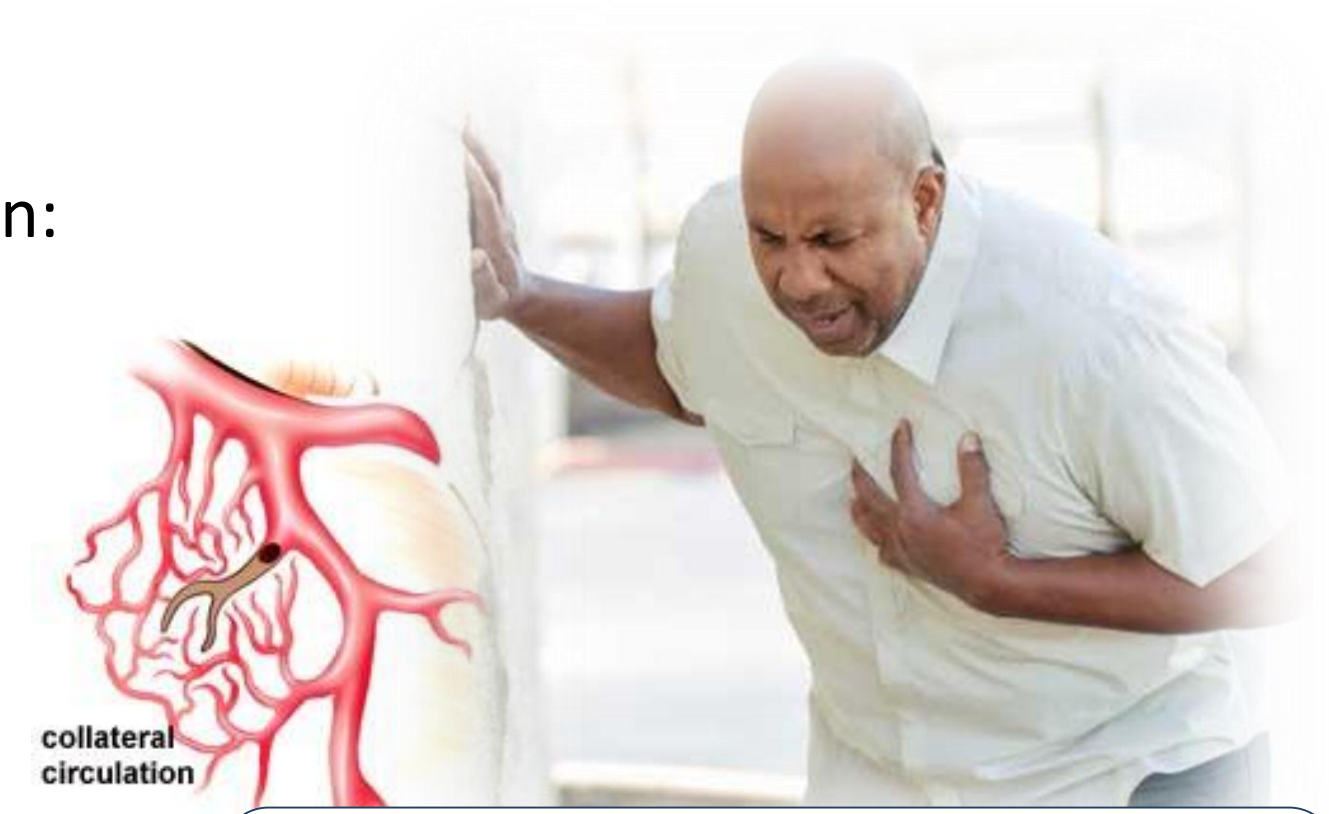
- Coronary heart disease
  - Most common form of heart disease and a leading cause of death in adults in Canada
  - Ischemia
  - Infarction

Atherosclerosis

- Provides a locus for the formation of a fixed blood clot
  - Caused by calcium precipitate from the bloodstream into the arterial walls, causing **arteriosclerosis**, a condition in which thickening and stiffening of the arterial walls greatly reduces the elasticity of the arteries
- Atherosclerosis is the main underlying cause of angina, MI, heart failure, aortic aneurysm and dissection, peripheral vascular disorders, and stroke.

Atherosclerosis

- Effects
  - The severity is dependent on:
    - Time of onset
    - Degree of obstruction
    - Pt's ability to produce collateral circulation



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.

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Cardiovascular Pathophysiology

STABLE ANGINA

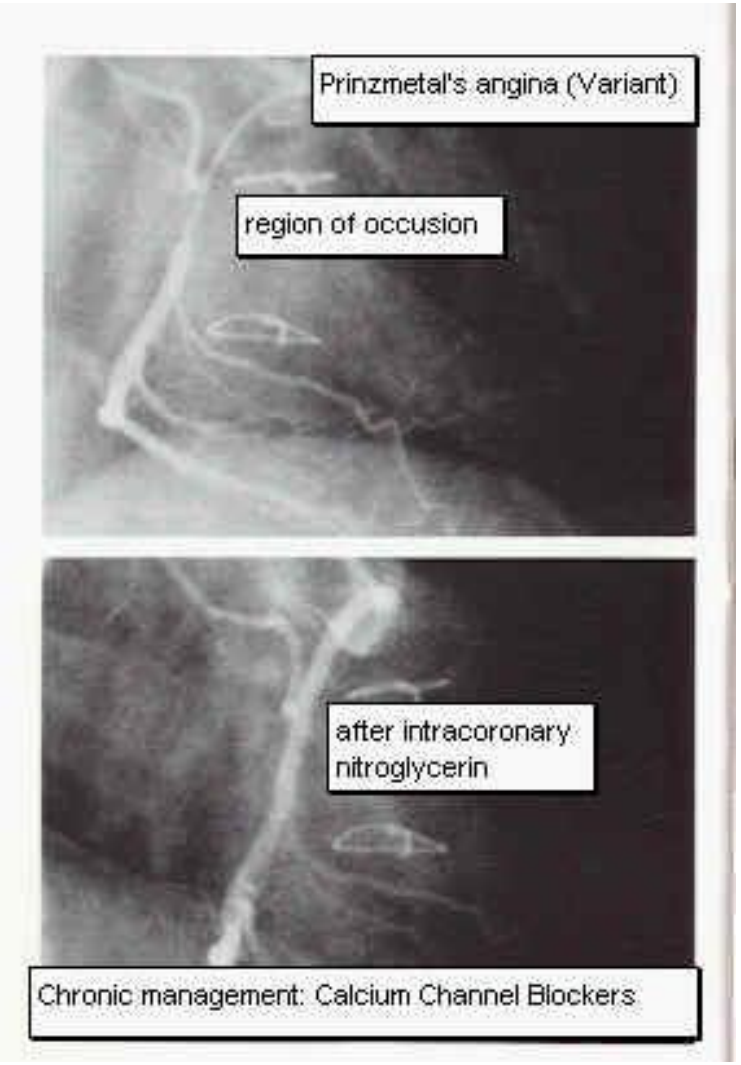
Angina Pectoris

- Etiology
  - Cardiac muscle becomes ischemic
  - 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

Angina Pectoris

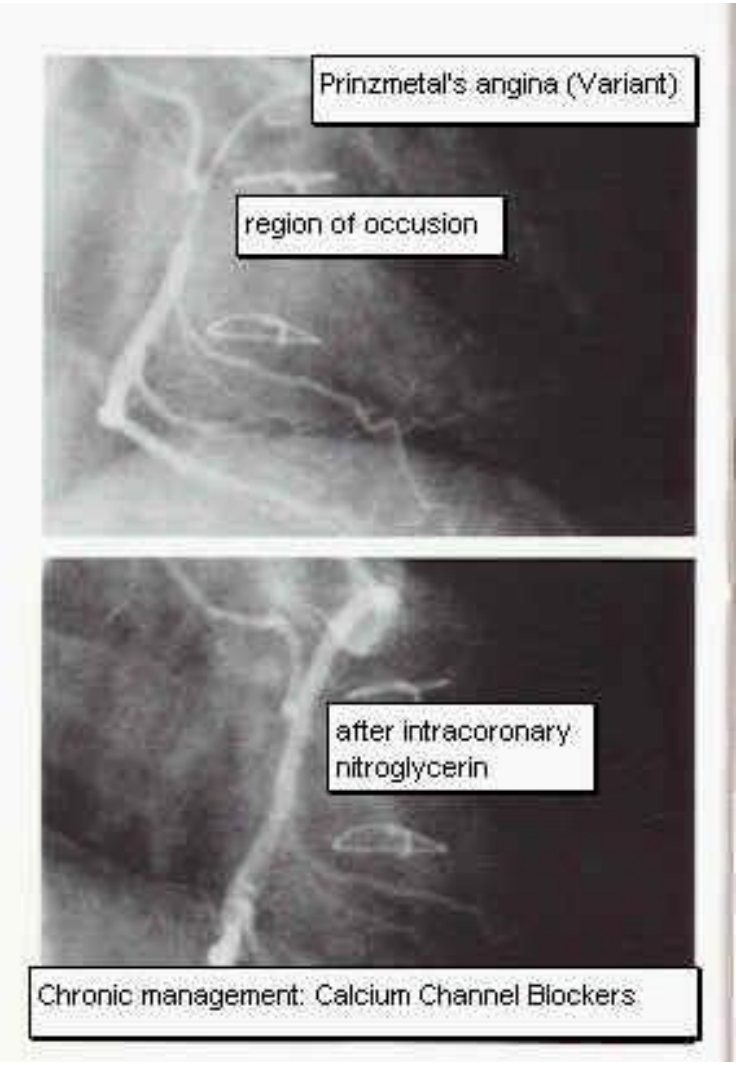
- Precipitating Factors
  - Atherosclerosis tends to be major cause
  - Emotional stress
  - Increased activity
- Note:
  - If increased O_2 demands are not met, may lead to ischemia of the muscle
  - This ischemia may result in arrhythmias or infarction

Prinzmetal's Angina



- Also known as Variant Angina Pectoris
- Caused by temporary spasm of coronary vessels and may or may not include atherosclerosis
  - About ⅔ 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”
- Recurrent pattern of pain after exertion
  - Predictable location, intensity, and duration
  - Patients with chronic, stable angina often take NTG or some other nitrate agent to relieve pain.
- 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)

Acute Coronary Syndrome (ACS)

- Continuum of cardiac disorders causing myocardial ischemia or infarction
  - Unstable angina
  - Non–ST-segment elevation myocardial infarction (NSTEMI)
  - ST-segment elevation infarction (STEMI).

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
- Much more serious than stable angina
  - Greater degree of obstruction of the coronary arteries
  - High risk of imminent AMI
  - Benefits from early and aggressive treatment to prevent infarction

Unstable Angina

- 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

Management

- Place pt at rest (Sitting or lying)
- Emotional Support
- Administer O_2 (as required)
- Baseline Vitals
- Administer Aspirin (160 – 325 mg PO)
- Administer NTG (0.4 mg SL, q 3-5 min with BP > 100/50)
- Initiate IV
- Monitor (3 lead, 12 lead)
- Transport

Acetylsalicylic Acid (ASA)

Classification

- Antiplatelet, NSAID, antipyretic, analgesic

Mechanism of Action

- Inactivates cyclooxygenase (COX) enzyme which decreases the production of prostaglandins in the tissue, blocking the pain receptors sensitivity and decreases inflammation. Due to this inactivation of COX, thromboxane A2 production is also inhibited, which is responsible for platelet aggregation

Indications

- Acute Coronary Syndrome
  - Acute MI
  - Unstable angina

Acetylsalicylic Acid (ASA)

Contraindications

- GI bleed
- Asthmatics sensitive to ASA
- Hypersensitivity

Dosage

- 160 mg – 325 mg PO (Chewed)

Nitroglycerine

Classification

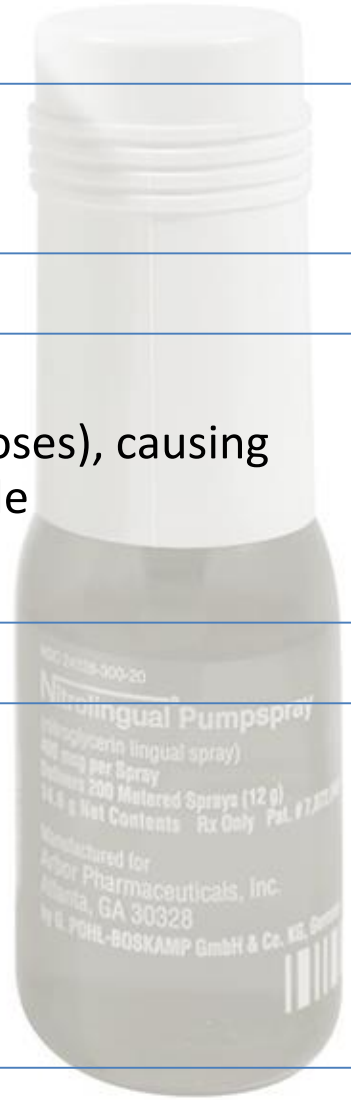
- Anti-angina, vascular smooth muscle relaxer, vasodilator

Mechanism of Action

- Relaxes vascular smooth muscle, there by dilating the veins and arterioles (at higher doses), causing blood pooling, which reduces the preload thus decreasing workload of the heart muscle
- Reduces left ventricular systolic wall tension, which decreases afterload

Indications

- Possible ischemia due to ACS:
  - Unstable angina
  - AMI
- Pulmonary edema/CHF



Nitroglycerine

Contraindications

- Hypotension (< 90 mmHg)
- Severe bradycardia/tachycardia (< 50 or > 150 bpm)
- Increase ICP or intracranial hemorrhage
- Patients taking erectile dysfunction medications
- Viagra within 24 hours
  - Cialis, Levitin, Staxyn within 36 hours

Dosage

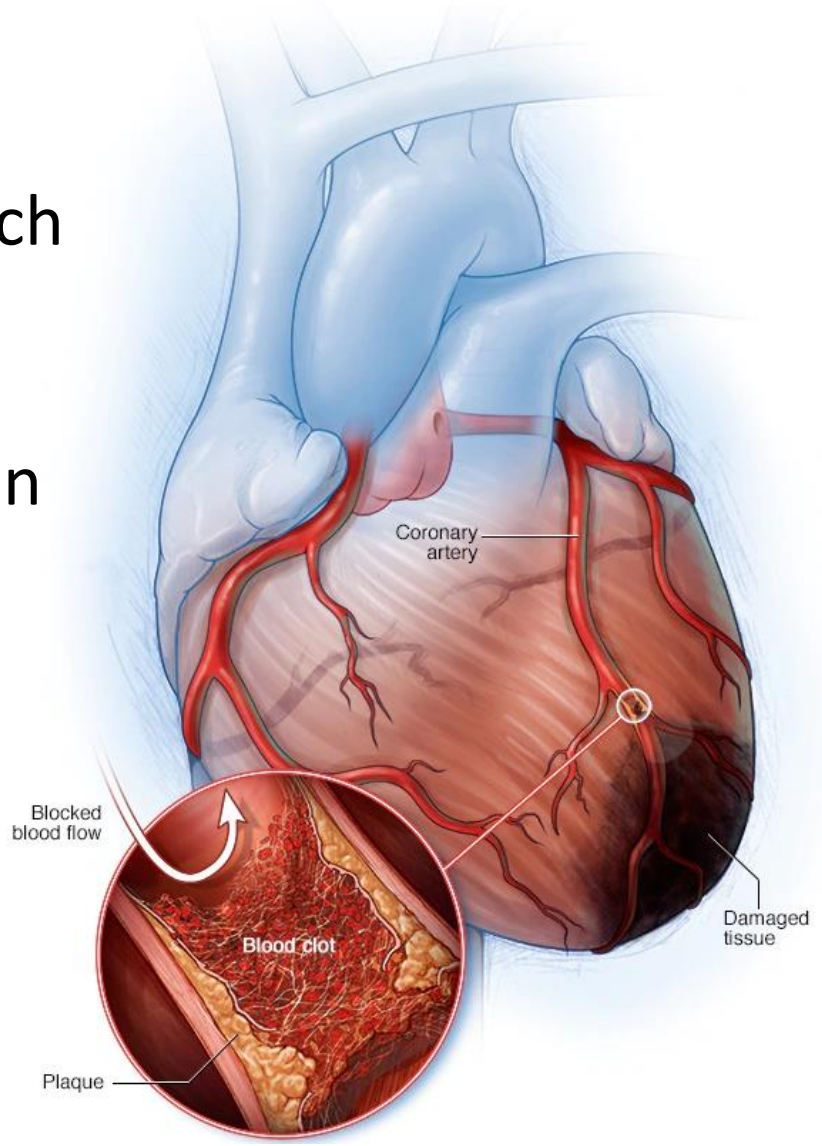
- 0.4 mg SL q 3 - 5 min

Acute Myocardial Infarction (AMI)

- Portion of the cardiac muscle is deprived of coronary blood flow long enough that portions of the muscle die.
  - Plaque rupture and thrombus formation
  - Spasm of a coronary artery
  - Reduction of overall blood flow
- Classified as either NSTEMI or STEMI (only an ECG differentiates them)

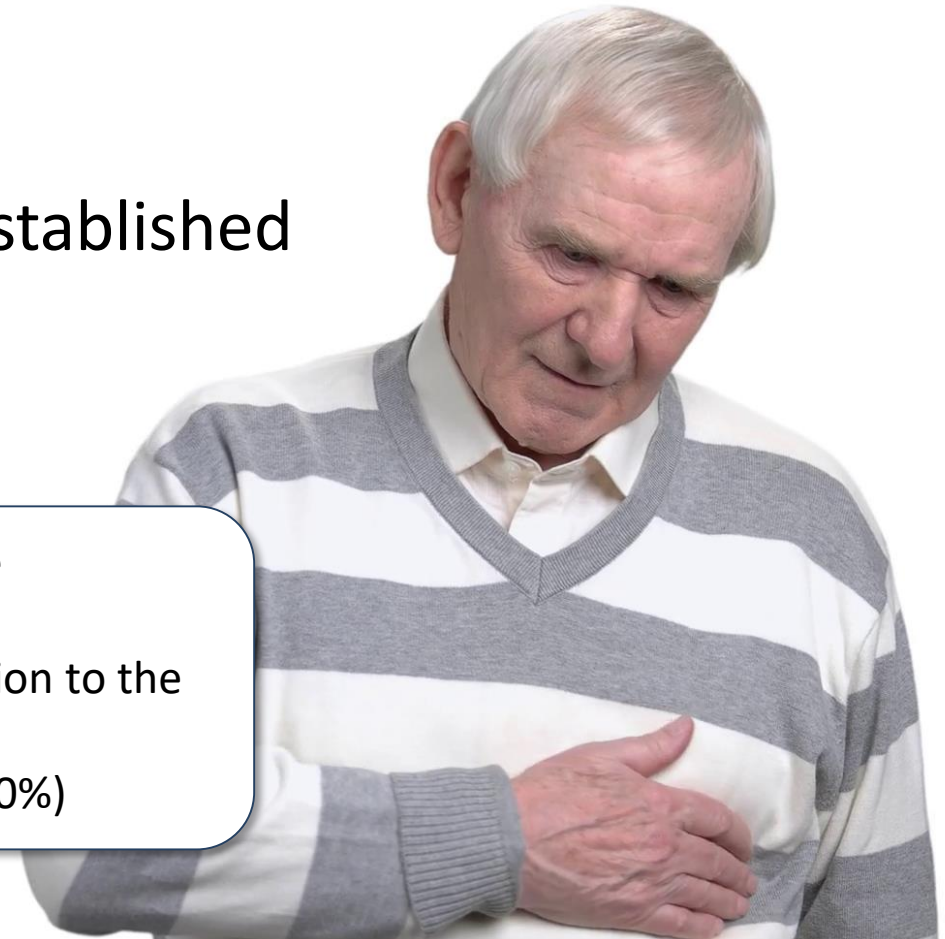
Myocardial Infarct (MI)

- 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



Myocardial Infarct (MI)

- Size of infarct is determined by:
  - the metabolic needs of the tissue
  - presence of collateral circulation
  - duration of time before reperfusion is established



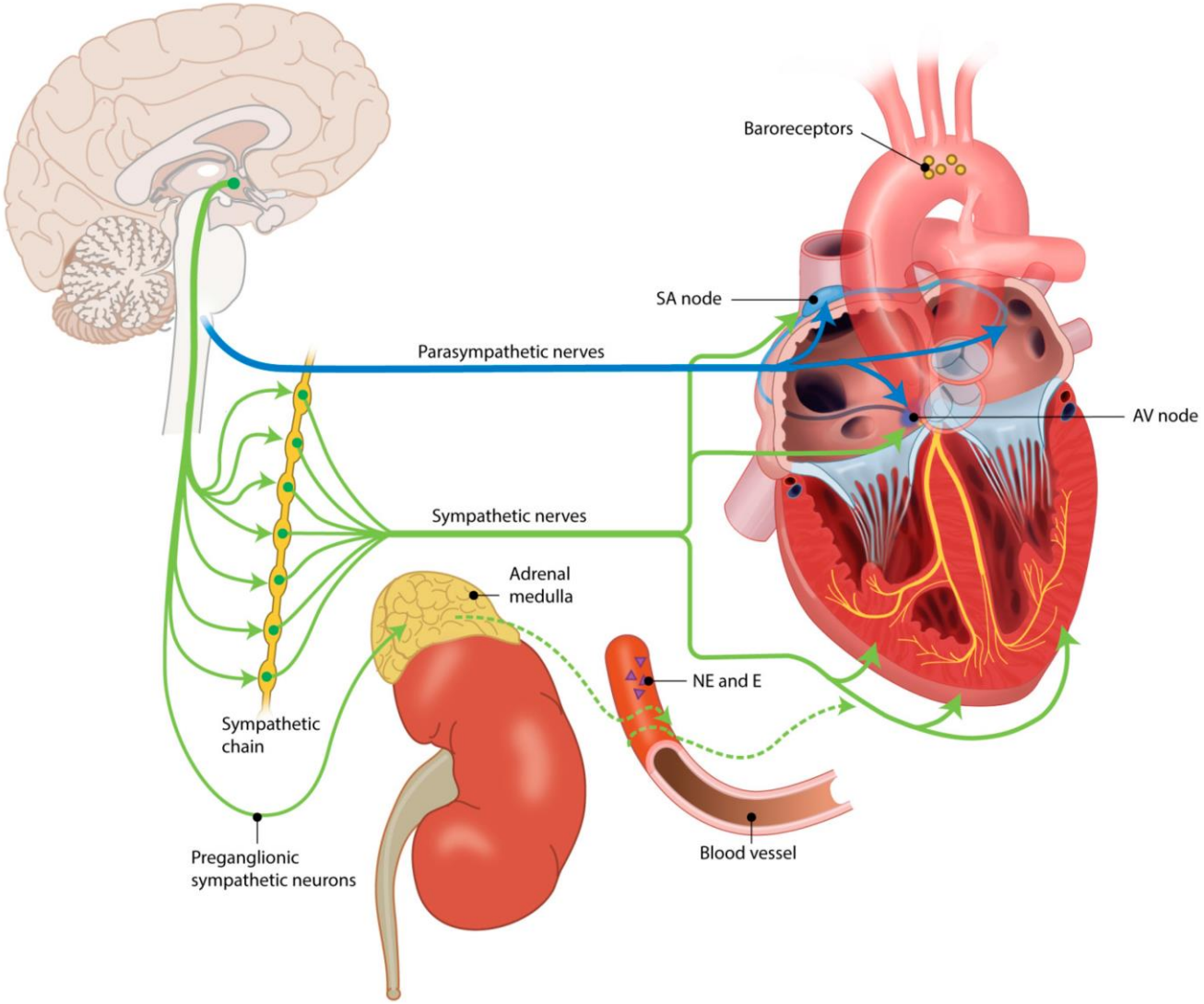
- Most AMI's occur in the L Ventricle or the interventricular septum (40 – 50%)
- R ventricle MI is usually a result of occlusion to the RCA (30 – 40%)
- Lateral wall is usually L circumflex (15 – 20%)

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

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



MI

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

ACS

- Not all chest pain is caused by cardiac ischemia or infarction.
- Conditions that can be mistaken for angina or AMI:
  - Pulmonary embolism
  - Pneumothorax
  - Pneumonia
  - Pericarditis
  - Aortic dissection
  - Gastroesophageal reflux disease
  - Gastritis
  - Peptic ulcer disease

Signs and Symptoms

- Chest Pain
  - Pain of MI typically felt just beneath the sternum or on the left side of the chest
  - Described as burning, heavy, pressure, tightness, squeezing
  - May radiate to shoulders, jaw, arm, neck and back
  - Patient unconsciously clenches a fist when describing the pain (Levine sign)
  - May also describe as pressure or discomfort
  - Patient may mistake the pain for indigestion

Signs and Symptoms

- Not every AMI patient has chest pain—silent MI
  - People with diabetes, older people, and heart transplantation patients
  - Present with symptoms related to a drop in cardiac output: sudden dyspnea, pulmonary edema, hypotension, confusion, profound fatigue, or feeling weak and dizzy

Signs and Symptoms

- Women may present differently from men.
  - Nausea
  - Light-headedness
  - Epigastric burning
  - Sudden onset of weakness or unexplained tiredness

Many women may not recognize these symptoms as potentially cardiac.

- Heart disease accounts for one-third of all female deaths in Canada.
- Women are more likely to die of AMI than men.

Signs and Symptoms

- Anxious
- Cool to the touch
- Diaphoresis
- Syncope
- Dyspnea
- Anorexia, nausea, vomiting, or belching; hiccups occasionally
- Profound weakness
- Dizziness
- Palpitations perceived as the heart skipping a beat
- Feeling of impending doom

Prehospital Management of ACS

- Overview
  - Stress response causes surge of catecholamines (epinephrine and norepinephrine).
  - Peripheral circulation is in a state of severe vasoconstriction.
  - The heart's need for oxygen soars when it is in a state of oxygen deprivation.
  - This cycle can quickly lead to dysrhythmias and death.
  - Treatment is time sensitive, with the longest delay during the phase from onset of symptoms to patient recognition, therefore your care must begin immediately.
  - Goal of treatment is to try to limit the size of the infarct.

Prehospital Management of ACS

- On arrival at the scene
  - Assessment and management for possible ACS should occur simultaneously.
  - Calm the patient and place him or her at rest.
  - Determine the presence of possible ACS.
- Initial treatment
  - Put the patient physically and emotionally at ease.
  - Decrease the amount of work that the heart must do (semi-Fowler position or sitting up if there is pulmonary edema).

Prehospital Management of ACS

- Confirm possible ACS.
  - Perform a 12-lead ECG
  - Rapid acquisition of the 12-lead ECG before the secondary assessment and before anti-ischemic therapy is preferred.
  - Consider rapid transport if patient has evidence of a STEMI.
    - Given risk of sudden ventricular dysrhythmias, patients should be transported with defibrillation pads on their chest.

Prehospital Management of ACS

Management

- Place pt at rest (Sitting or lying)
- Administer O_2 (as required)
- Baseline Vitals
- Monitor (3 and 12 leads)
- Administer Aspirin (160 mg PO)
- Administer NTG (0.4 mg SL, q 3 - 5 min with BP > 100/50)
- Initiate IV
- Treat dysrhythmias as required
- Transport

Reperfusion Techniques for STEMI

- Majority as a result of thrombus formation at the site of a ruptured atherosclerotic plaque
  - Thrombus occludes the coronary artery.
  - "Time is myocardium."
  - Immediate angioplasty, or percutaneous coronary intervention (PCI), is preferred to fibrinolysis when it can be accomplished within 90–120 minutes from first medical contact.
  - If you can reach a hospital within 60–90 minutes of first contact, and if local guidelines allow, it may be appropriate to transport hemodynamically stable patients directly to a cardiac interventional facility.
  - Attempt quick reperfusion by either PCI or fibrinolysis.
  - Know how to recognize STEMI on an ECG and which area hospitals carry out emergency PCI (or fibrinolysis).
  - Provide early notification (along with 12-lead ECG results) to the facility.

Reperfusion Techniques for STEMI

- Fibrinolysis
  - One way to reperfuse the blocked coronary artery is to break up the occluding blood clot with medications, restoring circulation to the ischemic heart.
  - Fibrinolytic therapy seeks to administer, during the early hours of MI, an agent that will activate the body's own internal system for dissolving clots.
    - Recanalization: reopening the artery
    - Reperfusion: allowing the resumption of blood flow through the artery
    - If agent capable of promoting clot dissolution is given intravenously, it affects clot anywhere in the body, which can lead to uncontrolled bleeding.

Reperfusion Techniques for STEMI

- Fibrinolysis (continued)
  - PCI is preferred to fibrinolytic therapy when it is timely (less bleeding risk and greater blood flow restoration).
  - Fibrinolytic therapy can be performed in virtually every ED in Canada; paramedics can safely administer in settings with long transport times to the closest ED.
    - Reduced time to administration of fibrinolytics decreased mortality rates when given to patients with STEMI.
    - Even in EMS systems that do not give fibrinolytic therapy: their ability to identify candidates for such therapy has a decisive role in helping ED personnel administer it with success.

Reperfusion Techniques for STEMI

- Fibrinolysis (continued)
  - Need to be as certain as possible that patient is having a STEMI to determine the appropriateness of administering fibrinolytic agents
  - Exclusion criteria are used to identify patients for whom the risk of fibrinolytic therapy is unacceptably high (eg, those likely to experience hemorrhagic complications).

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.

Anticoagulant Therapy

- 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

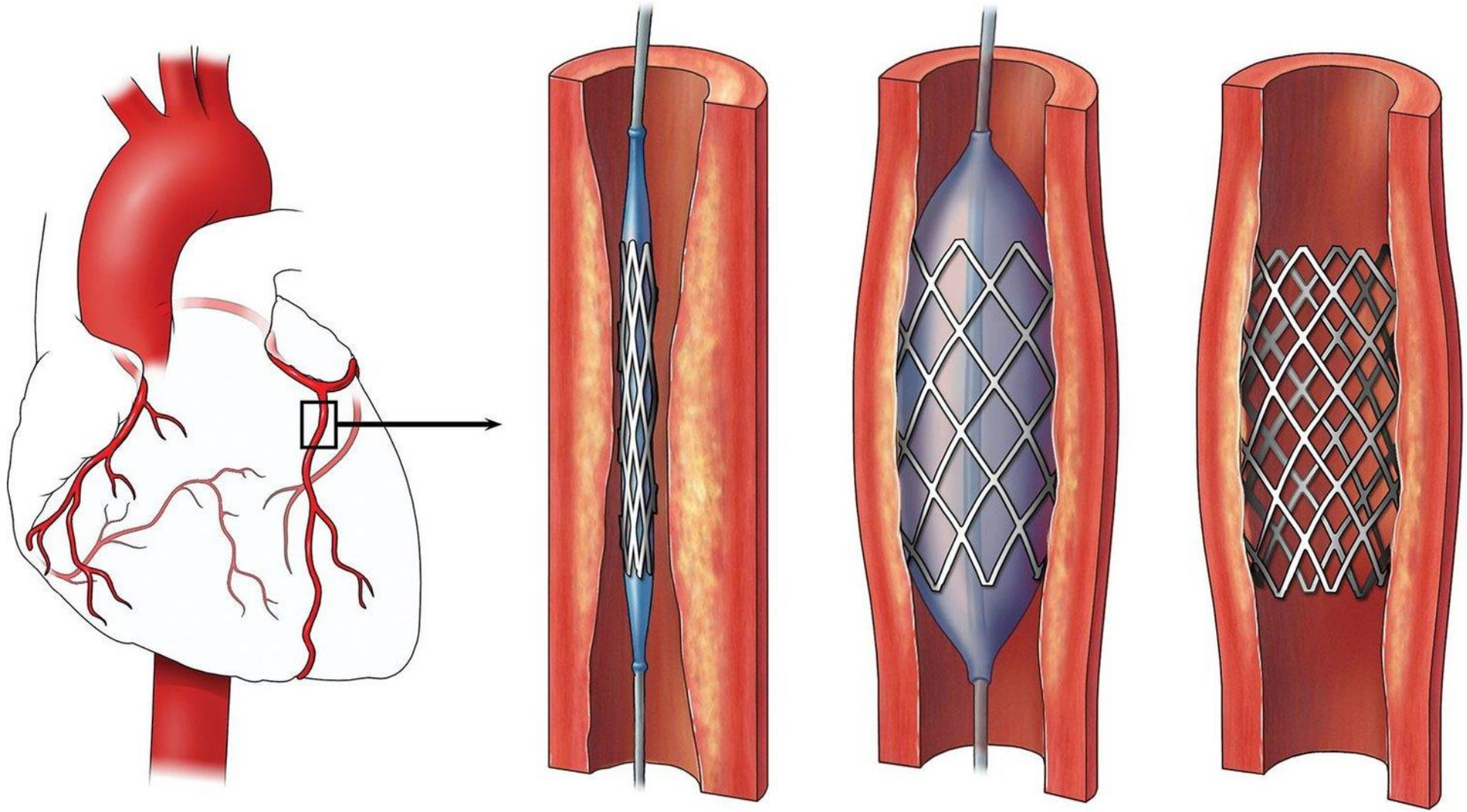
Fibrinolytic Therapy

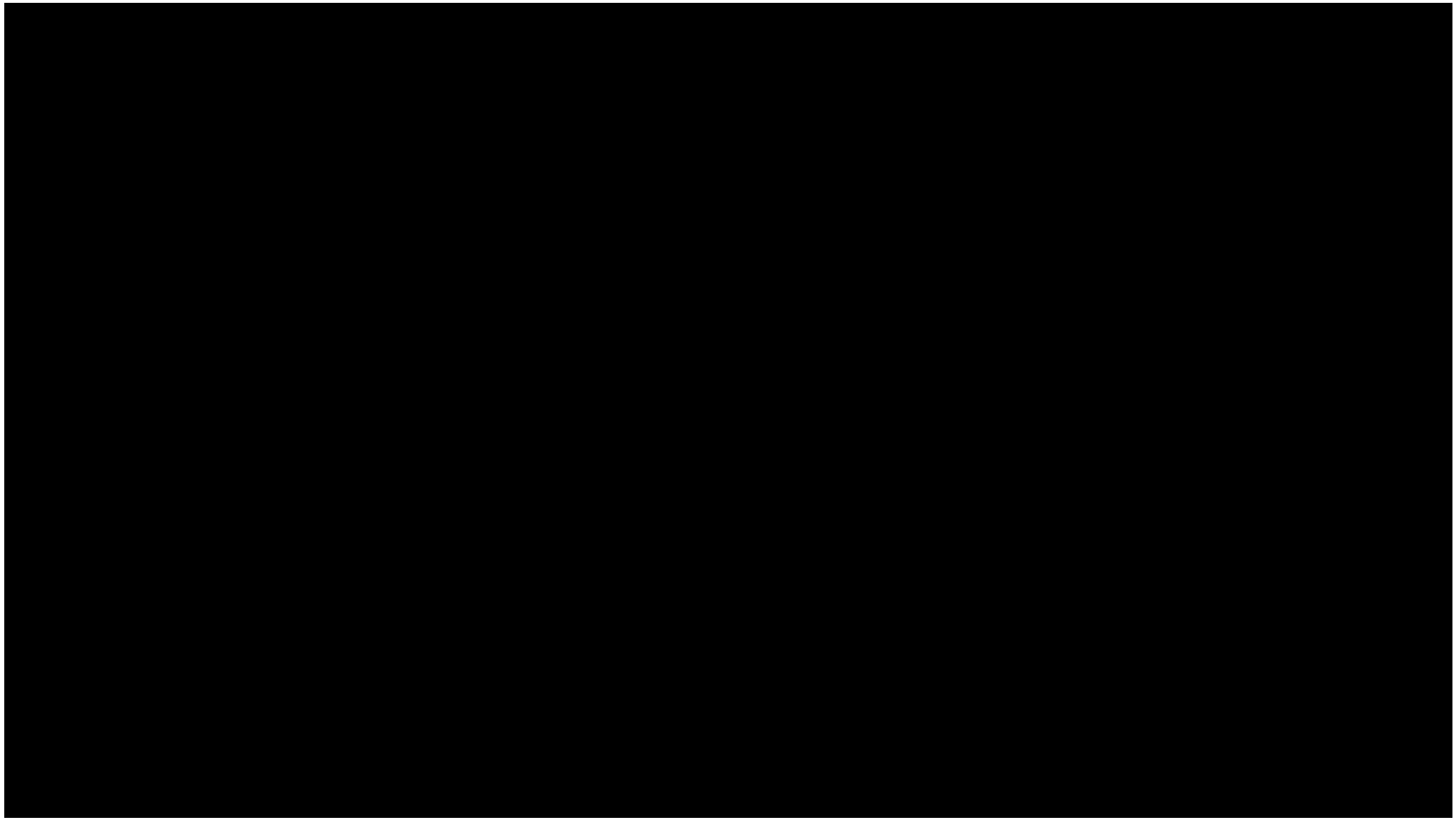
- Relative Contraindication
  - Pregnancy or post partum state
  - Uncontrolled HTN
  - Major surgery within last 3 weeks
  - Intercranial tumor
  - AAA
  - CPR
  - Trauma in last 2 to 4 weeks
  - Use of anticoagulants
  - Terminal illness
  - Other possible criteria based on hospital/EMS protocols

Percutaneous Intervention (PCI)

- Alternative to fibrinolysis
- Emergently opening the blocked coronary artery by mechanical means with a balloon-tipped catheter
- Better outcomes than fibrinolytic therapy
- Target is to have the artery opened by the balloon within 90 to 120 minutes from first medical contact.
- In most systems, paramedics must alert interventional teams early so they can prepare the interventional suite or delay the start of an elective procedure to make room for an emergency.

Percutaneous Intervention (PCI)





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Cardiovascular Pathophysiology

HEART FAILURE

Heart Failure

- Heart failure describes several clinical syndromes where the hearts mechanical performance is so compromised that cardiac output cannot meet demand.

Heart Failure

- 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

Right vs Left Failure

Systolic vs Diastolic

High vs Low Output

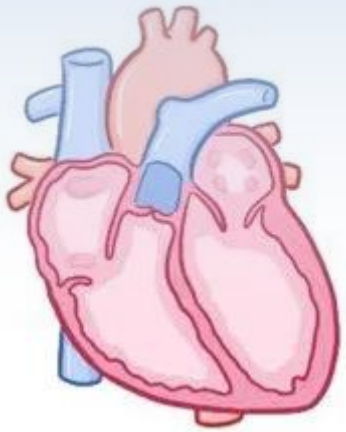
Low Output	High Output
Reduced pumping efficiency (↓Cardiac Output) • Most commonly caused by a depressed ejection fraction	Volume demand exceeds what the heart can pump • Due to a high metabolic state or shunting of blood that increases myocardial oxygen demand
Examples: • Dilated cardiomyopathy • Chronic hypertension • Valvular heart disease	Examples: • Hyperthyroidism • AV Fistulas • Anemia • Pregnancy

Systolic vs Diastolic

Systolic Heart Failure

Ventricles can't pump hard enough during Systole (Impairment of myocardial contraction)

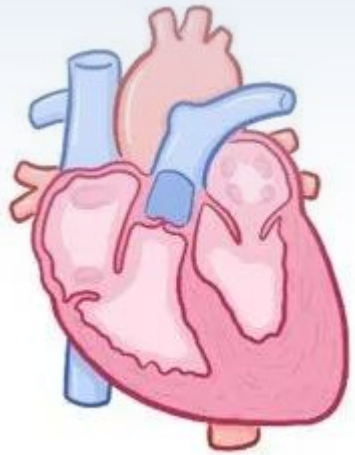
- Increased afterload
- Damaged myocytes (necrosis)



Diastolic Heart Failure

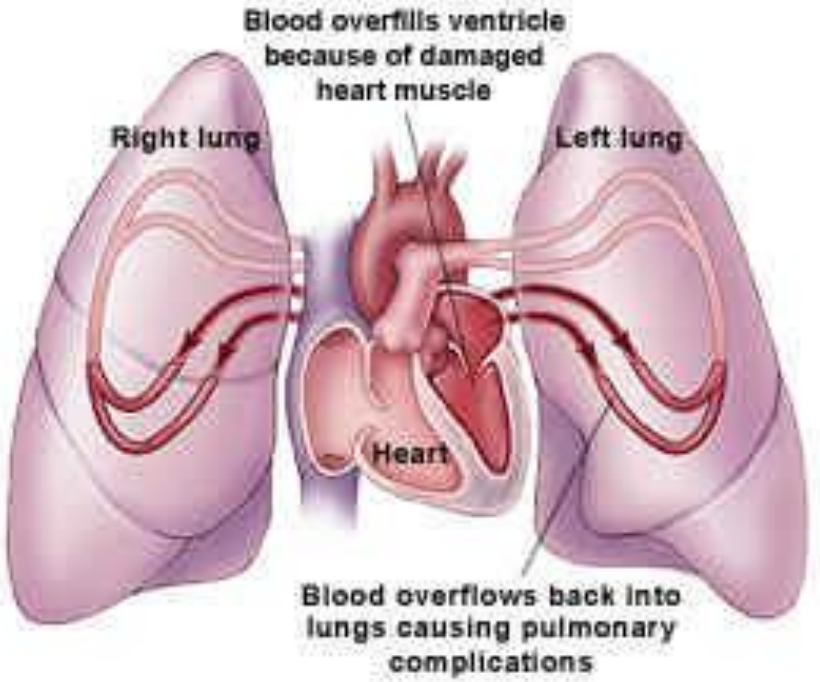
Not enough blood fills into the ventricles during Diastole (Impairment of myocardial relaxation)

- Myocardial ischemia
- Hypertrophy



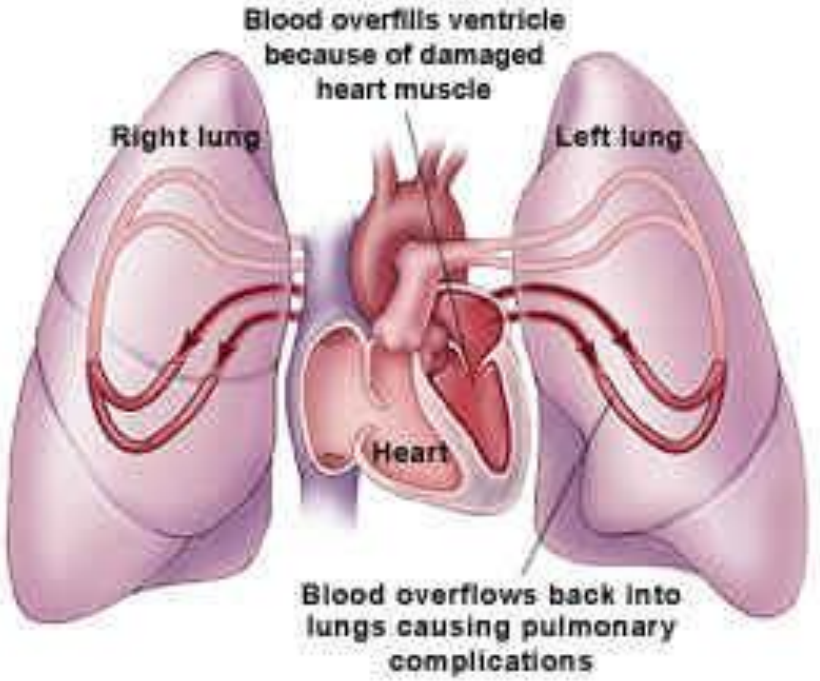
Left Ventricular Failure

- 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)



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
- May be as a result of:
  - AMI or Ischemia
  - Valve damage
  - Hypertension



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)

Left Ventricular Failure

Management

- Aimed at
  - decreasing venous return to the heart
  - Decreasing M_vO_2
  - Improving oxygenation
- Primary Survey (ABC's, RBS)
- Place pt at rest (Sitting with legs down)
- Baseline Vitals (Include auscultation)
- Administer O_2, 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

Left Ventricular Failure

Pharmacology

PCP

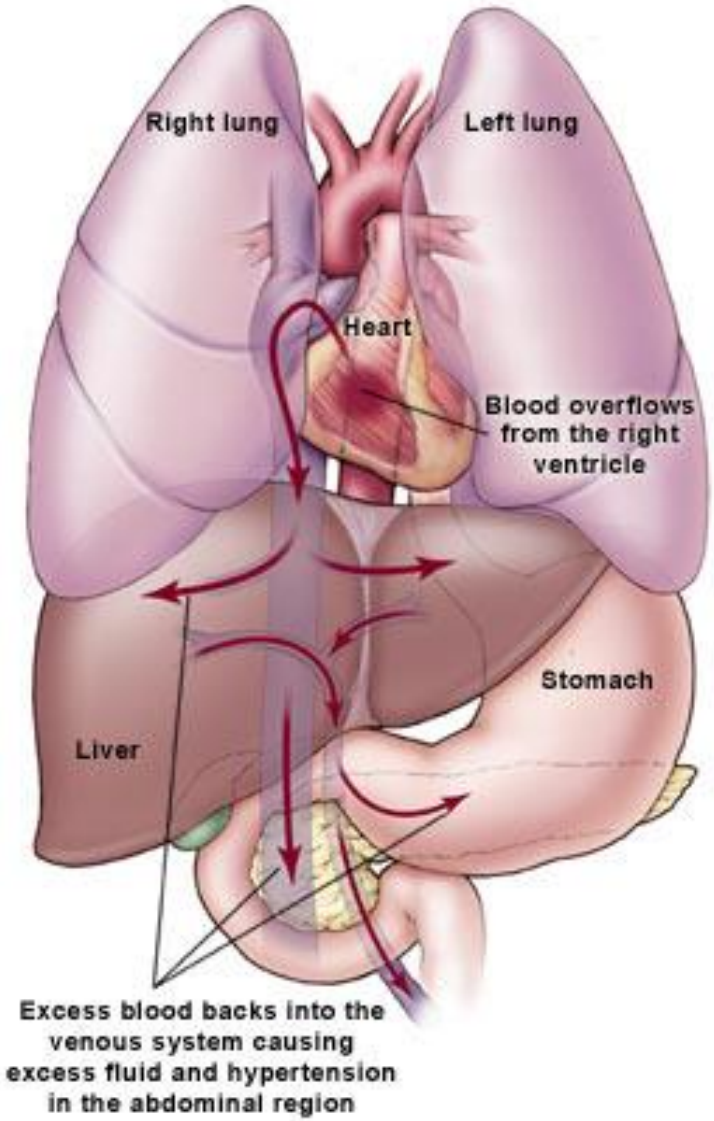
- NTG (0.4 mg SL, q 3 – 5 minutes)
  - Decrease preload and afterload thus decreasing M_vO_2
- Ventolin (5 mg Aerosol - if wheezing present and decompensating)

Right Ventricular Failure

- 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

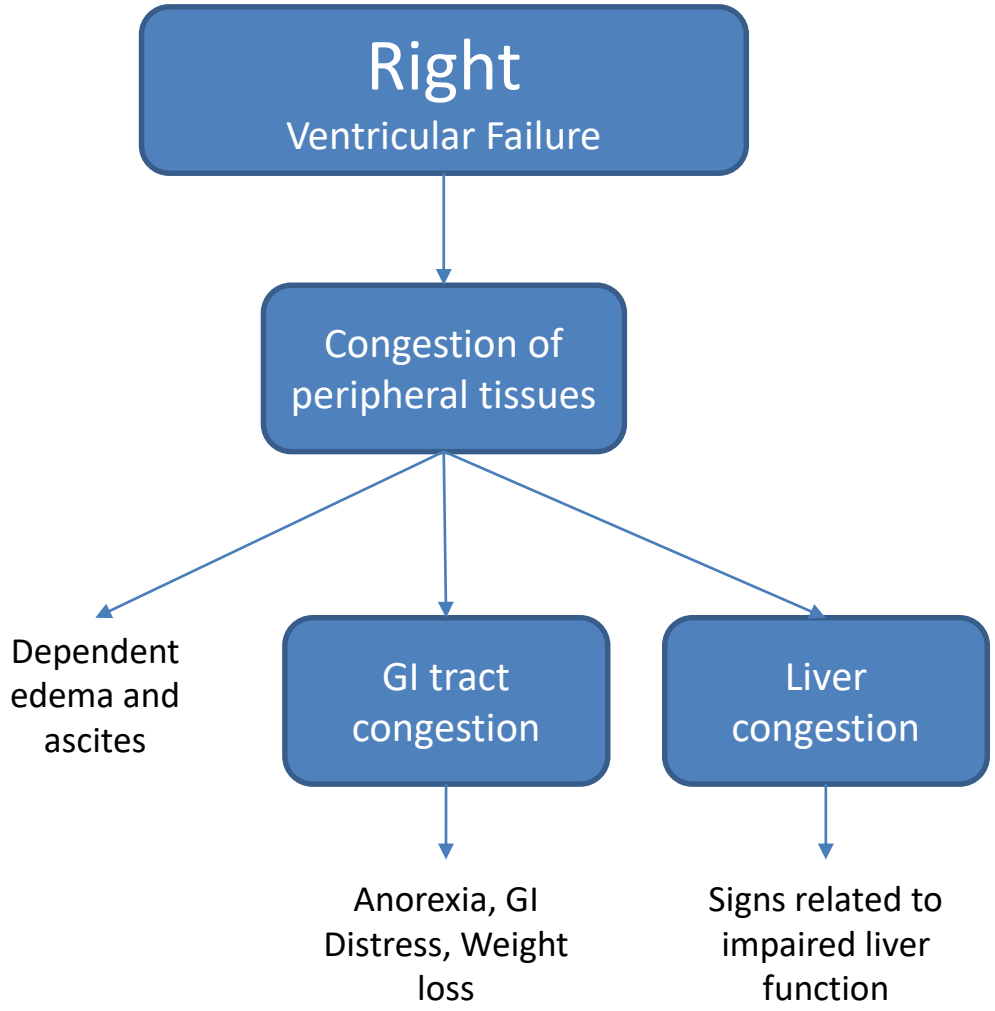
- Chest pain, may have RUQ pain
- Dyspnea
- Hypotension
- Tachycardia
- Venous congestion
- Engorged liver and/or spleen (hepatomegaly, splenomegaly)
- May see hepatojugular reflux
- Distend Neck Veins
- Peripheral Edema (may be pitting)
  - feet and hands, entire body
  - sacral area for the bedridden
- 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)

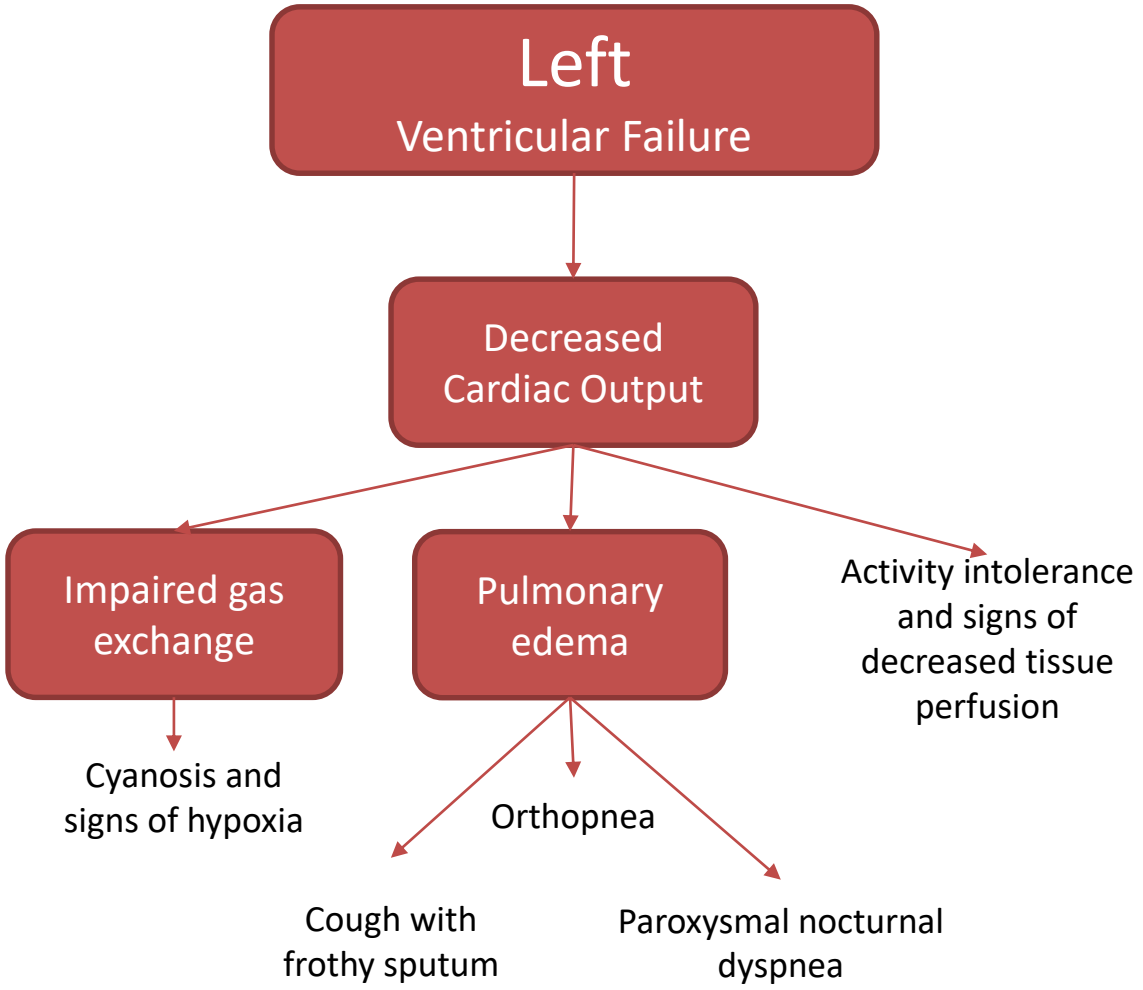
Right Ventricular Failure

Management

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

Heart Failure





Cardiomyopathy

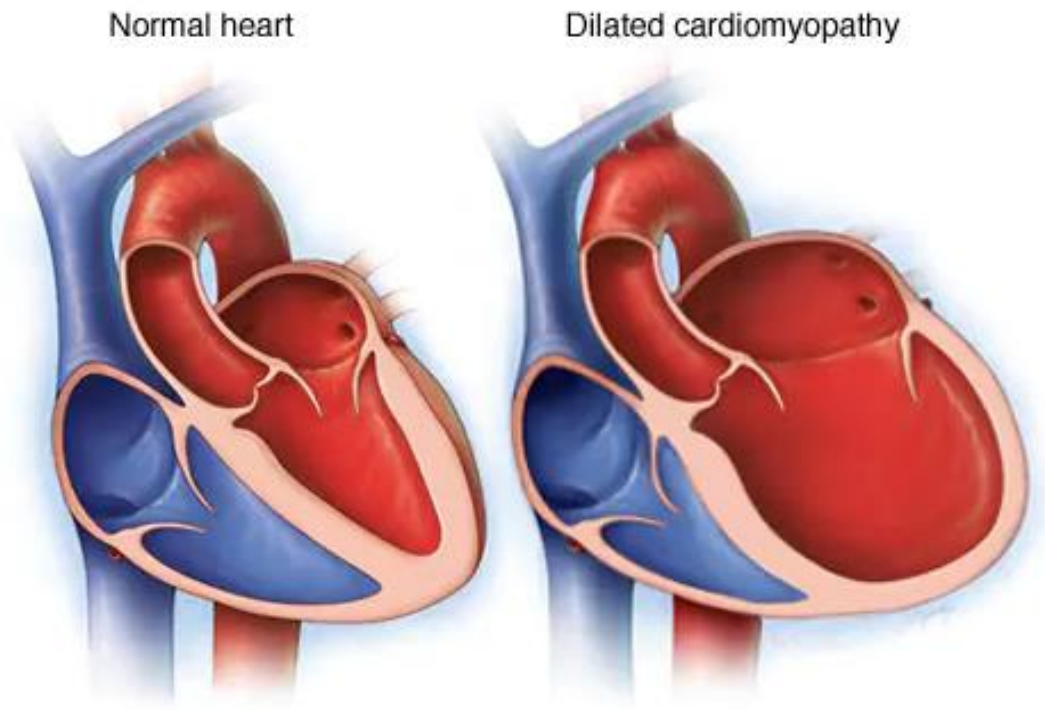
- Cardiomyopathy is a disease of the Heart muscle, causing it to become enlarged, thickened or rigid.
- As the condition progresses the heart becomes weaker, less able to pump blood or maintain an electrical rhythm.
- Often contributes to heart failure or arrhythmia or valve pathology.

Cardiomyopathy

- Three main presentations of Cardiomyopathy:
  - Dilated
  - Hypertrophic
  - Restrictive

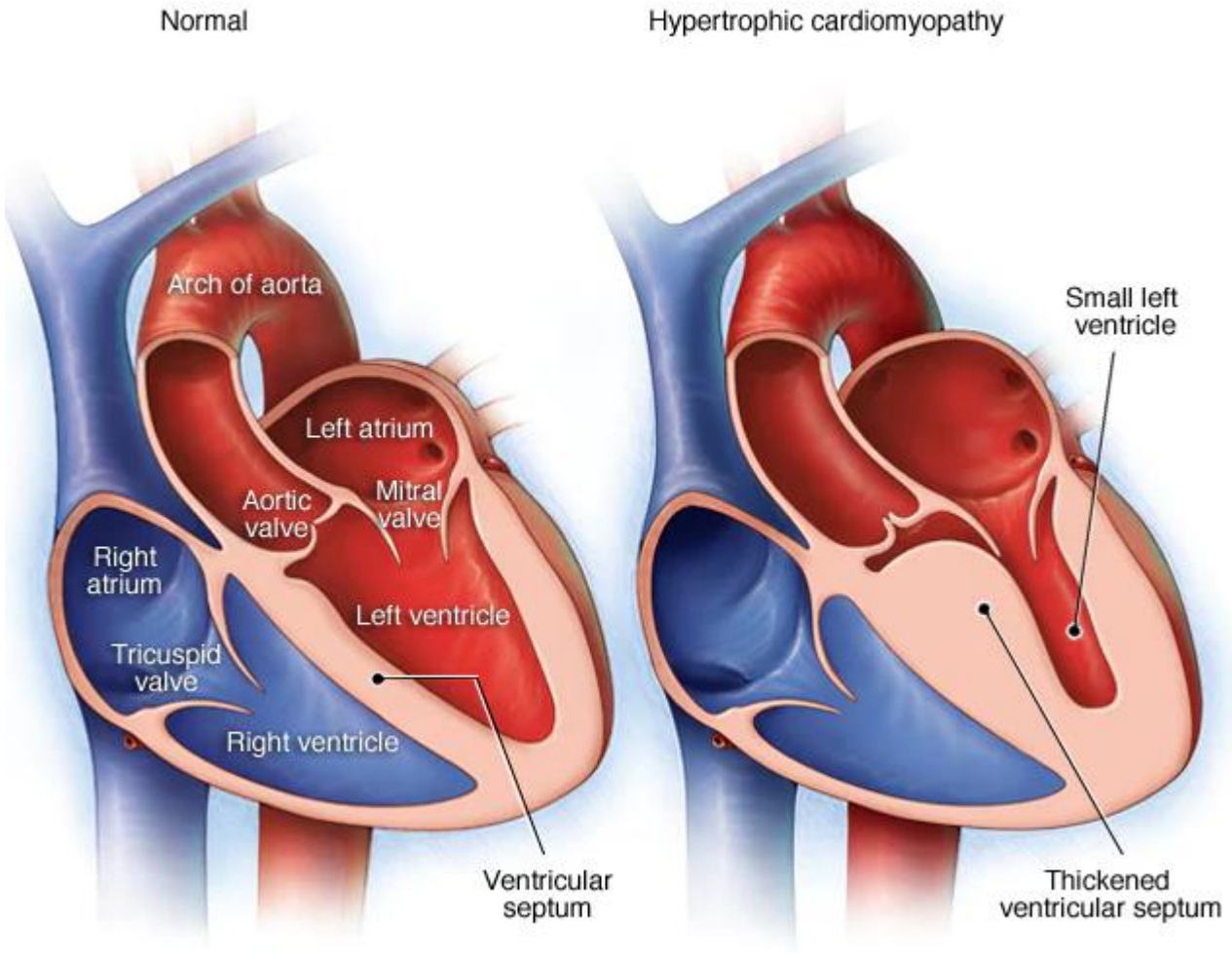
Cardiomyopathy: Dilated

- Condition where the ventricles stretch and become thinner leading to heart failure.
- Reduced contractility leads to diminished systolic pressure and stroke volume.



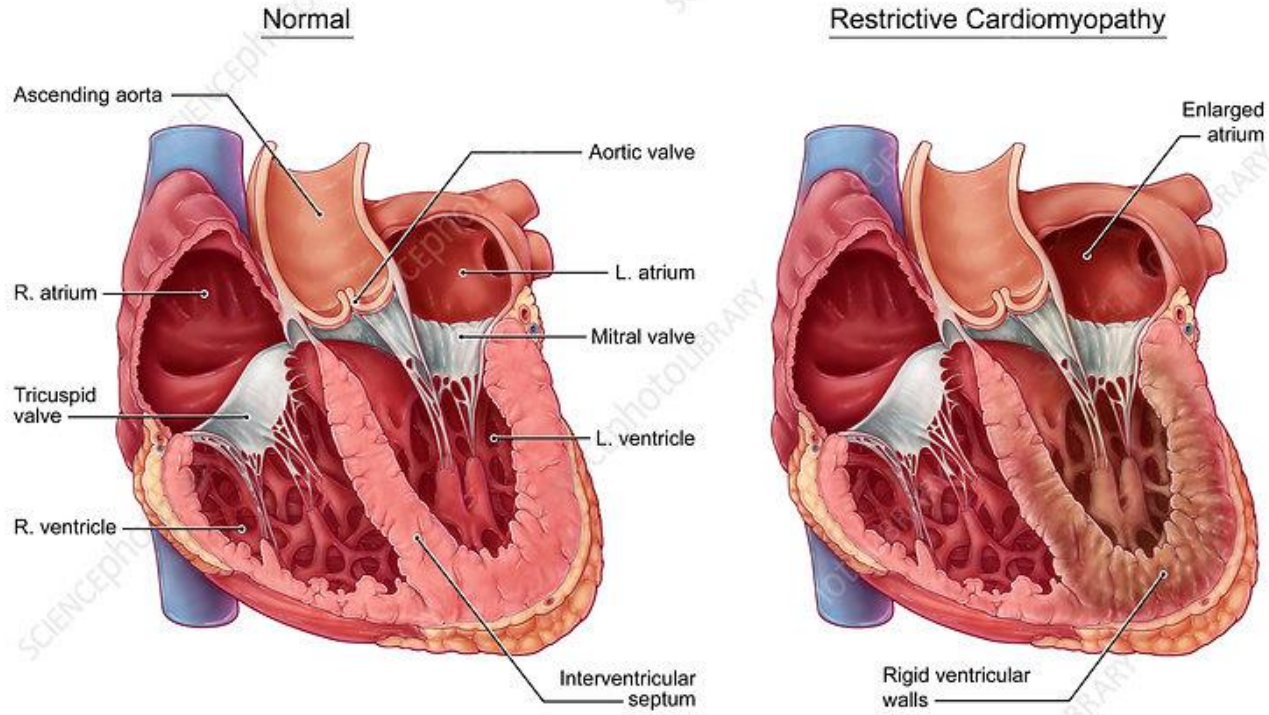
Cardiomyopathy: Hypertrophic

- Occurs when heart muscle cells enlarge and cause the walls of the ventricles to thicken.
- Reduces blood flow from the ventricle, leakage from the mitral valve, increase blood pressure and cause arrhythmia.



Cardiomyopathy: Restrictive

- Ventricles become rigid due to abnormal tissue (usually scar tissue) replacing heart muscle.
- Impedes ventricular filling and increases filling pressures.
- Often leads to CHF, cardiomegaly or dysrhythmia



Cardiomyopathy

Pathophysiology	Dilated	Hypertrophic	Restrictive
Major symptoms	Fatigue Weakness Palpitations	Dyspnea Angina Fatigue Syncope Palpitations	Dyspnea Fatigue
Conduction defects	Intraventricular	Nonspecific	Atrioventricular
Dysrhythmias	Sinoatrial tachycardia Atrial Ventricular	Atrial Ventricular	Tachydysrhythmia
Associated conditions	Alcoholism Pregnancy Infection Nutritional deficiencies Toxic exposure	Genetic defect	Infiltrative disease
Eventual effect	Left Heart Failure	Left Heart Failure	Right Heart Failure

Cardiogenic Shock

- 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

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

Right Ventricular Failure

Management

- Primary Survey (ABC's, RBS)
- Place pt at rest (Supine or semi-Fowler if dyspneic)
- Baseline Vitals
- Administer O_2 as required, 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)

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Cardiovascular Pathophysiology

HYPERTENSION

Hypertension

- A chronic condition where BP is consistently greater than 130/80 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 (Generalized)

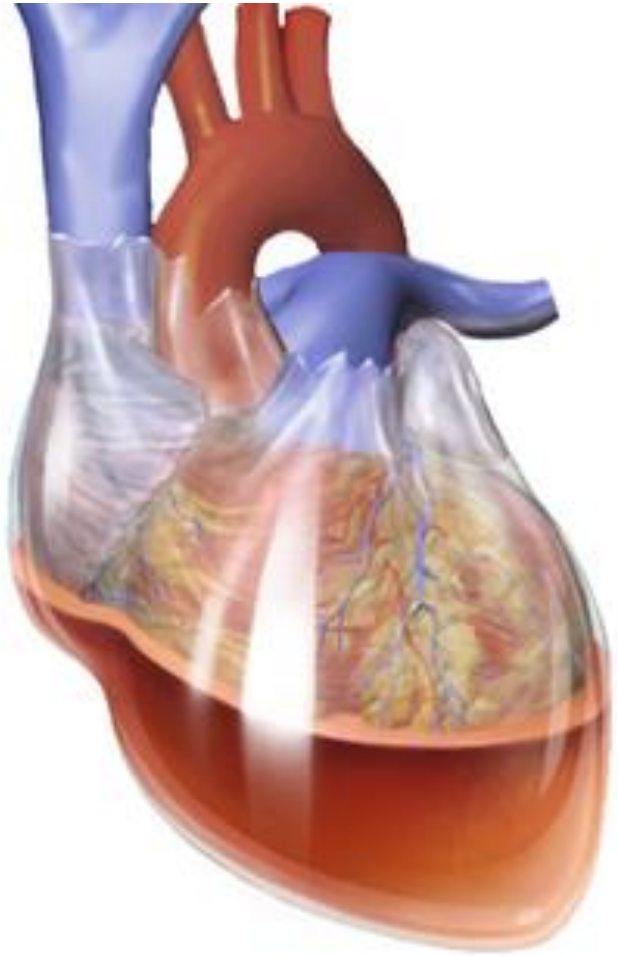
- Paroxysmal nocturnal dyspnea
- SOB
- ALOC
- Vertigo
- Headache
- Epistaxis
- Tinnitus (ringing in the ear)
- Changes in visual acuity
- N/V
- Seizures
- ECG changes

Cardiovascular Pathophysiology

PERICARDIAL TAMPONADE

Pericardial Tamponade

- Defined as impaired diastolic filling of the heart by increased intrapericardial pressure and volume
- Excessive fluid accumulates within the pericardium.
  - Limits the heart's ability to expand fully after each contraction thus decreasing SV
- Maybe
  - Gradual in onset (neoplasm, infection)
  - Acute (trauma, CPR)



Pericardial Tamponade

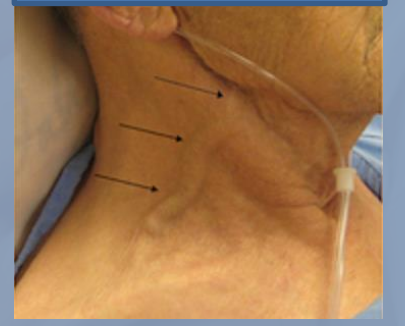
Signs and Symptoms

- 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)
- Narrowing pulse pressure
- Pulsus paradoxus
- Faint or muffled heart sounds

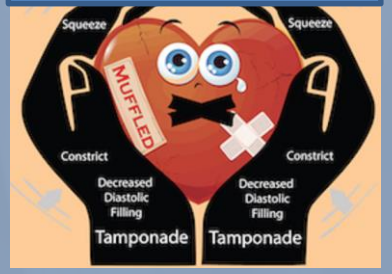
Beck's Triad

Hypotension

JVD



Muffled Heart Sounds



Pericardial Tamponade

Management

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

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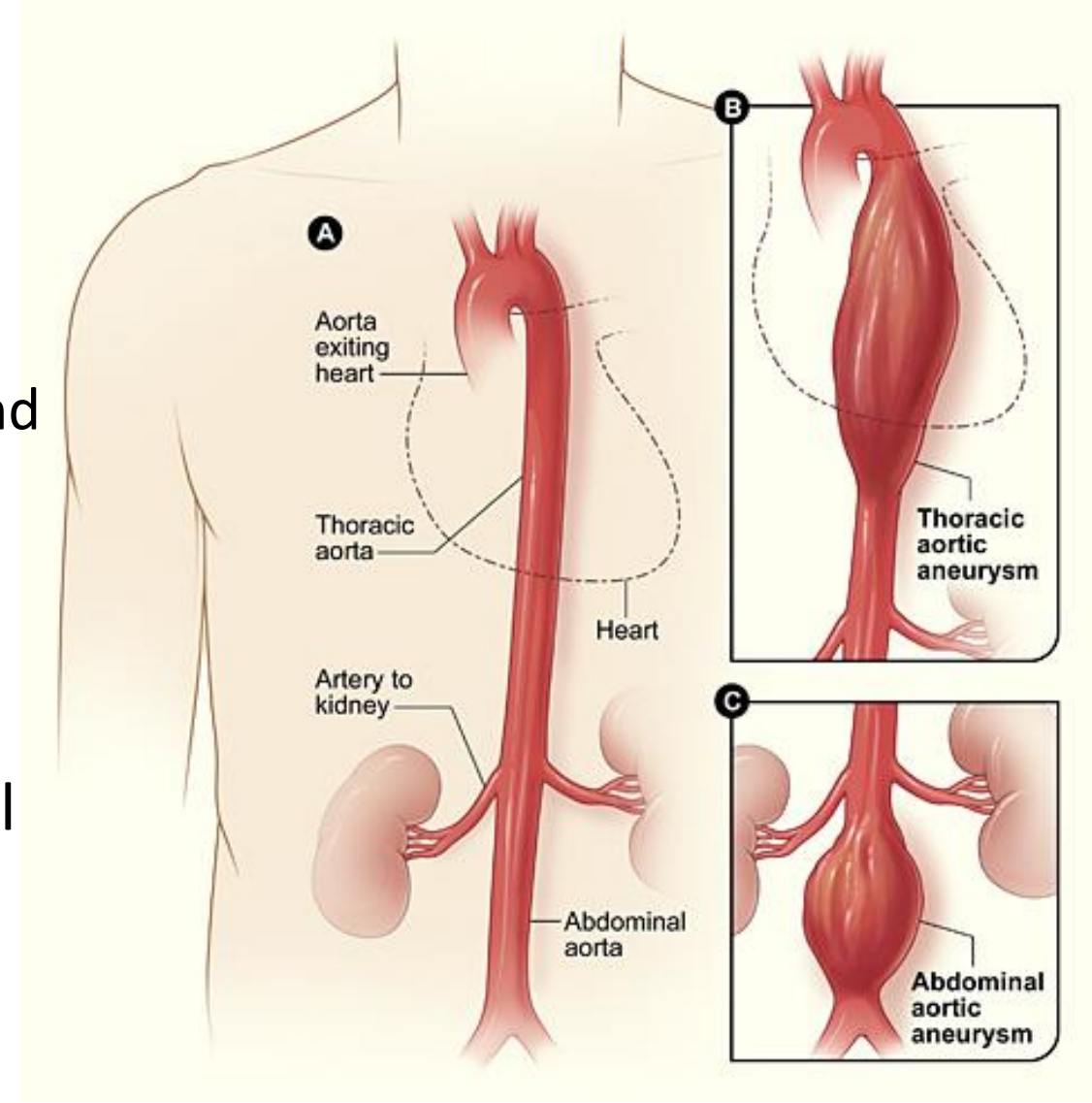
Cardiovascular Pathophysiology

AORTIC EMERGENCIES

Aortic Aneurysm

- 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
- Most common concern is an expanding or ruptured aneurysm of the abdominal aorta
- Dissection is a tear in the innermost wall of the artery, which leads to blood flow between the layers of the artery



Acute Dissection

- Degenerative changes in the media of the aorta
- Pronounced with advancing age, in people with chronic high blood pressure, and in younger patients with connective tissue disorders
- May lead to a disruption of the underlying intima
- Once intima is torn, dissection often begins:
  - A jet of blood forces into the torn arterial wall
  - Creates a false channel between the intimal and medial layers of the wall
- May prevent the valve from closing
- If the valve is affected, coronary blood flow will likely be affected as well.

Signs and Symptoms

- Typical patient is a middle-aged or older man with chronic hypertension.
- Most common chief complaint is chest pain.
- Pain comes on suddenly.

Table 30-13 AMI Versus Dissecting Aortic Aneurysm

	AMI	Dissecting Aortic Aneurysm
Onset of pain	Gradual, with prodromal symptoms	Abrupt, without prodromal symptoms
Severity of pain	Increases with time	Maximal from the outset
Timing of pain	May wax and wane	Does not abate once it has started
Location of pain	Substernal; back is rarely involved	Back is often involved, between the shoulder blades
Clinical signs	Peripheral pulses equal	BP discrepancy between arms or a decrease in the femoral or carotid pulse

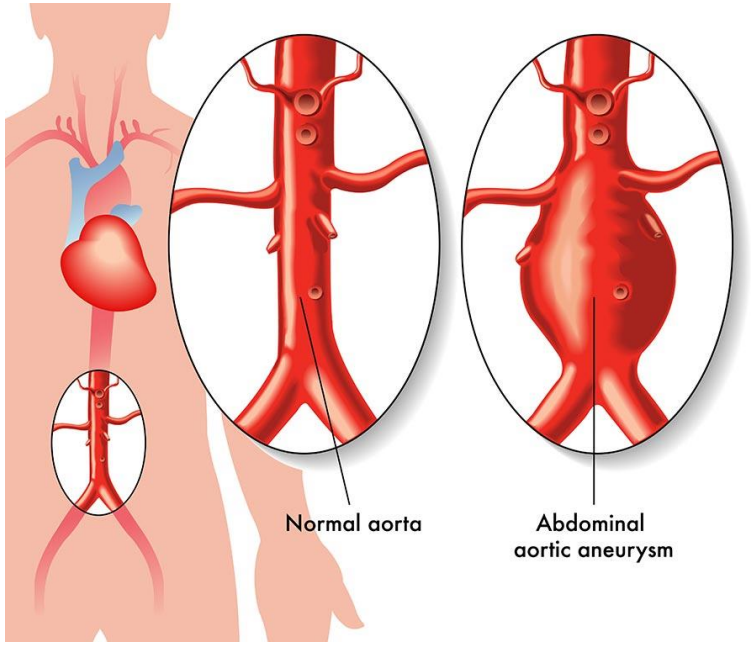
Abbreviations: AMI, acute myocardial infarction; BP, blood pressure

Signs and Symptoms

- Other signs and symptoms depend on the site of the intimal tear and the extent of the dissection.
- Disruption of flow through the brachiocephalic or left subclavian artery is likely to produce a difference in blood pressure between the two arms.
- Disruption of blood flow into the left common carotid artery may produce signs and symptoms of a stroke.
- Death is nearly always a result of aortic rupture into the pericardium and resultant cardiac tamponade.
- Occurs more commonly in older patients with a history of hypertension

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 as required, assist if necessary
- Initiate IV (TKVO but if ruptured fluid challenge)
- Attach monitor
- Transport

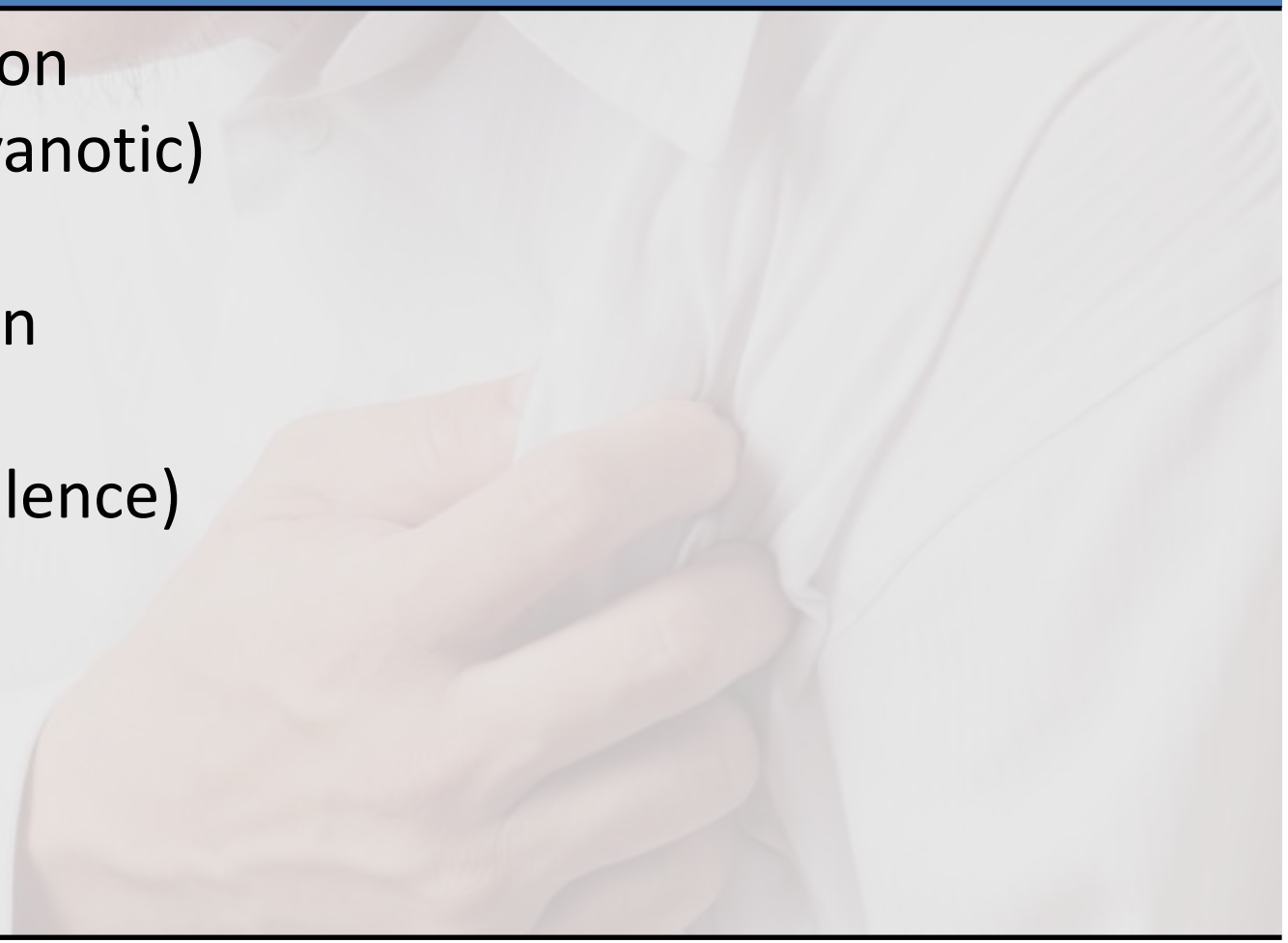
Arterial Occlusion

- 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

Cardiovascular Pathophysiology

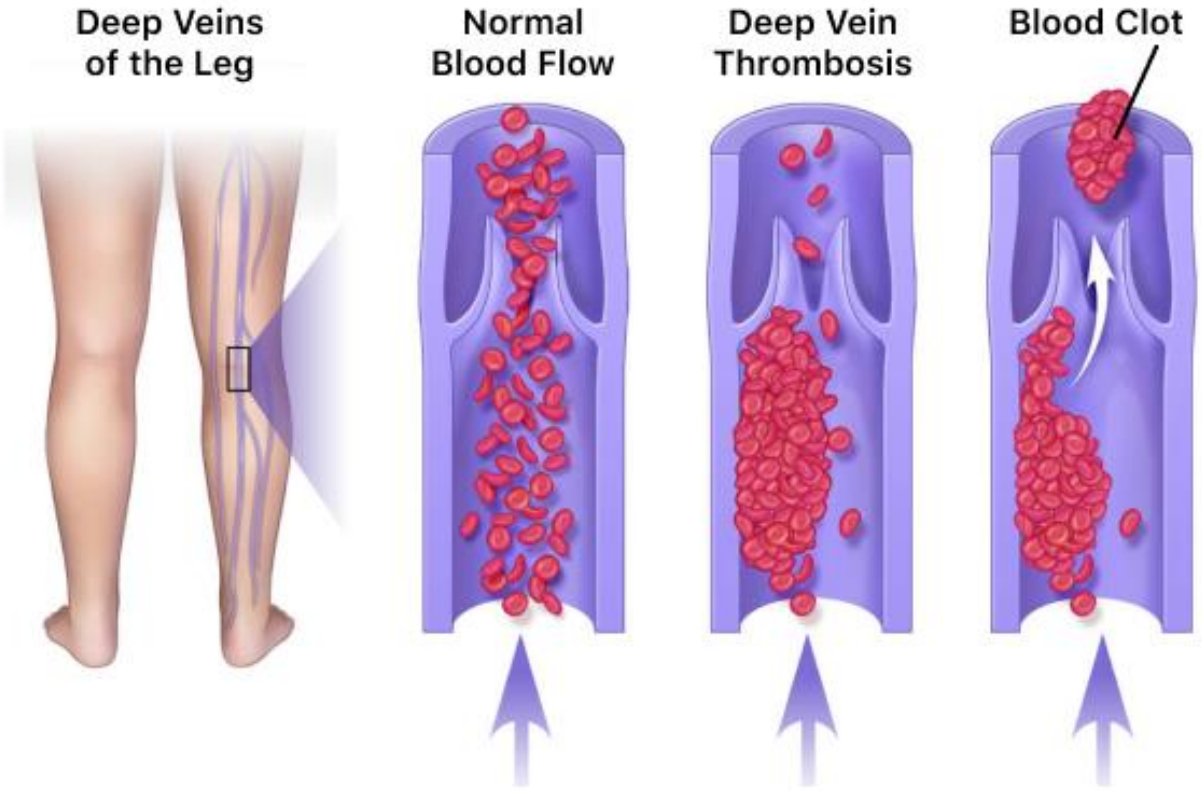
PERIPHERAL VASCULAR DISEASE

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

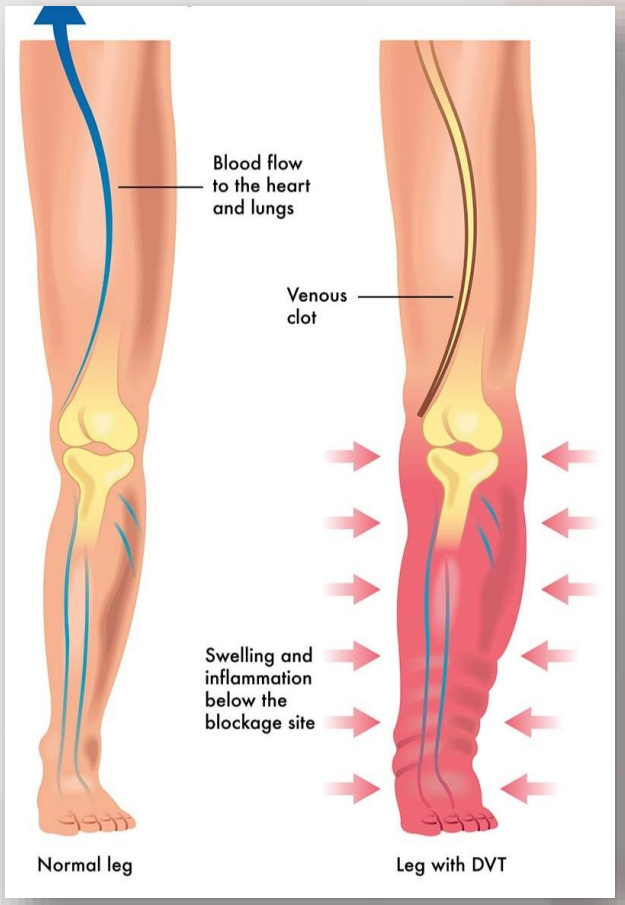
- Occlusion of the deep veins
- Most common in the lower extremities
- Risk factors
  - Recent trauma
  - Advanced age
  - Recent MI
  - Inactivity
  - CHF
  - Cancer
  - Oral contraceptives
  - History of thrombus
  - Obesity
  - Smoking



Arterial Occlusion

Signs and Symptoms

- Pain
- Edema
- Warmth
- Erythema
- Cyanosis
- Tenderness



Management

- Immobilization and elevation of the extremity

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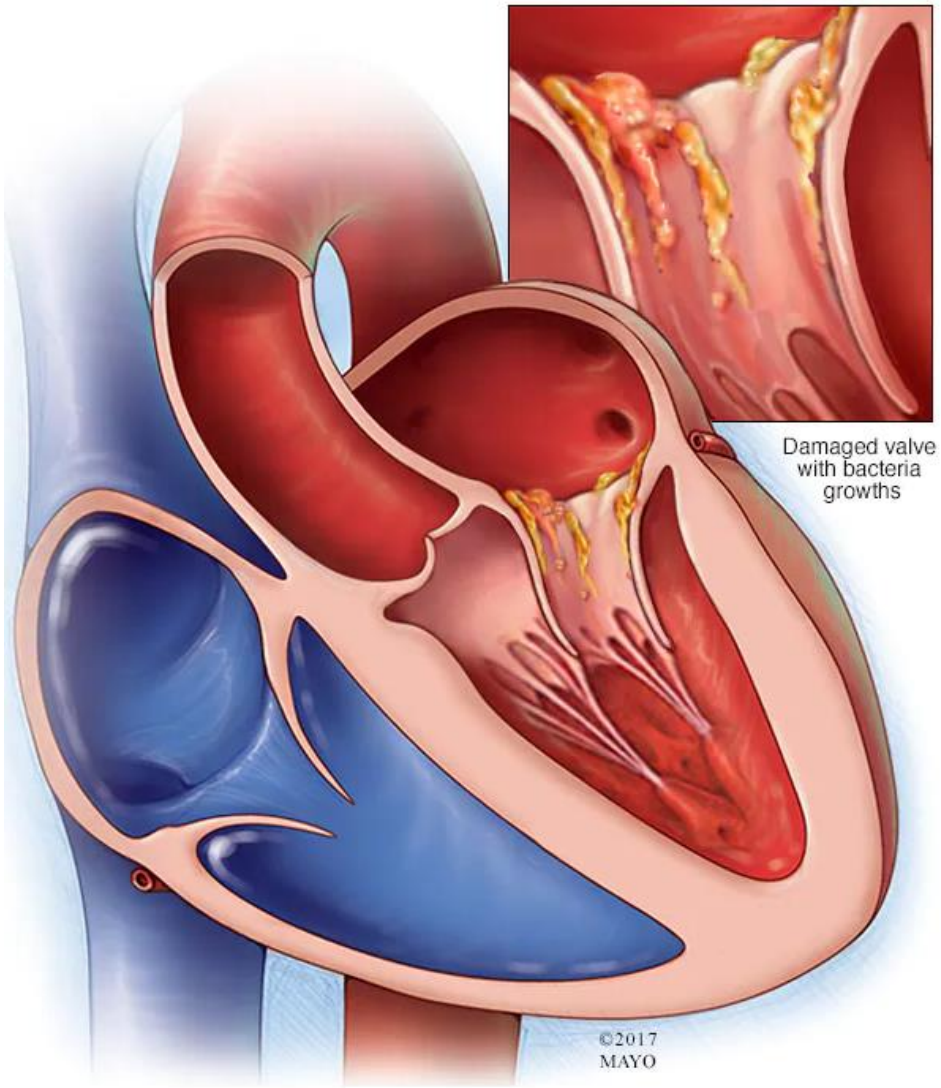
Cardiovascular Pathophysiology

INFLAMMATORY DISORDERS

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.



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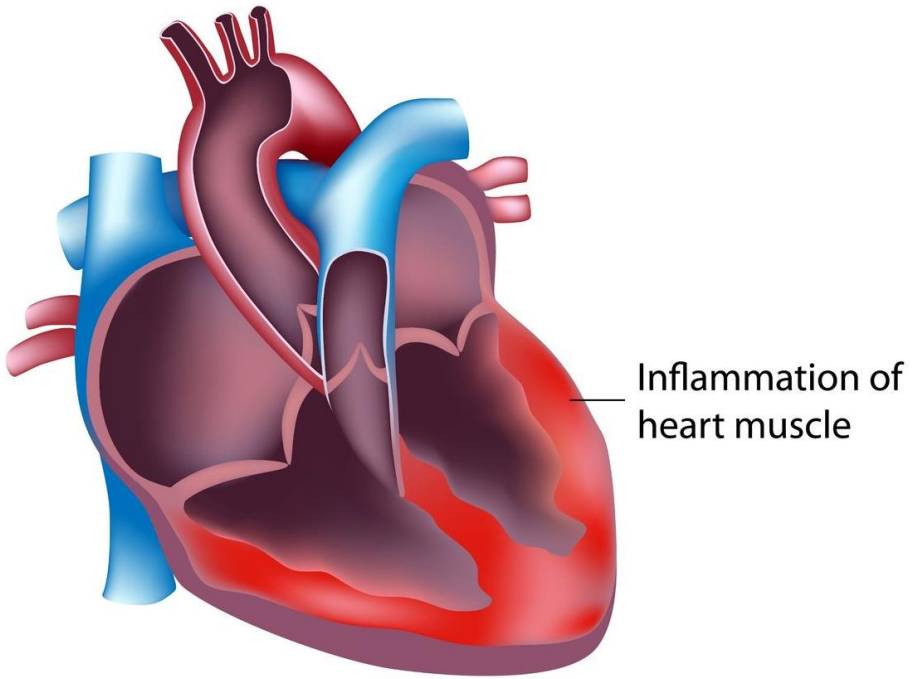
Endocarditis

Signs and Symptoms	Management
• 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.	• 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

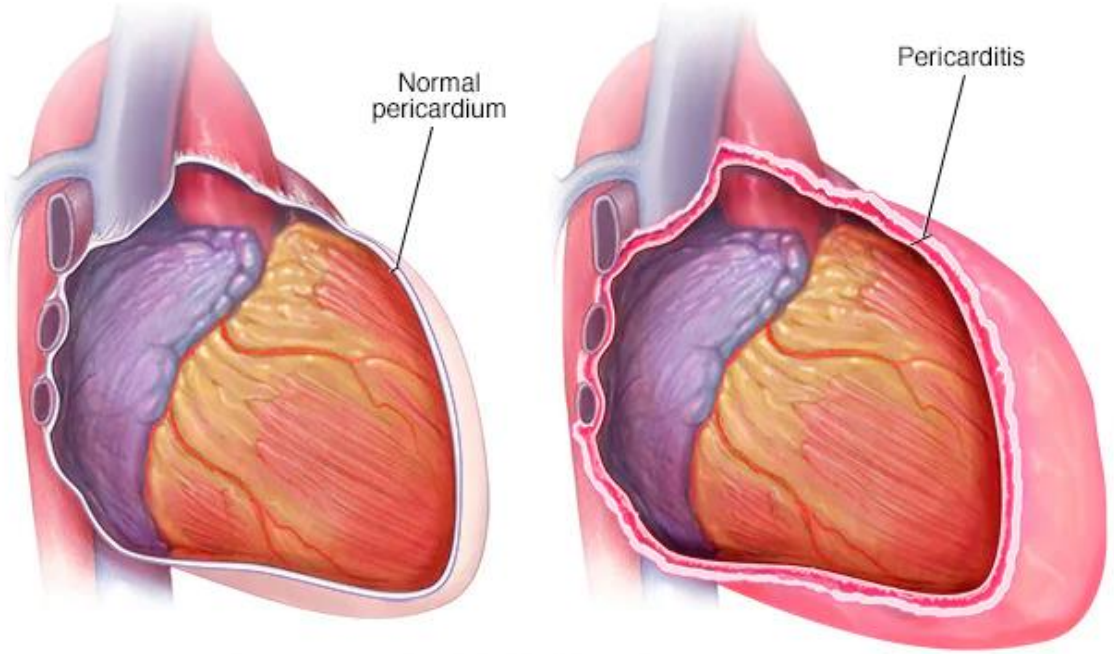


Myocarditis

Signs and Symptoms	Management
• 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	• Symptoms generally clear up with rest and time. • Management of chest pain and arrhythmias is most important. • 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 (buildup of toxins)
  - Tumors (Cells from tumor may metastasize to the pericardium)
  - Radiation therapy
  - Tuberculosis
  - Overactive immune system (rheumatoid arthritis and lupus)



Pericarditis

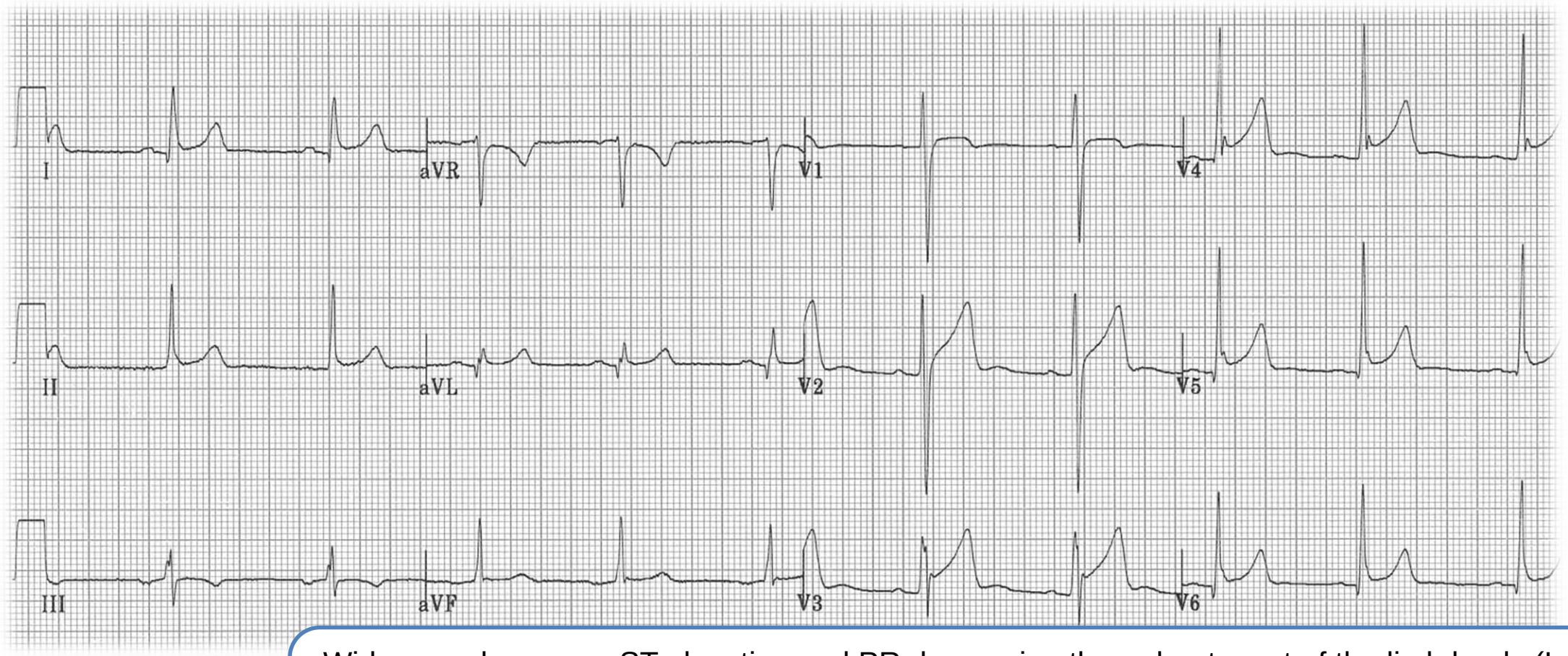
Signs and Symptoms

- 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

Management

- Anti-inflammatory agents (Aspirin)
- NSAIDs (ibuprofen, Motrin, Aleve)
- If pericardial effusion is significant treat as tamponade

Pericarditis



- Widespread concave ST elevation and PR depression throughout most of the limb leads (I, II, III, aVL, aVF) and precordial leads (V2-6)
- Reciprocal ST depression and PR elevation in lead aVR
- Sinus tachycardia is also common in acute pericarditis due to pain and/or pericardial effusion