Anatomy of the Heart

Right Chamber: Provides oxygen-poor blood through the Lungs

Left Chamber: Provides oxygen-rich blood through the Body

Right Atrium: Oxygen-poor blood (from body) travels to the right *AV (Tricuspid) Valve*

Right Ventricle: Oxygen-poor blood (right atrium) travels to the *Pulmonary Artery* via the *Pulmonary (Semilunar) Valve*

Left Atrium: Oxygen-rich blood (from lungs) travels to the left ventricle through the left *AV (Bicuspid/Mitral) Valve*

Left Ventricle: Oxygen-rich blood (left atrium) travels to the *Aorta* via the *Aortic (Semilunar) Valve*

Heart Valves: Allows blood to flow in one direction

High Pressure Behind Valve: Valve opens

High Pressure In Front of Valve: Valve closed; does NOT open, ONLY opens in one direction

Chordae Tendineae: Tendinous fibers that connect AV valves to *Papillary Muscles*, preventing **Everting** of the AV valves during ventricular contraction

Connective Tissue: Dense fibrous ring that surrounds each heart valve and separates the atria from the ventricles, providing an anchor for the heart valves/cardiac muscle

Anatomy of the Heart (cont)

Endocardium: Thin layer of endothelial tissue surrounding the inside of the heart chamber

Myocardium: Middle layer linked by *Intercalated Disks*, forming 2 contacts: *Desmosomes and Gap-Junctions*

Desmosomes: Responsible for clustering cells

Gap-Junctions: Allow low-resistant electrical flow between muscle cells, forming a *Functional Syncytium*

Epicardium: Thin outer membrane containing a small volume of *Pericardial Fluid*

Pericardial Fluid: Fluid that prevents heart from getting friction

Mechanical Events of the Cardiac Cycle

Systole: Contraction and Emptying

Diastole: Relaxation and Filling

End-Diastolic Volume: Blood volume left at the end of *Diastole* (Maximum amount of blood held in chamber during the cycle)

Isovolumetric Ventricular Contraction: Chamber is closed (no blood can enter/-leave); pressure **Increases**

End-Systolic Volume: Blood volume left after *Systole* (Complete ejection)

Stroke Volume: Amount of blood pumped out **(End Diastolic Volume - End Systolic Volume)**

Mechanical Events of the Cardiac Cycle (cont)

Isovolumetric Ventricular Relaxation: Chamber is closed (no blood can enter/-leave); Pressure **Decreases**

Regulation of Cardiac Output

Cardiac Output (C.O.): Blood volume pumped (per minute) depending on the **heart rate** and **stroke volume**

Heart Rate: Regulated by **Parasympathetic** and **Sympathetic** Nervous Systems

Stroke Volume: Volume of venous blood return **(Intrinsically)** and the **Sympathetic Nervous System (Extrinsically)**

C.O. Equation: C.O. = H.R. x S.V.

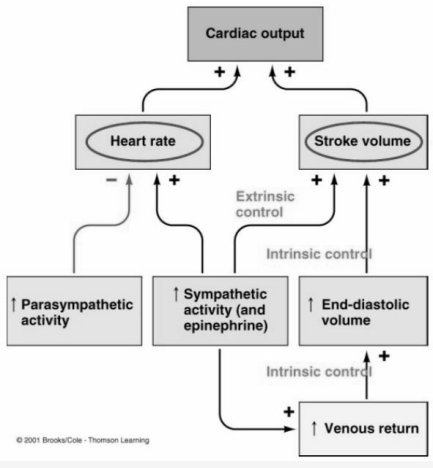
Heart Rate Regulation: Mainly controlled by autonomic input, affecting the hypersensitivity of SA node

Parasympathetic: **Vagus Nerve** contributes to the SA/AV nodes and the contractile cells

Parasympathetic Input: Mediated by Acetylcholine (ACh), a neurotransmitter

Acetylcholine: Causes heart rate to decrease

Parasympathetic and Sympathetic Inputs



Blood

Blood Flow: Calculated by **Pressure Gradient** and **Resistance and Viscosity**

Equation: F = (Delta) P/R

Flow Rate (F): Blood volume going through a vessel **(Proportional to r^4)

Pressure Gradient (P): Difference of pressure from the beginning compared to the end of a vessel

Resistance (R): Resistant of flow **(Proportional to 1/r^4)** *(3 Factors)*:

(1) Blood Viscosity: Blood friction due to plasma protein (conc.) and red blood cell count

(2) Vessel Length: Blood friction along vessel walls **(Proportional to length)**

(3) Vessel Radius: Blood friction along vessel walls **(Inversely Proportional to 1/r^4)**

Baroreceptors

Baroreceptor Reflex: Regulates cardiac output and total peripheral resistance

Baroreceptors: Mechanoreceptors detect blood pressure through force of the pressure

Baroreceptor Reflex (High BP): Decrease heart rate, stroke volume, arteriolar, and venous vasodilation

Baroreceptor Reflex (Low BP): Increase heart rate, stroke volume, arteriolar, and venous vasodilation

Local Physical Control

Temperature: Inversely proportionate to Arteriolar Smooth Muscle Tone

Myogenic Response: Arteriolar smooth muscle contract when stretched

Intrinsic (Local) Metabolic Changes (Control)

Local Metabolic Changes: Cause dilation in smooth muscle tone via **Mediators (Nitric Oxide)**

O2 Concentration: Decreases as metabolism increases (Inverse)

CO2 Concentration: Increases as metabolism increases (Proportional)

pH: CO2 increases and blood pH lowered by Lactic Acid

Extracellular K+ Concentration: Increased neuronal activity exceeding Na+/K+ ATPase

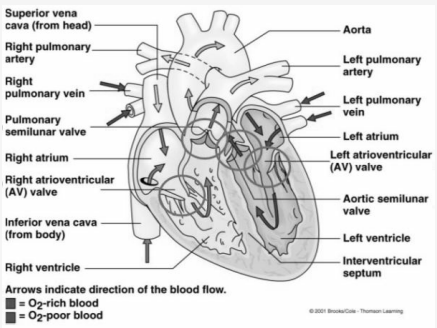
Osmolarity: Increase solute concentration

Adenosine: Released when metabolism is increased

Prostaglandins: Derived from fatty acid metabolism

Histamine Release: From damaged tissues, causing vasodilation and inflammation

Anatomy of the Heart



Electrical Activity of the Heart

Autorhythmicity: Self-generated rhythmic activity from cardiac muscle cells that produce *Pacemaker Activity*

Pacemaker Cells: Clustered into **Nodes**, regulating rate and coordination of contractions

Pacemaker Activity: 1% of cardiac muscle (*Autorhythmic and Intrinsically*) that self-generate AP at a regular frequency via *Pacemaker Potentials*

Contractile Cells: 99% of the cardiac muscles that are responsible for pumping but **DO NOT** self-generate AP

Autorhythmic Cells: Cyclically generate AP (through heart) to trigger rhythmic contractions

Nodes: Clusters of cells that produce pacemaker activity

Sinoatrial (SA) Node: Cluster of pacemaker cells (right atrium), firing **70** AP per minute *(Fastest)*

Atrioventricular (AV) Node: Cluster of pacemaker cells (right atrium), firing **50** AP per minute (*Slower*)

Bundle of His: Pacemaker cells (AV node) that branches to the left/right ventricles

Purkinje Fiber: Small pacemaker cells (*Bundle of His*), spreading through ventricular myocardium, firing **30** AP per minute *(Slowest)*

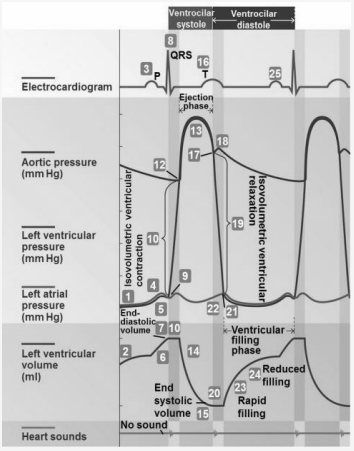
Interatrial Pathway: Cardiac cells that carry pacemaker activity (Right atrium -> Left atrium)

Electrical Activity of the Heart (cont)

Internodal Pathway: Cardiac cells that transmit pacemaker activity from the SA node to the AV node

AV Nodal Delay: 100ms delay in AV Node conduction, ensuring that the ventricles contract after atrial contraction

Mechanical Events of the Cardiac Cycle Diagram



Parasympathetic Release of Acetylcholine

SA Node: Higher permeability to K+, delaying inactivation of K+ channels, causing **Greater Hyperpolarization** and **Slow K+** of pacemaker potential

AV Node: Higher permeability to K+, reducing excitability and delay response to input from SA Node

Atrial Contractile Cells: Reduces Ca2+ permeability and strength of contraction

Sympathetic Release of Norepinephrine

Sympathetic Nerves: **Increase** heart rate by supplying the SA/AV Nodes and ventricles via **Norepinephrine** through **Beta-Adrenergic** receptors

SA Node: Lower K+ permeability speeds up inactivation K+ channels leading to **Less Hyperpolarization** and faster K+

AV Node: Increase conduction velocity, reduce AV nodal delay, and slightly increase Ca2+ permeability

Bundle of His & Purkinje Fibers: Similar actions as the AV Node

Atrial/Ventricular Contractile Cells: Increase contractile strength via increasing Ca2+ permeability

Basic Organization

Arteries: Large vessels that carry blood away from the heart

Arterioles: Small (Diameter) vessel branches from arteries that lead to the organs

Capillaries: Smallest (Diameter) vessels formed when arterioles branch

Venules: Vessels that form when capillaries join together

Veins: Large (Diameter) vessels formed by merging venules

Microcirculation: Collection of arterioles, capillaries, and venules

Functions

Arteries: Channel for low resistance blood flow due to **Pressure Reservoir**

Pressure Reservoir: Driving force during **Ventricular Diastole** due to elasticity of artery walls **(Elastin)**, which can expand and store pressure

Arterioles: Vascular resistance in circulatory system regulates cardiac output and arterial pressure, both **Intrinsically and Extrinsically**

Capillary Exchange: Exchanges materials between blood and interstitial space

Interstitial Fluid: Same composition as arterial blood

Exchange: Through (1) **Diffusion** and (2) **Bulk Flow**

Diffusion: Blood moving down its concentration gradient

Bulk Flow: Maintains fluid balance between blood and extracellular space; permits flow of plasma **(NOT proteins/blood cells)**

Ultrafiltration: Bulk flow in **tissues**

Reabsorption: Bulk Flow in **capillaries**

Veins: Reservoir for blood and channel for blood flow to heart

Venous Capacity: Volume of blood veins can withstand

Venous Return: Volume of blood entering each atrium per minute

Factors That Influence Bulk Flow

(1) Capillary Blood Pressure (P_C): Pushes fluid from capillaries to the interstitial fluid

(2) Plasma-Colloid Osmotic Pressure (π_p): Draws water into capillaries from interstitial fluid via protein concentration

(3) Interstitial Fluid Hydrostatic Pressure (P_{IF}): Pushes fluid into capillaries from the interstitial fluid

(4) Interstitial Fluid-Colloid Osmotic Pressure (π_{IF}): Draws water out of capillaries to the interstitial fluid

Ultrafiltration: Positive net pressure

Reabsorption: Negative net pressure

Net Pressure: $(P_C + \pi_{IF}) - (P_{IF} + \pi_p)$

Blood Pressure Abnormalities

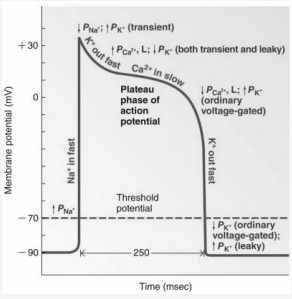
Hypertension : High BP above 140/90 mmHg

Primary Hypertension: 90% of cases, unknown

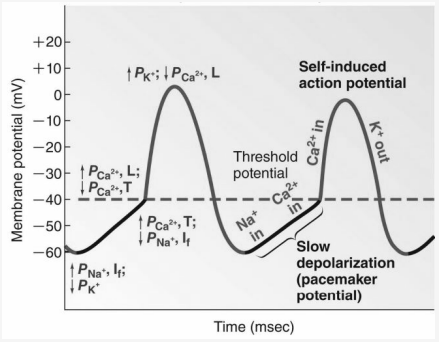
Secondary Hypertension: 10% of cases, occurs 2nd to 1st

Hypotension: Low BP below 100/60 mmHg

Pacemaker Activity of Cardiac Cells



Pacemaker Activity of Cardiac Autorhythmic Cells



Electrical Activity of the Heart (Cont.)

Excitation-Contraction Coupling: Ca^{2+} entry into the cytosol differs from that in skeletal muscle cells

Dyhydropyridine Receptors: Voltage-gated Ca^{2+} channels in cardiac T-Tubules

AP Invades T-Tubule Membranes: *Dyhydropyridine Receptors* open, letting Ca^{2+} enter the cytosol

Ca^{2+} Entry: Triggers more Ca^{2+} release from the SR; both sources of cytosolic Ca^{2+} drive contraction strength based on Ca^{2+} concentration (Proportional to # of cross-bridges)

Electrocardiogram (ECG): Records heart's electrical activity (AP) via surface voltage differences to detect abnormalities

ECG Waveforms (P-Wave): *Depolarization* of the *Atria*

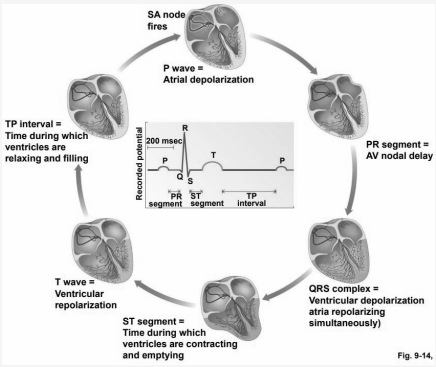
ECG Waveforms (QRS-Complex): *Depolarization* of the *Ventricles*

Electrical Activity of the Heart (Cont.) (cont)

ECG Waveforms (T-Wave): *Repolarization* of the *Ventricles*

ECG Waveforms (PR-Segment): AV Node Delay

Electrocardiogram Waveforms



Heart Sounds

Lub: *Low-pitched*, soft and relative long **(AV Valves closes)**

Dup: *High-pitched*, sharp and short sound **(Semilunar Valves closes)**

Murmurs: Uncommon heart sounds (cardiac disease) from turbulent blood flow through broken valves

Stenotic Valve: **Does NOT OPEN Completely**; Producing **Whistling** sounds

Insufficient Valve: **Does NOT CLOSE Properly**; Producing **Swishing** sounds

Stroke Volume Regulation

Extrinsically: **Sympathetic Nervous System** controls neural activity

Intrinsically: **Venous Blood Return** (Volume)

Function of Both Factors: Increase SV by raising contraction strength

Intrinsic Control: The **Frank-Starling Law of the Heart** (End-diastolic volume and SV)

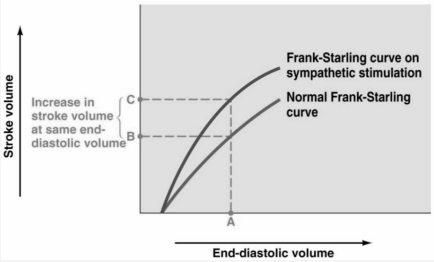
Frank-Starling Curve

Sympathetic Stimulation: Shifts the Frank--Starling curve to the left

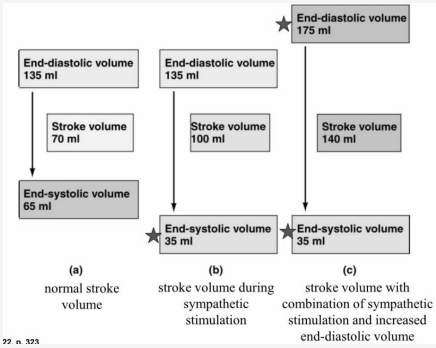
At End-Diastolic Volume: Increase Ca2+, which increases contractile force and SV

Heart Failure: Decrease cardiac contractility and shifts curve downward and to the right

Frank-Starling Curve (Graph)



Volume Differences



Sphygmomanometer

Sphygmomanometer: Used to measure **Systolic** and **Diastolic** pressure by listening to sound of blood

Pressure >120 mmHg: No blood flow and no sound

Pressure between 120-180 mmHg: Turbulent blood flow and intermittent sounds

Pressure <80 mmHg: Smooth blood flow and no sound

Pressure Throughout the Systemic Circulation

Left Ventricular Pressure: Between 0 mmHg **(During Diastole)** to 120 mmHg **(During Systole)**

Arterial Pressure: Fluctuates between 120 mmHg **(Systolic)** to 80 mmHg **(Diastolic)**

Arteriolar Pressure: Large pressure drop **(Systolic to Diastolic convert to Non-Pulsatile Pressure)**

Pressure: Decline at slower rate (When blood flows through capillaries and venous system)

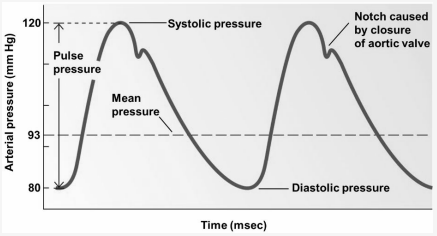
Arterial Pressure

Pulse Pressure: Difference between **Systolic and Diastolic pressure**

Mean Arterial Pressure: Regulated by blood pressure reflexes

Mean Arterial Pressure Diastolic Pressure + 1/3 Pulse Pressure

Arterial Pressure (Graph)



Factors Influencing Venous Return

Sympathetic Activity: Causes vasoconstriction, increasing venous pressure/return

Skeletal Muscle Activity: Skeletal muscles contract, squeezing veins and increases venous pressure

Venous Valves: Prevent backflow (In lumen of large veins)

Respiratory Activity: Chest pressure decreases during respiration, increasing pressure between veins and lower body/chest

Cardiac Suction: Below 0 mmHg during ventricular contraction, increasing venous pressure and pulling venous blood into atria