

Anatomy of the Heart

4 Chambers	Right AV valve (tricuspid) -> pulmonary(semilunar) valve -> left AV valve/bicuspid/mitral -> aortic/semilunar valve
Blood Circulation	
Pulmonary circulation	Chambers on the right pump oxygen poor blood to the lungs
Systemic Circulation	Chambers on the left pump oxygen rich blood to body tissues
Right atrium	receives oxygen poor blood from inferior and superior vena cava
Right Ventricle	receives oxygen poor blood from the right atrium and pumps blood through the pulmonary valve into pulmonary artery
Left atrium	receives oxygen rich blood from pulmonary circulation via the left and right pulmonary veins
Left Ventricle	receives oxygen rich blood from left atrium and pumps blood through aortic valves into aorta
Chordae Tendinae	tendonous fibers attached to the inside edges of AV valves and base of ventricles via papillary muscles, prevents valves from everting
Connective Tissue	separates atria for ventricles providing attachment of heart valves

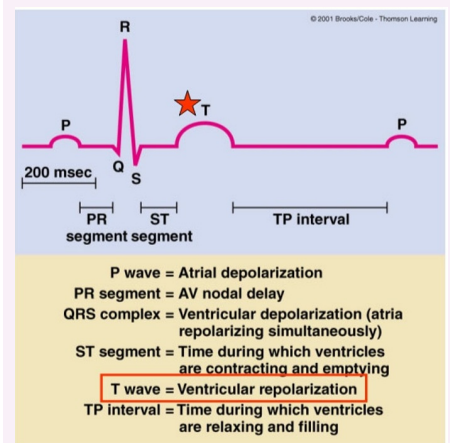
Electrical Activity

Autorhythmicity	heart muscle is capable of generating its own rhythmic electrical activity
Pacemaker activity	spontaneous, rhythmic generation of electrical impulses by specialized heart cells (like those in the sinoatrial node) that initiate and regulate the heartbeat, ensuring consistent cardiac contraction and blood circulation
SA Node	generates 70 AP per min, located in the wall of the right atrium near superior vena cava
AV Node	50 AP per minute, located at the base of the right atrium follows the SA node
Bundle of His	specialized pacemaker cells originating at AV node projecting into left and right ventricles
Purkinje Fibers	30 AP per min, spread throughout ventricular myocardium
Interatrial Pathway	specialized cardiac cells that conducts pacemaker activity from the right atrium to the left atrium
Internodal Pathway	pathway of specialized cardiac cells that conducts pacemaker activity from SA to AV nodes
AV nodal delay	Pacemaker activity is conducted relatively slowly through the AV node resulting in a delay of approximately 100 ms

Electrocardiogram Waveforms

P wave	Depolarization of the atria
QRS complex	depolarization of the ventricles
T wave	repolarization of the ventricles
PR segment	represents AV nodal delay

Electrocardiogram Waveforms



Mechanical Events of the Cardiac Cycle

Systole	Contracting and emptying
Diastole	relaxation and filling
End Diastolic Volume	volume of blood in chamber at end of diastole, equivalent to max amount of blood chamber holds during cycle
Isovolumetric contraction	period of time during contraction when chambers stay closed increasing chamber pressure during this periods
End systolic volume	amount of blood remaining in the chamber at the end of systole

Mechanical Events of the Cardiac Cycle (cont)

Stroke volume	amount of volume blood pumped out of the chamber with each contraction
Stroke volume equation	EDV - ESV
Isovolumetric relaxation	period of time during relaxation when the chamber remains closed and therefore no blood can enter or leave, chamber pressure decreases then
Lub	closure of AV valves
Dup	Closing of the semilunar valves
Murmurs	abnormal heart sounds from turbulent flow of blood through malfunctioning valves
Stenotic valve	stiff narrow valve that doesn't open completely, abnormal whistling sound
Insufficient valve	structurally damaged valve that does not close, abnormal swishing sound
Rheumatic fever	an auto-immune disease triggered by streptococcal bacteria that leads to valvular stenosis and insufficiency

Regulation of Cardiac Output

Cardiac Output	HR - SV
Heart rate regulation done by	parasympathetic and sympathetic nervous systems

Regulation of Cardiac Output (cont)

Stroke Volume	regulated intrinsically by volume of venous blood returning to the ventricles and extrinsically by the sympathetic nervous system
Parasympathetic	Vagus Nerve to the SA and AV nodes and to the contractile cells of the atria
Parasympathetic NTs	ACh and Muscarinic receptors
Effects of Parasympathetic Release of ACh	Increases permeability of SA nodal cells to K ⁺ in the SA node leading to greater hyperpolarization and slowing of the K ⁺ component of the pacemaker potential, in AV node increases permeability of AV nodal to K ⁺ and in atrial contractile cells, shortens duration of cardiac fiber AP reducing Ca ⁺⁺ permeability
Sympathetic	Norepinephrine through beta adrenergic receptors
Effects of Sympathetic influence on HR	SA node - less hyperpolarization, acceleration of the K ⁺ component, av node slowing increase in Ca ⁺⁺ permeability

Regulation of Cardiac Output (cont)

Stroke Volume Regulation	Extrinsically regulated by neural control and intrinsically by the volume of venous blood returning to heart
Intrinsic control	direct correlation between end-diastolic volume and stroke volume
Heart failure	inability of CO to meet demands of the body

Basic Organization

Arteries	composed of large vessels that carry blood from the heart
Arterioles	small diameter vessels that arise from the branching of arteries
Capillaries	smallest diameter vessels that are formed when arterioles branch
Venules	the vessels that form when capillaries join together
Veins	large diameter vessels formed by merging of venules
Microcirculation	name given to collection of arterioles, capillaries and venules

Blood Flow

Blood flow	determined by pressure gradient in the vessels and resistance to flow caused by friction and viscosity of the blood
Blood flow equation	$F = \Delta P / R$



Blood Flow (cont)

F	Flow rate, volume of blood passing through a vessel per unit of time
Delta P	Pressure gradient - difference in pressure between the beginning and end of the vessel
Resistance	depends on blood viscosity, vessel length, vessel radius
Blood viscosity	friction developed in blood determined by the concentration of plasma proteins and number of circulating RBCs
Vessel length	friction between blood and the inner surface of a vessel is proportional to the vessel length
Vessel radius	friction between blood and the inner surface of a vessel is inversely proportional to the 4th power of the vessel radius
Pressure reservoir	Serves as a driving force during ventricular diastole, elasticity of the of artery walls smooth muscle, collagen, elastin
Pulse Pressure	pressure difference between systolic pressure and diastolic pressure
Mean Arterial Pressure	pressure that is monitored and regulated by BP reflexes

Intrinsic (local control)

intrinsic control	factors intrinsic to an organ or tissue
Local metabolic changes	factors derived from metabolic activity causing dilation. smooth muscle tone is controlled by release of mediators such as NO
O2 concentration	reduced O2 during metabolic demand
CO2 concentration	increased CO2 during metabolic demand
pH	increases in CO2 and or lactic acid lowers blood pH
Extracellular K+ conc.	increased neuronal activity that outpaces the Na+/K+ ATPase
Osmolarity	increased solute concentration resulting from metabolic activity
Adenosine	released in Cardiac muscle in response to metabolic demand
Prostaglandins	produced from teh metabolism of fatty acids
Histamine release	release when tissues are damaged and leads to vasodilation accompanying an inflammatory response
local physical control	temperature and myogenic response
Temperature	arteriolar smooth muscle tone is inversely proportional to temperature
Myogenic response	arteriolar smooth muscle responds to stretch by contracting