Cardiac Anatomy and Physiology

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Objectives

• Describe the size, shape, location, and orientation of the heart.

• Describe the normal anatomy of the heart and peripheral circulatory system.

• Name and describe the location of the cardiac valves.
• Describe the anatomy of the coronary arteries and the veins.
• Describe the differences between the structural and functional aspects of the arterial and venous systems.
• Describe the structure and function of capillaries.
• Describe the course of blood flow through the heart and lungs.

• Describe the cardiac cycle.

• Define the following terms.

• Stroke volume
• preload

• afterload
• cardiac output

• Starling’s law
• Describe the innervation of the heart.
• Name major electrolytes that affect cardiac function.
• Describe the electrical properties of the heart.
• Describe the normal sequence of electrical conduction through the heart.
• Describe the normal sequence of electrical conduction through the heart.

• Describe cardiac depolarization and repolarization.

• Name three areas of the heart with pacemaking capabilities, and list the intrinsic rates of each.
Review of Basic A & P
Anatomy and Physiology

• Location
  – Between Lungs
  – Mediastinum
  – Base - 2nd intercostal
  – Apex - left & downward
  – Apex & right ventricle rest on diaphragm
  – Posteriorly

• Size and Dimensions
  – 10-12 cm long
  – 9 cm wide
  – 6 cm thick
  – Weighs 300 grams
  – Size of owner’s fist
Cardiovascular Anatomy

• Anatomy of the Heart
  – Location and Size of the Heart
Organ Membrane

• Pericardium - Double Sac (2)
  – Visceral - inner, serous layer (epicardium)
  – Parietal - (outer) attached to pleura & diaphragm
Heart Wall

• Myocardium
  – lined inside by endocardium
  – folding of endocardium form valves
  – endocardial lining is continuous w/the lining or arteries, capillaries & veins
  – specialized muscle cells found only in heart
  – muscle fibers 1/3 smaller than others
  – very dependant on calcium for contraction
Myocardium

- Muscular layer of ventricle wall contain myofibrils composed of:
  - actin
  - myosin
  - Ability to contract
Cardiovascular Anatomy

- Tissue Layers
  - Endocardium
  - Myocardium
  - Pericardium
    - Visceral Pericardium
    - Parietal Pericardium
    - Pericardial Fluid
Heart Chambers

• Atria
  – right and left
  – less muscular

• Ventricles
  – right and left
  – more muscular
Heart Chambers

• Septum
  – interatrial
  – intraventricular
  – both: connective tissue and muscle
Heart Valves

• Atrioventricular (AV) Valves
  – Tricuspid - 3 flaps
  – Bicuspid - 2 cusps
  – both are anchored by Chordae Tendineae to papillary muscles of ventricles

• Semilunar valves
  – Pulmonary - proximal end of pulmonary artery
  – aortic valve - proximal end of aorta
Cardiovascular Anatomy

- **Valves**
- **Atrioventricular Valves**
  - Tricuspid Valve
  - Mitral Valve
- **Semilunar Valves**
  - Aortic Valve
  - Pulmonic Valve
- **Chordae Tendonae**
Cardiovascular Anatomy

- Blood Flow
  - From the Body
    - Right Atrium
  - To the Lungs
    - Right Ventricle
  - From the Lungs
    - Left Atrium
  - To the Body
    - Left Ventricle
Describe the flow of blood through the circulatory system beginning at the right atrium.
• Unoxygenated blood flows into the right atrium from the Superior Vena Cava, the Inferior Vena Cava and from the heart itself through the Coronary Sinus
• Blood then travels through the tricuspid valve into the right ventricle
• Which pumps the blood through the pulmonic artery and to the lungs where it is exchanges carbon dioxide for oxygen
• The oxygenated blood travels through the pulmonary veins into the left atrium
• From there it is pumped through the mitral valve into the left ventricle through the aortic valve and into the systemic circulation
Cardiac Circulation

- Right Coronary Artery (RCA)
- Posterior Descending Artery (branch of RCA)
- Left Anterior Descending (LAD)
- Left Circumflex Artery (LCA)
Cardiovascular Anatomy
– Coronary Circulation
• Collateral Circulation
Coronary Arteries

- Originate in the aorta just above the aortic valve
- Empty into the coronary sinus
Right Coronary Artery

- Originates from right side of aorta
- **Inferior wall** of the left ventricle
- SA node in 50% population
- AV node in 90% population
- Right ventricle
Posterior Descending Artery

- Posterior fascicle of the left bundle branch
- posterior wall of the left ventricle
- left bundle branch
Left Anterior Descending

• Originates from left side of aorta
• “widow maker”- associated with sudden death
• Anterior wall of the left ventricle
• Intraventricular septal artery (septum)
• RBBB, LBBB, both fascicles
Left Circumflex Artery

- Lateral wall of left ventricle
- SA node in 45% of patients
- AV node in 10% patients
- Posterior wall of left ventricle
Cardiac Physiology

- Nervous Control of the Heart
  - Sympathetic
  - Parasympathetic
  - Autonomic Control of the Heart
    - Chronotropy
    - Inotropy
    - Dromotropy
  - Role of Electrolytes
Nervous System

- The sympathetic system influences both the atrium and ventricle
- The parasympathetic system primarily influences the atria
- When might vagal maneuvers be effective?
- Why?
- What does Atropine do?
Review

• Where is the heart located?
• What is its function?
• How is the heart organized?
• What are the layers of the heart?
• What sac surrounds the heart?
Review

- What is the average size and shape of the heart?

- Describe the cardiac cycle- diastole and systole- we’ll talk more about this later!

- Which ventricle ejects more blood?
Valve Disorders

- Murmors - abnormal sounds
- Valvular insufficiency
- Stenosis
Valvular Insufficiency

Growth of scar tissue on the valves as a result of disease (rheumatic fever). Valves do not seal and blood leaks back (gurgitation) Causes detectable turbulence
Stenosis

Valvular growths narrow openings & interfere w/blood flow. Increased pressure is required to force blood thru. Causes heart to work harder. Valves are roughened which causes turbulence and murmur
Functional murmurs

Common in the young, especially during exercise. Not pathological and considered normal
Great Vessels - not boats :) 

- Vena Cava (2)
- Pulmonary artery (2)
- Pulmonary veins (4)
- Aorta

(collective name for large vessels attached to base of heart)
Blood Vessels

- Vessels of the peripheral circulation are made up of several layers
- tunica intima - innermost lining, one cell thick
- tunica media - elastic fiber and muscle, thicker in arteries
- tunica adventitia - outermost, fibrous lining
- lumen - inside cavity
Poiseuille’s Law

• Amount a vessel can transport is directly related to its diameter
• Blood flow through a vessel is directly proportional to the 4th power of the vessel’s radius
Arterial System

• Carries blood from the heart
• Functions under high pressure
• Larger vessels- arteries
• Arterioles
• Capillaries- points of connection between arterial and venous systems, exchange of gases
Venous System

- Transports blood back to heart
- Low pressure
- One way valves
- Capillaries drain into venules
- Then into veins
Cardiovascular Anatomy

• Anatomy of the Peripheral Circulation
  – General Structure
    • Poiseuille’s Law
  – Arterial System
    • Arteries, arterioles, and capillaries
  – Venous System
    • Capillaries, venules, and veins
Terminology

• Venous return - amount of blood flowing into the right atrium each minute from the systemic circulation
• Cardiac output - Amount of blood pumped into the aorta each minute by the heart
  • CO = SV X HR
  • about 4-8 L/ min
Con’t

• Stroke volume- amount of blood ejected from a ventricle with each heart beat- usually 60-100ml
• Affected by:
  • preload- force exerted by the blood on the walls of the ventricles at the end of diastole
  • afterload- resistance against which ventricle must pump to eject blood
• contractility
Frank Starling Law: greater the volume of blood in the heart during diastole, more forceful the contraction

Ejection fraction: percentage of total ventricular volume ejected during each myocardial contraction

- normal > 50% (>70%)
- impaired function < 40%
Con’t

• Blood pressure - mechanical activity of the heart reflected by the pulse and BP. Measures the force exerted on the walls of the arteries.

• Peripheral resistance - resistance to the flow of blood determined by vessel diameter and tone.

• $BP = CO \times PR$
Review

- Cardiac output =
- SV is affected by…
- Frank Starling’s Law affects….
- Define
  - preload
  - afterload
- BP reflects …
Cardiac Cycle

- The heart performs its pumping action over and over in a rhythmic sequence:
- Atrial Diastole- the atrium is relaxed allowing blood from the body and lungs to fill the atrium
- As the atria fill with blood, the pressure rises above that in the ventricles, forcing the tricuspid and mitral valves to open allowing blood to fill relaxed (diastole) ventricles
Con’t

• Then the atria contracts (systole) filling the ventricles to capacity. This “atrial kick” accounts for 30% of the cardiac output.

• What is atrial fibrillation and how might it affect patients?

• Pressure in the atria and ventricles equalize and the tricuspid and mitral valves begin to close

• Then the ventricles contract (systole) causing ventricular pressure to rise and the aortic and pulmonic valves open
Cardiac Physiology

• The Cardiac Cycle
  – Diastole
  – Systole
  • Ejection Fraction

• Stroke Volume
  – Preload
  – Cardiac Contractility
  – Afterload

• Cardiac Output
Heart Cells

- Myocardial- mechanical
- Pacemaker- electrical
Cardiac Physiology

– Cardiac Conductive System
• Properties
  – Excitability
  – Conductivity
  – Automaticity
  – Contractility
Excitability

• Irritability
• Shared by all cardiac cells
• Ability of cardiac muscle cells to respond to an external stimulus (chemical, electrical, mechanical)
Conductivity

• Ability of cardiac cell to receive an electrical stimulus and conduct that impulse to an adjacent cell
• All cardiac cells possess this characteristic
Automaticity

• Ability of cardiac pacemaker cells to **spontaneously initiate** an electrical impulse
• SA node, AV node and Purkinje fibers usually possess this characteristic
Contractility

• Ability of cardiac cells to shorten, causing muscle contraction in response to electrical stimulus
• Can be enhanced by medications- digitalis, dopamine, epinephrine
Heart

• Composed of cylindrical cardiac cells that partially divide at their ends. These connect with branches of adjacent cells forming a branching and anastomosing network of cells called syncytium. At the junctions where branches join are specialized cellular membranes called intercalated discs that permit very rapid conduction of electrical impulses from one cell to another.
Cardiac Physiology

- **Electrophysiology**
  - **Cardiac Muscle**
    - Atrial
    - Ventricular
    - Excitatory and Conductive Fibers
      - Intercalated discs
      - Syncytium
  - **Atrioventricular Bundle**
    - Depolarization
• Cardiac cells are enclosed in a semi-permeable cell membrane that allows certain charged chemical particles (ions) such as Na+, K+, Ca++ to flow in and out of cells resulting in contraction and relaxation of the heart
Cardiac Physiology

- Cardiac Conductive System
  - Components
    - Sinoatrial Node
    - Internodal Atrial Pathways
    - Atrioventricular Node
    - Atrioventricular Junction
    - Bundle of His
    - Left and Right Bundle Branches
    - Purkinje Fibers
Electrical Pathway

- Originates in the SA node
- Travels down the atrial intranodal and intraatrial pathways
- Slows at the AV node allowing the atria to contract and empty
- Travels to the Bundle of HIS
- Travels more rapidly to the bundle branches
Bundle Branches

• The left common bundle branch further divides into two major divisions the left posterior and left anterior fasicle
• The impulse then travels to the Purkinje fibers causing the ventricles to contract
Electrical Conduction System

- What is the primary function of the electrical conduction system of the heart?
- The SA node is located?
- What is its purpose?
- Its intrinsic rate is?
- The intrinsic rate of the AV node/ventricles is?
Electrolytes

• Cardiac Function - electrical & mechanical is influenced by electrolyte (imbalance)
  – sodiums major role is depolarization of myocardial cells
  – calcium major role is depolarization phase of myocardial pacemaker cells & in myocardial contractility
  – hypercalcemia - increases contractility
  – hypocalcemia - decrease contractility & increases electrical irritability
Electrolytes (cont.)

- Potassium - major role is repolarization phase
- hyperkalemia - decreased automaticity & conduction
- hypokalemia - increased irritability
POLARIZATION
resting membrane potential

• Resting state with no electrical activity
• Inside the cell is more negative than outside
• K+ - primary intracellular anion
• K+ slowly leaks out of cell
• Leaving negative charged ions inside
DEPOLARIZATION

• Cardiac muscle cell is stimulated causing the inside of the cell to become more positive
• Na+ diffuses into cell resulting in greater positive charge inside the cell
• Change in charge occurs from the endocardium to the epicardium
REPOLARIZATION

• After the cell depolarizes diffusion of Na+ stops
• K+ is allowed to diffuse out of cell leaving negative ions in the cell
• Occurs secondary to outward diffusion of K+
• Occurs from epicardium to endocardium
Escape Pacemakers & Latent Pacemakers

• Depolarize Automaticity
  – ischemia, hypoxia, acidosis, electrolyte imbalance
  – the farther the escape pacemaker from SA node, slower it’s firing rate
  – escape or ectopic beats
Enhanced Automaticity

- Abnormal condition of latent pacemaker cells when their firing rate is increased beyond their inherent rate
Enhanced Automaticity

• Cell membrane becomes abnormally permeable to Na
• Na leaks into cell = sharp rise in phase 4 = depolarization
• Causes atrial, junctional & ventricular ectopic beats

• Causes
  – increased catecholamines
  – dig toxicity
  – hypoxia, hypercarbia
  – ischemia, infarction
  – hypokalemia, hypercalcemia
  – Starling’s Law
  – Atropine
Re-Entry

- Condition in which the progression of an electrical impulse is delayed or blocked in the conduction system
- Impulse is conducted normally through rest of system
- Delayed impulse goes through & goes back into cells already depolarized
Review

• Name two types of cardiac cells.
• Describe their function.
• Define automaticity.
• Depolarization is the same as contraction.
• T / F
Phases of Cardiac Action Potential

- Phase 0- represents rapid depolarization
- Begins when the cell receives an impulse
- Na+ moves rapidly into the cell through fast Na channels
- K+ leaves the cell
- Ca++ moves slowly into the cell through slow Ca channels
- Cell depolarizes and cardiac contraction begins
Cardiac Action Potential

- Phase 1 - early repolarization
- Early, brief period of limited repolarization
- Fast Na channels partially close slowing influx of Na+ and exit of K+
- Results in a positive charge within the cell
Cardiac Action Potential

• Phase 2- Plateau occurs secondary to the slow influx of Ca++ through slow Ca++ channels
• Continued exit of K+ through K+ channels allows cardiac muscle to sustain increased period of contraction
• ABSOLUTE REFRACTORY PERIOD
Cardiac Action Potential

- Phase 3- late and rapid repolarization
- Cell rapidly completes repolarization as K+ quickly exits
- Slow channels close stopping influx of Ca++ and Na+
- RELATIVE REFRACTORY PERIOD
Cardiac Action Potential

- Phase 4- resting membrane potential
- Excess Na+ inside cell
- K+ outside cell
- Na+/ K+ pump activated
- Transports Na+ out and K+ in
- Polarized
Cardiac Physiology

– Cardiac Depolarization
  • Resting Potential
  • Action Potential
  • Repolarization
Fast Response Action Potential

- Occurs in atria, ventricles and Purkinje fibers
- Occurs secondary presence of many voltage sensitive Na+ channels
- Allow rapid influx of Na+
Slow Response Action Potential

• SA and AV nodes have slow Ca++ and slow Na+ channels

• Occurs normally in the SA and AV nodes but can occur abnormally anywhere in the heart

• Usually secondary to ischemia, injury and electrolyte imbalance
Concentrations of Extracellular Ions

- Hyperkalemia (K+)- heart becomes extremely dilated and flaccid, decreased HR
- Hypercalcemia (Ca++)- spastic contractions
- Hypocalcemia (Ca++)- flaccid, resembles hyperkalemia
Review

• What is the significance of “absolute” refractory period?
• Relative refractory period?
• Why is this important?
• During polarization the inside of the cell is more (negatively/positively) charged.
• Does an electrical impulse create a pulse?
• Why or why not?
• The electrical impulse stimulates the mechanical cells to contract if they respond the heart contracts and pumps blood = pulse
Review

• Discuss the cardiac cycle
• What is the electrical conduction pathway?
OBJECTIVES

• Name the common chief complaints of cardiac patients.
• Describe appropriate history and physical assessment goals for cardiac patients.
ASSESSMENT

• Systemic approach
• Check for responsiveness
• Open airway- patent, suction if needed
• Check for breathing- look, listen, feel
• Check circulation at Carotid & Radial simultaneously
  • skin- color, quality, temperature
  • pulse- rate & quality
  • capillary refill
Common Presenting Symptoms

- Chest pain or discomfort
- Shoulder pain
- Neck pain
- Dyspnea
- Syncope
- Palpitations
Occlusion Data

- CP at exertion- 70-85% occlusion
- CP at rest 90% occlusion
- CP that does not resolve with NTG = 100% occlusion
Chest Pain

• C.P. or discomfort most common presenting symptom of cardiac disease
• C.P. most common presenting symptom of myocardial infarction
• May be atypical especially in the elderly and women...if in doubt err on the side of the patient!!
OPQRST

- O-nset, what were you doing?
- P-rovocation, initiates pain, better/ worse
- Q-uality, crushing, pressure, tightness
- R-adiation to neck, jaw, shoulder, arm(s)
- S-everity, scale 1-10
- T-time of onset, intermittent/ constant
History

- Exact location
- Duration
- Factors precipitating pain - exercise, stress
- Associated symptoms - nausea, dyspnea
- Aggravating/ alleviating
- Similar episodes
Dyspnea

• Due to the interrelationship between the heart and respiratory system
• May be the only symptom of AMI
• May be primary symptom of pulmonary fluid congestion secondary to CHF
History

- Duration
- Onset - sudden/ progressively worse
- Aggravates/ alleviates dyspnea
- previous episodes
- Associated symptoms
- Past medical history
- Differential diagnosis
Syncope

- Interruption of blood flow to the brain
- Cardiac problems major cause
- May be the only presenting symptom of cardiac disease, especially elderly
- Usually result from dysrhythmias if cardiac etiology
- For example…explain
Palpitations

- Sensation that the heart is pounding, racing or skipping a beat
- Usually related to irregular or rapid heart rate
History

- Circumstances of occurrence
- Duration of occurrence
- Associated symptoms
- Frequency of occurrence
- Previous episodes
Significant PMH

• Prescription medications
• Medical history
  • heart attack
  • angina
  • heart failure
  • hypertension
  • diabetes
• Allergies
Differential Diagnosis

• Aortic dissection
• Acute pericarditis
• Acute myocarditis
• Pulmonary embolism
Physical Examination

• Primary Survey
• Secondary Survey
• JVD - 45% elevation
• peripheral edema
• scars, pacemaker, nitroglycerin patch
• breath sounds - adventitious sounds
• heart sounds
Heart Sounds

• S1- first sound produced by closure of AV valves (tricuspid, mitral) during ventricle systole
• S2- second sound produced by by closure of the aortic and pulmonary valves
• S3- third sound, associated with CHF
• S4- occurs immediately prior to S1 associated with increased atrial contraction
• Carotid Artery Bruit- turbulent blood flow through a vessel indicative of partial blockage
• Pulse- rate and regularity, equality
• Skin- pale and diaphoretic, indicative of peripheral vasoconstriction and sympathetic stimulation
• mottled often indicates chronic cardiac failure
Angina

• Chest discomfort or other related symptom caused by myocardial ischemia
• Myocardial ischemia occurs when the heart’s demand for O2 exceeds its supply from the coronary circulation
• Pain occurs secondary to the stimulation of nerve endings caused by a build up of lactic acid and CO2 in ischemic tissue
Characteristics

• Typically- heaviness, pressure, squeezing, constriction or pain

• Common sites- upper part of chest, beneath sternum radiating left arm, epigastric; beneath sternum radiating to neck and jaw; epigastric radiating to neck and jaw; left shoulder; intrascapular

• Diaphoresis, nausea, vomiting, weakness
Cause

- Decreased myocardial O2 supply
- OR
- Increased myocardial O2 demand
- IF
- Process is not reversed and blood flow restored severe myocardial ischemia may lead to cellular injury and eventually, infarction
Management

• Reduce O2 demand
• Increase blood flow to the myocardium

• What are some methods used to achieve this?
- O2, rest- increase supply, decrease demand
- Beta blockers- reduce heart rate
- NTG- increase blood flow by dilating coronary arteries
Stable

• Remains relatively constant and predictable in terms of severity, presentation, character, precipitating events, response to therapy

• Transient episodes of pain or discomfort related to activities that increase myocardial O2 demand

• Duration- 2-5 minutes, occasionally 5-15 min., > 30 min uncommon
Unstable

- Intermediate severity between stable angina and acute myocardial infarction
- Usually a result of plaque rupture or thrombus formation in a coronary artery and causes a sudden decrease in myocardial blood flow; spasm, HTN, hyperthyroidism, hypertrophic cardiomyopathy
Unstable Angina Con’t

- Usually 60-80 yo who have 1(+) CAD risks - HTN, hyperlipdemia, smoking, diabetes
- Symptoms at rest and usually > 20 min
- Progressive worsening of preexisting stable angina
- New onset (< 2 mo.) severe with minimal exertion
- Increased frequency- up to 30 min, radiates widely, may be accompanied by dyspnea
ECG Changes

- Myocardial ischemia delays repolarization so ECG may result in temporary changes in the ST segment and T wave
  - ST segment- depression
  - Ischemia present through full thickness of myocardium- inverted T wave results
  - Ischemia present only in the subendocardial layer T wave remains positive because direction of repolarization remains from the epicardium to the endocardium
Not Characteristic of Angina

- Sharp, stabbing pain brought on by respiratory movement or coughing
- Primary location of discomfort in mid- or low abdomen
- Pain reproducible with movement or palpation
- Constant pain lasting several hours or intermittent pain lasting only seconds
- Pain radiating to lower extremities
AMI

- Diagnosis is based on at least two of the following criteria:
  - Clinical history of ischemic type of chest discomfort
  - Changes on serially obtained ECG’s
  - Rise and fall in serum cardiac markers
Clinical Presentation

• Chest discomfort suggestive of ischemia-Levine’s sign
• May be accompanied by:
  • Nausea, vomiting
  • Dyspnea
  • Diaphoresis
  • Weakness, dizziness, anxiety
ECG Changes

- Injured myocardium does not function normally, affecting both muscle contraction and conduction of electrical impulses
- Injured myocardium depolarizes incompletely and remains electrically more positive than the uninjured surrounding it
- ST elevation in leads affecting injury
- Q waves of > 1/3rd R wave
Review

- A decreased supply of oxygenated blood to a body part or organ?
- Describe the pain pattern typically associated with chronic stable angina.
- Describe the characteristics of unstable angina.
- What ECG changes would expect to see in a patient experiencing angina? Why?
• When assessing a patient what other possible conditions may a AMI mimic?
• The most common presenting symptom of MI is?
• Name some other Sx.
Dissecting Aneurysm

- Pain usually described as ripping or tearing
- Substernal and radiating to the back between the scapula
- Pulses in lower extremities may be diminished or absent
- Pulses of LA & RA may be markedly different
- HTN with “shocky” appearance
Pulmonary Embolism

- Sudden onset of severe unexplained dyspnea
- Tachypnea
- History of recent immobilization
- Possible JVD, decreased BP
Pericarditis

- Usually younger patients with out cardiac risk factors
- Dyspnea
- Tachycardia
- Fever
- Malaise
- Weakness
- Chills
- Chest Pain
Con’t

- Increased severity with flat and improves while sitting up and leaning forward
- Rarely radiates down arm
- Often pleuritic and increases with inspiration
- May last for hours or days
- Pericardial friction rub
- ST segment elevation in most if not ALL leads
Scenario

• You are called late at night to the home of a 58 yo executive, who is complaining of a severe pain in his chest. He is pale, cool and diaphoretic. He is also nauseated.
History

• Has had back pain for several days which he thought was a strain and has been taking OTC pain relievers- ASA, ibuprofen
• Chest pain started about 30 minutes ago
• “Sharp, tearing pain” constant- 10/10
• Radiating through to his back
• Meds- ASA, NTG, some blood pressure pills
• PMH- angina, HTN
• Allergies- PCN
Vital Signs

- HR: 110 regular ST
- BP: 110/90
- RR: 18 and unlabored
- SpO2: 97%
Physical Exam

- **HEENT** - normocephalic, atraumatic
- No JVD, No TD, PERL
- **CHEST** - /= good excursion, normal resp effort
- **HEART** - RRR
- **LUNGS** - CTA bilaterally
- **ABD** - obese, SNT, +N,-V
- **BACK** - unremarkable
- **EXTREMITIES** - PMS - weak pulses in lower extremities, feet cold, No pedal edema, MAEW
Differential Diagnosis

• Describe the steps you took to arrive at this conclusion.

• Significance
Scenario

• Your patient is a 68 yo female complaining of severe SOB. She denies any pain or other complaints. She appears to be in acute respiratory distress. Skin- cool, clammy.
History

- SOB came on quite suddenly about 10 minutes ago while she was watching television.
- She smokes 1 pack/day since she was 20 yo.
- Denies C.P., nausea, vomiting.
- Meds- arthritis pain killers  NKDA
- PMH- arthritis, the weather has been severe and she has been unable to get around and do much due to her arthritis so she sure is glad she has cable. She even sleeps on the couch! Her daughter brings meals and empties the bed pan. By the way could you empty it while you are here?
Vital Signs

- HR- 140 regular ST
- BP- 100/70
- RR- 32 labored
- SpO2- will not pick up
Physical Exam

- HEENT- normocephalic, atraumatic
- No JVD, No TD, PERL
- CHEST- stable, = rise, moderate resp effort with some accessory m. use, speaks in short sentences
- HEART- RRR   LUNGS- CTA bilaterally
- ABD- SNT, -N, -V
- EXTREMITIES- PMS =/present x 4, no pedal edema, no clubbing, peripheral cyanosis present, MAE
Differential Diagnosis

• WHY?
• SIGNIFICANCE
Scenario

• A 30 yo male is c/o chest pain and wants you to take him to his doctor. He has a fever and chills feels weak all over. Skin- warm, dry.
History

- Patient has had some chest pain for a day or two
- Substrenal with no radiation and it hurts like @#!$ you moron or why would he be calling?! He can’t even like down to sleep now and he’s exhausted.
- He awoke this afternoon sitting up in his chair with a fever and chills
- Meds- None Allergies- NKDA
- PMH- admits to some IV drug abuse occasionally
Vital Signs

- HR: 110 regular, ST
- BP: 120/80
- RR: 18, sl. Shallow
- SpO2: 96%
Physical Exam

- HEENT - normocephalic, atraumatic
- No JVD, NO TD, PERL
- CHEST - rise with sl shallow resp., pain on deep inspiration
- HEART - RRR  LUNGS - CTA bilat
- ABD - SNT
- EXTREMITIES - PMS =/present x4, No pedal edema, MAEW
Differential Diagnosis

- Why?
- What might you expect to see on an ECG?
- What might you hear while auscultating the lungs/heart?
- Significance
Scenario

• You arrive at the home of a 42 yo female c/o substernal chest pain. She appears anxious and apprehensive. Skin- pale, cool, diaphoretic.
History

• She has been having substernal chest pain for about an hour. It started while she was balancing her checkbook.

• She states it is not really pain but feels as if an elephant is sitting on her chest and she can’t quite catch her breath. 8/10

• The discomfort radiates to her left shoulder and she has been nauseated

• Meds- None  Allergies- Codeine

• PMH- None, never had anything like this happen before
Vital Signs

- HR- 100 sl. Irregular
- BP- 130/92
- RR- 18 unlabored
- SpO2- 99%
- While taking her VS she apologizes profusely as she vomits on your shoes
Physical Exam

- **HEENT**: normocephalic, atraumatic, No JVD
- **CHEST**: =/ good excursion, normal resp effort  
  **HEART**: sl. irregular, clear
- **LUNGS**: CTA bilat
- **ABD**: SNT, +N, +V x1
- **EXTREMITIES**: PMS =/present x 4, No pedal edema, MAE
Differential Diagnosis

• WHY?
• SIGNIFICANCE