“Take On Me”:

A Cardiology Primer

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SBHPP 2024

agenda

Cardiac Anatomy

Cardiac Physiology

Cardiac Pharmacology

What is “Normal”

What is “Abnormal”

Later today…

“Beat It”: Understanding Arrythmias

This session will go over the appearance of common arrythmias as well as concerning arrythmias, identify key aspects to look for to distinguish between arrythmias, and discuss intervention strategies to consider.

“Under Pressure”: How to Manage Hypertension

This session will define the stages of hypertension, review guideline directed medical therapy recommendations for hypertension, and discuss monitoring recommendations such as labs to be obtained as medications are titrated.

“Every Little Thing She Does is Magic”: Cardiac Murmurs Demystified

This session will guide the attendee through the identification of murmurs through auscultation, how to determine murmur severity, and when interventions should be considered.

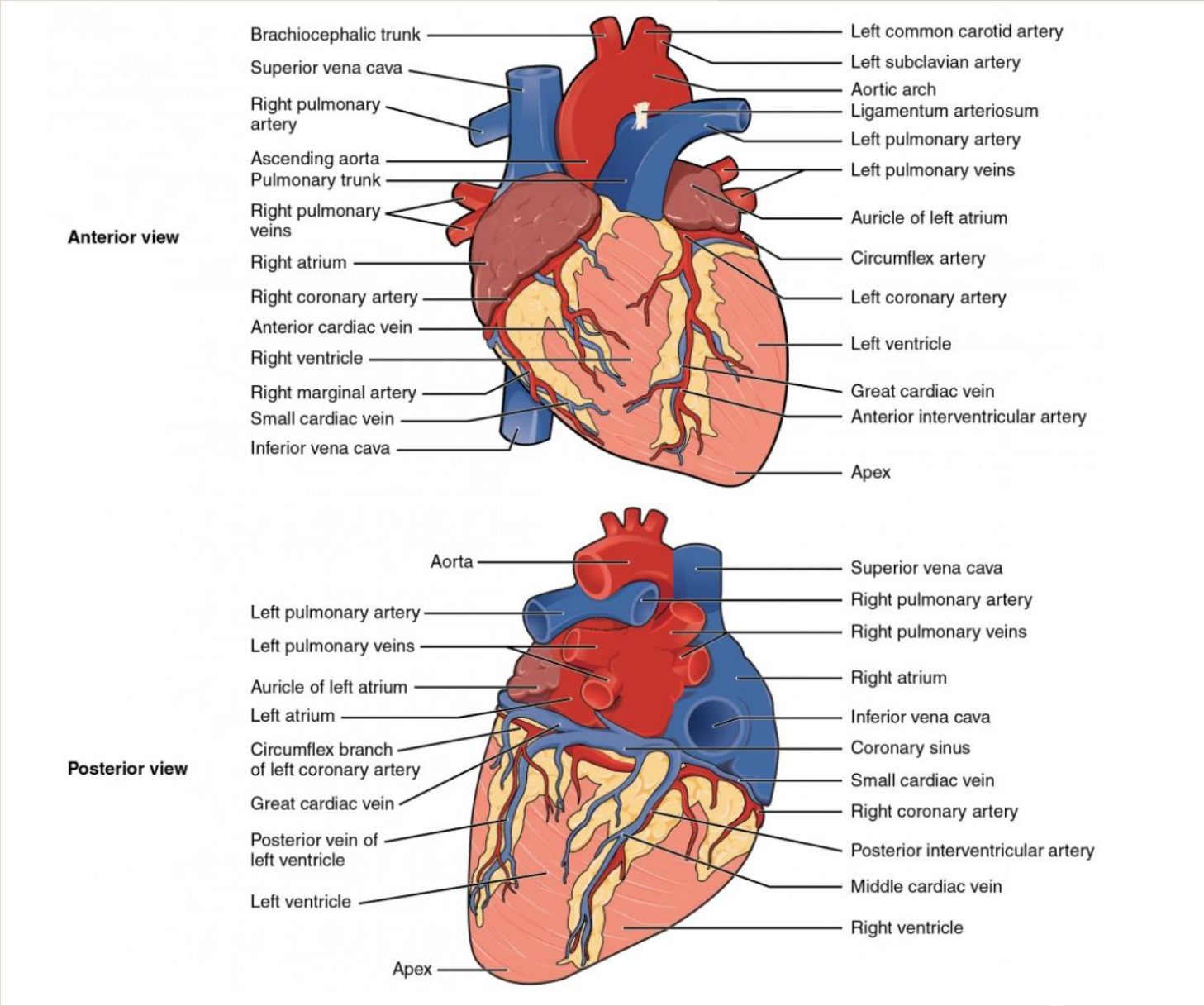
“Billie Jean is not my Cardiologist”: How to manage the heart failure patient

This session will delve into the different types of cardiomyopathies, different stages of heart failure, interventions to consider,

kɑːrdiˈɑːlədʒi– [car.di.ol.ogy]

- “the study and treatment of disorders of the heart and the blood vessels”
- “a medical specialty and a branch of internal medicine concerned with disorders of the heart”
- “the study of the heart”
- *Note: the Greek word “cardia,” means heart and “logy” means “study of.”*

Anterior/Posterior Views



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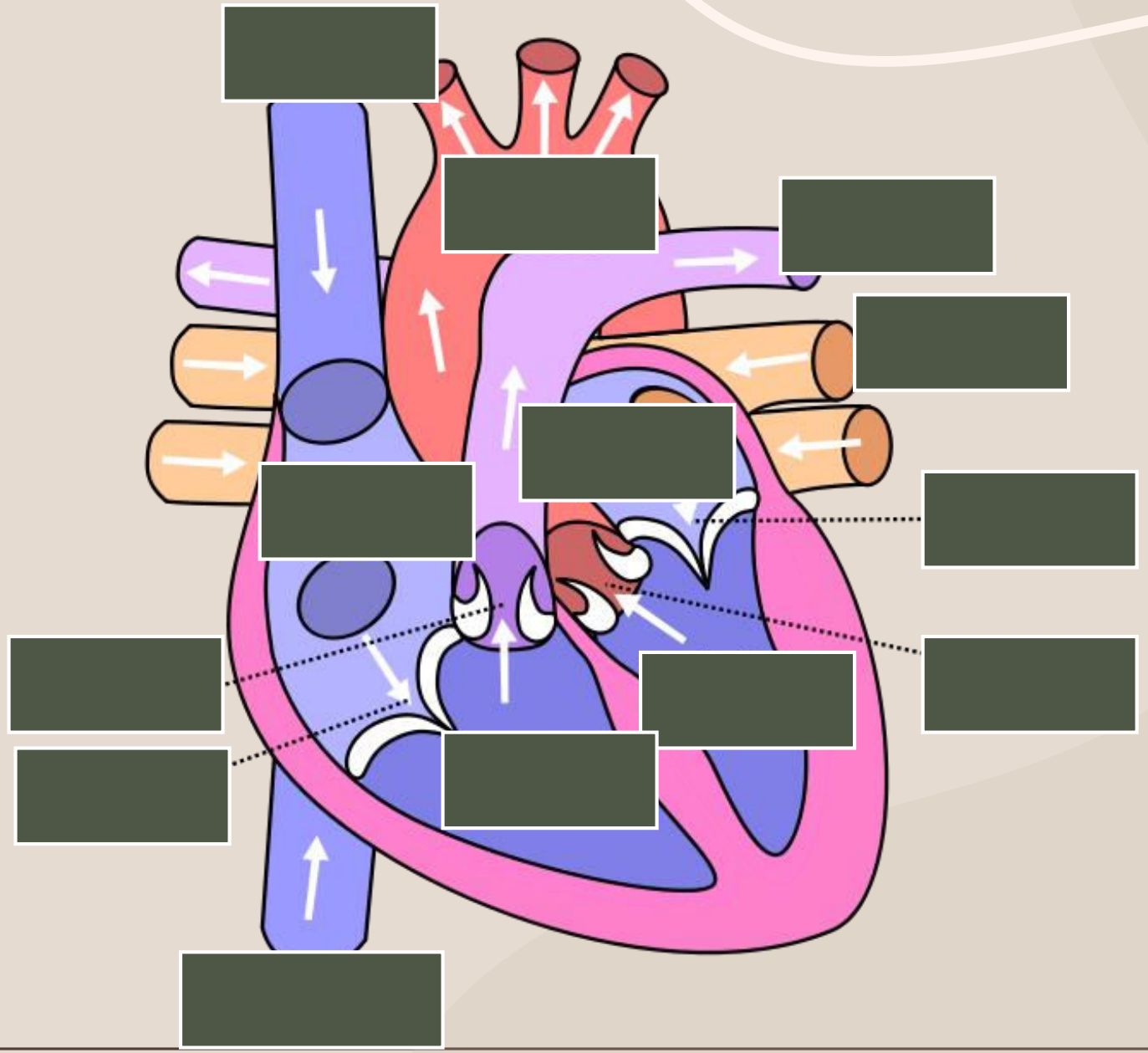
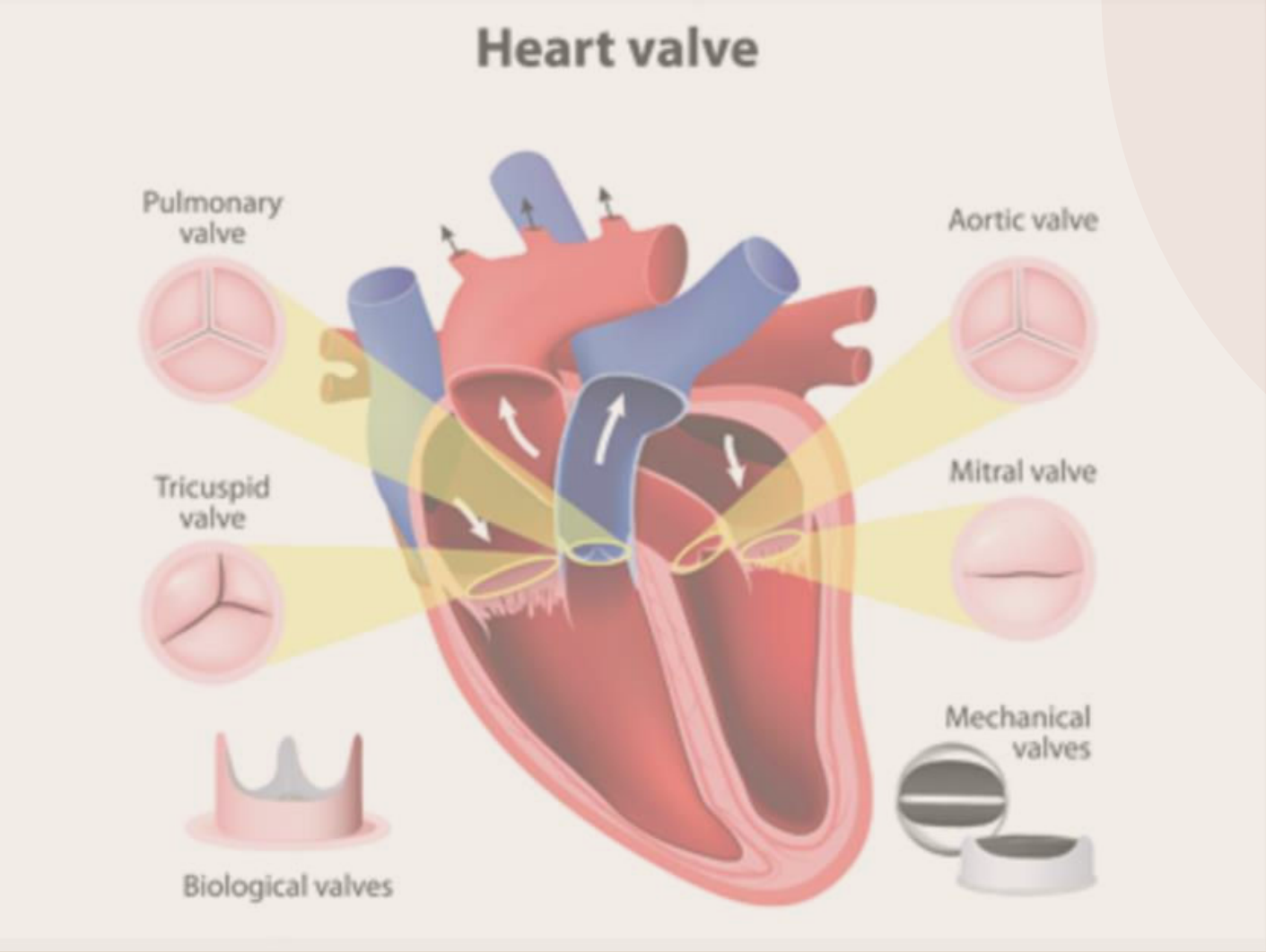


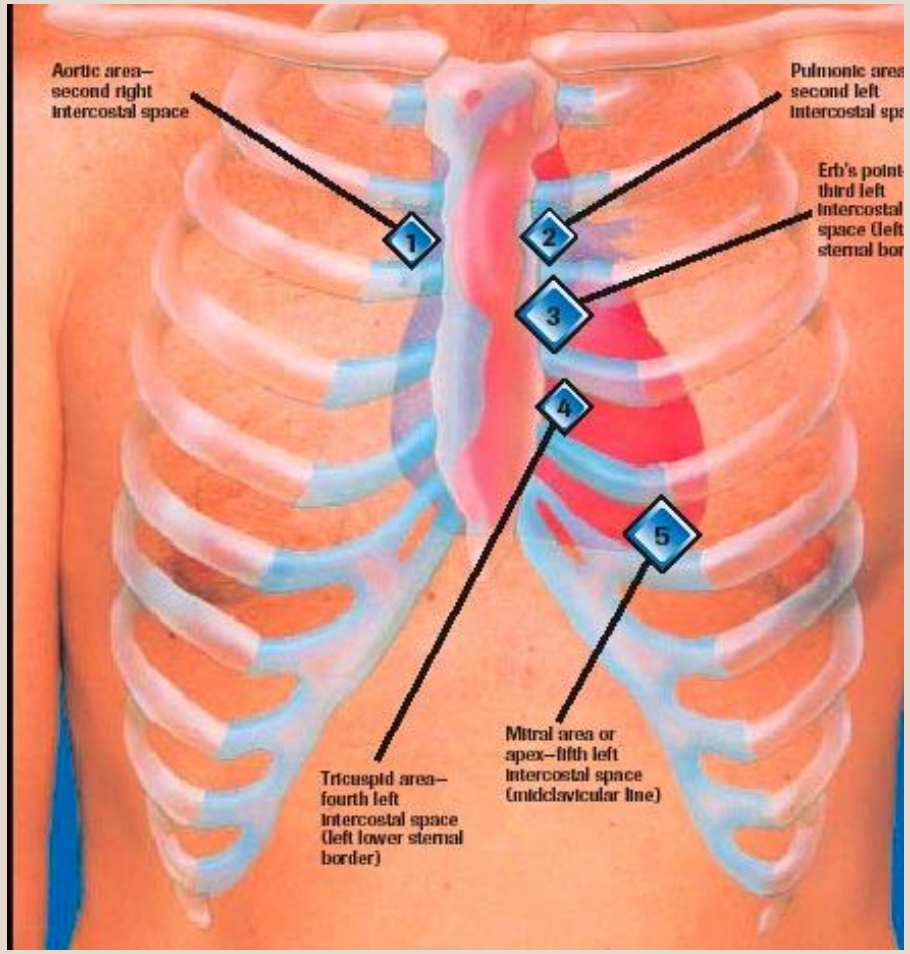
Image used here and throughout slides:
http://commons.wikimedia.org/wiki/File:Diagram_of_the_human_heart_(cropped).svg

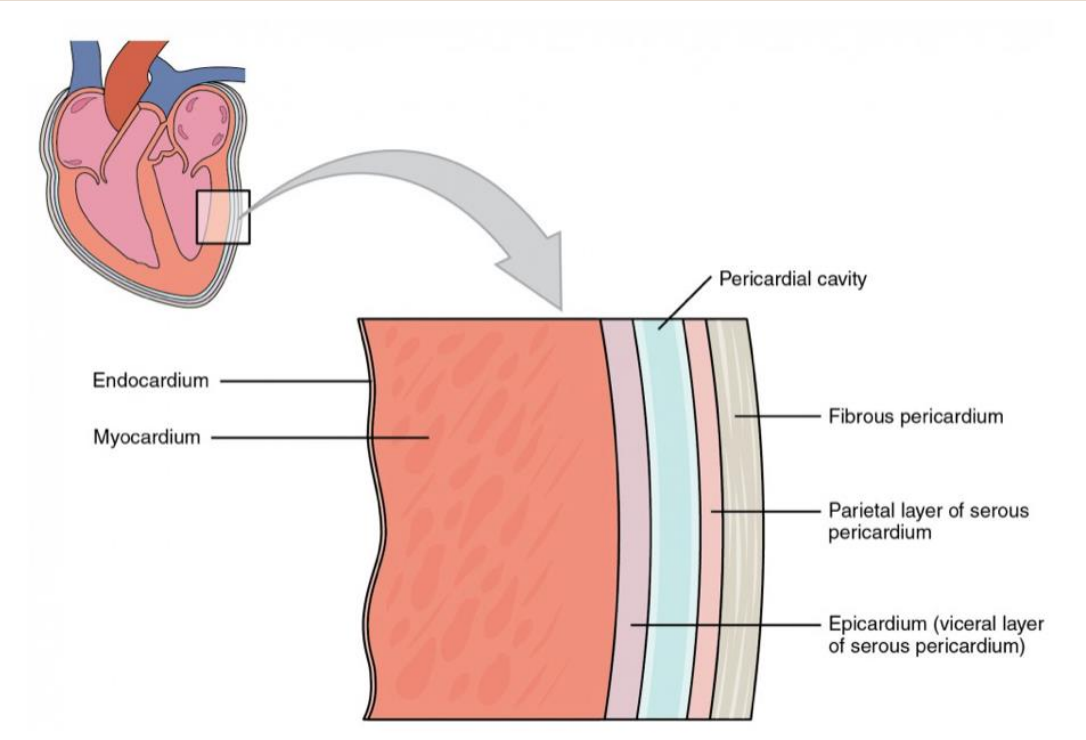
Heart Valves

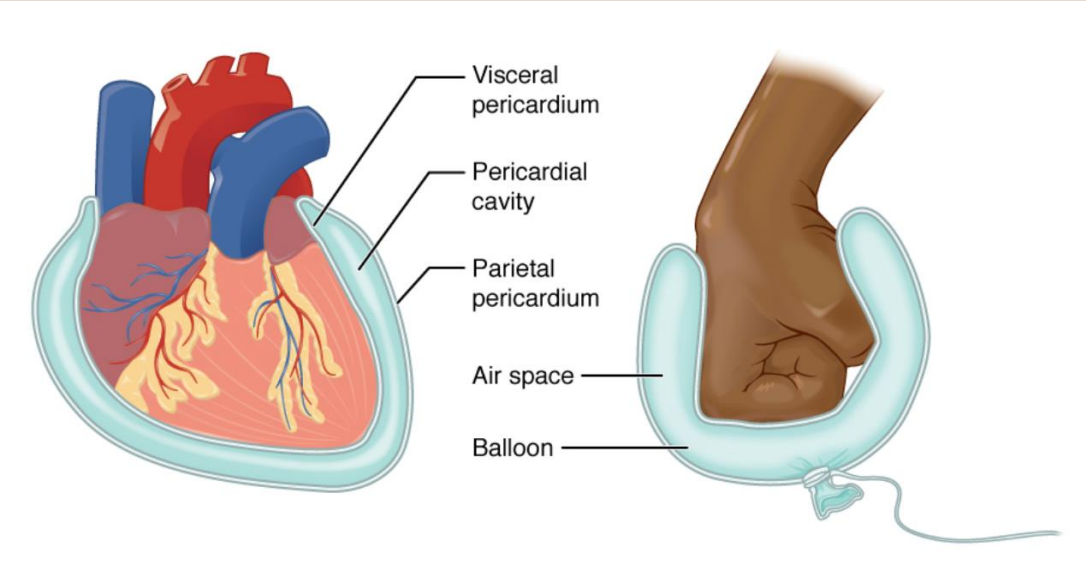


Auscultation

https://www.youtube.com/watch?time_continue=198&v=dBwr2GZCmQM&feature=emb_logo





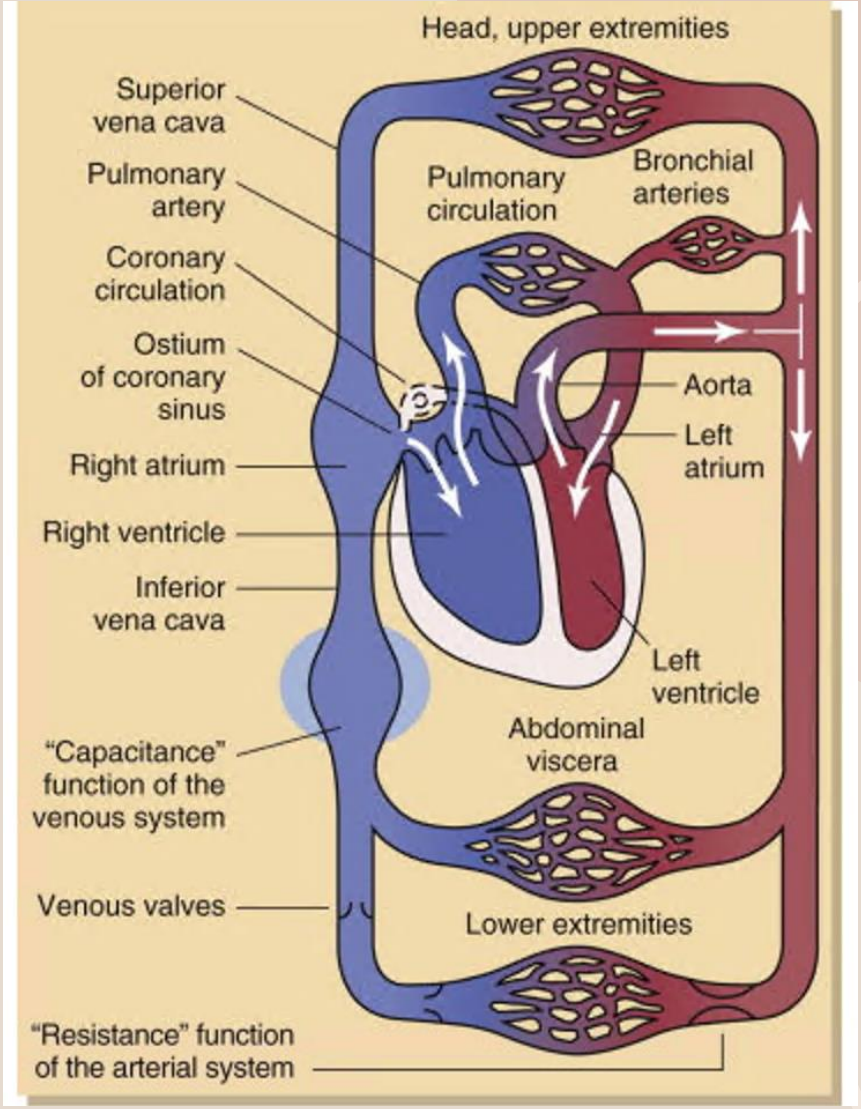


Heart Membranes

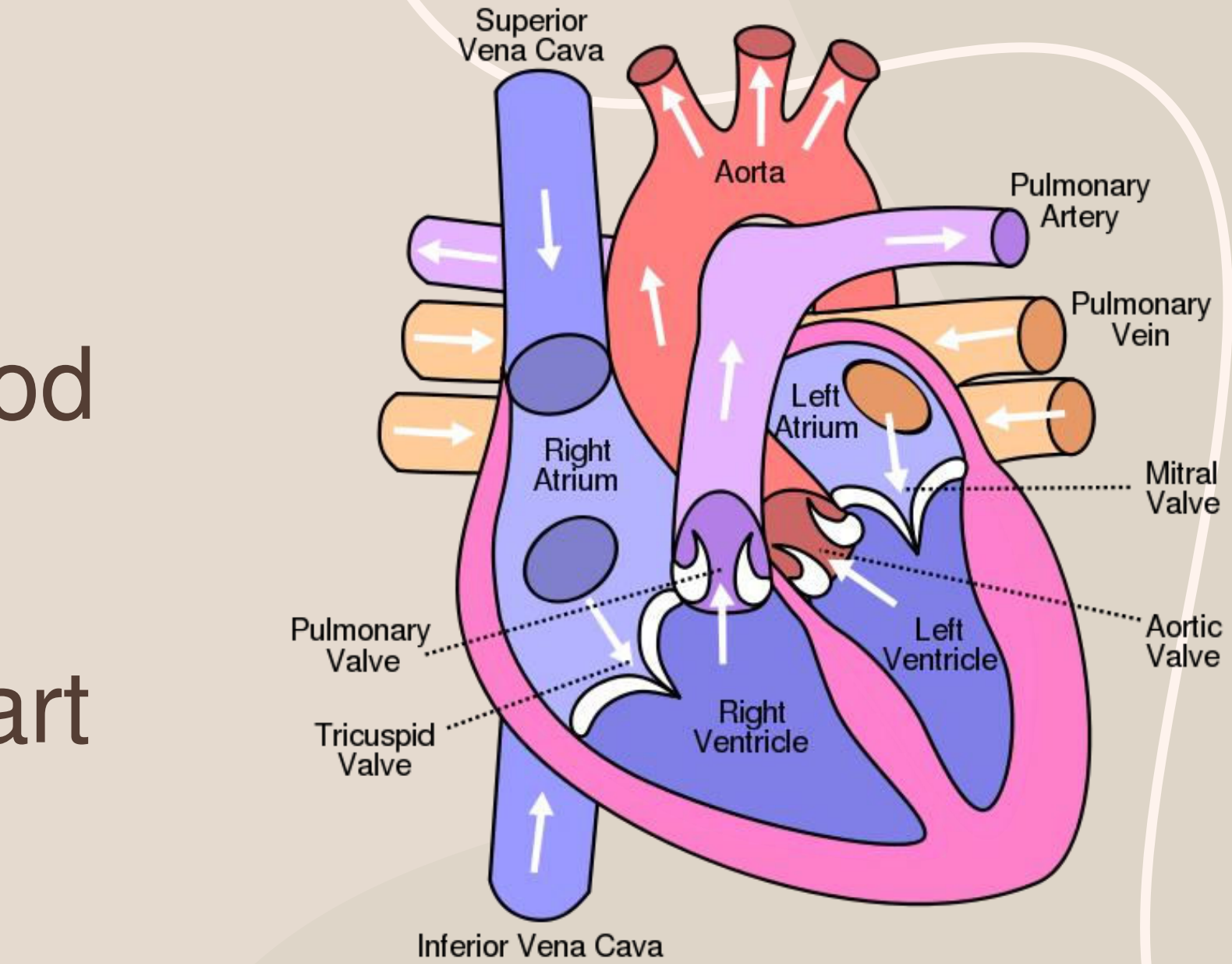
- Pericardium: thin, double-layered membrane surrounding the heart
  - Inner layer = (visceral pericardium)
    - adheres to the outer surface of the heart
  - Outer layer = (parietal pericardium)
    - Attaches to the sternum, vertebral column, and diaphragm to stabilize the heart in the chest
- Epicardium: outer surface of the heart
- Pericardial space: filled with a small amount of fluid (<50 mL)
  - lubricates contact surfaces, limits direct tissue-surface contact during myocardial contraction.
  - Note: too much fluid in this space (pericardial effusion) can cause impaired ventricular filling and abnormal septal movement

Circulatory System

- 4 chambers
- Atria: low-pressure chambers, store blood during ventricular contraction (systole)
- Atria fill ventricles with blood during ventricular relaxation (diastole)
- Ventricles pump blood through lungs (right ventricle) and to the peripheral tissues (left ventricle).
- LV = thicker than RV (to generate the higher systemic pressures for perfusion)

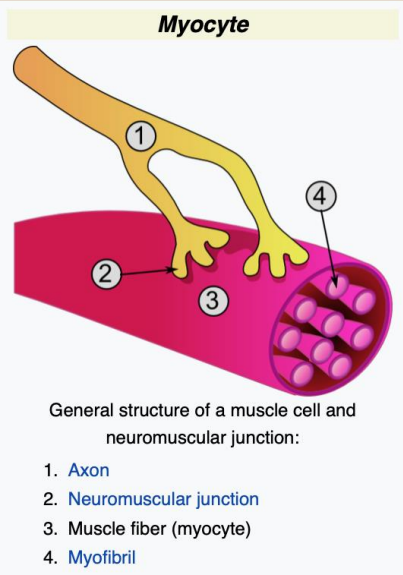


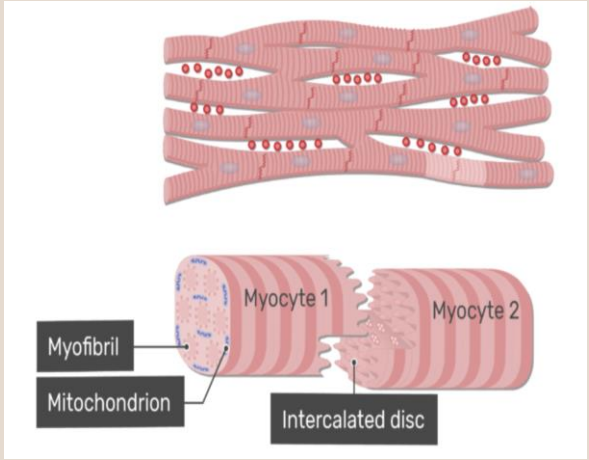
Review: how blood flows through the heart

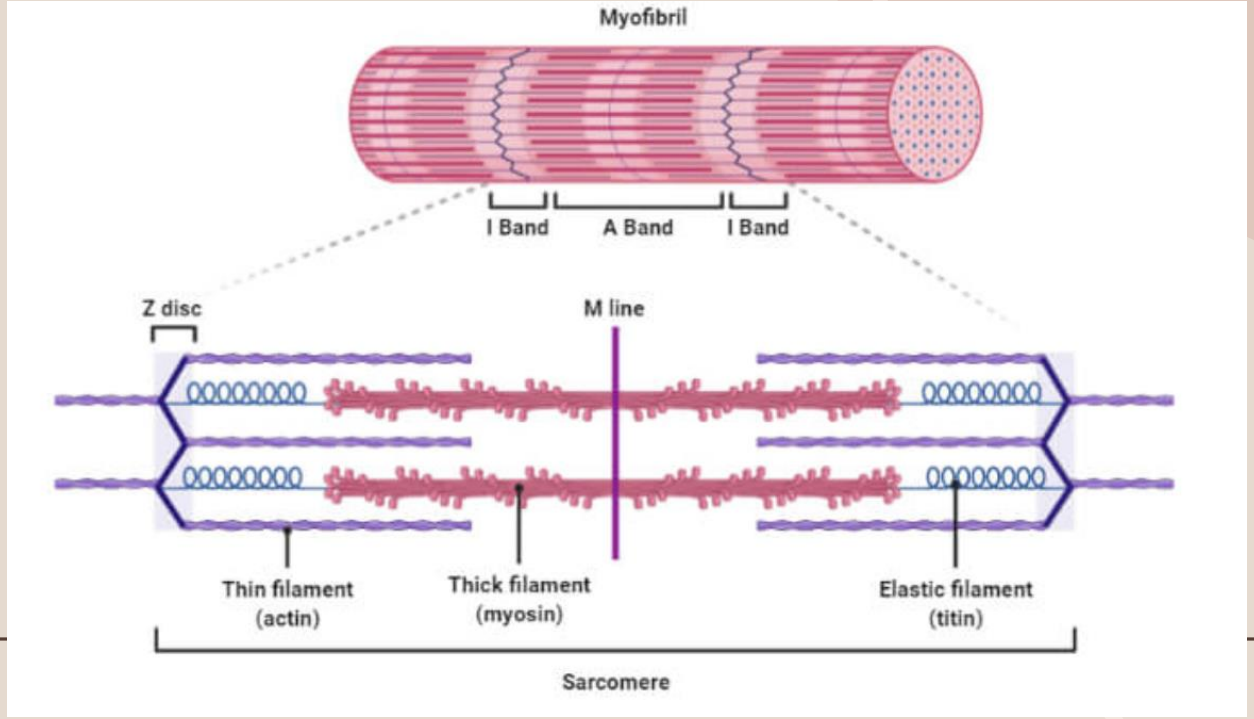


Myocardium and more…

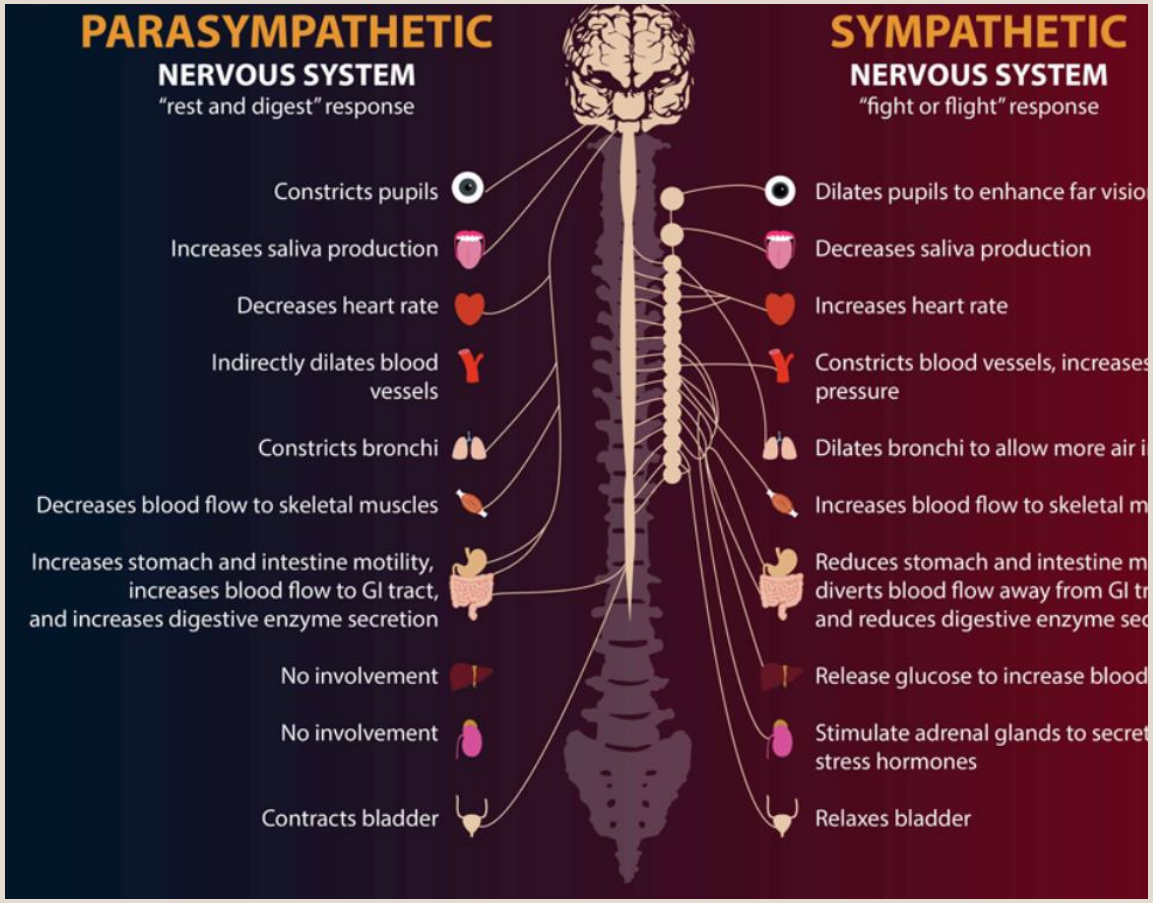
- Atrial and ventricular myocytes
  - specialized, branching muscle cells
  - connected end to end by intercalated disks
  - (disks cause tension between cells)
- Myocytes have more mitochondria to make:
  - adenosine triphosphate (ATP)
- sarcomeres (made of **<u>actin</u>** and **<u>myosin</u>**)
- When an electrical impulse is cardiac my they mecha contract





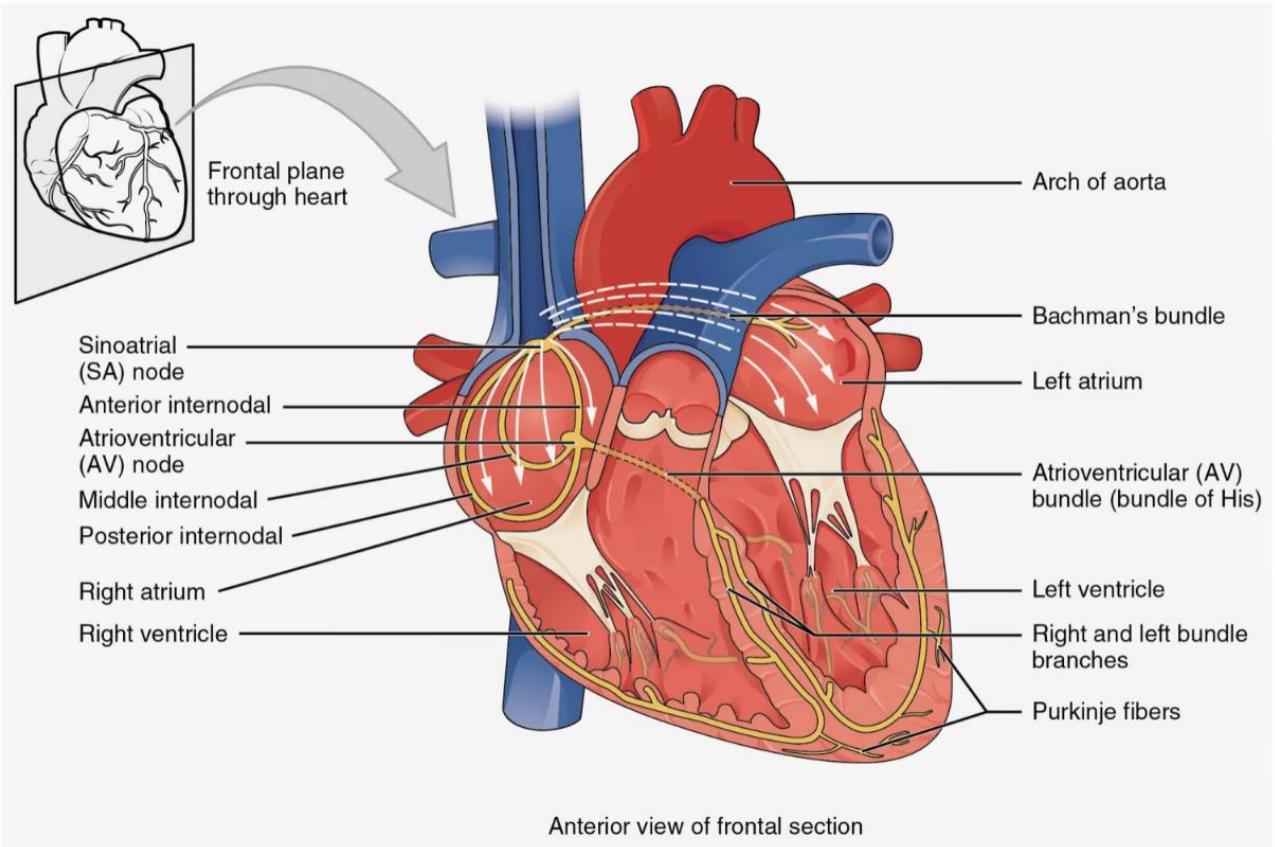


Neural Innervation



- **<u>Autonomic</u> nervous system**:
  - control system that acts mostly unconsciously
  - regulates cardiac function (and other bodily function: digestion, RR, etc.)
  - two main divisions: sympathetic and parasympathetic
  - **<u>Sympathetic</u>** system:
    - increases HR (chronotropy)
    - increases force of myocardial contraction (inotropy)
    - signals go through nerves that reach the SA node, AV node, epicardial vessels, and myocardium
  - **<u>Parasympathetic</u>** system:
    - decreases HR
    - decreases contractility
    - signals go through nerves that reach the SA node, AV node, epicardial vessels, and myocardium

Signals to Squeeze

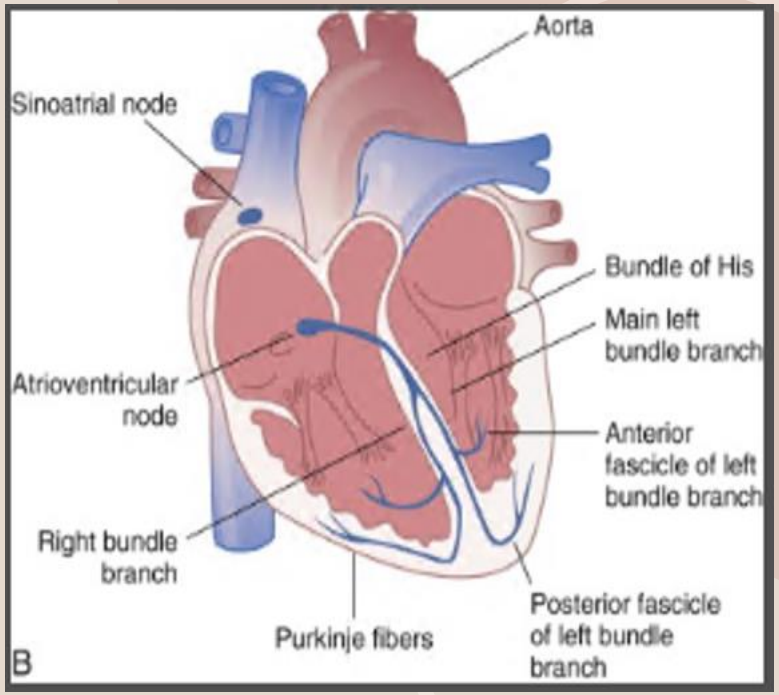


- SA regulated by the **autonomic nerves** of peripheral nervous system
  - Parasympathetic and sympathetic autonomic nerves send signals to the SA node
    - Speed up (sympathetic) HR
    - Slow down (parasympathetic) HR
- SA to AV impulses have 1/10 sec delay (during which atria contract, dump blood into ventricles)
- AV sends impulses to ventricles via bundle of His through left and right bundle branches to Purkinje fibers

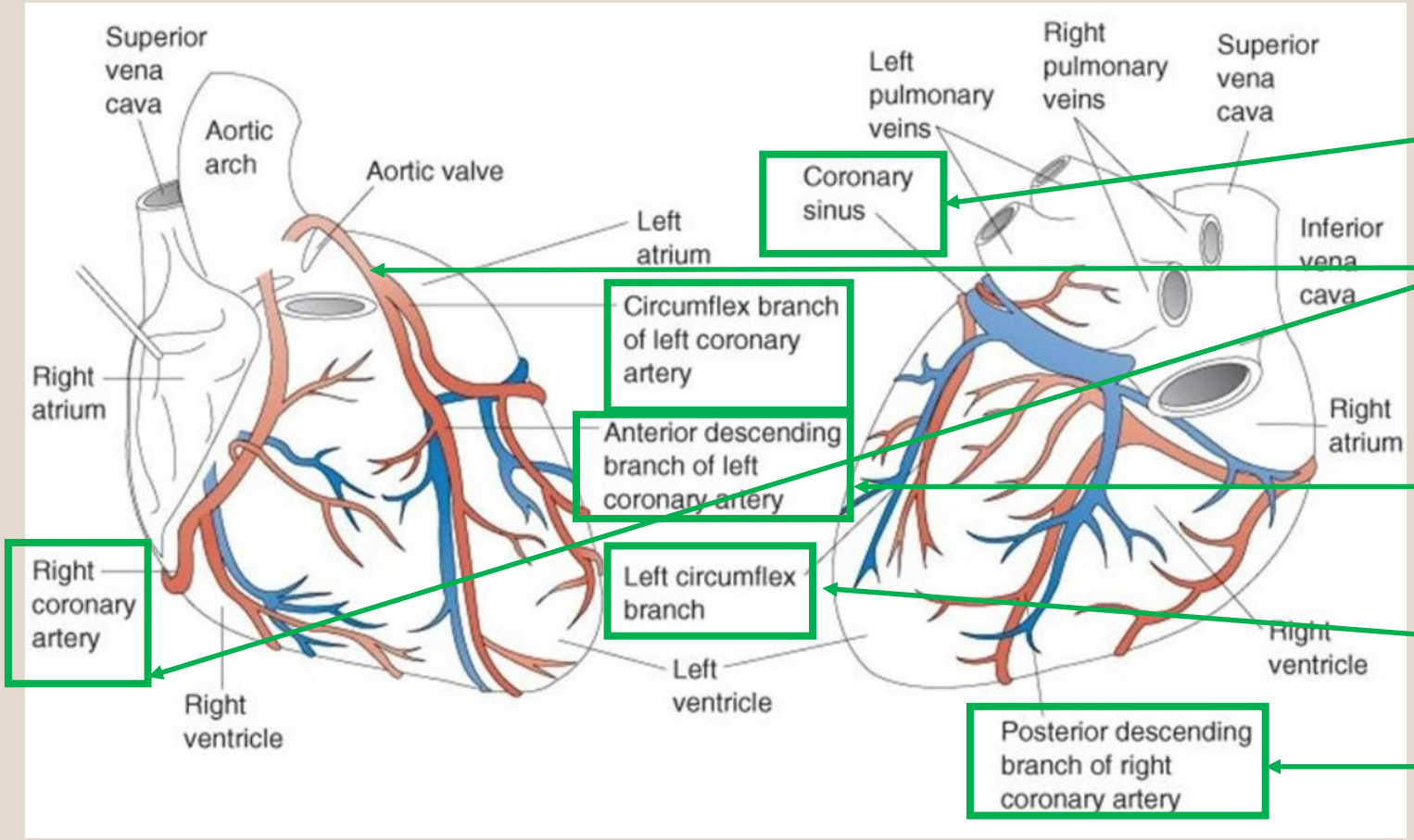
Image source: https://www.thoughtco.com/heart-nodes-anatomy-373242

Conduction System

- Sinoatrial (SA) node:
  - Specialized pacemaker cells, in the RA between the SVC and the RAA
  - Gets blood from a branch of the RCA (60% of the population) or a branch of the LCx (40%)
  - Electrical impulse from SA goes to AV node via tracts within the atria
- Atrioventricular (AV) node:
  - Located at inferior aspect of RA, between the coronary sinus and the TV
  - Gets blood from a branch of the RCA (90% of the population) or a branch of the LCx (10%)
- Electrical impulse goes from AV node through His-Purkinje system to ventricles
  - Divides into the LBB and RBB and ends at the Purkinje cells
- Purkinje cells:
  - ~~Stimulate myocytes to contract or squeeze~~
  - RBB and LBB get blood from branches from the LAD



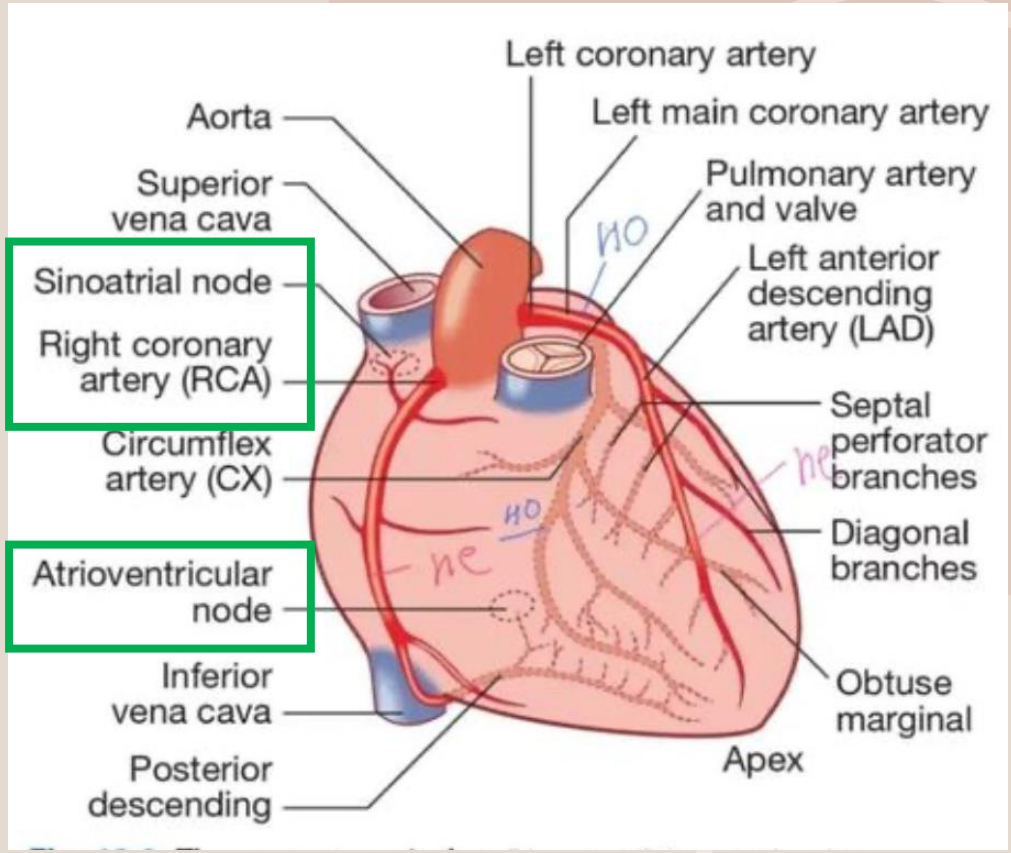
How does the heart get blood?



- Venous drainage from the heart enters the right atrium through the coronary sinus.
- The heart receives its blood supply through the LCA and RCA (originate at the *sinuses of Valsalva)*
- Left main coronary artery divides into the LAD and LCx
  - LAD: perfuses anterior and anterolateral LV and to the anterior interventricular septum
  - LCx: perfuses the lateral aspect of the LV and the LA
- RCA: perfuses RA and RV

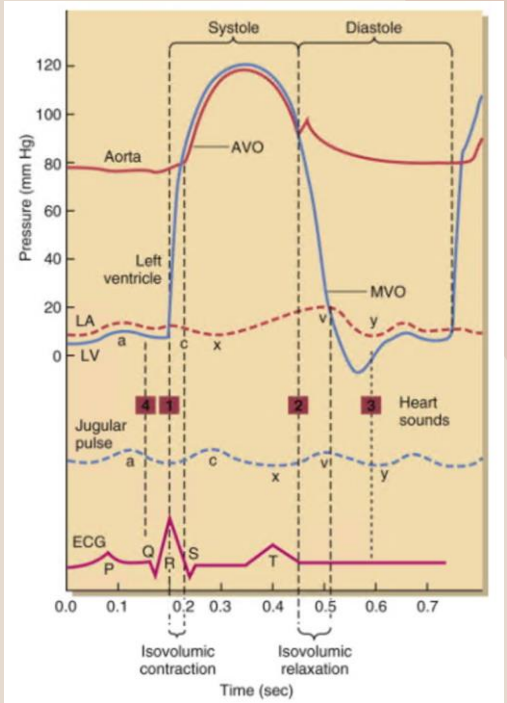
What about the coronary arteries?

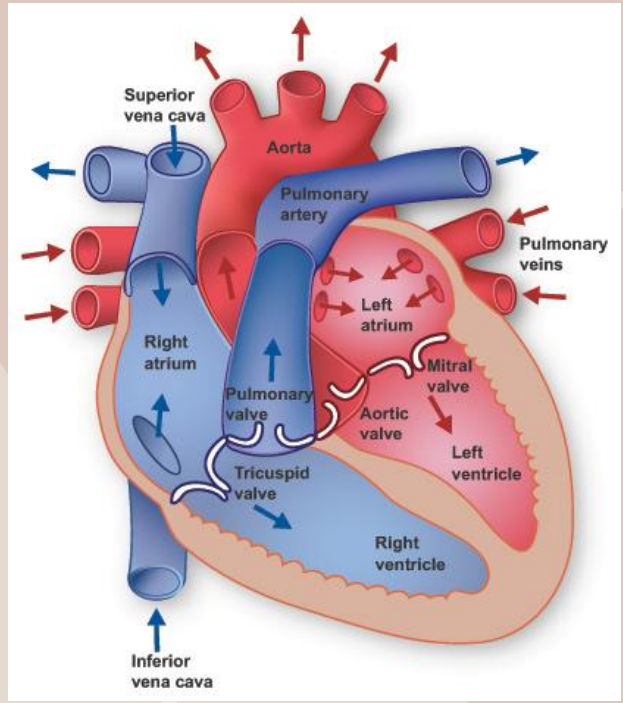
- Blood flow in the coronary circulation is reduced during systole (because the blood vessels are compressed by the contracting myocardium) so **most coronary flow is during diastole**.
- **Diastolic pressure = major pressure driving flow for coronary circulation**
- Note: tachycardia is bad because coronary flow is reduced when the diastolic filling time is abbreviated, and the MvO_2 (amount of oxygen used by the heart) increases with increasing HR
- When MvO_2 changes, coronary arteries dilate or constrict, changing vascular resistance and flow
- *(note: figure is anterior view of heart)*



Circulatory Physiology and the Cardiac Cycle

- Cardiac cycle: pressure changes in each cardiac chamber over time
  - systole (ventricular contraction) and diastole (ventricular relaxation)
- Cardiac valves open and close due to pressure gradients during systole and diastole
- Beginning of systole: ventricular pressure > atrial pressure, so TV/MV passively closes
  - As myocytes contract, intraventricular pressures initially rise (but no change yet to ventricular volume UNTIL intraventricular pressures > pressures in the aorta and PA
  - Now AV/PV can open, and ventricular ejection of blood occurs
- When intracellular calcium levels fall, ventricular relaxation begins
  - Now arterial pressures exceed intraventricular pressures, AV/PV close
  - No change to ventricular volume at start of ventricular relaxation UNTIL intraventricular pressures<atrial pressures at which point MV/TV valves open
  - Once MV/TV valves open, rapid ventricular filling occurs during diastole (blood in the atria dumps into the ventricle)

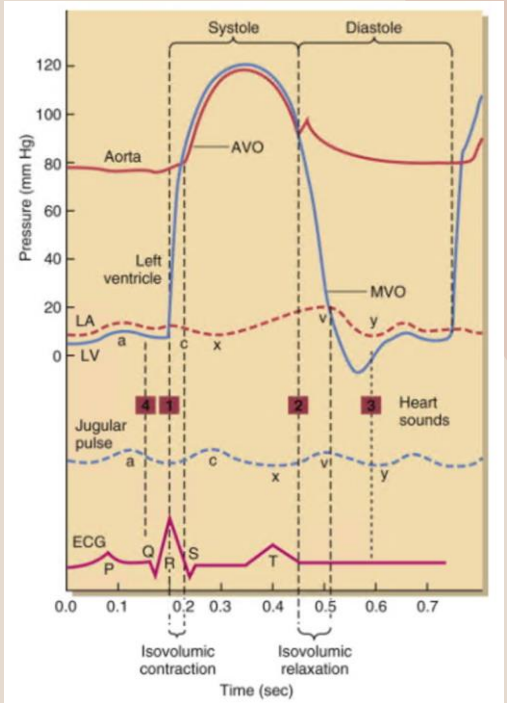


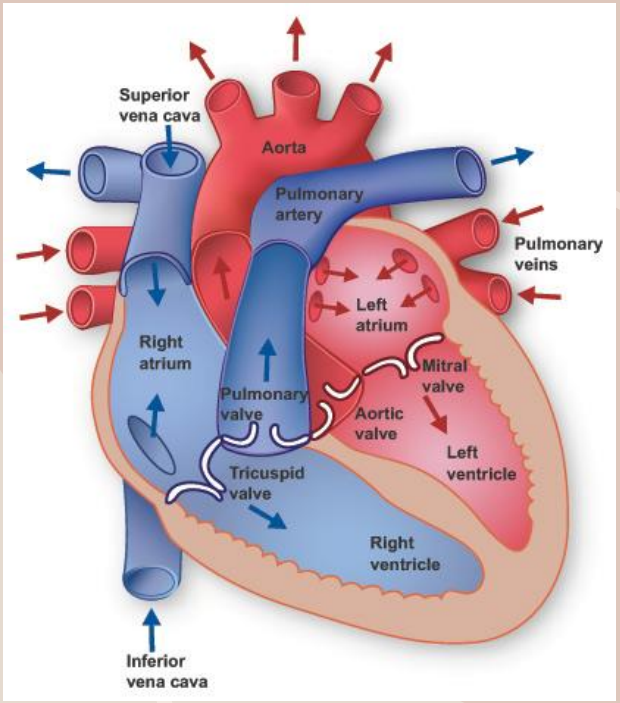


Heart Sound	Occurs during:	Associated with:
S1	Isovolumetric contraction	Closure of mitral and tricuspid valves
S2	Isovolumetric relaxation	Closure of aortic and pulmonic valves
S3	Early ventricular filling	Normal in children; in adults, associated with ventricular dilation (e.g. ventricular systolic failure)
S4	Atrial contraction	Associated with stiff, low compliant ventricle (e.g., ventricular hypertrophy; ischemic ventricle)

Circulatory Physiology and the Cardiac Cycle

- ECG events initiate and precede the mechanical (pressure) events, mechanical/pressure events precede the auscultatory events (heart sounds) (red boxes)
  - After the P wave, the atria contract to produce the a wave
  - QRS complex initiates ventricular systole
    - LV contraction and buildup of LV pressure
    - When LV pressure > LA pressure, MV closes and we hear the first heart sound
    - After isovolumic contraction and when LV pressure > aortic pressure, the aortic valve opens
  - When ventricular pressure is < aortic pressure, the aortic valve closes and we hear the second heart sound
  - LV pressure continues to fall during relaxation < LA pressure and the mitral valve opens





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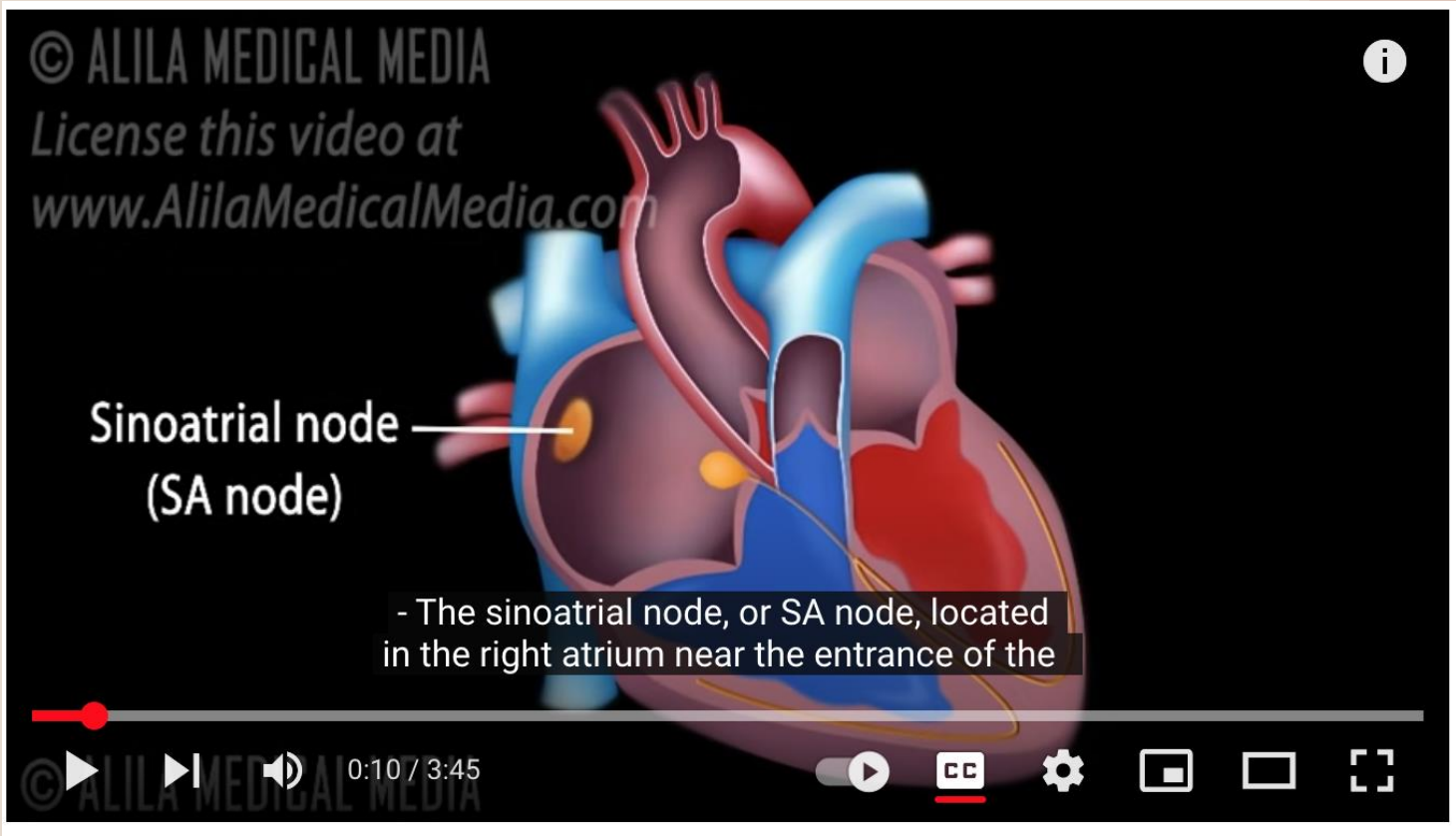
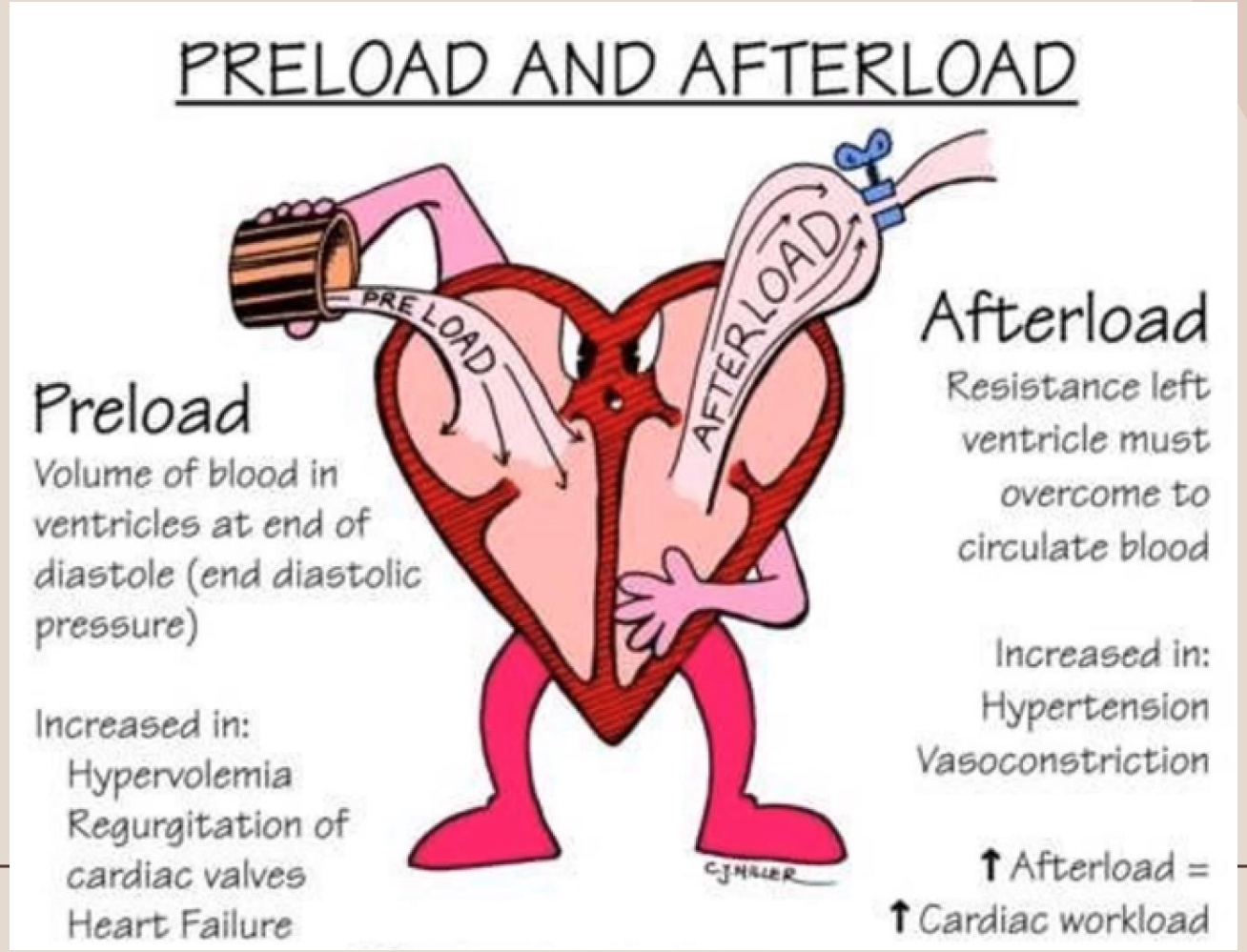
Putting it all together…

- https://www.youtube.com/watch?v=RYZ4daFwMa8

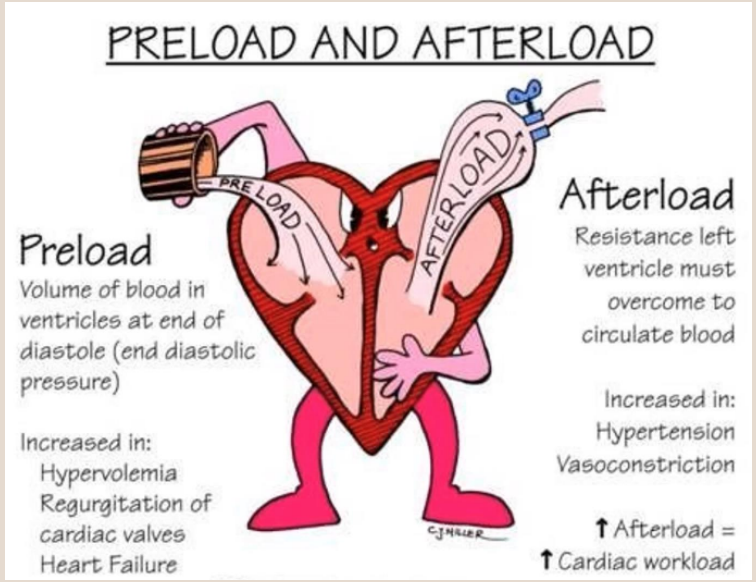


Cardiac Performance

- Cardiac output (CO) = blood ejected by the heart each minute
  - **CO=SV×HR (STARLINGS's LAW)**
- Normal CO is 4-6 L/min (can increase 4-6x during exercise!!)
- Cardiac index (CI) normalizes CO to body size (CI=CO/BSA), measured in L/min/m ^{2}



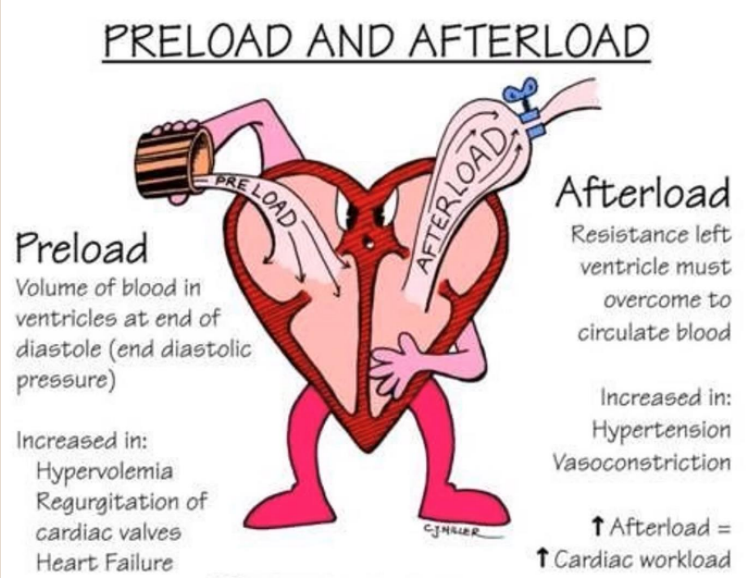
Components of Stroke Volume



1. **Preload** (volume of blood in ventricle at end of diastole; reflects venous return)
   - Ex: fluid infusion increases preload, diuretics or dilators (nitroglygerin) decrease preload
2. **Afterload** (force against which ventricles must contract to eject blood)
   - Main determinants:
     - Arterial pressure (as pressure increases, amount of blood that can be ejected decreases), dimensions of the ventricle. As the arterial blood pressure increases, the amount of blood that can be ejected into the aorta decreases.
     - Left ventricular width (ventricular wall hypertrophy is a compensatory mechanism to reduce afterload)
   - Ex: Angiotensin-converting enzyme (ACE) inhibitors and hydralazine drop BP by reducing afterload. Diuretics decrease left ventricular volume and size, reducing wall stress–associated afterload.
3. **Contractility** (inotropy) (force of ventricular contraction with constant preload and afterload)
   - Regulated through stimulation of:
     - Cathecholminergic (epinephrine, norepinephrine, and dopamine) receptors
     - Intracellular signaling cascades (phosphodiesterase inhibitors)
     - Intracellular calcium levels (affected by levosimendan and, indirectly, by digoxin (increases contraction by inhibiting Na-K ATPase thus increasing Ca).

- ~~Many antihypertensive medications (β blockers, calcium channel antagonists~~ (amlodipine, nicardipine, verapamil, diltiazem, etc.) interfere with adrenergic receptor activation or intracellular calcium levels, which can decrease the strength

Factors affecting cardiac performance

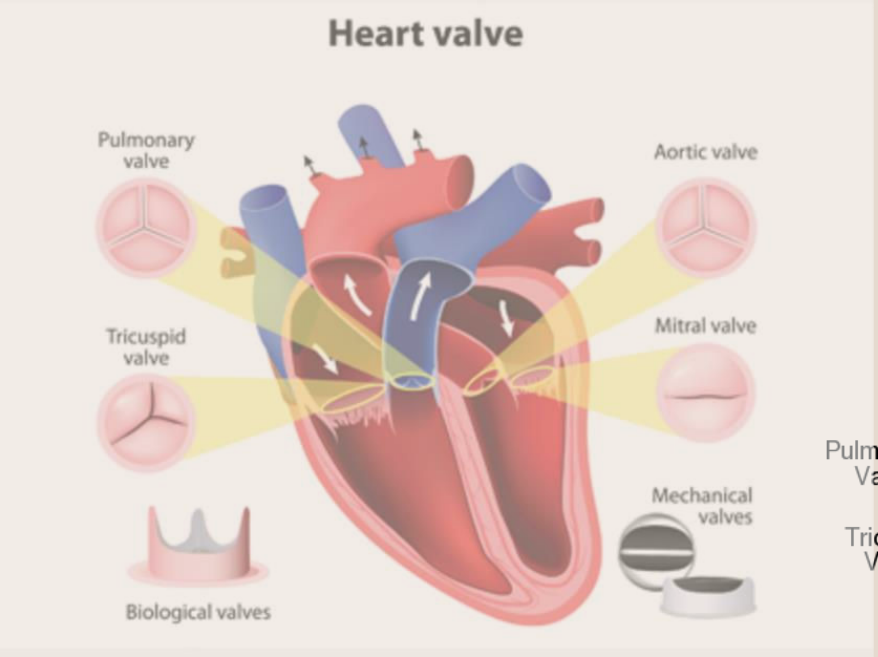


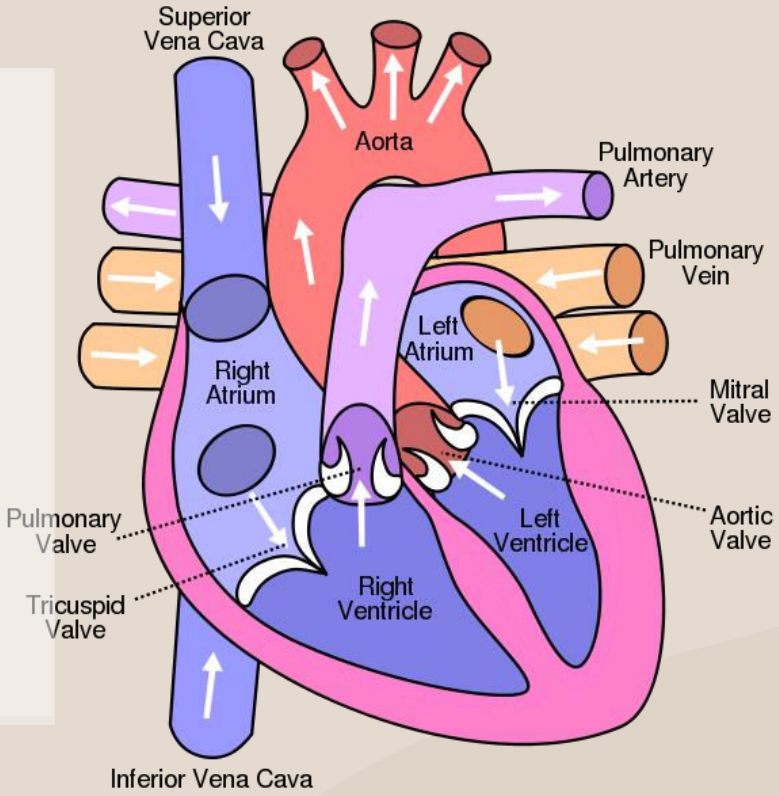
PRELOAD (LEFT VENTRICULAR DIASTOLIC VOLUME)
Total blood volume
Venous (sympathetic) tone
Body position
Intrathoracic and intrapericardial pressures
Atrial contraction
Pumping action of skeletal muscle
AFTERLOAD (IMPEDANCE AGAINST WHICH THE LEFT VENTRICLE MUST EJECT
Peripheral vascular resistance
Left ventricular volume (preload, wall tension)
Physical characteristics of the arterial tree (elasticity of vessels or presence of outflow obstruction)
CONTRACTILITY (CARDIAC PERFORMANCE INDEPENDENT OF PRELOAD OR AF
Sympathetic nerve impulses
Increased contractility
Circulating catecholamines
Digitalis, calcium, other inotropic agents
Increased heart rate or post-extrasystolic augmentation
Anoxia, acidosis
Decreased contractility
Pharmacologic depression
Loss of myocardium
Intrinsic depression
HEART RATE
Autonomic nervous system
Temperature, metabolic rate
Medications, drugs

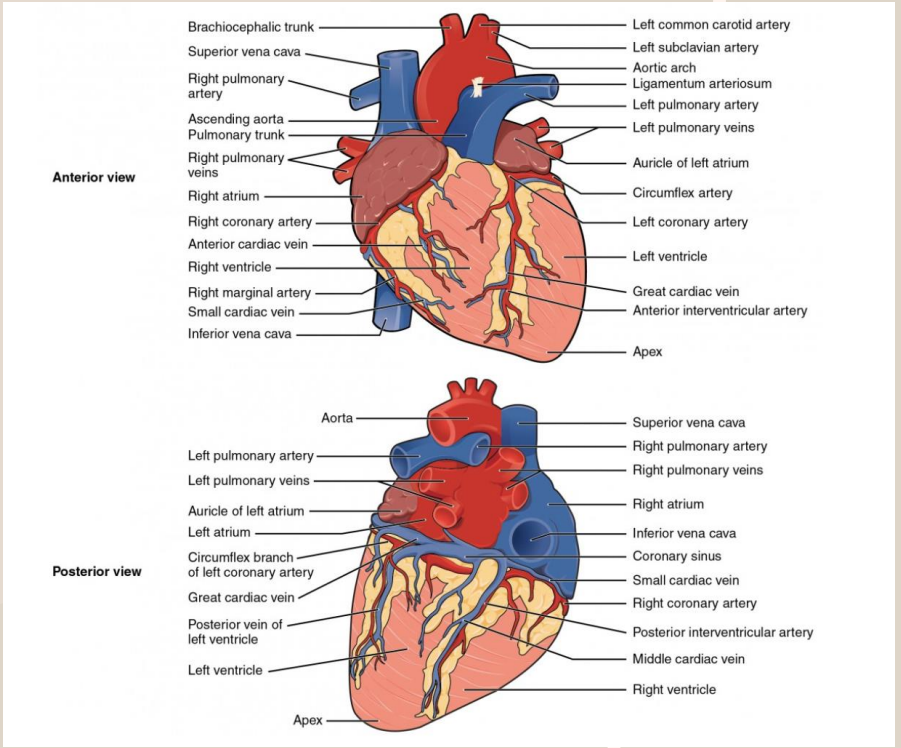
Physiology of the Systemic Circulation

- Poiseuille's law: describes relationship between pressure and flow in a vessel (flow through a tube is proportional to the pressure difference between the ends of the tube)
- The most important determinants of blood flow in the cardiovascular system are **arterial radius** and **systemic vascular resistance (SVP)**
  - Small changes in arterial radius can cause large changes in flow to a tissue or organ
  - SVR is the total resistance to flow caused by changes in the radius of resistance vessels (small arteries and arterioles) of the systemic circulation
- Autonomic nervous system alters systemic vascular tone through sympathetic and parasympathetic innervation as well as metabolic factors
- Neural regulation of BP occurs primarily by baroreceptors (stretch-sensitive nerve endings)
  - An increase in systemic BP increases the firing rate of the baroreceptors.

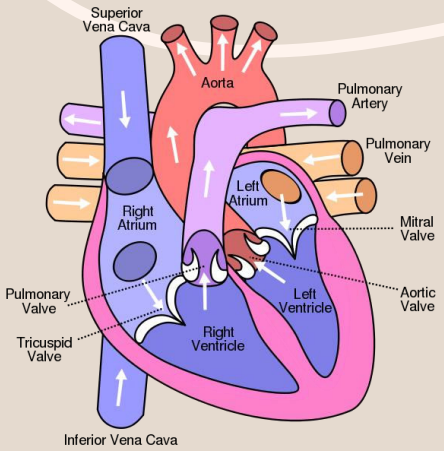
What is normal…

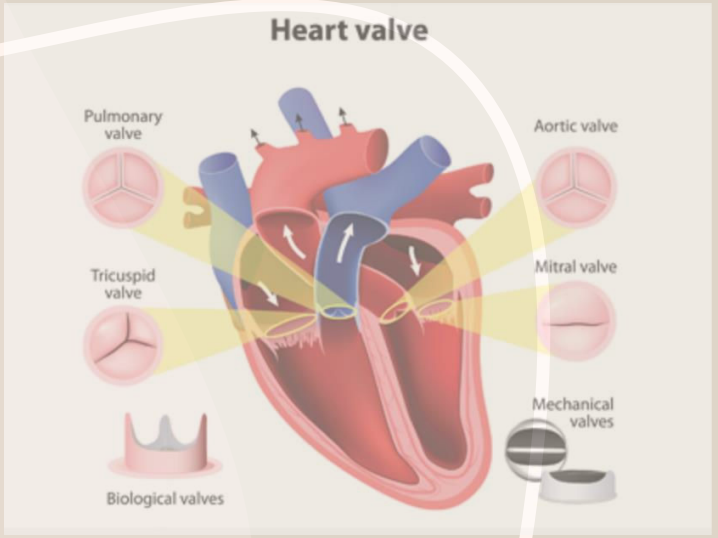




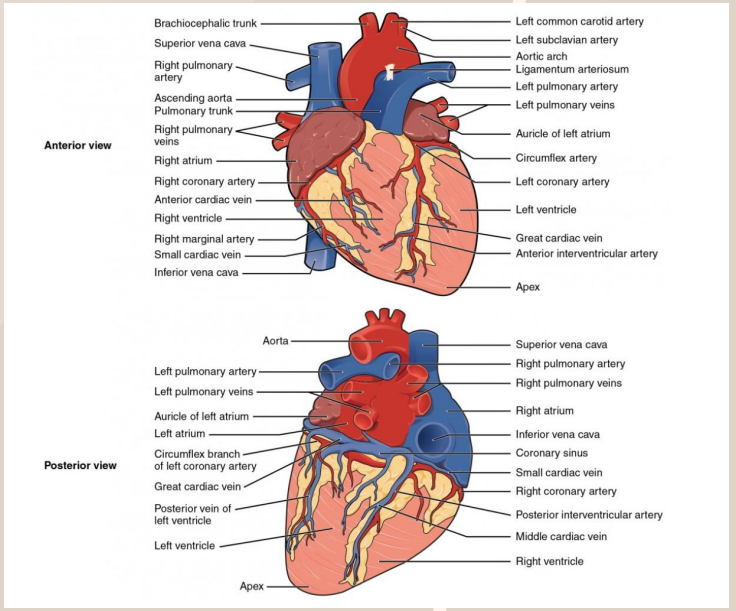


What is abnormal…

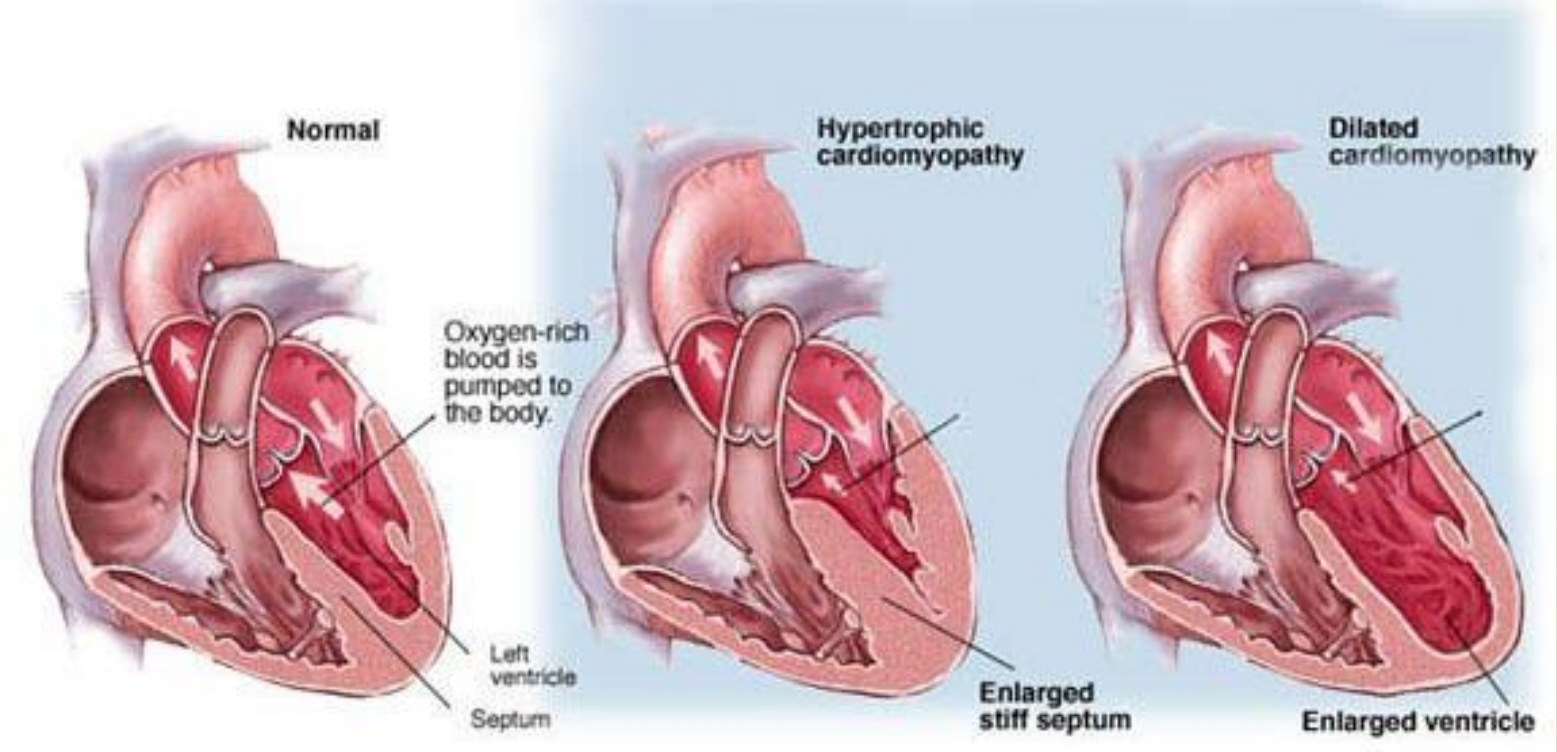




- Coronary artery disease (CAD)
  - Patients may present with angina, acute coronary syndrome (such as MI), or may be silent
- Congestive heart failure
  - HFrEF, HFpEF, cardiomyopathy (systolic dysfunction), diastolic dysfunction (d/t Htn, amyloid, others)
- Stroke
  - Cerebral hypoperfusion (carotid disease, emboli, bleeding)
- Peripheral arterial disease (PAD)
  - See often with CAD
- Valvular heart disease
  - We will discuss this later today
- Congenital heart disease



What is abnormal: Compensatory Mechanisms



- Myocardial hypertrophy or dilation (heart muscle remodeling/reconstruction)
  - LVH with AS
  - LV dilation with cardiomyopathy (usually final stages of HF)

What is abnormal…congenital considerations

- Ventricular septal defect
- Atrial septal defect
- Tetrology of Fallot
- Single ventricle
- Pulmonary valve stenosis
- Patent Ductus arteriosus
- Dextro-transposition of great arteries
- Aortic valve stenosis

Chest Pain: cardiovascular

TABLE

Table 3.1

Cardiovascular Causes of Chest Pain

Condition	Location	Quality	Duration	Aggravating or Alleviating Factors	Associated Symptoms or Signs
Angina	Retrosternal region: radiates to or occasionally isolated to neck, jaw, shoulders, arms (usually left), or epigastrium	Pressure, squeezing, tightness, heaviness, burning, indigestion	<2-10 min	Precipitated by exertion, cold weather, or emotional stress; relieved by rest or nitroglycerin; variant (Prinzmetal) angina may be unrelated to exertion, often early in the morning	Dyspnea; S_3, S_4, or murmur of papillary dysfunction during pain
Myocardial infarction	Same as angina	Same as angina, although more severe	Variable; usually >30 min	Unrelieved by rest or nitroglycerin	Dyspnea, nausea, vomiting, weakness, diaphoresis
Pericarditis	Left of the sternum; may radiate to neck or left shoulder, often more localized than pain of myocardial ischemia	Sharp, stabbing, knifelike	Lasts many hours to days; may wax and wane	Aggravated by deep breathing, rotating chest, or supine position; relieved by sitting up and leaning forward	Pericardial friction rub
Aortic dissection	Anterior chest; may radiate to back, interscapular region	Excruciating, tearing, knifelike	Sudden onset, unrelenting	Usually occurs in setting of hypertension or predisposition, such as Marfan syndrome	Murmur of aortic insufficiency; pulse or blood pressure asymmetry; neurologic deficit

View full size

Benjamin I. J. Griggs R. C. Wing E. J. & Fitz J. G. (2016). Andreoli and carpenter's cecil essentials of medicine (9th ed.). Elsevier/Saunders.

Chest Pain: non-cardiovascular

TABLE

Table 3.2

Noncardiac Causes of Chest Pain

Condition	Location	Quality	Duration	Aggravating or Alleviating Factors	Associated Symptoms or Signs
Pulmonary embolism (chest pain often not present)	Substernal or over region of pulmonary infarction	Pleuritic (with pulmonary infarction) or angina-like	Sudden onset (minutes to hours)	Aggravated by deep breathing	Dyspnea, tachypnea, tachycardia; hypotension, signs of acute right ventricular heart failure, and pulmonary hypertension with large emboli; pleural rub; hemoptysis with pulmonary infarction
Pulmonary hypertension	Substernal	Pressure; oppressive	—	Aggravated by effort	Pain usually associated with dyspnea; signs of pulmonary hypertension
Pneumonia with pleurisy	Located over involved area	Pleuritic	—	Aggravated by breathing	Dyspnea, cough, fever, bronchial breath sounds, rhonchi, egophony, dullness to percussion, occasional pleural rub
Spontaneous pneumothorax	Unilateral	Sharp, well localized	Sudden onset; lasts many hours	Aggravated by breathing	Dyspnea; hyperresonance and decreased breath and voice sounds over involved lung
Musculoskeletal disorders	Variable	Aching, well localized	Variable	Aggravated by movement; history of exertion or injury	Tender to palpation or with light pressure
Herpes zoster	Dermatomal distribution	Sharp, burning	Prolonged	None	Vesicular rash appears in area of discomfort
Esophageal reflux	Substernal or epigastric; may radiate to neck	Burning, visceral discomfort	10-60 min	Aggravated by large meal, postprandial recumbency; relief with antacid	Water brash
Peptic ulcer	Epigastric, substernal	Visceral burning, aching	Prolonged	Relief with food, antacid	—
Gallbladder disease	Right upper quadrant; epigastric	Visceral	Prolonged	Spontaneous or after meals	Right upper quadrant tenderness may be present
Anxiety states	Often localized over precordium	Variable; location often moves from place to place	Varies; often fleeting	Situational	Sighing respirations; often chest wall tenderness

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What is abnormal…

- Dyspnea
  - Can be cardiac or pulmonary (more often)- think when does it occur?
- Palpitations
  - Skipping, racing, fluttering- associated symptoms? Drugs? Arrythmias?
- Syncope
  - Inadequate blood flow, drop in CO, other reasons, sometimes associated with seizure
- Edema
  - Can be cardiac or liver related (or kidney or thyroid), travels up legs,
- Cyanosis
  - Blueish skin, low oxygen levels, vasoconstriction, Raynaud's, congenital heart disease
- Others
  - Cough, nausea, vomiting, reduction in ability to perform ADLs, others

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Physical Exam/VS considerations

- JVP assessment- 45 degrees
- NIBP- have they been sitting? Are they relaxed?
- Palpate pulses
- Chest wall abnormalities- pectus?
- Is PMI displaced? Is there a thrill?
- Auscultate and palpate
  - S3 can be sometimes heard in kids and young adults, S4 sometimes in older patients
  - Murmur present? (slides on this later)

References

Numerous images courtesy of:

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thank you

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