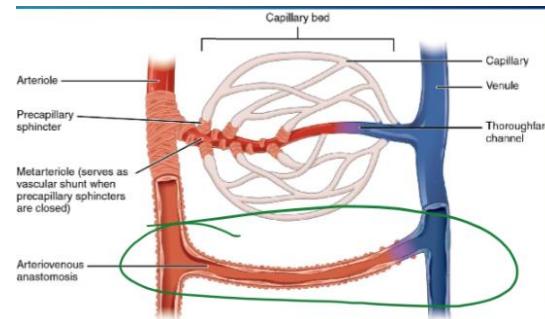


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Cardio Histology

- 2 main systems of circulation: **Systemic** (throughout the body) and **Pulmonary** (to and from the lungs, back to the heart)
- Portal Systems = 2 Capillary networks connected by a vein (Capillary → Vein → Capillary)
 - o **Hepatic Portal and Hypothalamic-Hypophyseal Portal**
- **Arteriovenous Anastomoses** = Shunt to bypass the capillary beds to decrease heat loss.
 - o Closed shunt: Capillaries open, heat lost
 - o Open shunt: Capillaries closed, heat conservation
 - o Found in fingertips, nose, lips, and erectile tissue

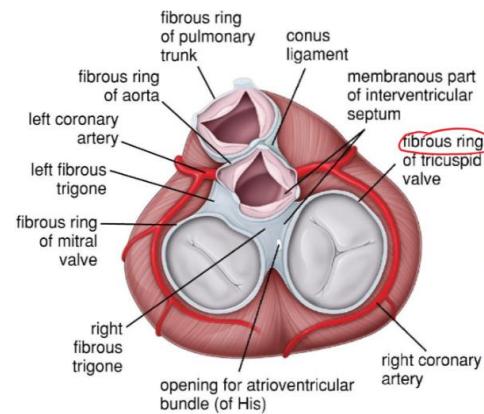
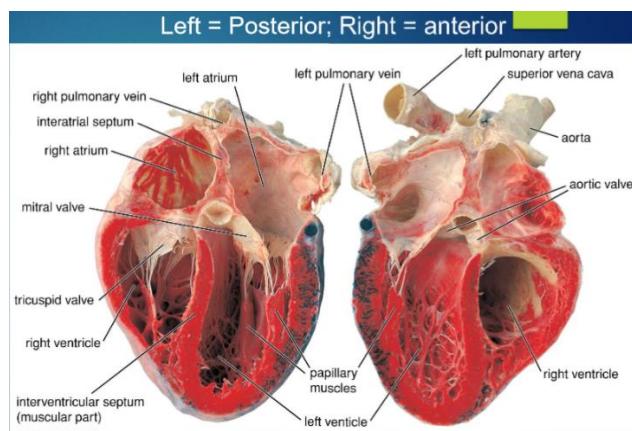


Heart

- Located in Thoracic cavity in the middle mediastinum surrounded by sternum, vertebral column, diaphragm, and lungs (AKA Anterior and Posterior Mediastinum's biiiiiiittttchhhh)
- Sits in a serous pericardium sac (2 layers, outer parietal layer and inner visceral layer) which attaches to neighbouring organs
 - o Parietal Layer = Simple Squamous mesothelium
 - o Serous fluid produced from both visceral and parietal mesothelial cells fills pericardial cavity.
- Has 4 chambers: 2 atria, 2 ventricles
 - o Deoxy blood enters Right Atrium → Right Ventricle → Pulmonary circulation
 - o Oxy blood enters Left Atrium → Left Ventricle → Systemic circulation
- Has 4 valves: 2 arteries, 2 atrioventricular (prevents backflow)

Layers of the Heart		
Epicardium (visceral serous pericardium)	Myocardium (middle Layer)	Endocardium (Inner most Layer)
= Simple Squamous epithelium -Underlying Connective tissue - Home to nerves and vessels	Cardiac Muscle <ul style="list-style-type: none"> - Striated, Involuntary - Branched cells, single nucleus - Intercalated discs Thicker in Ventricle than Atrium <ul style="list-style-type: none"> - b/c pumping against 2 high pressure systems 	3 Layers: Inner: Simple Squamous Endothelium + C.T. Middle: C.T. + Smooth Muscle Cells Deep (beside myocardium): C.T

- AV valves = Fibrous rings (part of fibrous skeleton of heart) + Valve



Interventricular Septum	Interatrial Septum
<p>2 portions:</p> <ol style="list-style-type: none"> Membranous portion (primarily connective tissue) Myocardial tissue (cardiac muscle surrounded by endocardium) 	<ul style="list-style-type: none"> Much thinner Cardiac muscle flanked by endocardium Looks whiter than interventricular because it has more fibrous tissue, and less muscle

Valves		
Spongiosa Layer	Fibrosa Layer	Ventricularis Layer
Blood vessel side/ Atrial Loose CT (loosely arranged collagen and elastic fibres) Lots of proteoglycans <ul style="list-style-type: none"> Absorb vibrations Give flexibility and plasticity 	Core of the valve <ul style="list-style-type: none"> Dense irregular CT Fibrous extensions from ring 	Ventricle side of valve <ul style="list-style-type: none"> Dense CT and many elastic fibers Continues into chordae tendinea in AV valves

Conduction

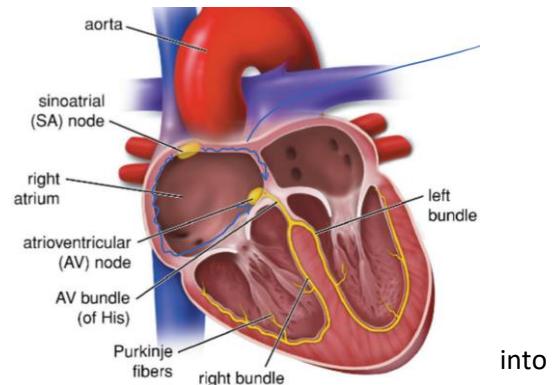
- Rhythmic contractions are **self-generated** (not dictated by nerves)
- Controlled by 2 nodes (modified cardiac muscle fibres) – **Smaller, no intercalated discs**

Sinoatrial (SA) Node:

- Generates initial impulse
- **Pacemaker of the heart** = Fastest rate of depolarization
- Spreads impulse along **modified CMF** through atria

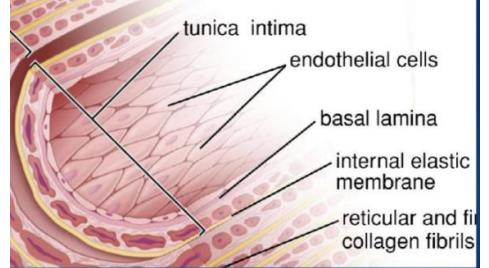
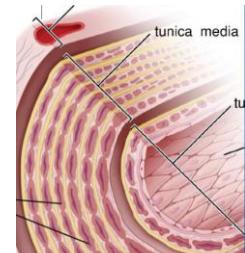
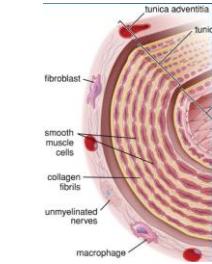
Atrioventricular (AV) Node:

- Picks up the impulses from the modified atrial CMF
- Conduction fibres (modified CMF; **larger than regular ventricular CMF**) carry impulse through Bundle of His
- Bundle of His divides into L and R branches and then further Purkinje Fibres



into

Heart Rate		
Parasympathetics	Acetylcholine	Decrease H.R.
Sympathetics	Norepinephrine	Increase H.R.
Hormones	Norepinephrine & Epinephrine	Increases force of contraction
CNS		
Baroreceptors	(High pressure receptors – Arterial pressure)	Located on <ul style="list-style-type: none"> - Carotid Sinus (Internal carotid artery) - CN IX - Aortic Arch - CN X
Chemoreceptors	Detect change in O ₂ , CO ₂ tension, pH	Located on <ul style="list-style-type: none"> - Carotid Bodies - Aortic bodies (bifurcation)
Volume Receptors	(Low Pressure sensors – central venous pressure)	Located on: <ul style="list-style-type: none"> - Walls of Atrium and Ventricle, sense sensory to CNS about heart distension

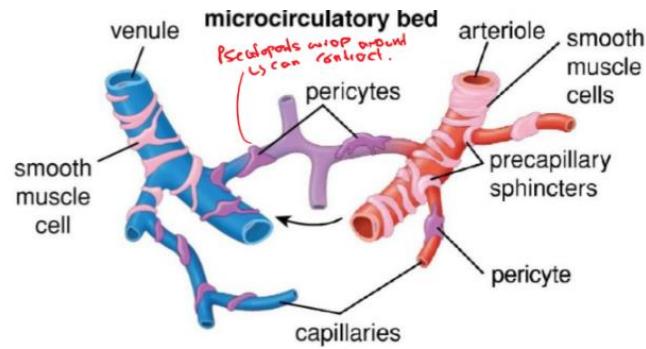
Layers of the Vessels		
Tunica Intima	<p>Innermost layer of vessel Made of</p> <ul style="list-style-type: none"> a) simple squamous epithelium (endothelium) b) basal lamina c) subendothelial loose C.T. sometimes with smooth muscle cells within <p>In arteries there is also an internal elastic membrane. Thin layer of fenestrated elastic material</p>	
Tunica Media	<p>Vascular smooth muscle cells Variable levels of elastin, reticular fibres, and proteoglycans depending on artery or vein and size Outer layer made of elastin</p>	
Tunica Adventitia	<p>Outermost layer Made of C.T – longitudinal collagen and a few elastic fibres Thickness depends on vessel or artery</p>	
Large Arteries		1. Tunica Intima - SMC 2. Tunica Media – Lots of SMC, Elastic Fibres (no fibroblasts) 3. Tunica Adventitia - Thin
Large Vein		1. Tunica Intima – No SMC 2. Tunica Media – Few SMC, collagen, fibroblasts 3. Tunica Adventitia – Thick, Collagen + elastic fibres, longitudinally arranged SMC

Vascular Endothelium:

- **Simple Squamous Epithelium**
 - o Flattened, elongated, polygonal shaped with long axis in direction of blood flow to decrease sheer forces
- **Function:**
 - o Maintain **selective permeability** (O_2 , CO_2 , Glucose, Amino Acids)
 - o Maintain non-thrombotic barrier by **releasing Prostacyclin, and NO** to prevent coagulation
 - o Secrete **vasoconstrictors and dilators**
 - o Control lymphocyte-endothelial adhesion during immune response
 - o Synthesize and release growth factors
 - o Converts **Angiotensin I to Angiotensin II** in Renin-Angiotensin System (Blood pressure control)
- **Damaged by:**
 - o High levels of LDL cholesterol
 - o Hypoglycemia (diabetes)
 - o Hypertension
 - o Smoking
 - o Bacterial and Viral infection

Capillaries

- Only a Tunica Intima layer (no TM or TA)
 - o TI only endothelial cells and basal lamina (No CT)
- Pericytes
 - o Cells with branching cytoplasmic arms that surround capillary
 - o Covered by basal lamina
 - o Contractile units that modulate capillary blood flow



Continuous Capillaries	Fenestrated Capillaries	Discontinuous Capillaries
CT + all 3 muscle types Tight junctions, complete basal lamina impermeable Found in skin, lungs, and CNS	Selectively permeable Found in Endocrine glands, gall bladder, kidney, GI, Pancreas	Very permeable Spot connections between cells Found in: Liver, Spleen, Bone Marrow
a		

Lymphatic Capillaries

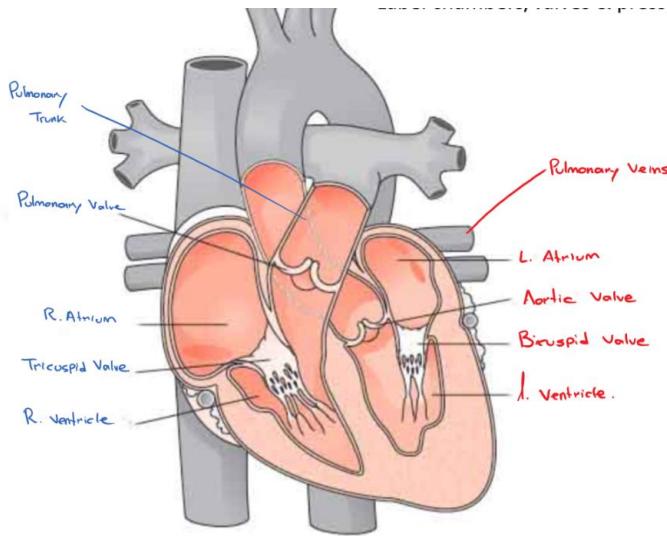
- Smallest of the lymphatic system
- Blind ended (closed) tubes
- Empty into lymphatic vessels
- Numerous in CT under epithelium of skin and mucous membranes
- Unidirectional -> **only uptake, no delivery**
- Lined with endothelium + discontinuous basal lamina
 - o = **Highly permeable**

Lymphatic Vessels

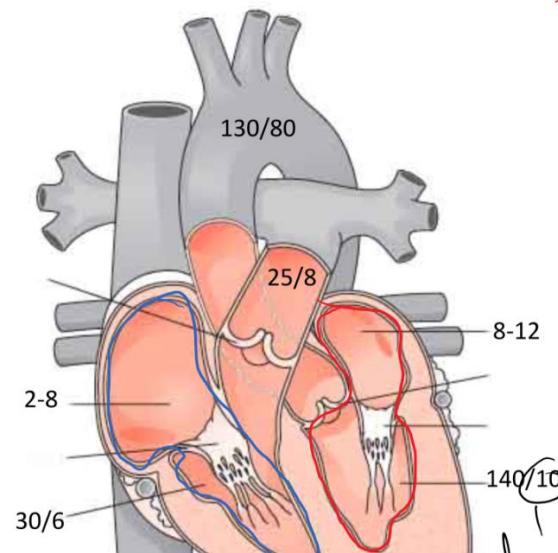
- Tight junctions + continuous lamina = **no leakage**
- Contains SMC
- Thicker walls, and has valves to fight gravity
- Skeletal muscle compression forces lymph through system

Cardiac Physiology

Structures



Pressures (don't memorize values, just trends)



Pressure Differential: R. Atrium & R. Ventricle pressure <<<< L. Atrium & L. Ventricle

- Heart has to pump blood a much larger distance, so the contraction and pressure in the left ventricle has to be greater than right.

Shunts	
Hole between Left and Right Ventricles	<p>Some O₂ blood would get recirculated through lungs, some through normal circulation</p> <ul style="list-style-type: none"> - More blood than normal enters pulmonary circulation. Left ventricle has to work harder to get enough blood around body. <p>Results:</p> <ul style="list-style-type: none"> - Left → Right shunt (b/c of pressure difference) - ↑ Pulmonary blood flow - L. Ventricular hypertrophy - L. atrium & L. Ventricle overload and eventual fail
Hole between Left and Right Atria	<p>Some O₂ blood enters right atrium and gets recirculated through pulmonary circulation again, some goes through normal circulation</p> <ul style="list-style-type: none"> - More blood than usual enters pulmonary circulation, this time more blood in the right ventricle <p>Results:</p> <ul style="list-style-type: none"> - Left → Right shunt (b/c of pressure difference) - ↑ Pulmonary blood flow - R. Ventricular hypertrophy

Valves:

- Open when upstream pressure is greater than downstream pressure
- Sound generated when valve slaps closed

	Open
Tricuspid	R. Atrial Pressure > R. Ventricular Pressure
Pulmonary	R. Ventricular Pressure > Pulmonary Artery Pressure
Mitral	L. Atrial Pressure > L. Ventricular Pressure
Aortic	L. Ventricular Pressure > Aortic Artery Pressure

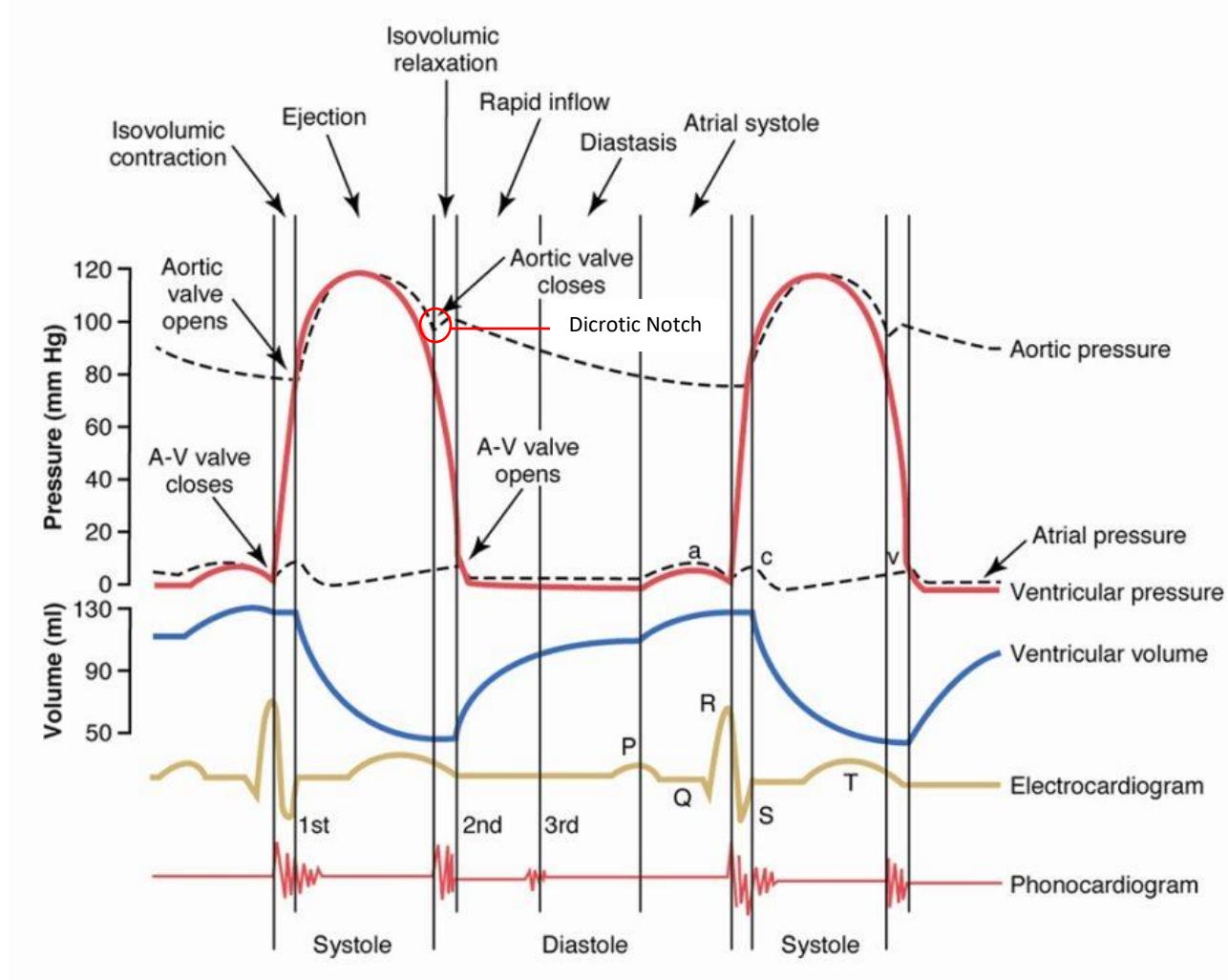
Cardiac Cycle:

Diastole: Ventricular Relaxation, filling

Systole: Ventricular Contraction, eject blood into body

Isovolumic Contraction = Both valves are closed and Ventricular pressure ↑

Wiggers Diagrams



Heart Sounds:

S1	Closure of AV valve High frequency Caused by vibrations in valves and walls
S2	Closure of Semilunar valves (Aortic and Pulmonary) High pitched Physiological Splitting: - During inspiration heard as 2 sounds (dub dub) Enhances venous return to right heart & lungs. Prolongs R Ventricle ejection and PV closure Reduced venous return to left heart, L. Ventricle ejection shortens and AV closes earlier - Expiration heard as 1 sound (dub)
S3	Early diastole during rapid ventricular filling Can be normal in children In adults can indicate volume overload w/ congestive heart failure
S4	Late diastole Atria contracting and ejecting against stiffened ventricle Indicative of cardiac disease

Murmurs:

= Sound generated by turbulent flow through heart

5 possibilities for cause of a murmur:

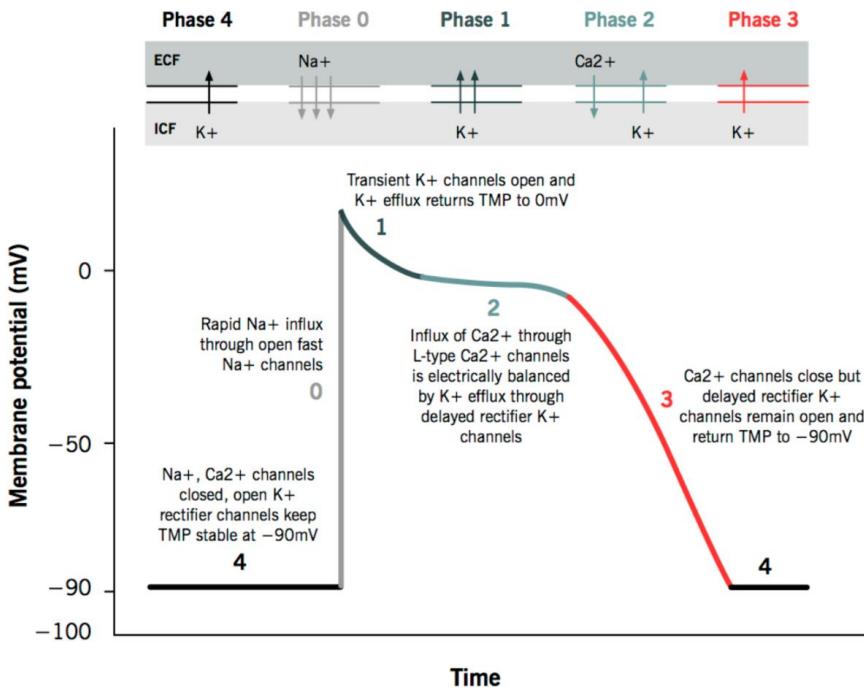
1. **Partial Obstruction** ([valve stenosis](#))
2. **Increased/disturbed flow** through normal structures (aortic systolic murmur in anemia)
3. Ejection into **dilated chamber** or vessel (aneurysmal dilation of aorta)
4. **Regurgitant** flow back through incompetent valve (mitral regurgitation)
5. Abnormal **shunting** from high to low pressure chamber (ventricular septal defect)

Examples:

Valvular murmur coinciding with ventricular systole	Stenosis of aortic or pulmonary valve Incompetent bicuspid or tricuspid valve (regurgitation)
Valvular murmur coinciding with ventricular diastole	Stenosis of bicuspid/tricuspid valve Incompetent Pulmonary or Aortic valve

Cardiac Action Potentials

Working Myocardium



Phase 0:

-Fast Na⁺ open → Large influx in Na⁺ (I_{Na})

Phase 1:

-Na⁺ channels inactive rapidly,
-Transient K⁺ channels with outward current partially depolarize membrane

Phase 2 (Plateau):

-Ca²⁺ channels open (slow inward current I_{si}) but inward current I_s is balanced by Transient outward current of K⁺
-At end of phase Transient Inward K⁺ closes and is replaced by Rapid and Slow K⁺ rectifiers (I_{kr} , I_{ks})

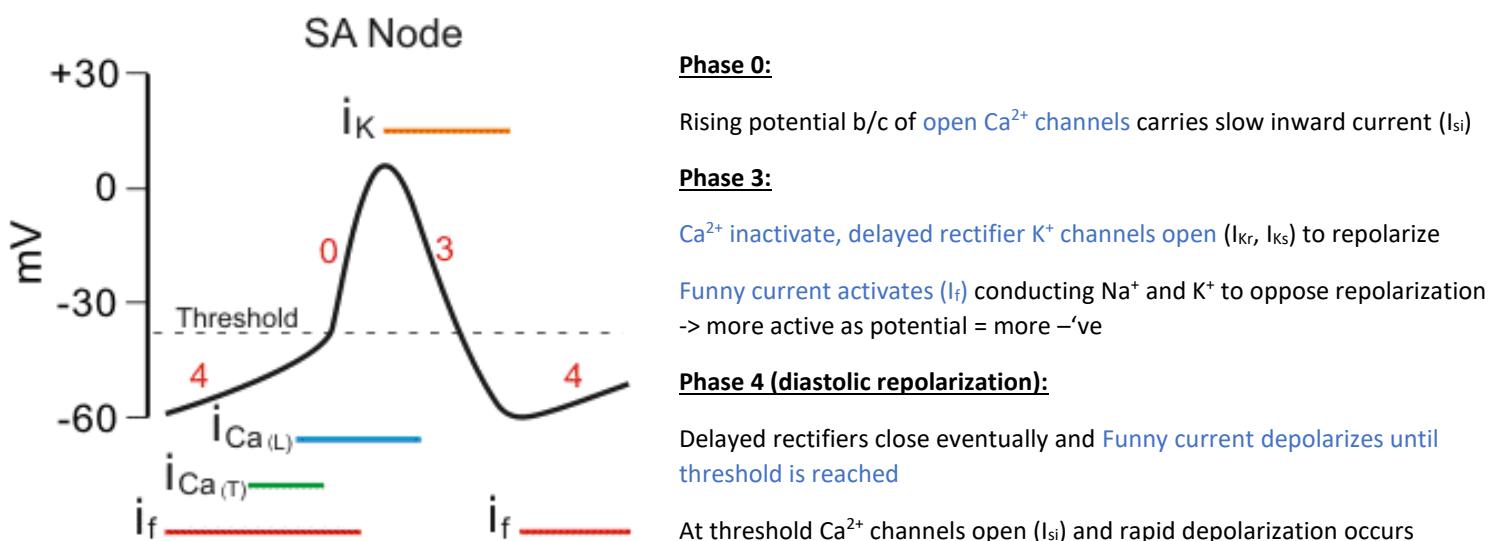
Phase 3:

-Ca²⁺ inactivates, K⁺ rectifiers fully active to pump out K⁺ → rapid repolarization
-inactivated channels close (Fast Na⁺, Ca²⁺, Transient K⁺)

Phase 4:

-inward rectifiers fully active and bring potential back to -90mV

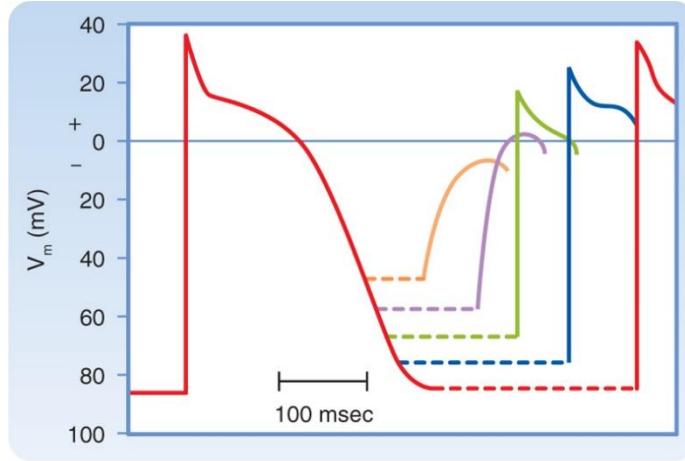
Pacemakers in AV and SA Nodes



Purkinje Cells and Bundle of His

- Potentials look like the working myocardium, but have i_f currents that act as pacemakers (slow phase 4 depolarization)
- Intrinsic frequency much slower than nodes
- Bundle of His has complex potential morphology
 - o Nodal phenotype proximal to atria and transitions to a Purkinje phenotype in the ventricles

Refractory Period



During refractory period Na^+ channels are “inactivated” NOT closed. In this state they cannot go straight to activation without first entering the closed state. This prevents cumulative AP firing and tetanus in the heart.

Rate and rise of the action potential is proportional to the # of closed Na^+ . Longer the refractory period lasts, the more channels close (allowing for more to consequently open)



Conduction Velocity

3 Factors:

1. Size of cells

- Smaller = \uparrow resistance to flow of current = Slower velocity
- Pacemakers are smallest, Purkinje cells are largest

2. # of gap junctions

- \uparrow #'s = Faster velocity
- Pacemakers have least, Purkinje cells have most

3. Rate of Action Potential rise

- Faster the rise, faster the conduction
- Pacemakers have slowest rise, Purkinje cells have fastest rate

SA Node	>	Atrial Cells	>	AV Node	>	Bundle of His	>	Purkinje Cells	>	Ventricular Cells
Slow Small Cells \downarrow gap junction Allows complete Atrial filling		Fast Larger cells \uparrow Gap junctions		Slow Small cells \downarrow Gap Junctions Allows complete ventricular filling		Allows rapid and uniform ventricular contraction Most Gap junctions Largest				

Physiology of Cardiac Electrical System

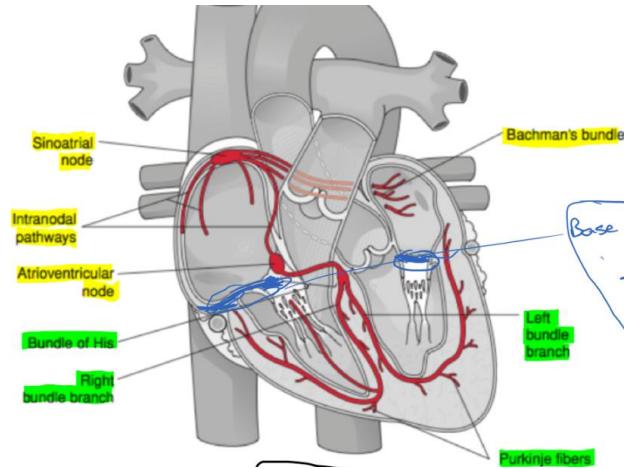
Electrical Conducting System

= System of fast conducting specialized cardiac muscle cells (NOT nerves)

- These cells don't contract, just transmit electrical signals
- Current leaks out of gap junctions and causes the cells around to contract.

Atrial Conducting System		Sinoatrial Node (SA) Intranodal Pathway Bachman's Bundle Atrioventricular Node (AV)
Ventricular Conducting System		Bundle of His Right and Left Bundle Branches Purkinje Fibres

1. AP spontaneously develops, reaches threshold fastest in SA Node
 2. Travels down atrial conducting system towards AV node
 3. Slows down at AV, allowing atria to fill and contract before signal carries on into Ventricular conducting system
- Base of atria = **Cardiac skeleton**. Prevents passage of electrical current/signal from passing directly into ventricle without AV Node



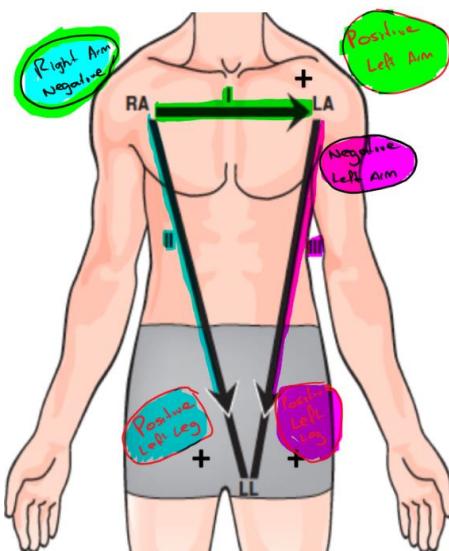
All conducting cells are capable of spontaneous depolarization, but each zone is at a different rate

- Allows for backups to keep heart beating, and directionality of signal and contraction
- If conduction is blocked above AV node (no SA) = HR slower, AV spontaneously depolarizes for contraction
- If SA still works but electrical block in between SA and AV = Uncoordinated contractions (Atria 60-100, ventricle 40-50). This isn't fatal, Atrial contraction only spills a little bit of blood into ventricle in normal situation anyway, most just passively drains into ventricle.

1. **SA Node:**
 - a. 60-100 bpm (Fast, steep phase 4 slope on AP curve)
2. **AV Node:**
 - a. 40-50 bpm (slower, shallower phase 4 slope)
3. **Purkinje Fibres:**
 - a. 20-30 bpm (Slowest, flattest phase 4 slope)

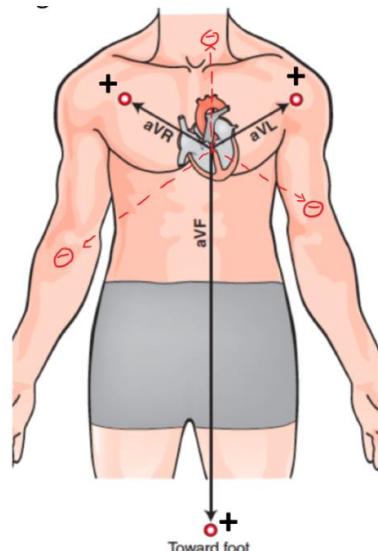
ECG Leads

Bipolar Limb Leads:

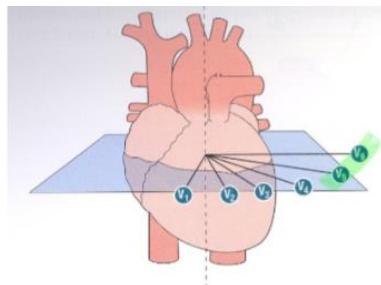
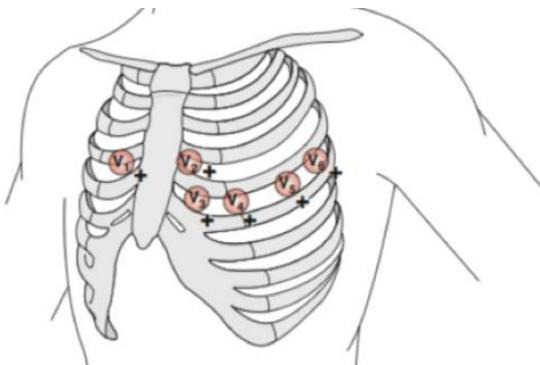


Lead I	Right Arm (-) -> Left Arm (+)	1L
Lead II	Right Arm (-) -> Left Leg (+)	2L's
Lead III	Left Arm (-) -> Left Leg (+)	3L's

Unipolar Limb Leads:



aVR (Right)	Right Arm +
aVL (Left)	Left Arm +
aVF (Left Foot)	Left Leg +

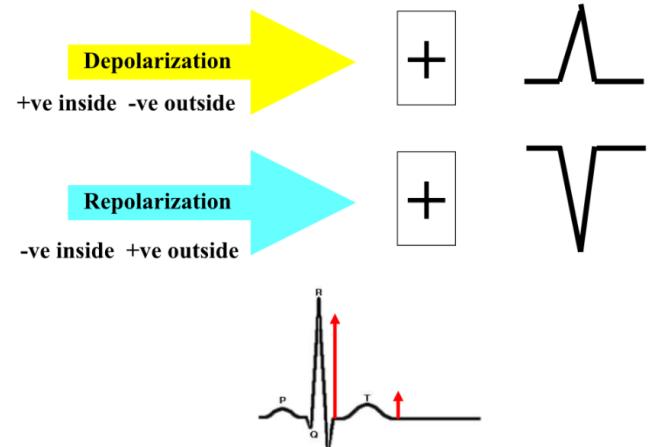
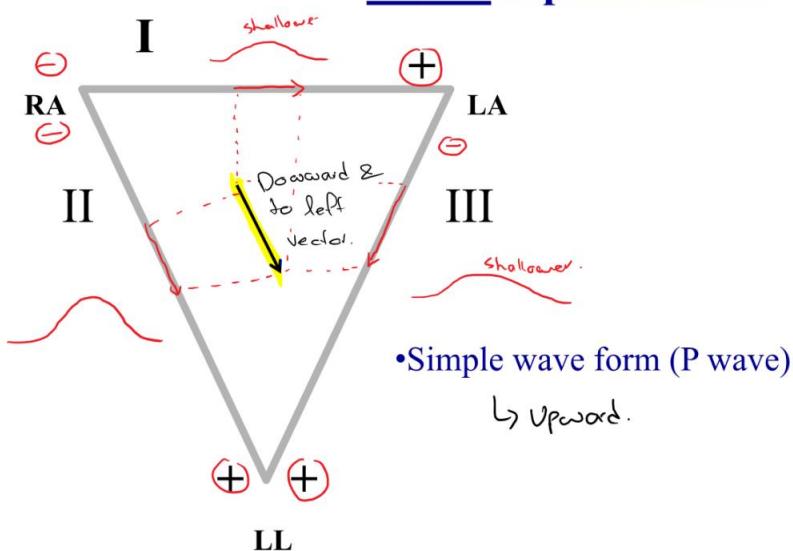


6 Precordial leads to measure ECG in horizontal plane:
(V₁, V₂, V₃, V₄, V₅, V₆)

- No ECG readings as AP travels through conducting system (insufficient current) = Flat line
- P, QRS, T waves represent current in atrial and ventricular muscle (contracting muscles)

Depolarization towards +ve	Upward Deflection	
Depolarization away from +ve	Downward Deflection	
Repolarization towards +ve	Downward Deflection (opposite sign)	
Repolarization away from +ve	Upward Deflection	

Atrial Depolarization



ECG Interpretations

P Waves	Upright P wave – SA Node (95%) Inverted P wave – Atria, AV Node, Ventricle No P wave – Not SA or Atria
PR Interval	Normal – Rules out AV Node and Ventricle
Baseline	Wavy w/ P waves & QRS – No Fibrillation Wavy w/o P waves – Atrial Fibrillation, atrial origin Wavy w/o QRS – Ventricular Fibrillation, ventricular origin
QRS	Narrow – Rules out Ventricle Wide – Could be diseased Purkinje Fibres
Rate	Atrial Rate > 200 – Not SA Node Ventricular Rate > 200 – Not SA Node, or Atria
Ratio	More P than QRS – Conduction Block P waves vary in shape and size – 2+ origins of P waves P waves + QRS w/o PR interval – 2 origins

Arrhythmia	Rate	Rhythm	P Waves	PR Interval	QRS
Sinus Bradycardia	< 60 bpm	Normal	Normal	Normal (3-5 squares)	Narrow (< 3 squares)
Sinus Tachycardia	>100 bpm	Regular	Normal	Normal	Narrow
Atrial Fibrillation	Any	Irregular, no pattern	None, wavy baseline	No P waves	Narrow
Atrial Flutter	Any/Flat	Regular or Irregular	More P than QRS, non-stop flutter waves	Sometimes normal, sometimes random	Narrow
1st Degree Block	Any	Regular	Normal	Long (> 5 squares)	Narrow
2nd Degree Mobitz Type I	Any	Regular w/occasional missing beats	Normal	Increasing with each beat until misses QRS, then resets	Narrow
2nd Degree Mobitz Type II	Any	Regular w/occasional missing beats	Normal	Constant	Narrow
3rd Degree Block	Ventricular rate slow	Regular, sometimes irregular	More P than QRS, P not associated with QRS	N/A – P waves not associated with QRS	Narrow or wide
Junctional Rhythm	40-60 bpm	Regular	Inverted or absent	Short, or N/A. Sometimes in front of or behind QRS	Narrow
Supraventricular Tachycardia	>100, >150	Regular	Not easily identified	No P waves typically present	Narrow
Ventricular Tachycardia	>100	Regular	Not easily identified	If P waves are present, not associated with QRS	Wide
Ventricular Fibrillation	0	Indeterminant	None	N/A	None

Cardiac Arrhythmia's

Normal Cardiac Rhythm	<ul style="list-style-type: none"> - P waves precede QRS (1:1) ratio - P waves are all the same shape and size - PR interval is normal and constant.
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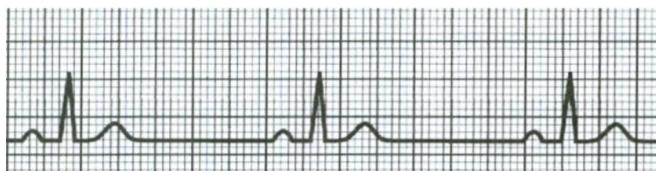
Brady Arrhythmia

= Slow HR for given conditions

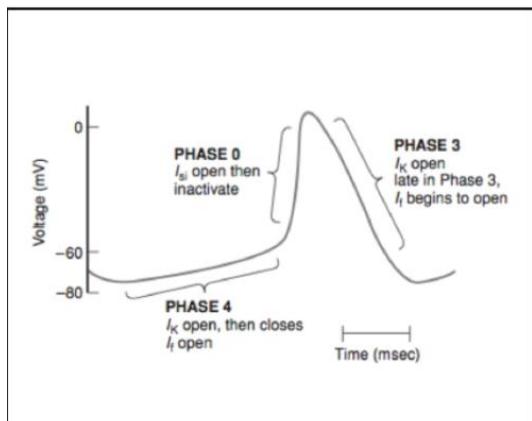
- Normal = 60-100bpm

Count number of large squares on ECG between R wave peak.
Divide 300 by the number = beats/min

Sinus Bradycardia (<60bpm)



Profound outpouring of parasympathetic vagal action. Slows the HR.



HR = 42bpm

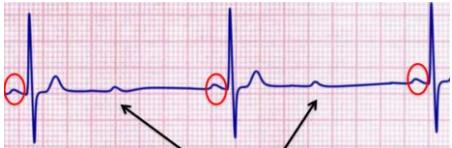
↑ Parasympathetic action

↓ phase 4 slope, ↓ Resting membrane potential, ↑ time to threshold

- Slows down HR -> takes longer for AP to cross threshold

Bradycardia	Bradyarrhythmia
HR < 60bpm at rest Can't be for pathological reason (sleep, waking, vasovagal response)	Rhythm that is abnormally slow AND b/c of electrical issue with heart Eg: Sinus node dysfunction <ul style="list-style-type: none"> - Problem with impulse generation or atrial conduction - Chronic slow HR or sinus pausing (HR ceases for short periods)

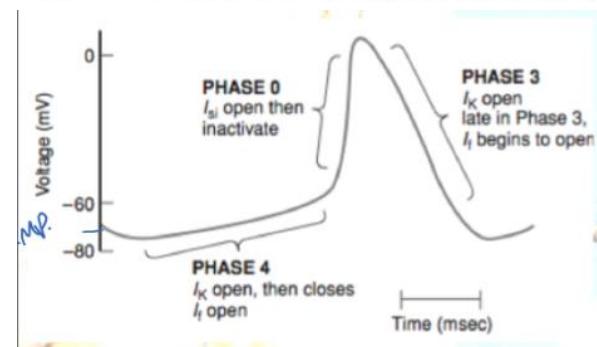
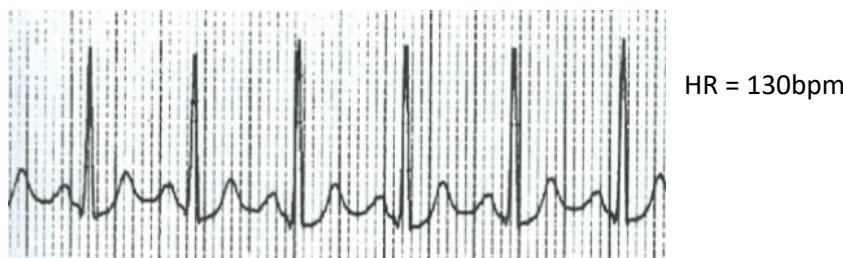
Conduction Blocks

1st Degree	Long PR interval (3-5 little squares) HR ~60bpm	
2nd Degree Mobitz Type I (Wenckebach Block)	1 st PR = Normal 2 nd PR = Longer 3 rd PR = Longest 4 th P with no QRS Reset Progressively longer PR intervals	
2nd Degree Mobitz Type II	PR constant and unchanging QRS randomly drops sometimes	
3rd Degree	P waves occur more than QRS – Atrium contracts @ same time as ventricle. Atrium not able to pump blood into ventricle and get pressure wave bouncing back up through Jugular Ventricle and Atrium independently conducting at their own rhythms. Nothing gets from atrium into ventricle P waves ~ 100/min QRS ~ 45/min	

Tachy Arrhythmia

= Abnormally fast rhythm given conditions

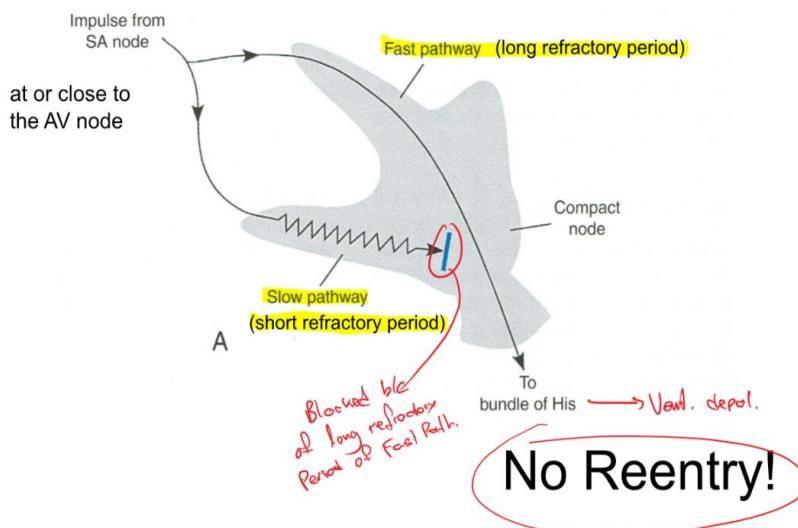
Sinus Tachycardia (>100 bpm)



- ↓ Parasympathetic drive (\uparrow Sympathetic)
- \uparrow Phase 4 slope, \uparrow Resting membrane potential, \downarrow Threshold
- Reach threshold AP faster for faster HR

AV Nodal Re-entry Circuits

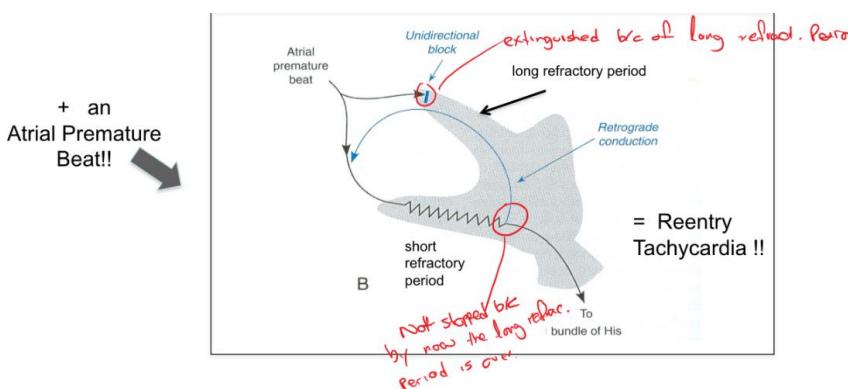
3 Requirements	<ol style="list-style-type: none"> 2 parallel pathways for AP (structural or electrolyte compositional) Differing conditions of refractory period and conduction velocity Arrival of a premature atrial beat
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Fast pathway: Transmits signal rapidly, but with a long refractory Period

Slow pathway: Transmits signal slowly, but with a short refractory period

- Normally signal splits through fast and slow refractory pathways
- Pathways merge together later into normal refractory zone, but by the time the slow pathway reaches merge, the fast pathway has already depolarized the area -> now in refraction = slow pathway blocked
- No Re-entry

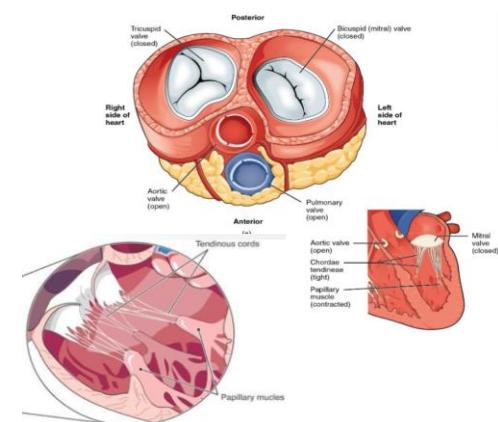


- Premature atrial AP runs down and splits into slow and fast pathways.
- Fast pathway is still in refractory period, so signal is blocked there. But carries on through slow pathway (b/c of its short refraction)
- At merger the signal splits again, one goes down to ventricle, and the other goes back up through the fast pathway (wrong direction) b/c by now it's done its refractory period.
- Signal loops back into slow pathway and the cycle continues.

Heart Valve Diseases

= Valves have incorporated cusps that open and close (usually only one way) during an increase in pressure in their corresponding chamber.

- Mitral Valve = 2 cusps (bicuspid)
- All other valves = 3 cusps (tricuspid)
- Chordae tendineae hold flaps closed, preventing prolapse of the cusps. Attached to papillary muscles that create the tension needed to keep the valve closed



3 Basics Problems	
Regurgitation	Occurs when valve doesn't close properly, blood flows back into chamber Often do to prolapse (mainly affects mitral valve)
Stenosis	Thickening or fusing of valve cusps (may be calcified) Prevents valve from opening fully (\downarrow blood flow)
Atresia	Valve lacks opening for blood (blind ended tube)
preload	Volume of blood in ventricle immediately prior to contraction With leaky valves, a little extra blood enters ventricle = \uparrow Preload = Ventricular dilation over time to accommodate extra volume \downarrow Preload = \downarrow Stroke Volume = \downarrow Blood Pressure
Afterload	Resistance to left ventricular ejection (Aortic pressure to overcome) When \uparrow Afterload is sustained, ventricles hypertrophy and dilate to meet increased contraction demands. - If continues farther, O ₂ demands are unmet and L. ventricular failure, angina, myocardial infarction, and heart failure may occur. \uparrow Blood Pressure = \uparrow Afterload = \downarrow Stroke Volume
Contractility (Inotropy)	Measure of cardiac performance describing quality of myocardial contraction. Ischemic heart = \downarrow contractility due to \downarrow O ₂ for cell function maintenance. \uparrow contractility = \uparrow Stoke Volume

	Description	Symptoms
Pulmonary Valve Stenosis	Stenosis of pulmonary valve, most often congenital Results in Right Ventricular hypertrophy (must work harder to get blood through)	Murmur Cyanosis Dyspnea Dizziness Upper Thorax pain Developmental disorders
Rheumatic Fever	Inflammatory disease (autoimmune) developed because of inadequate treatment of Strep throat or Scarlet fever. Develops autoantibodies against tissues in heart, joints, skin and brain	
Bacterial Endocarditis	Inflammation of endocardium, often involving heart valves (caused by bacterial infection) Endocardium becomes damaged and bacterial can infect heart valves and lining	Characterized by vegetation lesions (mass of platelets, fibrin, microcolonies of bacteria)
Aortic Regurgitation	Incompetent Aortic valve = backflow from aorta into left ventricle. Etiology: Aortic Root Dissection - Tear in wall of aorta, blood flows btwn layers of vessel walls. Aortic Root Dilation - Indicative of aortic aneurysm Myxomatous Degeneration - Pathological weakening of C.T - Mitral Prolapse Marfan Syndrome - Genetic C.T. disorder Rheumatologic disorders - SLE, Rheumatoid arthritis etc	Exertion dyspnea Orthopena - (shortness of breath when lying flat. Due to pulmonary edema with left sided heart failure) Paroxysmal Nocturnal Dyspnea - (Attacks of severe shortness of breath and coughing at night) Palpitations Chest Pain Gradual increase in Left Ventricular volume (dilation) and Stroke Volume - LV hypertrophy and chamber enlargement

Aortic Valve Stenosis	<p>Most common and most serious Progressive disease ->Damage to valve restricts flow -> increased resistance to eject into aorta ↑afterload and ↑ LV stress (pressure)</p> <p>Patient becomes symptomatic when ventricle can no longer compensate</p> <p>Etiology: Degenerative - Wear and tear with calcification over time Bicuspid aortic valve - Leads to hemodynamic stresses (fibrosis and calcification) Rheumatic Fever</p>	<p>Angina Syncope Dyspnea Decreased exercise/activity tolerance High mortality rate without surgery</p>
Aortic Valve Calcification	<p>Risk Factors like atherosclerosis</p> <p>3 Processes:</p> <ol style="list-style-type: none"> 1. Lipid accumulation - LDL Oxidation 2. Inflammation - Macrophage and T cell mediated (IL-1β) and growth factor β1 3. Calcification 4. Stenosis 	
Metabolic Syndrome	<p>Presence of at least 3 of 5 conditions:</p> <ol style="list-style-type: none"> 1. Abdominal Obesity 2. ↑ Blood Pressure 3. ↑ Fasting plasma glucose (diabetes) 4. Dyslipidemia (↑ triglycerides, ↓ HDL) <p>Associated with increased risk for CV disease and type 2 diabetes</p>	

Prosthetic Heart Valves

- Prior to surgery we address all dental disease/infections to avoid bacteremia
- Consider hospitalization and IV sedation if patients are unstable

	Mechanical Valves	Bio-prosthetic
	<p>3 basic types:</p> <ul style="list-style-type: none"> - Bi-leaflet - Mono-leaflet - Caged ball valves <p>Don't have the same functional properties as human valves, but last a long time</p>	<p>Typically pig valves</p> <p>Functional properties like human valves:</p> <ul style="list-style-type: none"> - Hemodynamics - Resistance to thrombosis <p>Wear down faster than mechanical valves</p>
Considerations	<p>Lifelong anticoagulation (warfarin) Lifelong antibiotic prophylaxis</p>	<p>Anticoagulation therapy (warfarin) for 1st 3 months after implantation Lifelong antibiotic prophylaxis</p>
Complications	<p>High risk for endocarditis Valve failure Valve endocarditis Valve thrombosis Thromboembolism Mechanical hemolytic anemia Systemic hemorrhage (anticoagulant related)</p>	

Bacterial Endocarditis

= Infection of the lining of heart and heart valves	
Prophylaxis	<p>Required if procedure involves manipulation of gingival tissues, periapical region of teeth or perforation of mucosa.</p> <p>Required for:</p> <ul style="list-style-type: none"> - Artificial heart valves - History infective endocarditis - Cardiac transplant develops valve problem - Certain specific serious congenital heart conditions <ul style="list-style-type: none"> (Unrepaired/incompletely repaired cyanotic congenital heart disease) (Completely repaired congenital heart defect with prosthetic material/device during 1st 6 months after procedure) (Repaired congenital heart defect with residual defect at the site or adjacent to site of prosthetic patch or device)
Prophylaxis Pharma	<p>Amoxicillin 2.0g (orally) 1hr before surgery</p> <p>If allergic to Penicillin antibiotics:</p> <ul style="list-style-type: none"> - Clindamycin 600mg (orally) 1hr prior to procedure OR - Cephalexin or Cefadroxil 2.0g (orally) 1hr prior to procedure OR - Azithromycin or Clarithromycin 500mg (orally) 1hr prior
Dental Procedures requiring Prop.	<p>All procedures involving gingival manipulation or periapical region of tooth or breaking of oral mucosa</p> <p>No Prophylaxis needed for:</p> <ul style="list-style-type: none"> LA in healthy tissue Radiographs Placement or adjustment of orthodontic appliances Bleeding from lips or mucosa (at home, non dental created)

Congestive Heart Failure

= inability of heart to fill and eject blood, or supply proper amounts of O₂ blood to meet bodies metabolic demands

Structural Factors	Functional Factors
Myocardial Damage (coronary artery damage)	Increased workload
Myocardial Infarction	Increased peripheral vascular resistance Hypertension

Cardinal Symptoms:

- **Dyspnea** (especially with exercise)
- **Orthopnea**
- **Fatigue**
- **Edema** (Pulmonary, peripheral, ascites)
- ↓ exercise

Symptom	Left-Sided Failure	Right-Sided Failure
Pitting Edema (legs, hands)	Mild – Moderate	Moderate – Severe
Fluid Retention	Pulmonary edema and pleural effusion	Abdomen
Organ enlargement	Heart	Heart + Liver
Neck Veins	Mild – Moderate ↑ JVP	Severe JVP, visible distention
Shortness of breath	Prominent Dyspnoea	Dyspnea present but not prominent
GI	Present, not prominent	↓ appetite, bloating, constipation, more prominent than LVF

Causes

Coronary Artery Disease	Impaired blood flow through coronary artery (atheroma)	<p>Presentations: Silent ischemia Angina pectoris Unstable angina MI Sudden cardiac death</p> <p>Diagnosis: ECG Symptoms Stress Testing Coronary angiography</p> <p>Prevention: Reduce hypercholesterolemia, Reduce hypertension Reduce Physical inactivity Prevent obesity Stop smoking</p>
Left Ventricular Dysfunction	<p>Sustained ↑afterload (hypertension, stenosis), ventricle walls hypertrophy to meet the ↑ pumping demands. Ventricle then dilates to counteract the ↓ in volume</p> <ul style="list-style-type: none"> - Ventricle ↓ stroke volume, & Ejection fraction <p>Body compensates, ↑ HR -> ↑ myocardial O₂ demand, but also ↓ O₂ supply</p> <ul style="list-style-type: none"> - More heart beats = ↑ energy needed, and ↓ perfusion during diastole. 	<p>Respiratory Symptoms:</p> <ul style="list-style-type: none"> - Left heart failure = blood backup into lungs -> Respiratory symptoms, and fatigue - Pulmonary vascular pressure increases -> fluid extravasates from capillaries into interstitium and alveoli = pulmonary edema <ul style="list-style-type: none"> ➔ ↓pulmonary compliance, ↑ work of breathing ➔ Poor blood gas transfer -> dyspnea <p>Resp Signs: tachypnea, increased work of breathing, recruitment of accessory muscles in breathing</p>
Pulmonary Heart Disease (cor pulmonale)		
	<p>2° to Left heart failure, pulmonic stenosis, pulmonary hypertension, tricuspid regurgitation</p> <ul style="list-style-type: none"> - ↓ LV efficiency -> blood backup in pulmonary vasculature that RV needs to pump against 	<ul style="list-style-type: none"> - Narrowing of arteries in lungs and fibrosis of capillaries -> ↑ BP in pulmonary arteries ↑ afterload of RV - Congestion of systemic capillaries = peripheral edema, ascites, hepatomegaly, ↑ JVP <p>JVP – Measure distance between sternal angle and top of JVP (should be <3cm above sternal angle)</p> <p>Congestive Hepatopathy - ↑ R Atrial Pressure = backflow into IVC and hepatic veins, congesting liver</p> <ul style="list-style-type: none"> - ↓ hepatic drainage - Blood stasis (accumulation of deoxy blood, accumulation of water, necrosis, collagen deposition, fibrosis)
Physical Exam		
Auscultation Cyanosis		<p>Crackles in lungs suggest pulmonary edema</p> <p>Late stage pulmonary edema</p> <p>Central: Around core, lips and tongue</p> <ul style="list-style-type: none"> - Cased by CHF, Acute Coronary syndrome, MI, Lung edema, Lung thromboembolism, Severe pneumonia, Acute Asthma <p>Peripheral: Extremities of fingers/toes</p>
Heart Murmurs		Indicative of valvular heart disease (aortic stenosis, mitral regurgitation)
Gallop Rhythm		Marker of ↑ blood flow/intracardiac pressure

Shock

= State where **Brain, Heart, Liver, and Kidneys** are not receiving enough O₂

Stage I	Low perfusion first detected, and compensatory mechanisms are activated Results: ↑HR + ↑SVR + RAAS Activation Very few symptoms, treatment can halt any progression
Stage II	Compensatory mechanisms start failing, perfusion drops and symptoms begin Symptoms: Confusion, disorientation (Cerebral hypoxia) Angina (Cardiovascular hypoxia) Treatment can reverse this stage
Stage III	Irreversible tissue damage Cardio function ↓, Renal Failure, Death

4 Kinds of Shock

Hypovolemic	↓ Venous return and Cardiac output - Heart can't pump enough blood due to fluid or blood loss Causes: - Sudden hemorrhage - Diarrhea, vomit, excess sweat or urination
Cardiogenic	↓ Function of heart - Myocardial Infarction - Arrhythmia - Valve problems - Ischemia - Myocarditis - Bacterial Endocarditis - Cardiac tamponade
Vasodilatory	↓ Systemic Vascular Resistance - Rapid drop in BP -> ↓ Tissue perfusion Causes: - Anaphylaxis (histamine enhanced vasodilation) - Septic Shock (toxin enhanced vasodilation)
Obstructive	Blocked circulation - Pulmonary Embolism - Myocardial Infarction - Cerebrovascular Accident - DVT

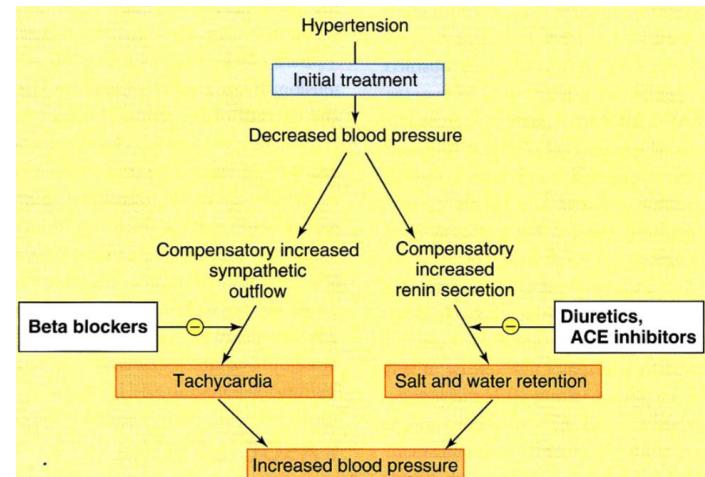
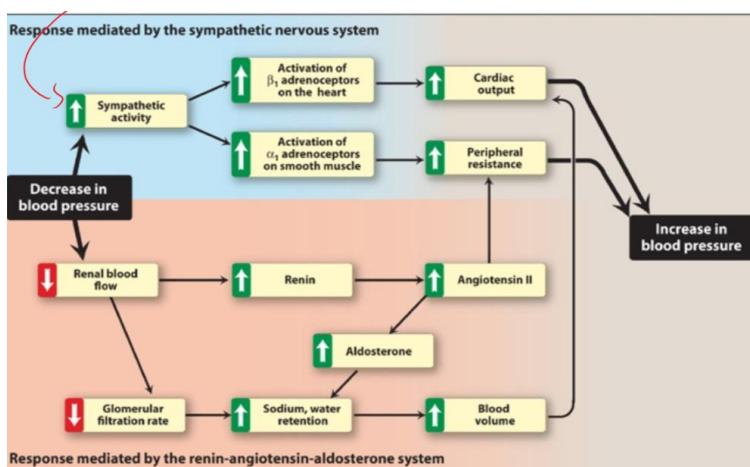
9 Signs and Symptoms of Shock

1. Systolic BP <90mmHg	5. Altered mentals (cerebral hypoxia)
2. ↑ HR (Sympathetic Stimulation)	6. ↓ Urine (↑ Aldosterone + ADH)
3. Weak Pulse (↓ Cardiac Output)	7. Thirsty (loss of ECF)
4. Cool, Pale, Clammy, Skin (Sympathetic constriction, and diaphoresis)	8. Acidosis (Lactic acid build up in absence of O ₂)
	9. Nausea (impaired blood flow to GI)

Cardiovascular Pharmacology

Hypertension

<p>Stepped Care Approach to treatment:</p> <ol style="list-style-type: none"> 1. Lifestyle Changes: ↓ Smoking, ↓ Stress, ↓ Weight, ↓ Salt, ↑ Exercise 2. Antihypertensive drug treatment: Diuretic, or beta-blocker, ACE inhibitor, Ca²⁺ channel blocker 3. ↑ Dosages and combinations of drugs: Add vasodilators/adrenergic drugs 4. More drug mixing 	<p>Monotherapy</p> <p>More popular than stepped care</p> <ul style="list-style-type: none"> - Simpler - Better compliance - Low toxicity and interactions <p>Works well for mild-moderate hypertension</p>
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When give too high a dose of antihypertensive drugs, the body can over compensate the other way to bring BP back to normal. Causing hypotensive symptoms:

- Light headed
- Dizziness
- Muscle spasms

We can counter act this counter action by adding more drugs that will decrease the systemic effects increasing blood pressure back up

Diuretics

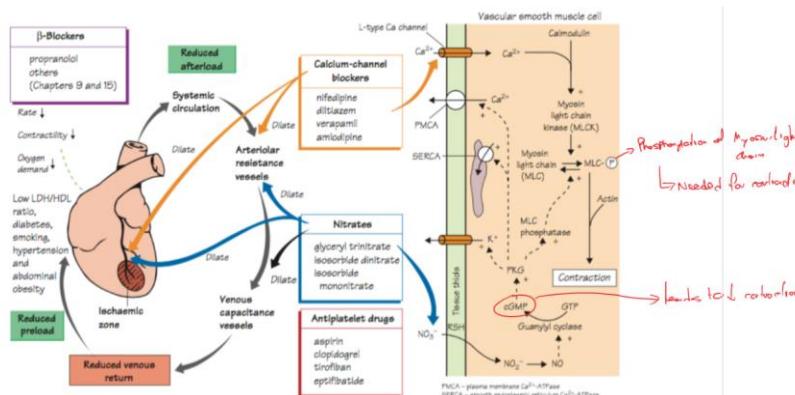
	Thiazide Diuretics	Loop Diuretics	Potassium-Sparing Diuretics
MOA	Inhibit the resorption of Na ⁺ at distal renal tubule -> reduce water retention NCC channels (Na, Ca antiporter)	Similar to thiazide diuretics but act on ascending loop of Henle to prevent Na ⁺ resorption NKCC channel	Similar again to Thiazides, but by acting at end of the nephron it prevents the body from compensating and resorbing Na ⁺ which would result in hypokalemia (for every Na ⁺ resorbed by the body, K is lost)
Drugs	Metolazone Bendroflumethiazide Hydrochlorothiazide	Furosemide Bumetanide	Spironolactone Amiloride Triamterene

Smooth Muscle Control

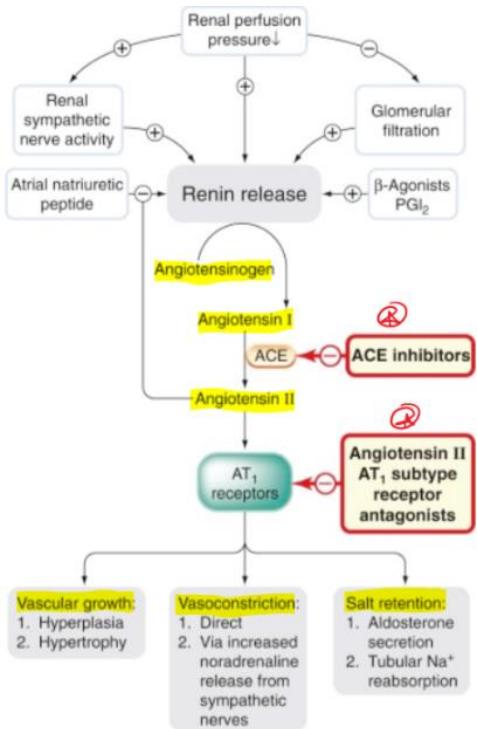
Controlled by mediators secreted by sympathetic and vascular endothelium, and circulating hormones

Smooth muscle contraction: Initiated by ↑ [Ca²⁺] → myosin Light chain kinase phosphorylates myosin

	Stimulation (Vasoconstriction)	Relaxation (Vasodilation)	α ₁ - Blockers
MOA	<ol style="list-style-type: none"> Release intracellular Ca²⁺ Depolarize membrane, open voltage gates Ca²⁺ channels → Ca²⁺ influx 	<ol style="list-style-type: none"> Directly Inhibit Ca²⁺ entry through voltage gated channels (Eg: nifedipine) Indirectly by hyperpolarizing membrane (potassium channel activators), preventing opening of voltage gated Ca²⁺ entry ↑ Intracellular cAMP inactivates myosin light-chain kinase and aids Ca²⁺ efflux ↑ intracellular cGMP opposes induced increases in [Ca²⁺] 	α_1 found in peripheral vessels . <ul style="list-style-type: none"> - Stim = Vasoconstriction + ↑ SVR Selective blockers → vasodilation. Effective when combined with β-blockers or diuretics
Drugs	Dopamine Dobutamine	Ca-channel blockers: <ul style="list-style-type: none"> - Nifedipine - Diltiazem - Verapamil - Amiodipine Nitrates: <ul style="list-style-type: none"> - Glyceryl trinitrate - Isosorbide dinitrate - Nitroglycerine 	Prazosin Terazosin Doxazosin
Side Effects		Nitroglycerine <ul style="list-style-type: none"> - Acute circulatory failure - Orthostatic hypotension - Myocardial insufficiency (from obstruction) - ↑ Intracranial pressure - ↑ Intraocular pressure - Severe anemia 	Orthostatic hypotension CNS depression (↓ alertness)
Drug Interactions			NSAIDs - ↓ antihypertensive effects Epinephrine - ↑ antihypertensive effect & reflex tachycardia -> Epinephrine starts binding to β instead of α-> more epi release



Renin- Angiotensin System



1. Renin released in response to sympathetic activation, and ↓ BP
2. Renin activates angiotensinogen into Angiotensin I
3. ACE converts Angiotensin I into Angiotensin II

AT ₁ Receptor	
Generalized vasoconstriction Noradrenaline release (↑ Sympathetic effects) Proximal tubule resorption of H ₂ O Cardiac and vascular cell growth	
AT ₂ Receptor (not important for BP regulation)	
Expressed in fetus and specific regions in adult brain Involved in growth development	

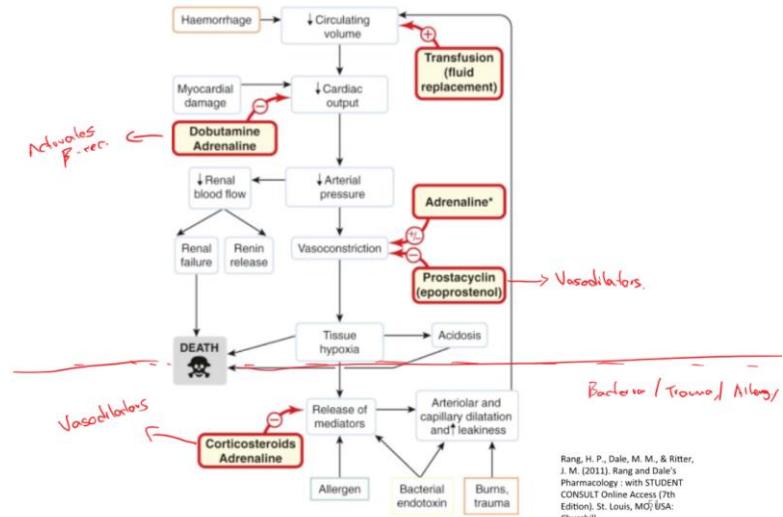
	ACE Inhibitors	Angiotensin Receptor Antagonist (ARB)	Aldosterone Antagonists
MOA	Bind to the active site of ACE and prevent the binding and subsequent activation of angiotensin II <ul style="list-style-type: none"> - ↓ Sympathetic effects - ↓ Na⁺ + H₂O retention - ↑ Vasodilation - ↑ Bradykinin (causes cough) 	Competitively inhibit angiotensin II at receptor sites (AT ₁ and AT ₂) <ul style="list-style-type: none"> - Less effective than ACE Inhibitors though 	Aldosterone increases resorption of water and Na in distal tubules and collecting ducts ACE inhibitors ↓ AT II -> ↓ Aldosterone
Drugs	Suffix -pril Captopril Enalapril Lisinopril	Suffix -sartan Losartan Valsartan Candesartan	Spironolactone (K ⁺ sparing diuretic also) Suffix - enone Eplerenone Canrenone Finerenone
Side Effects	Dry cough Renal damage (with pre-existing renal disease) Contra-indicated in pregnancy (fetal kidney damage)	<ul style="list-style-type: none"> - Reduces aldosterone levels (stimulated by ATII) -> Potassium retention - No Bradykinin release -> no cough 	-

Hypotension

= Systolic <90mm Hg, Diastolic <60mm Hg

Causes:

- Hemorrhage, burns, bacterial infection, acute myocardial damage, dehydration



	Dobutamine	Dopamine
MOA	<p>Direct interaction with α and β-adrenergic receptors</p> <ul style="list-style-type: none"> - Selective for β_1-adrenergic receptors <p>-'ve isomer = α_1 agonist -> marked pressor responses +'ve isomer = α_1 antagonist -> blocks effects of -'ve isomer Both isomers = β adrenergic agonist (+'ve more potent) -> inotropic effect to increase heart contractility and CO</p> <p>Mild β_2 agonist activity -> useful as a vasodilator</p> <p>- Doesn't act on dopamine receptors to induce the release of norepinephrine -> less prone to induce hypertension than is dopamine.</p>	<p>Metabolic precursor to NE and E</p> <p>[Low] - activate adenylyl cyclase -> ↑ cAMP</p> <ul style="list-style-type: none"> - ↑ glom. Filter rate, renal blood flow, Na^+ excretion <p>[High] - +'ve inotropic effect on myocardium (β_1 response)</p> <ul style="list-style-type: none"> - NE release -> ↑ HR, ↑ Systolic BP (min diastolic BP effects) - Activates vascular α_1 -> Vasoconstriction (↑ BP)

Ischemic Heart Disease

= Result of progressive myocardial ischemia -> persistently ↓ Blood flow

- Linked to atherosclerosis, hyperlipidemia and hypertension

Treatment goal:

- ↓ Myocardial demand for O_2 (↓ HR, ↓ Afterload, ↓ Contractility)
 - o β -Blockers, Ca Channel Blockers, ACE Inhibitors etc
- ↑ Myocardial O_2 Supply (Vasodilation, ↓ HR -> ↑ Diastolic:Systolic ratio improves time for coronary perfusion)

Factors affecting blood flow:

Vascular control by metabolites	$\downarrow \text{P}_{\text{O}_2}$ doesn't actually affect coronary vessels in vivo <ul style="list-style-type: none"> - Hypoxic -> Lactic acid -> $\downarrow \text{pH}$ -> Vasodilation - ATP -> ADP -> adenosine -> vasodilation
Neural and Humoral Control	Lots of sympathetic innervation, but little effects on coronary circulations <ul style="list-style-type: none"> - Large coronary vessels have α-adrenoceptors -> Vasoconstriction - Small vessels have β-adrenoceptors -> Vasodilation - Both systems cancel each other out = no significant effect

Angina:

Tx strategies

- ↑ O₂ delivery, ↓ O₂ requirement
- Nitrates, Ca²⁺ channel blockers, β-blockers

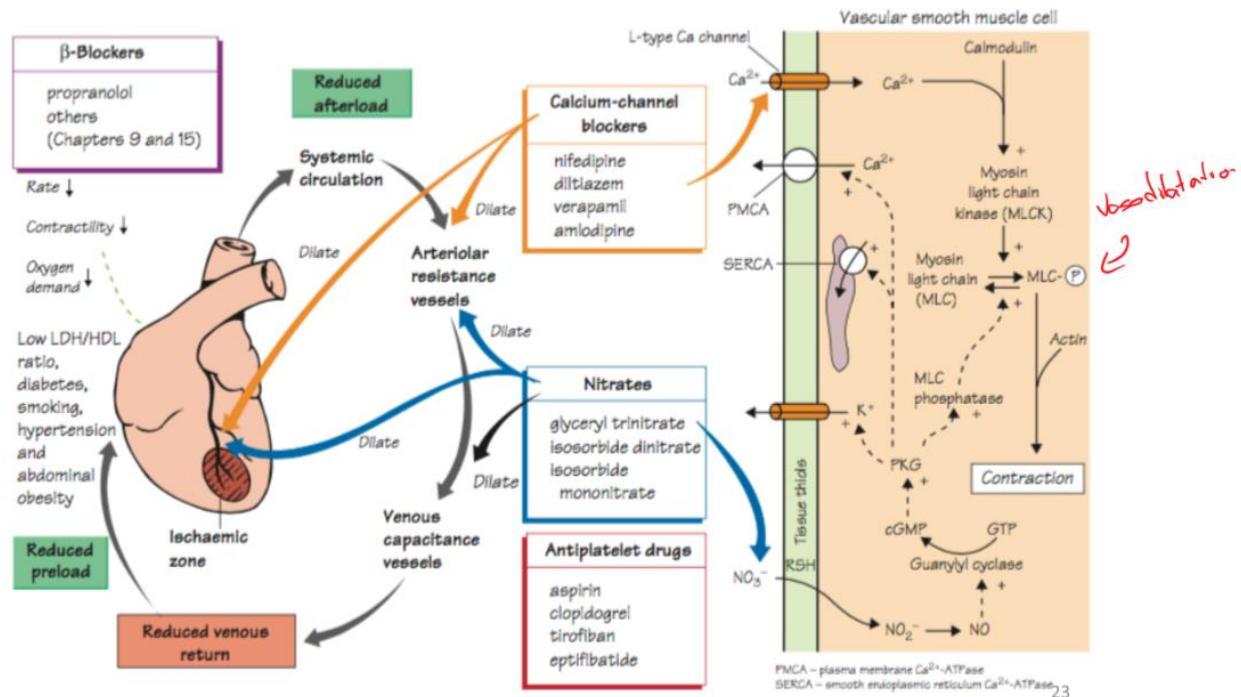
Type	Characteristics
Stable	Pain on exertion Tx: <ul style="list-style-type: none"> - Alter cardiac work load - Organic nitrates, β-blockers, Ca²⁺ antagonists
Unstable	Pain occurring with less exercise (eventually at rest) <ul style="list-style-type: none"> - Thrombus from ruptured atheroma w/o complete occlusion of the vessel. ↑ risk of infarction Tx: <ul style="list-style-type: none"> - ↓ risk of infarction = anticoagulants/Aspirin
Variant	At rest b/c of coronary artery spasm, associated with atheroma Tx: <ul style="list-style-type: none"> - Coronary vasodilation -> Nitrates, Ca²⁺ antagonists

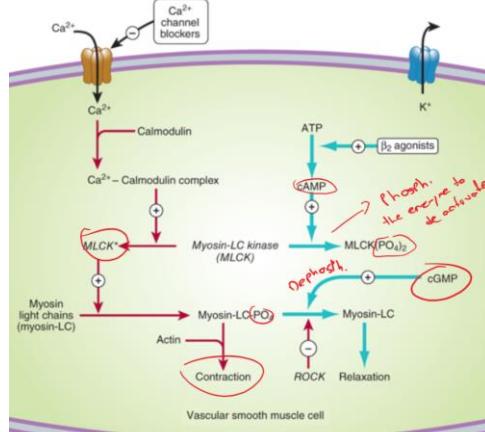
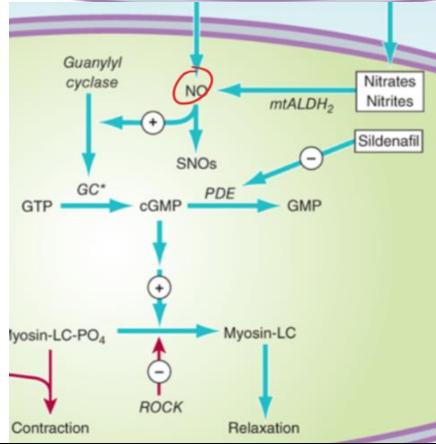
Controlling Vascular Tone

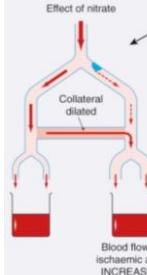
↑ cGMP	Facilitates dephos. of myosin light chains -> prevents binding of myosin to actin -> prevents contraction <ul style="list-style-type: none"> - Nitric Oxide (From Nitroglycerin)
Decrease intracellular Ca ²⁺	↓ Influx -> ↓ intracellular [Ca ²⁺] -> decrease contraction <ul style="list-style-type: none"> - Ca²⁺ channel blockers - β-blockers
Prevent depol. Of muscle cell	↑ Permeability of K ⁺ channels <ul style="list-style-type: none"> - Minoxidil
↑ cAMP in smooth muscle cell	↑ rate of inactivation of myosin light chain kinase -> ↓ rate of phosphor. Of MLC <ul style="list-style-type: none"> - β₂- agonist - Fenoldopam

	Nitrates Alone	Beta Blockers or Calcium Channel Blockers Alone	Combined Nitrates and β Blockers or Calcium Channel Blockers
Heart rate	Reflex increase	Decrease	Decrease
Arterial pressure	Decrease	Decrease	Decrease
End-diastolic pressure	Decrease	Increase	Decrease
Contractility	Reflex increase	Decrease	No change or decrease
Ejection time	Reflex decrease	Increase	No change
Net myocardial oxygen requirement	Decrease	Decrease	Decrease

	Nitrates	β -Blockers	Ca^{2+} channel blockers
MOA	Nitroglycerin forms free radical Nitric oxide <ul style="list-style-type: none"> - NO activates guanylate cyclase = \uparrow cGMP - cGMP dephospho. Myosin light chain = Relaxation 	Block β -Adrenergic receptors	Block the influx of Ca^{2+} in SMC and CMC
Effects	Vasodilation of large veins = \downarrow central venous pressure \downarrow Preload \downarrow Afterload Dilation of coronary arteries, \uparrow collateral flow to ischemic regions	\downarrow HR \downarrow Contractility \downarrow O ₂ Consumption \downarrow Peripheral resistance and central venous pressure <ul style="list-style-type: none"> - \downarrow coronary blood flow 	Vasodilation on resistance vessels <ul style="list-style-type: none"> - \downarrow Afterload Vasodilation of coronary vessels Heart: Antiarrhythmic \downarrow Contractility Impair AV conduction
Adverse Effects	Facial flushing Headaches Hypotension Fainting Reflex tachycardia	Risk of acute Hypertensive episodes if used with Epinephrine	Headache Constipation Ankle Edema Risk of cardiac failure or heart block Gingival enlargement May inhibit the metabolism of benzodiazepines (longer duration)
Drugs	Short acting: Nitroglycerin Long Acting: Isosorbide mononitrate Isosorbide dinitrate	Propranolol Atenolol Metoprolol	Nifedipine

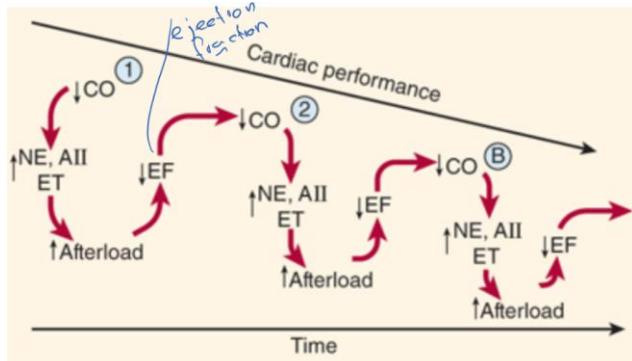


Drugs for Angina		
Strategy	Reason	MOA
↓ intracellular Ca ²⁺	↓ Ca ²⁺ influx -> prevent contraction, and automatic Ca ²⁺ nodal depolarization - ↓ contractility - ↓ HR	
↑ cAMP (β2 agonists)	↑ rate of inactivation of myosin light chain kinase (MLCK) -> ↓ phosph. of myosin -> ↓ actin binding	
Prevent depolarization	↑ permeability of K ⁺ channels	
↑ cGMP (Nitroglycerine)	Nitrates -> ↑ cGMP aids in dephosph. Of myosin light chain -> prevents binding of myosin to actin - ↓ contractility - ↓ HR	

Family	MOA	Effects	Adverse
Nitrates (Nitroglycerin) (isosorbide mononitrate)	Nitroglycerin -> Free radical NO -> activates guanylate cyclase -> ↑ cGMP -> dephosph. MLC	Dilation of large veins - ↓ preload Dilates coronary arteries - ↑ collateral flow to ischemic regions	 Effect of nitrate Collateral dilated Blood flow to ischemic area INCREASED
β-Blocker (Propranolol) (Atenolol) (Metoprolol)	Block β-receptors	Negative inotropic (↓ HR, ↓ contractility) - ↓ O ₂ demands ↑ peripheral resistance & Central Venous pressure -> ↓ coronary blood flow	Xerostomia Lichenoid Like Rxn
Ca²⁺ Blocker (Nifedipine)	Prevent opening of L-type Voltage gated Ca ²⁺ Channel	Vasodilation on resistance vessels - ↓ Afterload Vasodilation of coronary vessels Antiarrhythmic - ↓ contractility - ↓ AV node conduction Anti-Hypertensive	Headache Constipation Ankle Edema Gingival Hyperplasia

Congestive Heart Failure

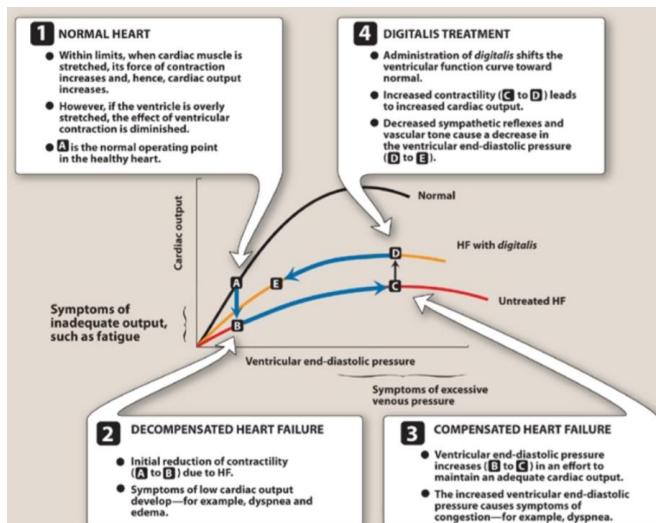
= CO insufficient to adequately perfuse tissues -> Right and Left heart failure -> Pulmonary congestion, Peripheral Edema



1. ↓ Cardiac Output
2. ↑ Sympathetic and Renin = Norepineph. Angiotensin II, and Endothelin
3. ↑ Afterload
4. ↓ Ejection Fraction
5. ↓ Cardiac Output

Damn...What a shitty cycle!

Digitalis Treatment



In heart failure = ↓ CO -> edema and dyspnea
Ventricular end diastolic pressure ↑ to boost CO

- Leads to symptoms of congestion (dyspnea)

Digitalis -> increases contractility -> ↑ CO

- ↓ sympathetic reflexes and vasoconstriction to bring end diastolic pressure back to normal.

	Digoxin	Digitalis
Family	Cardiac Glycosides	Cardiac Glycosides
MOA	Inhibit Na^+/K^+ ATPase -> ↑ Intracellular Na^+ and Ca^{2+} (Via Na^+ Ca^{2+} exchangers)	↑ Contractility -> ↑ Cardiac Output ↓ sympathetic reflex and vascular tone
Effects	Increase contractility force without an ↑ of HR Decrease O_2 consumption	↓ Vascular end diastolic pressure
Dental Implications	Short appointments, ↑ Stress Avoid vasoconstrictors (Epinephrine) Avoid muscarinic drugs (↓ bradycardia) Avoid Antimuscarinic drugs (Antagonist effects) Avoid broad spectrum antibiotics (increased GI absorption of digoxin) Avoid Epinephrine and Macrolide derived antibiotics (↑ Serum digoxin levels, and ↑ of digoxin toxicity)	
Other Effects	Purkinje System: AP shortened, ↓ membrane resistance ↑ K^+ conduction Shortened Atrial and Ventricular refractory period - Causes Ventricular fib at [high] AV Node: ↑ Vagal activity (muscarinic effects in heart) ↓ HR, ↓ AV conduction, prolongs AV refractory period - Bradycardia	

Congestive Heart Failure Drugs		
Family of Drugs	Drugs	Effects
Cardiac Glycosides	Digitalis Digoxin	↑ Contractility ↓ Diastolic Pressure ↓ AP time and refractory period ↓ HR, slow AV conductance
Thiazide Diuretics (↑ Fluid Excretion)	Hydrochlorothiazide	↑ H ₂ O excretion -> ↓ BP
Nitrates	Nitric Oxide (Nitroglycerin)	↓ Preload
Vasodilators Ca²⁺ channel Blockers α₁-blocker	Nifedipine, Amlodipine, Nicardipine Hydralazine , Prazosin	↓ Contractility ↓ HR ↓ SVR
ACE Inhibitors	Captopril, Enalapril	↓ Arterial and venous resistance Blocks formation of AT II ↓ Preload and ↓ Afterload
Sympathomimetic Drugs	Epinephrine	Activates cardiac β ₁ - Receptors - ↑ HR and O ₂ consumption

Cardiac Arrhythmias

- Affected by Acetylcholine (Ach) and Norepinephrine (NE)

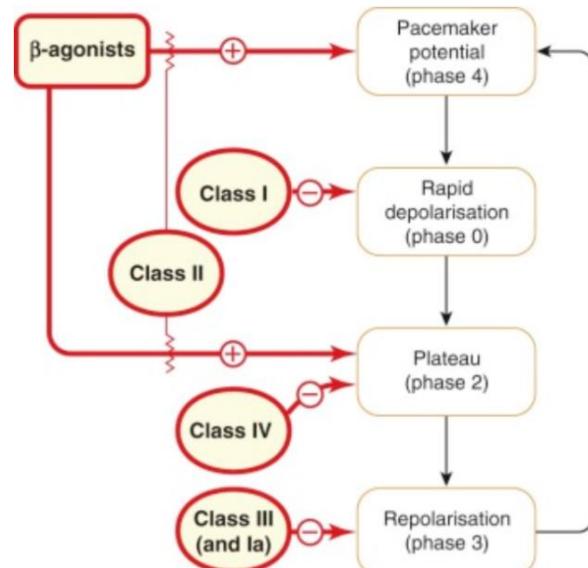
Agent	Effects
Acetylcholine	Parasympathetic (Vagal fibres) -> Release Ach -> Muscarinic receptor -> ↑ K ⁺ conductance = hyperpolarization (↓ HR)
Atropine	Blocks parasympathetic muscarinic receptor during sinus bradycardia -> ↑ HR
Norepinephrine	Sympathetic Fibres -> NE to pacemaker cells -> ↓ K ⁺ conductance = chronotropic effect (↑ HR) ↑ Ca ²⁺ conductance -> ↑ Contraction force (ionotropic effect)

Antiarrhythmic Drugs

Class		MOA	Effects	Drugs	
IA	Na⁺ Channel Blocker	Blocks open Na ⁺ Channels, reduces phase 3 K ⁺ current	Prolongs AP	Quinidine Procainamide Disopyramide	
IB		Blocks inactivated Na ⁺ Channels	Shortens AP	LA Lidocaine Mexiletine Phenytoin	
IC			Slows Conduction (No AP effects)	Flecainide	
II	β-Blocker	↓ Sympathetic action on heart	Slows AV Conduction	Propranolol Metaproterenol	
III	K⁺ Channel Blocker	Slows repolarization (phase 3) and prolong AP and Refractory Period in cardiac tissues	Slows Repol.	Bretylium (K ⁺ channel blocker) Amiodarone (Na, K, Ca channel blocker) Sotalol (K channel blocker & β-Blocker)	
IV	Ca²⁺ Channel Blocker	Shortens plateau phase	Causes gingival Hyperplasia ↓ Contraction force	Verapamil Diltiazem	
V	Random Mechs	Atropine (for sinus bradycardia) Epinephrine (For cardiac arrest in Asystole) Isoproterenol (For bradycardia by heart block) Adenosine (For supraventricular tachycardia b/c hyperkalemia)			

Dental Implications of Antiarrhythmic Drugs:

- Orthostatic hypotension
- Hypotensive syncope
- Stress induced arrhythmia
- Epinephrine use

**Oral Manifestations:**

Amiodarone – Pigmentation of skin and mucous membrane

Ca²⁺ channel blockers – Gingival hyperplasia

β-Blockers – Changes in salivary proteins

Lipid Lowering Drugs

Lipoproteins = Transport lipids and cholesterol → essential for life

Body produces more endogenous cholesterol than what we take in from diet

HDL	Good cholesterol <ul style="list-style-type: none"> - Transports lipids and triglycerides from periphery to liver 	
LDL	Bad Cholesterol <ul style="list-style-type: none"> - Transports lipids and triglycerides from liver and deposits in periphery (blood vessel walls etc) 	
VLDL	Bad Cholesterol <ul style="list-style-type: none"> - Precursor to LDL 	
Hyperlipidemia	↑ LDL & VLDL	
Atherosclerosis	Modifiable Risk Factors <ul style="list-style-type: none"> ↓ HDL, ↑ LDL (all about the ratio, not absolute #) - Smoking - Physical inactivity - Hypertension Pathophysiology <ul style="list-style-type: none"> - Chronic inflammation (injury response) - Endothelial dysfunction and damage - LDL infiltrates and oxidizes - Macrophages engulf LDL-> foam cells - Smooth muscle migration and collagen deposition -> forms fibrous cap Tx Strategies <ul style="list-style-type: none"> ↓ Cholesterol and LDL in diet ↓ Smoking, and alcohol <ul style="list-style-type: none"> - Alcohol ↑ Triglycerides and VLDL - Smoking -> toxins in blood damage endothelium Drugs 	Non Modifiable Factors <ul style="list-style-type: none"> - Age - Genetics - Gender - Diabetes mellitus

Family	Drugs	MOA	Effects	Side Effects
HMG-CoA Reductase Inhibitors (-Statins)	Lovastatin Simvastatin Pravastatin Atorvastatin Rosuvastatin	Specific, reversible, competitive inhibitors of rate limiting enzyme involved in endogenous cholesterol synthesis in Liver -> ↓ intracellular cholesterol = ↑ LDL receptor synth. Liver takes in more LDL to produce more cholesterol -> LDL clearance from periphery	↓ LDL Levels ↑ Endothelial function ↓ Platelet aggregation ↑ fibrinolysis Stabilize atherosclerotic plaque	May be toxic if liver disease present
Bile Acid Binding Resins	Cholestyramine Colestipol	Bind bile acids (and similar steroids) in intestine - Prevents reabsorption and recirculation - Excreted in feces Liver absorbs more LDL to produce more cholesterol for Bile	↓ absorption of exogenous cholesterol ↑ Metabolism of endogenous cholesterol into Bile Acids ↑ LDL receptors on liver cells -> to ↑ cholesterol and ↑ bile acids	Steatorrhea Bloating Constipation Impaired vitamin and drug absorption (Vit. K, Folic acid, Thiazide diuretics, warfarin)
Cholesterol Transport Inhibitor	Ezetimibe	Prevents absorption of dietary cholesterol and cholesterol excreted in bile Inhibits GI mediated transport of cholesterol	↑ LDL receptors on liver cells -> to ↑ cholesterol and ↑ bile acids	
Fibrates	Gemfibrozil Fenofibrate Clofibrate	Stimulate lipoprotein lipase (↑ LPL gene transcription) - ↑ clearance of triglyceride rich lipoproteins	↓ VLDL ↓ Triglyceride ↑ HDL (moderately)	
VLDL Synthesis inhibitor	Niacin	↓ ApoA1 catabolism	↓ VLDL Secretion from liver ↑ VLDL clearance ↑ HDL (major) ↓ Plasma triglycerides	Flushing - Limited with aspirin 30 mins prior to dose

Autacoids

Histamines

= Stored in mast cells, enterochromaffin cells, and neurons

- No therapeutic applications. Only receptor antagonists are useful

Histamine Receptors	Location
H ₁ Receptor	Smooth Muscle, Endothelium (blood vessels), Brain
H ₂ Receptor	Gastric mucosa, cardiac muscle, Mast cells, Brain
Effects	
Nervous System	Stimulant of sensory nerve endings -> Pain and Itching Appetite and satiety (H ₁ and H ₃ receptors)
Cardiovascular	Vasodilation ↑ capillary permeability ↓ BP (systolic and diastolic) ↑ HR (to compensate for ↓ BP)
Bronchiolar Smooth Muscle	Bronchoconstriction (H ₁ receptor)
GI Tract smooth muscle	Smooth muscle contraction (H ₁ Receptor)
Secretory Tissue	Stimulates gastric acid secretion (H ₂)
Metabolic	Uncertain in humans
Triple Response	Reddening -> Local vasodilation Edema -> ↑ permeability of post capillary venules Flare -> ↑ pain and itch in areas of reddening

Drugs:

Family	Drugs	MOA	Clinical Use
H₁ Antagonists	1st Generation Diphenhydramine (Benadryl) Hydroxyzine (Atarax) Promethazine (Phenergan)	Crosses blood brain barrier -> drowsy	Anti-allergy Anti-motion sickness Anti-morning sickness (pregnancy)
	2nd Generation Fexofenadine (Allegra) Loratadine (Claritin) Desloratadine (Clarinex) Cetirizine (Zyrtec)	Doesn't cross blood brain barrier -> non-drowsy	
H₂ Antagonists	Cimetidine Ranitidine Famotidine Nizatidine	Block H ₂ Receptors	GERD Stress Ulcers ↓ Nocturnal acid ↑ Healing

Eicosanoids

= Phospholipid derived mediator produced from arachidonic acid (AA)

- AA converted via **COX**, Lipoxygenase, P450 Epoxygenase, or Isoeicosanoid pathway

Prostanoids

AA -> COX oxidizes (COX-1, Constitutive enzyme; COX-2 induced by inflammation) -> **Prostaglandin** and **Thromboxane** production

PGE₂	Predominates in areas of acute inflammation	Works synergistically to ↑ effects of Histamine and Bradykinin
PGI₂	Vasodilators	
PGD₂	Released by Mast cells Vasodilators	

Vasoactive Peptides

Bradykinin	
Produces 4 classic symptoms of inflammation	
1. Redness	
2. Heat	
3. Swelling	
4. Pain	
Receptors	B ₁ – Inflammatory role B ₂ – Constitutive, in normal cells
Effects	Vasodilation ↑ Permeability Pain sensation Stimulate fluid secretion in airways and GI tract Contraction of intestinal and uterine smooth muscle
Substance P	
Tachykinin family Acts on mast cells -> histamine release	
Effects	Smooth Muscle contraction (venous, intestinal, bronchial) Neural activation Mucus secretion Vasodilation -> mediated by Nitric Oxide from endothelium

Infective Endocarditis Prophylaxis

Purpose	Prevent metastatic bacteremia and post surgical infections
Pharma Considerations	Ab must be at high concentration at target site BEFORE onset of bacteremia/surgery Loading dose must be used (2-4x maintenance dose)
Adverse Effects	Allergy and Toxicity to Ab Superinfections from resistant bacteria <ul style="list-style-type: none"> - Selection of Ab-resistant microbes - Induction of resistance gene transfer
Contraindications	At risk group cannot be defined to prevent overuse or abuse of Ab Efficacy is unreliable Bacteremia to be prevented rarely causes infection Prophylaxis directed at too many potential microbes rather than single one

Table 3 American Heart Association regimens for infective endocarditis prophylaxis²

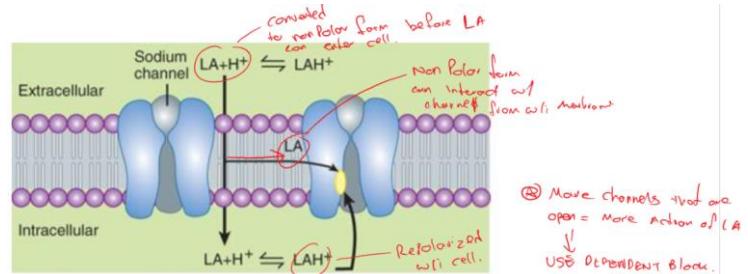
Patient group	Antibiotic	Route	Dose		Timing before procedure
			Adults	Children	
Standard general prophylaxis for patients at risk	Amoxicillin	PO	2 g	50 mg/kg	1 hour
Unable to take oral medication	Ampicillin	IV or IM	2 g	50 mg/kg	Within 30 minutes
Allergic to penicillin/amoxicillin/ampicillin	Clindamycin	PO	600 mg	20 mg/kg	1 hour
	Cephalexin or cephadroxil*	PO	2 g	50 mg/kg	1 hour
	Azithromycin or clarithromycin	PO	500 mg	15 mg/kg	1 hour
Allergic to penicillin/amoxicillin/ampicillin and unable to take oral medications	Clindamycin	IV	600 mg	20 mg/kg	Within 30 minutes
	Cefazolin	IV	1 g	25 mg/kg	Within 30 minutes

Note: IV = intravenous; PO = oral.

*Cephalosporins should not be used with penicillin or ampicillin in those with a history of anaphylaxis, angioedema or urticaria.

Local Anaesthetics

- Reversible block of nerve fiber conduction
 - o Weak Bases -> -> penetrate nerve non-ionized form -> once inside axon some ionized molecules form and block Na⁺ channels -> prevent AP generation



Use Dependant

- Degree of block is related to rate of nerve stimulation (LA only blocks Inactivated state of Na channels)
- LA molecules gain access to channel more readily when it is open (but inactivated) as opposed to resting and closed.
- LA needs to affect a sufficient number of nodes in an axon to block the impulse

Conduction is blocked in the following order:

Small myelinated axon -> non-myelinated axons -> large myelinated axons

- Nociceptive (pain) and sympathetic transmission blocked first

Vasopressors

Added to ↑ duration of pulpal anaesthesia, enhance depth of anaesthesia and ↓ concentration of LA in the blood (↓ systemic toxicity)

- By vasoconstricting. ↑ time LA remains in tissue, ↓ redistribution of drug to surroundings and ↓ [systemic]

Type of Nerve Fibre	Information	Myelin Sheath?	Diameter (micrometers)	Conduction Speed (m/s)
A- α	Proprioception	YES	13-20	80-120
A- β	Touch	YES	6-12	35-90
A- δ	Pain (mechanical and thermal)	YES	1-5	5-40
C	Pain (mechanical, thermal, and chemical)	NO	0.2-1.5	0.5-2

Transient Pain Signal
Slow Pain

13

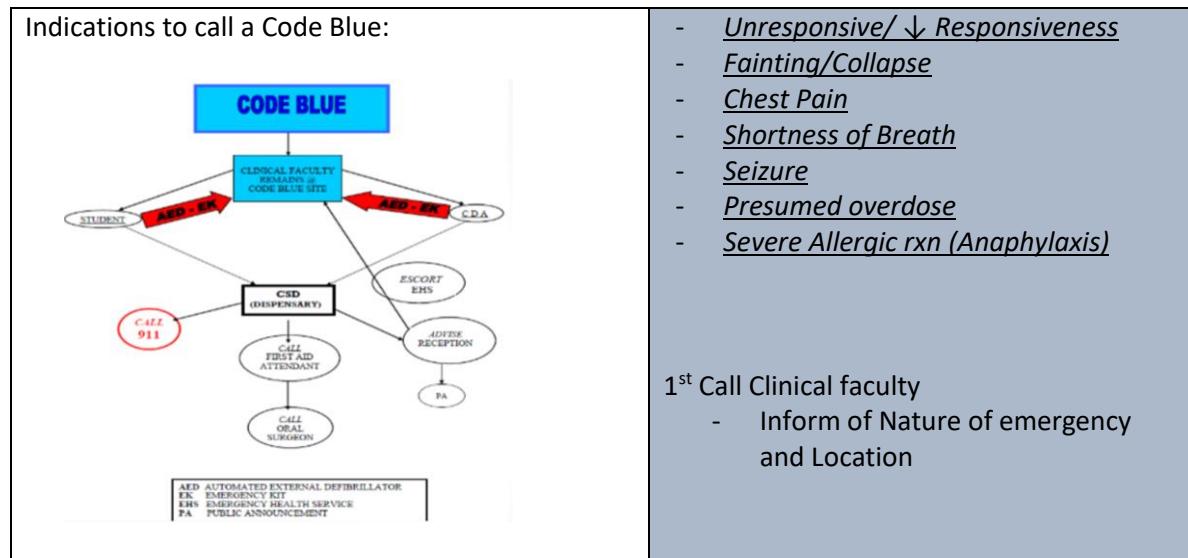
Vasopressors	Epinephrine/Norepinephrine Levonordefrin Phenylephrine Felypressin
MOA	Vasoconstriction by activating adrenergic receptors on the myocytes of blood vessels <ul style="list-style-type: none"> - Mostly α_1 effects -> (G protein coupled -> Ca channels open -> activates calmodulin MLCK - . Contraction)

Drug	α_1	α_2	β_1	β_2
Epinephrine	+++	+++	+++	+++
Norepinephrine	++	++	++	+
Levonordefrin	+	++	++	+

Medical Emergencies

1. Stop Procedure
2. Activate EMS (Code blue etc)
3. O₂ administration
4. Change chair position for patient comfort

5. Life support (C-circulation, A-Airway, B-Breathing)
6. Emergency Drugs



Equipment	Use
Epi-pen	Anaphylaxis
Benadryl	Mild Allergy (antihistamine)
Ventolin Inhaler (Salbutamol)	Acute Asthma
Glucagon	Hypoglycemia
Glucose tablets	Hypoglycemia
Nitroglycerin spray	Anginal chest pain
Aspirin	Suspected MI
Bag Valve Mask	Ventilation
Oral Airway	Ventilation
Stethoscope	BP measurement/chest auscultation
BP cuff	BP Measurement
Portable O ₂	Emergency O ₂

Essential Drugs

	Epinephrine	Oxygen
Dosages	0.5mg IM, Subcutaneous 0.3mg or 1mg IV	Nasal cannulae – 25-45% Simple face mask – 40-60 Non-rebreathing face mask – 90-100% Mouth to mouth – 16% Bag-valve mask with air – 21% Bag-valve with O ₂ – 75-95%
Indication	Anaphylaxis (0.5mg IM/subcutaneous or 0.3 IV) Asthma (0.5mg IM, 0.3mg IV) Cardiac Arrest (1mg IV)	Every emergency except Hyperventilation
Pharmacology	β -1 Agonist -> +ve Inotrope (↑ Contractility) +ve Chronotrope (↑ HR) β -2 Agonist -> Dilates bronchioles and vasculature α1 Agonist -> Vasoconstriction **high [Epi] = Vasoconstriction b/c ↑ # of α1 receptors Low [Epi] = Vasodilation b/c ↑ sensitivity of β receptors	

Syncope

= transient self-limited loss of consciousness due to ↓ blood flow to brain

- ↓ Pump function of heart
- ↓ Vascular tone -> ↓ BP delivery to brain
- ↓ CO in vessels

Cardiac Syncope

- Abrupt impedance of blood flow leading to systemic hypoperfusion and syncope

Causes	<ul style="list-style-type: none"> - Vasovagal -> fear, emotional stress, pain - Situational -> micturition, defecation, deglutition, cough, carotid sinus syncope - Orthostatic hypotension - Cardiac failure - Neurological & Psychiatric disorders - Medications
Pre-syncope Symptoms	<ul style="list-style-type: none"> - Dizziness - Lightheaded - Pallor - Palpitations - Nausea - Diaphoresis - Altered vision - Bradycardia - Hypotension - Pupil dilation - Peripheral coldness - Tachypnea
Influencing Factors	<ul style="list-style-type: none"> - Fasting - ↓ Food and Fluids - ↓ BP - Physical exercise exceeding energy reserves - Emotional stress - ↓ Sleep - Orthostatic hypotension

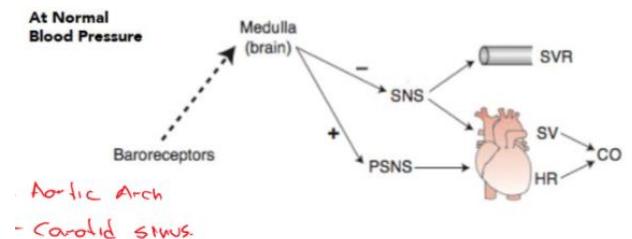
Coronary artery disease

- Aortic Stenosis, Mitral Stenosis
- Arrhythmia
- Hypertrophic cardiomyopathy
- Cardiac Tumor (Myxoma)
- Pulmonary Hypertension
- Pulmonary Stenosis
- Pulmonary Embolus

Vasovagal Syncope

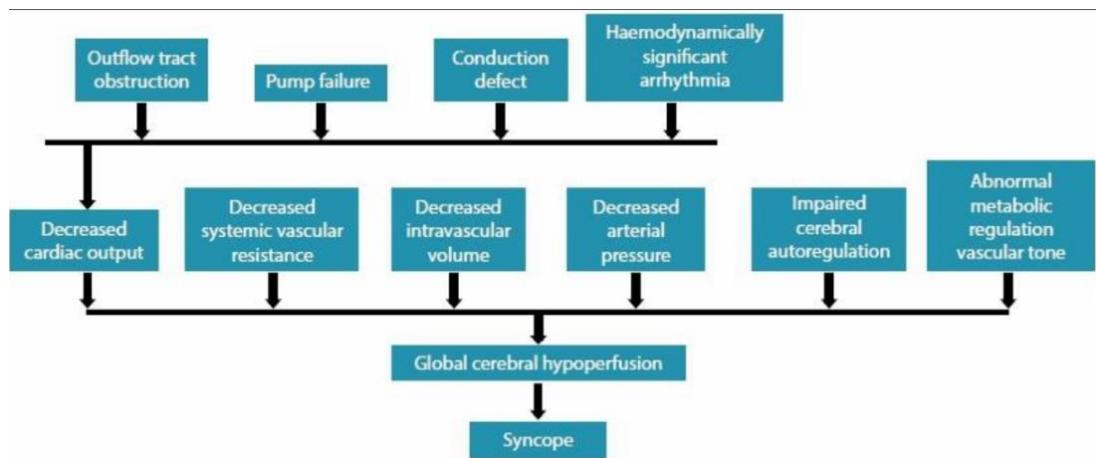
↑ Parasympathetic activity, ↓ Sympathetic activity -> ↓ HR,
 ↓ Contractility, ↑ Vasodilation

Sympathetic inhibition and PS stimulation always happening



Management	<ol style="list-style-type: none"> 1. Trendelenburg Position (supine with feet 15-30 degrees higher than head) 2. Assess consciousness 3. Establish airway 4. 100% O₂ 5. Monitor vitals 6. Cold Compress
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Pathophysiology



Cardiovascular Emergencies

Presents with:

- Severe Hypertension
- Chest Pain
- Dysrhythmia
- Cardiopulmonary Arrest

Cardiac Arrest

6 H's	6 T's
<ul style="list-style-type: none"> - Hypovolemia - Hypoxia - Hydrogen Ion (Acidosis) - Hypo-Hyperkalemia - Hypo-Hyperglycemia - Hypothermia 	<ul style="list-style-type: none"> - Tension Pneumothorax - Tamponade - Toxins - Trauma - Thrombosis

All lead either:

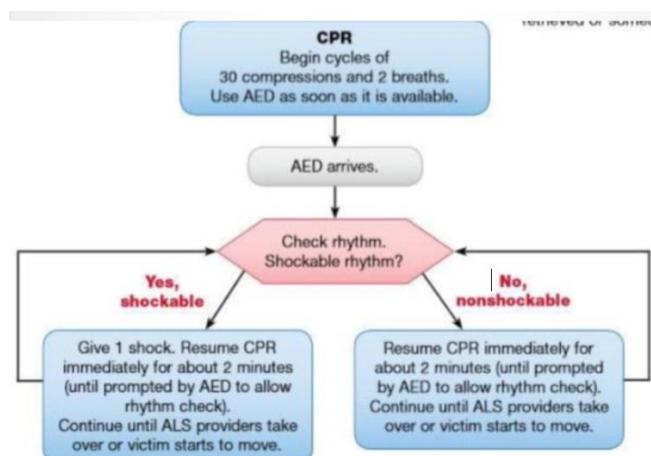
Shockable

- Ventricular Fibrillation
- Pulseless Ventricular Tachycardia

Not-Shockable

- Pulseless Electrical Activity
→ SA firing normally, but the ventricles (pump) are not pumping blood out somehow
- Asystole (NO AED)
→ No Electrical or Pump function

Tx: Pulseless VT or VF	<ol style="list-style-type: none"> 1. Chest compressions right away 2. Open airway and place airway device -> confirm O₂ and ventilation 3. Assess the 6H's and 6T's 4. Shock with AED once and resume CPR 5. Consider Epinephrine 1mg IV every 3-5 mins <p>If:</p> <p>Downtime > 4mins -> 5 cycles of CPR (30 compressions, 2 breathes), access if shockable rhythm.</p> <p>Downtime < 4mins -> 1 shock immediately, then compressions</p>
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Physiology of Blood Pressure

$$\text{BP} = \text{CO} \times \text{SVR}$$

SVR:

Vasodilation	Vasoconstriction
↓ Resistance = ↑ Blood flow = Warm and Pink patients + bounding pulse	↑ Resistance = ↓ Blood Flow = Cool and Pale patients + weak pulse

Cardiac Development

- Development begins in 3rd week -> heart is pumping by 4th week!
- Heart first forms “Straight heart tube” consisting of:
 - o **Atrial Pole** -> Atrium, Ventricle, Bulbus Cordis, Truncus Arteriosis
 - o **Venous Pole** -> right and left horns of sinus venosus
- Straight heart tube built of 3 layers
 1. Outer myocardial layer
 2. Inner endocardium
 3. Cardiac Jelly in the middle of the myocardium and endocardium

Heart Looping (week 4)

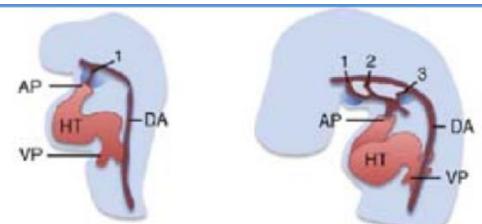
- Straight heart tube, twists and loops to form our heart shape with the different zones.

3 Heart Fields

Fields	Embryonic Structure	Final Structure	
1st Heart Field	Ventricle	Majority of L Ventricle	
2nd Heart Field	Bulbus Cordis	R Ventricle, Caudal parts of Atria Outflow Tract (Early Pulmonary Trunk)	<p>Sequence of Events in Looping</p> <p>Straight Heart Tube C-Shaped Loop S-Shaped Loop</p> <p>Blue = Bulbus Cordis Green = Ventricle Yellow = Atrium Red = Sinus Venosus</p> <p>Legend:</p> <ul style="list-style-type: none"> First heart field (Green) Caudal heart field (Red) Second heart field (Blue) Embryonic AVC (Yellow)
Caudal Heart Field	Caudal Heart Field	Epicardium and Aorta	
Embryonic AVC	Atrium	AV conduction fibers	

Pharyngeal Arches – Week 4

Initially Atrial pole is at 1st arch -> looping and rearrangement -> Atrial pole moves to 2nd and 3rd arches

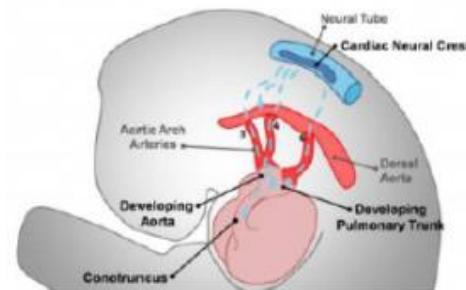


Cardiac Mesoderm		
Arch	Facial Muscle	Heart Muscle
1	Muscles of Mastication	Ventricular Myocardium
2	Muscles of facial expression, Soft Palate Muscles	Outflow Tract Myocardium
Cardiac Vessels		
3	Right Common Carotid Left Common Carotid	
4	Sections just before the Subclavian Ateries	
6	Ductus Arteriosus	

Neural Crest Cells – Week 5

- NCC Migrate from Arches 3, 4, 6 to 2nd Heart Field -> form cushions in Outflow Tract
 - o Develops into Interventricular Septum & Semilunar Valves
- Give rise to musculo-connective cells of the large artery walls

Embryo – Week 5: Migration of the Cardiac Neural Crest



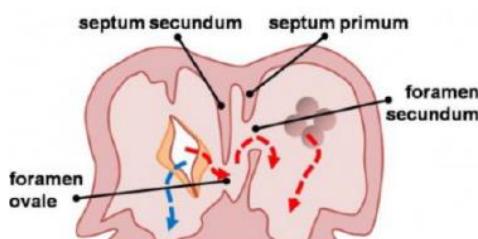
Outflow Tract

Gives rise to: Doral Aorta, Pulmonary Truck, Atrioventricular cushions (AVC)

- Doral Outflow Tract -> from **NCC** (Arches 3, 4, 6)
- Caudal Outflow tract -> from **Endocardial Mesenchyme**

Septation (following looping)

- As cushions from endocardial tissue grow larger and larger, they come together and fuse -> forming a wall
 - o Ventricular Septum is a complete fusion
 - o Atrial Septum is incomplete fusion consisting of 2 layers: **Septum Secundum** and **Septum Primum**
 - o Formation of the **Foramen Ovale**
 - Only allows **blood to flow from R->L Atrium**
 - Septum Secundum prevents Septum Primum from prolapsing -> One-way shunt.



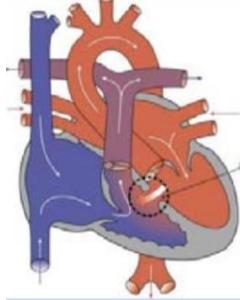
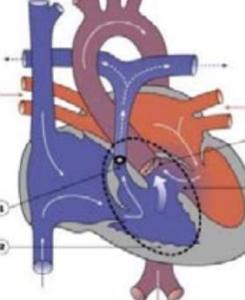
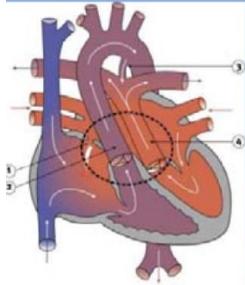
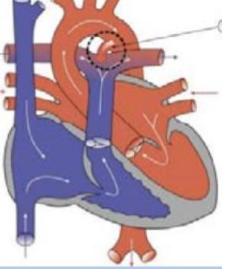
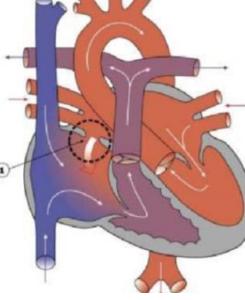
Conduction System

- Pacemaker-like myocardium gives rise to nodal regions and conduction pathways in heart
- Single myocardial stem cell can give rise to myocytes in 3 layers of the heart:
 - o Conductive cells
 - o Epicardium
 - o Myocardium

Weeks 5-8

- **Atrioventricular (Mitral + tricuspid) valves form**
 - o Valve endothelium transforms into valve mesenchymal cells
 - o BMP, TGF β and Wnt -> signals for cushion growth (similar to septation)
 - Leads to thick swollen tissue -> Congenital Valve Stenosis
 - Unknown how normal thin and functional valves form
- **Lip fusion occurs at this time**
 - o Can see correlation between cleft lip/palate and congenital heart defects

Congenital Heart Defects

	Ventricular Septal Defect	Tetralogy of Fallot	Transposition of great vessels	Patent Ductus Arteriosus	Atrial Septal Defect
					
% of CHD	25 Most Common	9-14	10-11	6-8	6-10
	<ul style="list-style-type: none"> - Possible it will close on its own - Arises from Septation issues - Causes Pulmonary Edema 	<ol style="list-style-type: none"> 1. Pulmonary Stenosis 2. Right Ventricular Hypertrophy 3. Dextroposition of Aorta 4. Ventricular Septal Defect 	<p>Looping problem</p> <ol style="list-style-type: none"> 1. ASD 2. Aorta from RV Deoxy blood through body 3. Patent Ductus Arteriosus 4. Pulmonary Trunk from LV <p>Oxy blood into lungs</p>	<ul style="list-style-type: none"> - May require surgery to tie it off - Usually closes at birth - Aortic blood shunts back into pulmonary - Left V - Hypertrophy and pulmonary congestion 	<p>Should close at birth.</p> <p>May require Dacron patch</p>

- Major side effect of [Down Syndrome](#) is [Congenital Heart Defects](#)
 - o If they have had surgery they may require prophylaxis for bacterial endocarditis
- Environmental Causes
 - o Maternal illness (Rubella, Diabetes, Influenzae)
 - o Drugs (Anticonvulsants, Ibuprofen)

PBL I – Toni Brackston

Definitions:

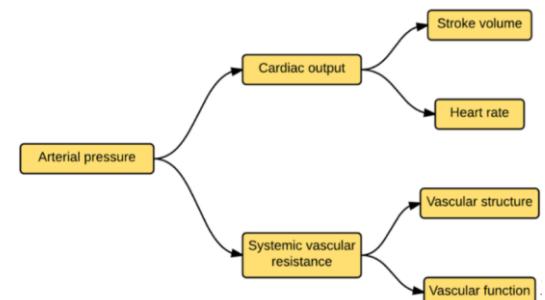
Acute Coronary Syndrome	Describes patients with unstable angina or myocardial infarction	CHD	Coronary heart disease
Acute MI	Acute myocardial infarction	CVD	Cardiovascular disease
Anoxia	Absence of O ₂ supply to tissue despite adequate perfusion	Hypoxemia	O ₂ deficiency in arterial blood
Angina Pectoris	Spasmodic, cramp-like, choking feels. Suffocating pain radiating from chest into arms and mandible	Hypoxia	O ₂ Deficiency in tissues and organs
Stable angina pectoris	Radiating chest pain due to MI. Predictable , reproducible, and responds to treatment	Ischemia	O ₂ deprivation accompanied by inadequate removal of metabolites due to inadequate perfusion
Unstable angina pectoris	Angina that is more frequent and severe, progresses and is not relieved by stopping exercise or medication . > 90% Occlusion of coronary artery	Ischemic Heart Disease	Heart disease secondary to insufficient blood supply to the myocardium
CAD	Coronary artery disease	Infarction	Area of necrosis in tissue caused by local ischemia , resulting from obstruction of circulation to area

Blood Pressure

Depends on:

1. **Cardiac Output (How much blood is pumped per beat)**
2. **Vascular Resistance**
3. **Total Blood Volume**

$$\text{BP} = \text{Cardiac Output} \times \text{Systemic Vascular Resistance}$$



Systolic BP	Highest pressure in arteries during ventricle contraction	Diastolic BP	Lowest arterial pressure when ventricles are filling up
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Hypertension

NEW CLASSIFICATION SYSTEM FOR HYPERTENSION (2017)			
BP Category	Systolic BP (mm Hg)		Diastolic BP (mm Hg)
Normal	<120	and	<80
Elevated	120-129	and	<80
Hypertension: stage 1	130-139	or	80-89
Hypertension: stage 2	≥140	or	≥90

Risk Factors	
Genetics	Age
Sedentary lifestyle	High Sodium Diet
Heavy alcohol consumption	Smoking
Stress	High cholesterol (LDL, VLDL)
Diabetes	Obesity

Primary:

- Many risk factors contribute, but does not have 1 cause. Not a result of any other disorders
- Can be hereditary

Environmental factors effect genetically susceptible youth (diet, Na^+ intake, obesity, stress), otherwise >65 is more likely to be affected

Secondary

Renal Failure	Damaged nephrons. Na^+ secretion altered = H_2O retention -> ↑ Blood volume and C.O ↑ Renin release -> Renin dependant H.T
Renovascular disease	Stenosis of vessel lumen -> ↓ renal perfusion Renin released -> ↑ Angiotensin & Aldosterone <ul style="list-style-type: none"> - Increase blood volume via Na^+ reabsorption - Systemic vasoconstriction and C.O
Primary Hyperaldosteronism	↑ Aldosterone (usually from adrenal hyperplasia or neoplasm) <ul style="list-style-type: none"> - Renal retention of Na^+ and water - ↑ Plasma volume and pressure
Pheochromocytoma	Catecholamine secreting tumors ↑ Epinephrine and norepinephrine <ul style="list-style-type: none"> - Systemic vasoconstriction and C.O ↑
Aortic Coarctation	"Like a kink in a garden hose" Narrowing of aorta (often distal to left subclavian) ↑ BP proximal to coarctation and ↓ BP distal (legs)

Untreated HT leads to organ damage in 2 processes:

1. Accelerates atherosclerosis in medium and large arteries
2. Remodels small arteries to ↑ wall thickness (inward hypertrophy)
 - a. Decrease parallel capillary networks
 - b. Increases total resistance

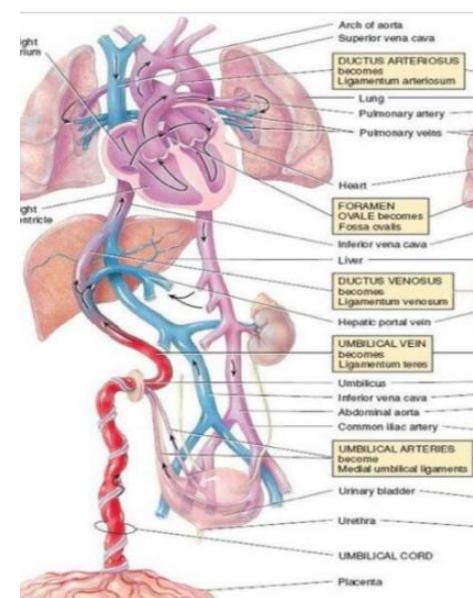
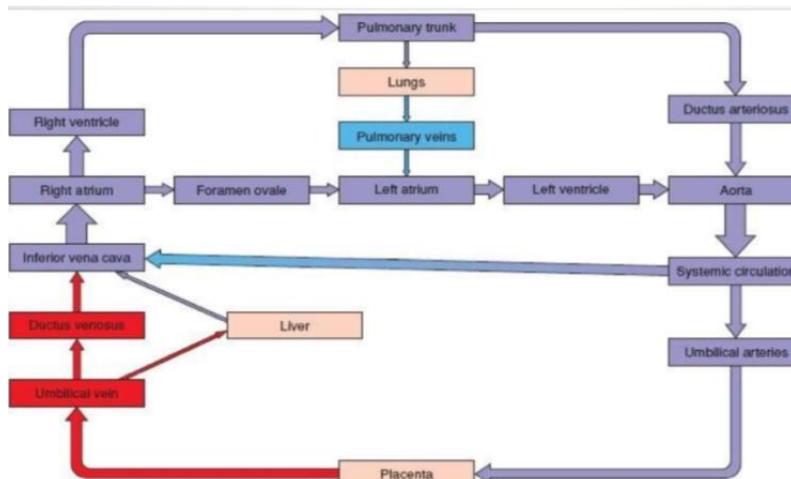
System	Tests ⁴⁵
Kidney	Microscopic urinalysis, albumin excretion, serum BUN and/or creatinine
Endocrine	Serum sodium, potassium, calcium, ?TSH
Metabolic	Fasting blood glucose, HDL, LDL, and total cholesterol; triglycerides
Other	Hematocrit, electrocardiogram, chest radiograph
Physical	Damage to heart, eyes, kidneys

PBL/DALE – Baby Lucas (Congenital Heart Disease)

Fetal Circulation:

4 alterations:

1. **Umbilical Vein**
 - Carries O₂ rich blood from mother in the placenta into fetal circulation
2. **Ductus Venosus**
 - Shunts blood away from fetal liver (slow moving through liver) and directly into the IVC where it can travel to the heart
3. **Foramen Ovale**
 - 1 way shunt connecting the Right Atrium to the Left atrium. Prevents blood from entering pulmonary circulation
4. **Ductus Arteriosus**
 - Shunt from the Pulmonary trunk draining into the Aorta, in case some blood did enter the pulmonary circulation.



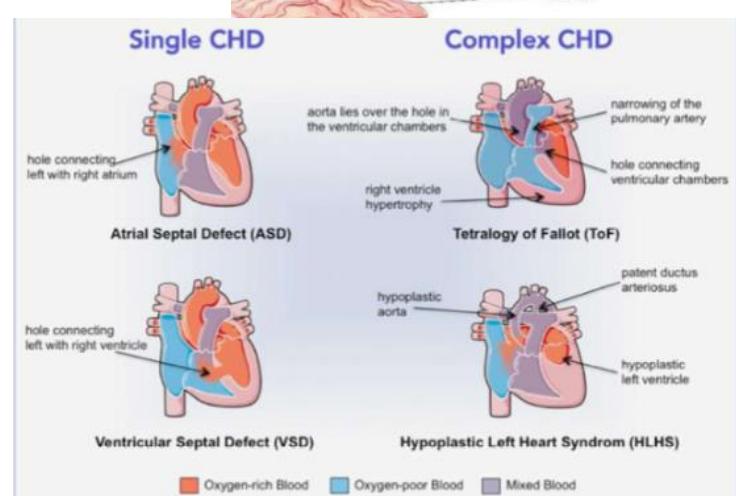
Congenital Heart Disease

Trisomy 21 is most common genetic factor of CHD

- Ventricular Septal Defect
- Patent Ductus Arteriosus
- Tetralogy of Fallot

Increased blood flow to Lungs:

- Patent Ductus Arteriosus (PDA)
- Atrial Septal Defect (ASD)
- Ventricular Septal Defect (VSD)
- Atrioventricular canal (AV Canal/AVC)



Congenital Heart Defects	
Physiologic Consequences	Asymptomatic heart murmur Abnormal Pulse Severe cyanosis Congestive Heart Failure
Signs and Symptoms	Tachycardia Tachypnea Dyspnea with Feeding Diaphoresis (Especially when feeding) Restlessness, Irritability Hepatomegaly
Presentations	High Pressure Shunt (Ventricular or greater artery) <ul style="list-style-type: none"> - Apparent days to weeks after birth Low Pressure Shunt (Atrial) <ul style="list-style-type: none"> - Apparent way later than HPS Large Left to Right Shunts (VSD, PDA) <ul style="list-style-type: none"> - ↑ Pulmonary flow and volume -> signs of heart failure and Failure to Thrive
Failure to Thrive	
= Arrested physical growth	<ul style="list-style-type: none"> - Height and Weight fall below 5th percentile - Downward change in growth across 2 major percentiles
Causes	Congenital Birth Defects (Down Syndrome) Endocrine disorders Brain damage (or CNS) Cardiopulmonary disorders Anemia GI disorders Chronic Infections Metabolism problems Pregnancy Complications
Coarctation of the Aorta	
= Narrowing of aortic lumen	Presentations <ul style="list-style-type: none"> Upper extremity hypertension Lower extremity Hypotension <ul style="list-style-type: none"> - ↓ Perfusion of abdominal organs and lower limbs Left Ventricular Hypertrophy Headache Heart Murmur ↑ Pressure proximal to obstruction
Pulmonary Stenosis	
= Thickening and ↓ function of the pulmonary valve	Signs and Symptoms <ul style="list-style-type: none"> Heart Murmur Cyanosis Dyspnea Dizziness Upper Thorax pain Edema in lower extremities (because of the eventual Right HF) ↑ Pulmonary pressure -> Pulmonary Hypertension
Effects	↑ Resistance = Right ventricular hypertrophy -> Right heart failure
Tetralogy of Fallot	
= Most common cyanotic condition	Key Features <ol style="list-style-type: none"> 1. Pulmonary Valve Stenosis 2. Ventricular Septal Defect 3. Overriding Aorta <ul style="list-style-type: none"> - Aorta shifts to right and sits directly above VSD. Receives blood from R and L ventricles 4. RV Hypertrophy

	<p>Tetralogy of Fallot</p>	
Eisenmenger Syndrome		
<p>= Deoxygenated blood enters systemic circulation -> Hypoxia</p>		
Effects	<ul style="list-style-type: none"> ↑ Pulmonary blood flow -> Pulmonary Vascular Disease - Unfixed Left-right shunt -> ↑ Pulmonary resistance -> switch to right-left shunting 	

PBL IV – Mr Blutarksy

- Review of information already in this package.

DALE I Wrap-up – Hailee Rostcross

BMI = Mass (kg) / Height (m)²

BMI Categories	
Underweight	<18.5
Normal weight	18.5 – 24.9
Overweight	25-29.9
Obese	30+

Lipids

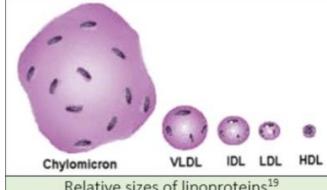
= H₂O insoluble organics, basic component of membranes

Examples:

Function	Energy Storage Signalling Membrane structure
<ul style="list-style-type: none"> - Fats - Waxes - Cholesterols 	<ul style="list-style-type: none"> - Vit. A, D, E, K - Mono- Di-, and Tri-glycerides - Phospholipids

Cholesterol

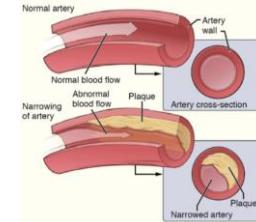
<ul style="list-style-type: none"> = Sterol, and essential component of all cell membranes to maintain integrity and fluidity - Precursor to Steroids, Bile Acids, and Vitamin D - Acquired via biosynthesis (75%) and diet (25%) - Synthesized in Hepatic cells, Intestines, Adrenal glands and Reproductive organs 	
Chylomicrons	Cary triglycerides from intestines to: Liver, Skeletal muscle, Adipose tissue Large size
Low-Density Lipoprotein (LDL)	BAD CHOLESTEROL High risk of heart disease Cholesterol core, formed in liver (converted from VLDL to LDL) Bind LDL receptors and taken up by cells Deliver cholesterol to peripheral tissues
VLDL	Carry triglycerides from liver to adipose (highest amounts of triglycerides)

HDL	Cholesterol from periphery (and atheromas) to liver for excretion in bile GOOD CHOLESTEROL
Relative Size	 <p>Relative sizes of lipoproteins¹⁹</p>

Atherosclerosis

= damage to endothelium of arteries leading to formation of atheroma (plaques)

- Plaques on the intima encroach on the lumen of **medium and large arteries**
- Contain **Lipids, Inflammatory cells, Smooth muscle and Connective tissue**



	Tunica Intima	Tunica Media	Tunica Adventitia
Elastic artery (conducting – Aorta, Pulmonary)	Endothelium + Weibel-Palade bodies, Basal Lamina, Sub-endothelial layer	Smooth muscle between 2 elastic membranes Thin external elastic lamina	Thin Fibro-elastic CT Vasa Vasorum Lymphatics Nerve Fibres
Muscular Artery (Distributing – Carotid, femoral)		Smooth Muscle Cells Thick external elastic lamina	
Arteriole (Microcapillaries)		1-2 Layers of smooth muscle cells	Loose CT Nerve Fibres

Weibel-Palade Bodies = storage granules that release vWF & P-selectin

Calcified Carotid Artery Atheroma (CCAA)

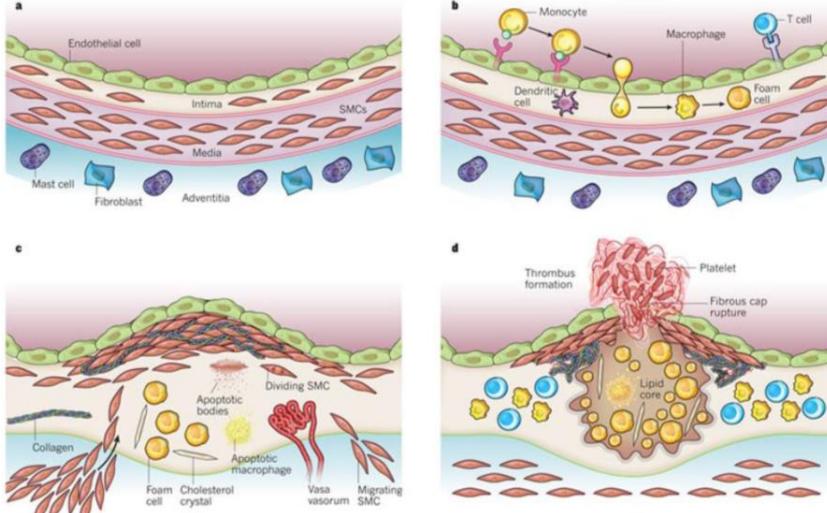
= Can find evidence of this in panoramic radiographs, NOT diagnostic though.

- Refer to physician if suspected
- Medical treatment if stenosis <50% or surgical if >50%

Pathophysiology (Atherosclerosis)

- Starts in utero
- **Reactive response to sheer forces of blood flow**
- Affects primarily **medium – large arteries and at bifurcations**

Steps	1. Irritation	Irritant present in vasculature (Hypertension, turbulent flow, toxins, LDL)
	2. Damage	Irritant damages endothelium (often can be sheer forces of blood at a bend or bifurcation in circulation)
	3. Lipids in Intima	LDL enters intima and oxidizes = fatty streak
	4. Recruitment + Adhesion	Monocytes recruited to damage site and adhere to endothelium
	5. Transmigration and Phagocytosis	Monocytes transmigrate into intima -> macrophages phagocytose oxidised LDL -> turn into foam cells = plaque
	6. Smooth Muscle Migration	Macrophage + T Cells cytokines (IFN- γ) recruit smooth muscle cells to area and creates a cap over the plaque
	7. SMC death	Necrosis of deep tissue smooth muscle
	8. Angiogenesis + Calcification	Microvasculature (vasa vasorum) forms around deep lesions and plaque becomes calcified
	9. Collagen decrease	Leukocytes reduce collagen production = weak cap structure
	10. Cap Rupture	Cap ruptures from sheer forces and weakening of structure -> induces thrombosis



Unstable Plaques	Stable Plaques
<ul style="list-style-type: none"> - High macrophage and thick lipid core + thin cap - <50% lumen narrowing can rupture spontaneously - Leads to: Unstable angina Myocardial Infarction CVA Cardiac Arrest <p>- Can turn unstable plaque into stable by ↓ lipid content of diet and ↓ Inflammation</p> <p>- Stability depends on: Lipid, SMC, inflammatory cell, CT and Thrombus proportion, Stress on cap, Size of lipid core.</p> <p>- Metalloproteinases + Collagenases from macrophages break down CT = thinner weaker plaque</p> <p>- T-cell cytokines inhibit SMC production of collagen</p>	<ul style="list-style-type: none"> - Thicker fibrous cap, fewer lipids - Narrow lumen by >50% - Increased size obstructs blood flow - Leads to: Stable, exercise induced angina Chronic Ischemia – Heart Failure, dysrhythmia Acute Ischemia – Angina Pectoris, TIA Infarction - MI or cerebral infarction

Risk Factors	Dyslipidemia Diabetes Cigarette smoking Family Hx Sedentary Lifestyle Obesity Hypertension Endothelial dysfunction Inflammation Age Male (<55), Post menopausal female (65+)
Symptoms	Asymptomatic for decades Symptomatic when blood flow is impeded enough (ischemia) <ul style="list-style-type: none"> - >75% blockage = Angina, TIA Diagnosis = Angiography, Ultrasonography, Imaging
Treatment	Lifestyle Modifications Dietary Modifications Physical Activity Antiplatelet drugs Antiatherogenic drugs Dyslipidemia, Hypertension and diabetes drugs

Hypertension

= Increased arterial wall tension = disturbed repair processes, aneurysm and endothelium damage

- Endothelial dysfunction inhibits **Nitric Oxide** production ([vasodilator and anti-inflammatory, anti thrombotic](#))
- **Endothelin** = [Vasoconstrictor](#), enhances SMC migration + growth. Stimulated by oxidised LDL

Diabetes

= Metabolic disorder characterised by hyperglycemia due to:

- [Defective Insulin Secretion \(Type 1\)](#)
- [Defective Insulin Action \(Type 2\)](#)

Hyperglycemia	<ul style="list-style-type: none"> - Oxidative stress (Directly injure endothelium) - Reactive O₂ Radicals (impair endogenous antioxidant defense system) - Glycosylated end products (Increases proinflammatory cytokines from endothelial cells)
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Glycosylated Hemoglobin

- RBC freely permeable to glucose, Glc irreversibly bind hemoglobin and lasts the lifecycle of RBC (120 days).
- Glycosylation state shows glucose levels for 2-3 months, more accurate reading!
- A1c test

Diagnosis	Fasting glucose : >7mmol/L 2hr Plasma glucose >11.1mmol/L Glycated hemoglobin (A1c) >6.5%	
Type 1 (Insulin Dependent)	Pancreatic β -cell destruction Prone to ketoacidosis Includes autoimmune process Begins at childhood (increases with age) Etiology: <ul style="list-style-type: none"> - Genetics: Linked to HLA gene on chromosome 6 - Autoimmune: Ab against islet cells or insulin itself - Environmental: Viral infections (mumps, rubella, Coxsackie virus) can trigger autoimmune - Idiopathic 	
Type 2 (Non-insulin Dependent)	Peripheral tissue insulin resistance Insufficient insulin production Etiology: <ul style="list-style-type: none"> - Genetics: Family history (38% risk with 1 parent, 60% risk with 2 parents) - Environmental: Obesity, Physical Inactivity 	
Gestational Diabetes Mellitus	Abnormal glucose tolerance with pregnancy <ul style="list-style-type: none"> - Often gets better after given birth - May lead to Spontaneous Abortion or large fetus 	
Long Term Complications	Microvascular <ul style="list-style-type: none"> Neuropathy Nephropathy (leads to renal failure) Retinopathy, cataracts Foot complications (ulcers, gangrene, arthritis) 	Macrovascular <ul style="list-style-type: none"> Myocardial Infarction Accelerated atherosclerosis Hypertension TIA CVA

Signs and Symptoms	Polyuria	Hyperglycemia exceeds renal reabsorption threshold - Increase peeing
	Polydipsia	↑ fluid loss triggers osmoreceptors - Intense Thirst
	Polyphagia	W/o dietary glucose absorption body breaks down proteins and fats – leads to negative energy balance - Increased appetite
	Oral Manifestations	- Gingivitis - Periodontal Disease - Xerostomia - Infection - Neuropathy - Poor wound healing - Candidiasis -> related to ↓ Immune function - Burning feeling in mouth - Dysgeusia - Sialosis

Smoking

- Highest risk factor for atherosclerosis
- Direct toxic effects (carbon monoxide) on vascular wall -> impaired endothelial function
 - o Impaired endothelium-dependant vasodilation by ↓ nitric oxide availability
 - o Causes vasoconstriction
- Decreased platelet sensitivity to NO -> ↑ platelet activation
- Decreases serum HDL, increases LDL
- Increases Oxidised LDL
- Increases WBC
- Increases inflammatory markers (CRP, Interleukin 6 and TNF-alpha)

Coronary Artery Atherosclerosis

= Impaired blood flow causing myocardial ischemia

Plaque rupture causes: Acute Coronary Syndrome (ACS), and Acute Myocardial Infarction (AMI)

Signs and Symptoms	<ul style="list-style-type: none"> - Chest Pain - Shortness of breath - Fatigue - Dizziness - Irregular H.R (Palpitations) - Heart Murmurs - Syncope - Leg edema - Diaphoresis - Stable Angina - Tachypnea - Xanthelasma
Angina	<p>= cardiac workload and myocardial O₂ demand exceeds ability of arteries to supply</p> <ul style="list-style-type: none"> - Stress and exertion make it worse - Relieved with rest or nitroglycerin - Presence of >75% stenosis of at least 1 major coronary artery <p>Tx:</p> <ul style="list-style-type: none"> - Aspirin, Nitrates, β-blockers, CCB, ACE Inhibitors, - Coronary angioplasty, Coronary artery bypass

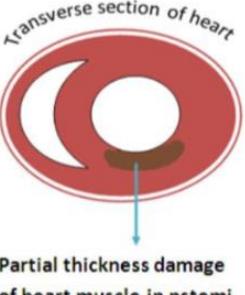
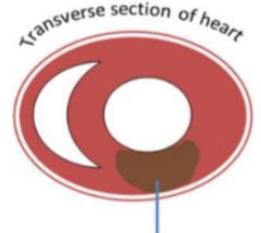
Acute Coronary syndrome

= Acute thrombus interrupting coronary flow causing:

- Unstable angina - > 90% occlusion
- Myocardial Infarction
- Sudden death

ST segment: = flat section between end of S wave and beginning of T wave

Represents between ventricular depolarization and repolarization

If coronary artery is partially blocked:	If coronary artery fully blocked
<ul style="list-style-type: none"> ○ No ST elevation on ECG ○ Cardiac biomarkers may be elevated (if not = unstable angina) 	<ul style="list-style-type: none"> ○ ST elevation on ECG ○ Cardiac biomarkers elevated
NSTEMI	STEMI
Complete Occlusion of minor coronary artery Partial occlusion of major coronary artery = Partial thickness damage of heart muscle  Partial thickness damage of heart muscle in nstem	Complete occlusion of major coronary artery = Full thickness damage of heart muscle (Transmural) Leads to: <ul style="list-style-type: none"> - Cardiogenic shock - LV Failure - Mitral regurgitation (papillary muscle damage) - Cardiac tamponade (from ventricular wall damage)  Full thickness damage of heart muscle in stemi

Cardiac Biomarkers:

- **Myoglobin:** Released from damaged tissue
- **Troponin I and Troponin T:** Released from cardiac muscle damage (necrosis)
- **Creatine Kinase-MB:** Not as specific as Troponin but rises in blood quickly so used as rapid indicator

Myocardial Infarction

Only used with evidence of myocardial necrosis Zone of reversible ischemia adjacent to infarcted tissue that responds to treatment (increases prognosis)	
Symptoms	Dental Treatments
Recent MI (< 1month)	Considered high risk , defer treatment If care is necessary: <ul style="list-style-type: none"> - IV line - Sedation - O₂ - Caution around vasoconstrictors - Nitroglycerin prophylaxis

Past MI (>1 month w/o symptoms)	<p>Intermediate risk</p> <ul style="list-style-type: none"> - Access vitals - Have nitroglycerin available - Provide profound LA (limit 2 cartridges of epinephrine) - Anxiety reduction (Sedation) - Good post op pain medication - No Ab prophylaxis if has pacemaker
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Hypertension Medications

	Category	Oral Manifestations
Hydrochlorothiazide	Diuretic	Xerostomia Lichenoid drug reactions
Metoprolol	β -blocker	Lichenoid drug reactions
Nitroglycerin	Vasodilator	Xerostomia
Prolonged Aspirin	NSAID	Gingival bleeding Petechiae Ecchymoses Prolonged Bleeding
Simvastatin	\downarrow Cholesterol from Liver	Xerostomia
Nifedipine	Calcium Channel Blocker	Gingival Hyperplasia
Diltiazem		
Ramipril	ACE Inhibitor	Cough Dysgeusia Lichenoid drug reaction

DALE II – Earnest Lillik (Hypertension)

Hypertension

BP depends on: Cardiac Output, Vascular Resistance, Total Blood Volume

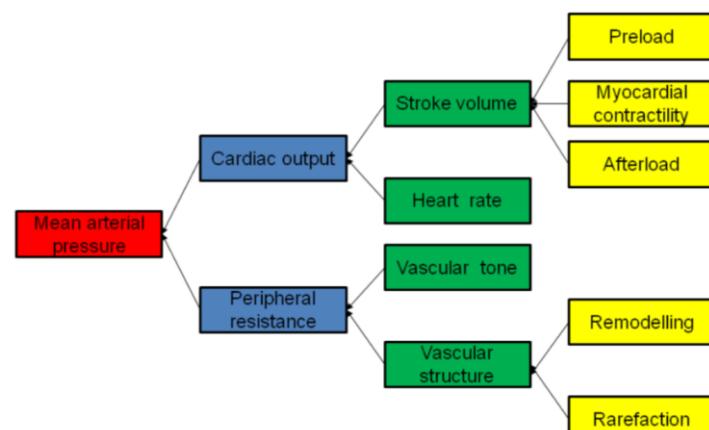
$$\text{BP} = \text{CO} \times \text{SVR}$$

Mean Arterial Pressure (avg. pressure in arteries)

$$\text{(MAP)} = ([2 \times \text{DBP}] + \text{SBP}) / 3$$

Untreated HT damages organs in 2 ways:

1. Accelerates atherosclerosis in medium-large arteries
 2. Small artery and arteriolar remodelling w/initial wall thickness and \downarrow compliance
 - o \downarrow lumen diameter, \downarrow parallel capillary networks (rarefaction)
- Ultimately \uparrow Resistance and BP and \downarrow perfusion



NEW CLASSIFICATION SYSTEM FOR HYPERTENSION (2017)

Category	Systolic (mmHg)	Diastolic (mmHg)
Normal	<120	and <80
Elevated	120-129	and <80
Stage 1 hypertension	130-139	or 80-89
Stage 2 hypertension	≥140	or ≥90
Hypertensive emergency/crisis	>180	and/or >120
Isolated systolic hypertension	>140	and <90

Body System	Cause
Renal	Renovascular HTN Renal parenchymal disease, glomerulonephritis, pyelonephritis, polycystic kidney
Endocrine	Primary hyperaldosteronism Pheochromocytoma Cushing's syndrome Hyperthyroidism/hyperparathyroidism Hypercalcaemia of any cause
Vascular	Coarctation of the aorta Renal artery stenosis
Drug induced	Oestrogens (pregnancy), MAOIs, Cocaine, Steroids, Lithium, Amphetamines, NSAIDs, Decongestants, Alcohol

	Diagnosis	Causes	Effects
Isolated Hypertension	Systolic: >140 Diastolic: >90	Hyperthyroidism Artery Stiffness Heart Valve Problems	Stroke Heart Disease Chronic Kidney Disease
White Coat Hypertension	BP >140/90 – 3 occasions at office BP <135/80 – Outside office	Anxiety	
Resistance Hypertension	Does not respond to appropriate management	Improper BP Measurement Volume Overload <ul style="list-style-type: none"> - Excess Na⁺ intake - Renal Disease Drug Induced <ul style="list-style-type: none"> - Non-adherence - Inadequate dosage - NSAIDs - Cocaine Obesity Excess Alcohol	

Hypertensive Crisis	HT Urgency: BP > 180/110 – NO Target organ damage HT Emergency: BP >180/120 – Impending or progressive organ damage		HT Urgency - Headache, Epistaxis, Faintness, HT Emergency - Chest Pain, Dyspnea, Neurological Deficit
Hypertensive Encephalopathy		>160 mm Hg cerebral vessels dilate (rather than remain constricted) - ↑ transmits directly to capillaries in brain = cerebral edema	Failure of cerebral autoreg. Of blood flow
Risk Factors			
Primary HT	<p>Modifiable</p> Obesity Sedentary Lifestyle Poor diet (\uparrow Na ⁺ , Cholesterol) \uparrow Blood Glucose (diabetes) Heavy alcohol consumption Smoking** Stress <p>Non-Modifiable</p> Family Hx \uparrow Age Genetics		

System	Tests
Kidney	Urinalysis, Albumin Excretion, Serum BUN, Creatinine
Endocrine	Serum Na, K, Ca, TSH
Metabolic	Fasting blood Glucose, HDL, LDL, total Cholesterol, Triglycerides
Other	Hematocrit, Electrocardiogram, Chest radiograph
Physical	Damage to heart, Eyes, Kidneys

UBC Dental Treatment Recommendations

Systolic mm Hg	Diastolic mm Hg	Medical risk factor ❤️	Dental treatment alteration
120-139	80-89	Yes/no	Routine dental care OK; discuss BP guidelines
140-159	90-99	Yes/no	Routine dental care OK; consider stress reduction protocol; refer for medical consult
160-179	100-109	No	Routine dental care OK; consider stress reduction protocol; refer for medical consult
160-179	100-109	Yes	Urgent dental care OK; consider stress reduction protocol; refer for medical consult
180-209	110-119	No	No dental treatment; refer for prompt medical consult
180-209	110-119	Yes	No dental treatment; refer for emergency medical treatment
>210	>120 *	Yes/no	No dental treatment; refer for emergency medical treatment

Dental Drug Interactions:

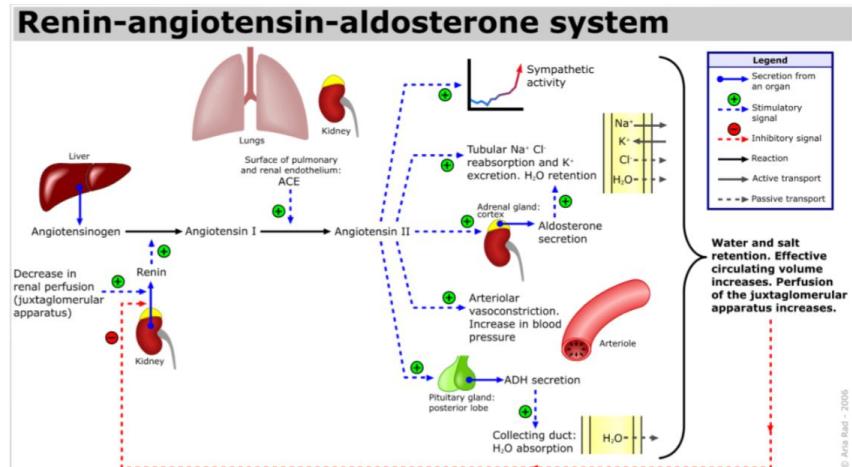
Drugs	Effects
Local Anaesthesia	β-blockers ↑ LA toxicity
Epinephrine	Cardio effects of Epi (↑ HR, ↑ Contractility etc) potentiated with non-selective β-blockers
NSAIDs	↓ effectiveness of diuretics, β-blockers, α-blockers, vasodilators, and ACE inhibitors
Oral Manifestations	
Thiazide Diuretics (Hydrochlorothiazide)	Xerostomia Lichenoid like rxns
β-Blocker (Metoprolol)	Lichenoid like rxn
Organic Nitrates (Nitroglycerin)	Xerostomia
NSAIDs	Gingival Bleeding, Petechiae, Ecchymoses, Prolonged bleeding times
Statins (Simvastatin)	Xerostomia
Ca Channel Blockers (Nifedipine)	Gingival Hyperplasia
ACE Inhibitors (Ramipril)	Cough, Dysgeusia, Lichenoid like rxns, angioedema, burning sensation in mouth
ARB's	Dysgeusia, Angioedema

Renin Angiotensin -Aldosterone System

- Potent controller of blood pressure
- Activated in response to ↓ Plasma Na, or ↓ circulating volume
- Renin released by Juxtaglomerular App from Sympathetic stim, ↓ osmolality of filtrate, ↓ Stretch (from ↓ BP)

Renin (Kidney)-> converts Angiotensinogen (liver) to AT I -> ACE (lungs) converts AT I to AT II

AT II Function	<ul style="list-style-type: none"> -↑ Sympathetic Activity (Positive chronotropic and ionotropic effects) -Na reabsorption, K excretion -Aldosterone Secretion (Also stimulates Na reabs. and K excretion) -Vasoconstriction (↑ SVR) -ADH Secretion (Water absorption)
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Hypokalemia

Symptoms	<p>Weakness and Fatigue (most common) Muscle Cramping and Pain Worsening diabetes control or polyuria Palpitations Psychosis, Delirium, Hallucinations, Depression Bradycardia with cardiovascular collapse Cardiac arrhythmia Acute respiratory failure from muscle paralysis</p>
↑ K⁺ Excretion	Aldosterone High Na ⁺ to collecting duct (diuretic) High urine flow (diuretics) High serum K ⁺
↓K⁺ Excretion	Aldosterone Deficiency Low Na ⁺ to collecting ducts Low urine flow Low serum K ⁺ levels Renal Failure

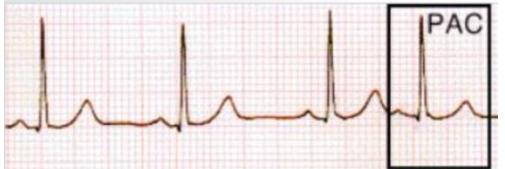
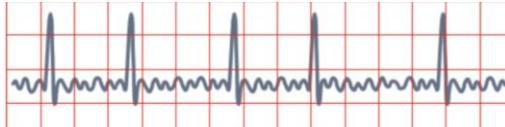
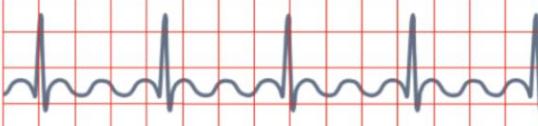
DALE III – Ms Gispin (ECG)

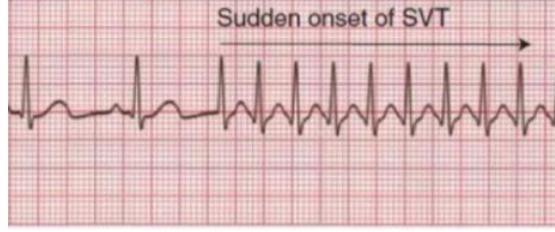
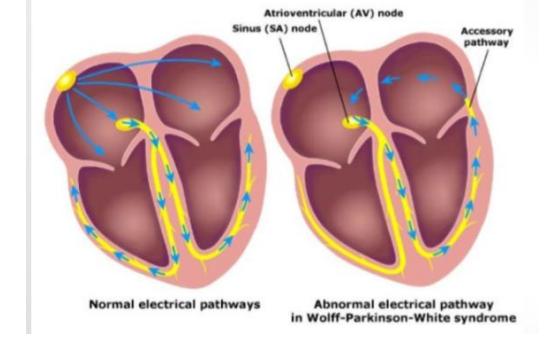
Arrhythmia

= Abnormal rhythm and/or speed of heart contraction (due to electrical problem of the heart)

3 Causes:

1. ↑/↓ automaticity
2. Triggered Activity
3. Re-Entry (most common)

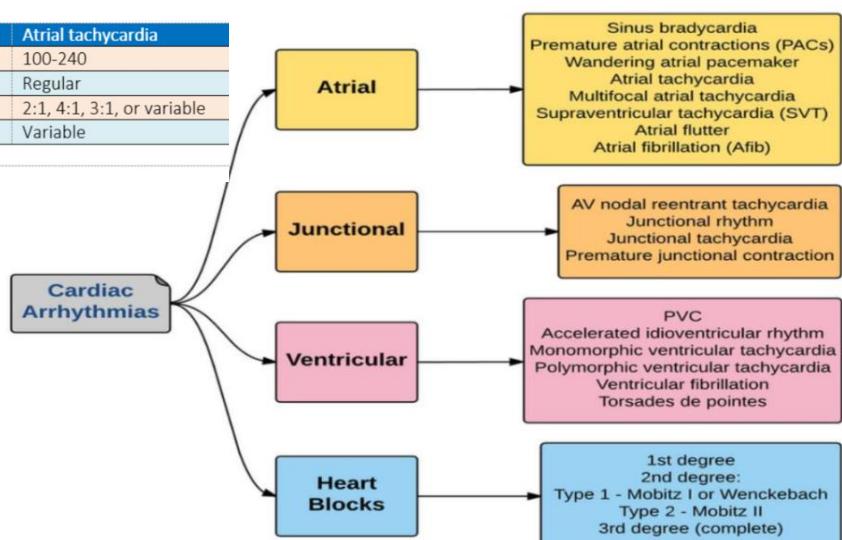
Premature Beats			
	Symptoms	Causes	
Atrial	Fluttering in chest "skipped a beat" feeling	Ectopic foci prematurely exciting myocardium <ul style="list-style-type: none"> - Extra P wave and QRS 	
Ventricular	Fluttering in chest "skipped a beat" feeling	Impulse generated in Ventricle <ul style="list-style-type: none"> - Wide QRS 	
Supraventricular Arrhythmia (originates in AV node, or Atria)			
Atrial Fibrillation (most common)	Rapid disorganized contractions <ul style="list-style-type: none"> - Wavy baseline on ECG not always leading to QRS 400-600 bpm <u>Complications</u> <ul style="list-style-type: none"> - Stroke - Heart Failure (V don't completely fill with blood) 	Ectopic pacemaker cells overriding SA node, Re-entry circuits <u>Risk Factors:</u> <ul style="list-style-type: none"> - ↑ BP - Coronary Heart D. - Heart Failure - Pericarditis - Sick Sinus Syndrome - Congenital Heart D - Rheumatic heart D - HT Risk Factors - Caffeine - Sleep Apnoea - Alcoholism - Hyperthyroidism 	
Atrial Flutter	Signal spreads through atria fast and regular <ul style="list-style-type: none"> - Baseline is regular but very fast 200-300bpm		 Rapid flutter waves, ventricular response irregular

Paroxysmal SV Tachycardia	Wolff-Parkinson-White Syndrome Regular rhythm P waves buried in T Can't measure PR QRS Normal Light Headedness, Anxiety, Dizziness, Palpitations	Sudden onset and ending Signal begins in atrial -> travels to ventricle -> Re-entry circuit back to atria.	 
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Ventricular Arrhythmia

Ventricular Tachycardia	Fast, Regular ventricular rhythm > few seconds = dangerous	Can occur also with micro level re-entry circuits	
Ventricular Fibrillation	Sudden death if not defibrillated right away	Disorganized signals make ventricle quiver instead of contract normally	
Long QT Syndrome		Genetics causing abnormal cardiac myocyte Na K channel depol. -> longer QT Can be acquired and caused by certain meds - Drug induced long QT	

	AF	Atrial flutter	Atrial tachycardia
Atrial rate (bpm)	> 400	240-350	100-240
Atrial rhythm	Irregular	Regular	Regular
AV block	Variable	2:1, 4:1, 3:1, or variable	2:1, 4:1, 3:1, or variable
Ventricular rate (bpm)	Variable	150, 75, 100, or variable	Variable
AV - atrioventricular; bpm - beats per minute			



CHADS₂ Score

- Used to estimate risk of stroke and whether or not treatment is required w/ anticoagulation therapy or antiplatelet therapy
- If score 2+ then use anticoagulation/antiplatelet therapy

C	Congestive Heart Failure	1
H	Hypertension (>140/90), or treated HT on meds	1
A	Age >75	1
D	Diabetes mellitus	1
S	Prior Stroke or TIA	2