Lecture 9: (33slides) Homeostasis. Autonomic

1) Autonomic system vs somatic nervous system.

- Effectors : - Somatic nervous systems stimulates skeletal mucles.
    - ANS innervates cardiac muscle, smooth muscle and glands.

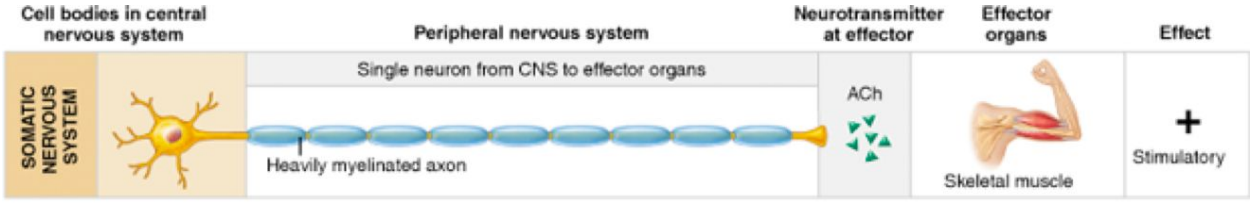
- Efferent pathways and Ganglia.

ANS uses a two-neuron chain to reach it effectors

- The Cell body of the first neuron, the preganlionic neuron, resides in the brain stem or spinal cord Its axon, the preganlionic axon, synapes with the second motor neuron.
- The postganlionic neuron (sometimes called the ganglionic neuron) is the second motor neuro. It cells body is in an autonomic ganglion outside the CNS. ITs axon , the post ganlionic axon, extends to effector organ.
  Preganglionic are thin, postganglionic are thinner.
  So, Conduction throught the autonomic efferent chain i slower than in somatic motor system. For most of theri course, may preganlionic and postganglioninc fivers are incroporateed into spinal or cranial nerves.
    - A. ganglia are motor ganglia, containing the cell bodies of motor neurons. Technically, they are sites of synapse and information .

Neurotransmitter Effects:

- Somatic motor neurons release acetylcholine (ACh)



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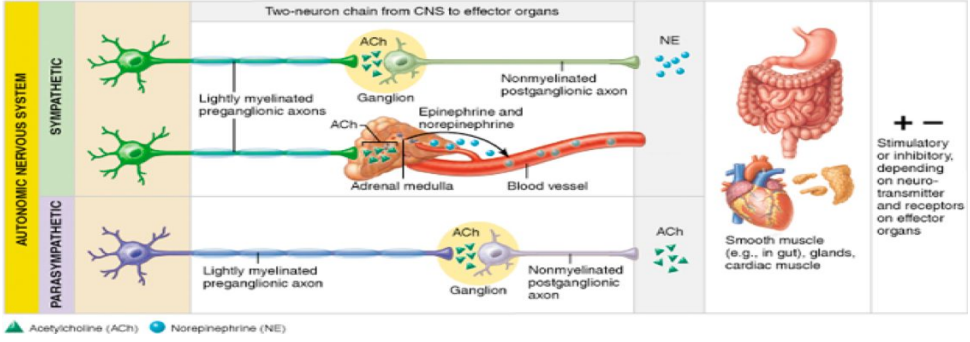
- ANS 2 neurontransmitter - norepinephrine (NE) : sympathetic fibers
    - ACh : by parasympathetic fibers.



Depends on the receptors on the target organ, the effect maybe excitatory(inrease) or inhibitory (decrease).

Homeostasis

DEF : ability of the body to maintain a relatively constant internal environment with changing external conditions. (Walter Cannon)
Why : - Cells and components of cells work best when specific conditions are maintained (optimal temperature, pH, O2, etc)
Differnt organ system work co operatively to promote the well being of the entire body and to maintain stable internal conditions.

Negative feedback Mechanisms

- Push the variable in the direction opposite to the original change.
- Set point is the numerical value measured at steady rate
- Minzimees changes form the set point of the system =>> Stabiility
- Exp : - body temperature (sweat glands + skeletal muscle0 , blood sugar.

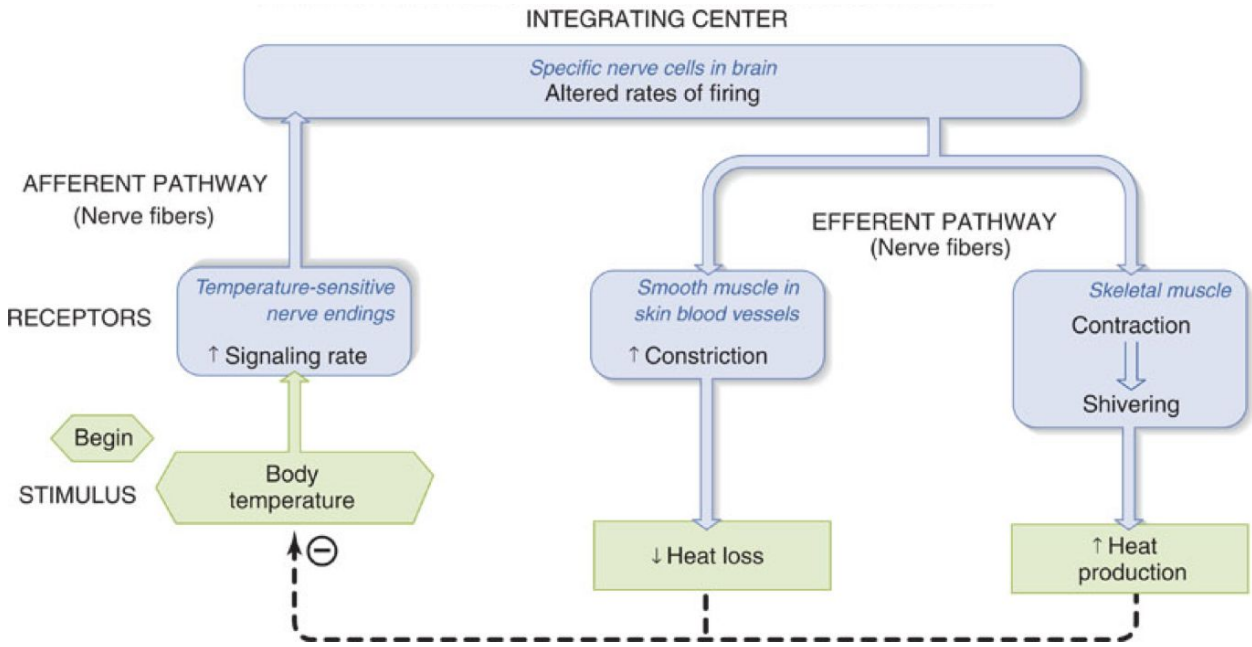
Positive feedback Mechanisms

- Accelerate a process, less common, push values away from the set point
- Exp: Blood clot, (platelet release chemicals to attract more platelets,), contraction in prenant woman.

Components of Homeostatic control system

- Multiple organs control 1 variable
- Singal relayed by : Nervous system + endocrine system
- Homeostasis is an example of reflex. (involuntary response to a particular stimulus), usually without our awareness.

- Reflex arc : pathway describing a reflex. (consists of receptor, afferent pathway, integrating center, efferent pathway, and effector.)



ANS (Start from slide 18)

Consists 2 part

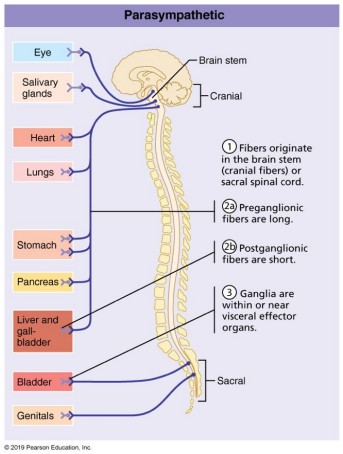
Parasympathetic division : - promotes maintenance functions, conserves energy.

Sympathetic division : - mobilizes body during activity.

Dual innervation: all visceral organs are served by both divisions , but these cause opposite effects.

- Function thi on roi
- Anatomical differences :

- Parasympathetic : - Origin: Craniosacral, orginate in brain stem and sacral spinal cord
  - Has long preganlionic and short post ganlionic fibers
  - Location of gangiia : Visceral effector organ



-Sympathetic: - Origin: - thoracic and lumbar regions of snial cord.
- Has short preganglionic and long posligangonic fibers
- Ganlia close to spinal cord.

- Sympathetic tone: - Blood vessel smooth muscle only ⇒ BP, constriction
  - Shunt blood, minimize injury.
  - Thermo regulation
  - Release of renin from kidney
  - Metabolic effects.

-Parasympathetic tone: - Dominate heart and smooth muscle of urinary tract organs, activate glands , NOT andrenal or sweat glands.
- Slow the heart,
- invlove in digestive and urinary tract.

Sympathetic division can be block by stress

Parasympathetic : - elicit short- lived and highly localized control over effectors. (ACh is quickly destroyed by acetylcholinesterase)
Sympathetic : Longerlasting with body wide effects

- NE is activated more slowly than ACh
- NE and epinephiine
- Take time to “come down” after traumatic event.

Levels of ANS control

1) Hypothamic controls

- Main integrative center of ANS activity
- Anterior : Parasympathetic functions, posterior-sympathetic
- Direct or indirect control through reticular system or spinal cord.
- Center of hypothalamus control : - heart activity, BP, body temperature, water balance, endocrine activity\
  - Emotional responses (rage, fear, pleasure) acitvated throught limbic system signal hypothalsus to activate fight or flight system

-2) Brain stem and spinal cord
- Brain stem reticular formation- direct influence over ANS
- Medullary centers regulate heart rate and blood vessel diameter
- brain controls muscles of pupil and lens
- Spinal cord controls deficatino and micturioing (peeing) - conscious override.

3) Cortical control

- May modify ANS but does so subconsciously
- Works through limmbic system structures on hypothalamic centers
- Voluntary cortical control of some visceral activities is possible

DISORDERS of ANS

- Hypertension : - Overactive sympatheic vasoconstircto rresposne to stress
    - Heart must work harder ,and artery wall are subjected to increase wear and tear
    - Can be treated with adrenergic receptor blocking drugs
- Ulcers : - Increase in sympathetic vasoconstrictor response to stress
    - Decreases blood flow to stomach wall.

Homeo stattic imbalance

- Autonomic neuropathy : damage to autonomic nerves that is a common complication of diabetes mellitus
- Sexual dysfunction in male (75% of males)
- Dizziness after standing, urinary incontinednce, sluggins eey puipl reactions, impaired sweating.

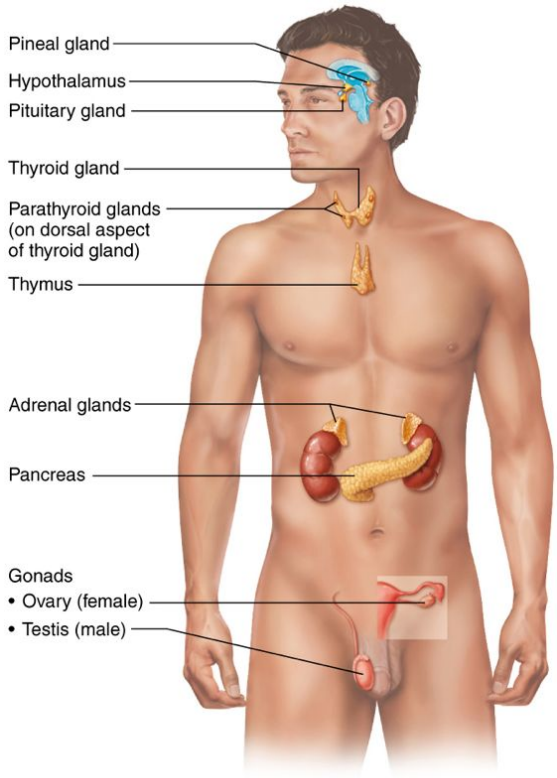
Maintain blood glucose levels -====> prevent diabetic neuropathy.

Lecture 10: Endocrine system.

- 16.1 The endocrine system is one of the body's two major control systems
- 16.2 The chemical structure of a hormone determines how it acts
- 16.3 Hormones act through second messengers or by activating specific genes
- 16.4 Three types of stimuli cause hormone release
- 16.5 Cells respond to a hormone if they have a receptor for that hormone
- 16.6 The hypothalamus controls release of hormones from the pituitary gland in two different ways
- ** Includes Focus Figure 16.1

Table 16.1 Comparison of Nervous and Endocrine Systems

NERVOUS SYSTEM	ENDOCRINE SYSTEM
Initiates responses **rapidly**	Initiates responses **slowly**
Short-duration responses	**Long-duration** responses
Acts via action potentials and **neurotransmitters**	Acts via **hormones** released into the blood
Acts at **specific locations** determined by axon pathways	Acts at **diffuse locations**—targets can be anywhere blood reaches
Neurotransmitters act over very **short distances**	Hormones act over **long distances**



Endocrine galnds

- Pitutarry
- Thyroid

- Parathyroid
- Adrenal
- Pineal glands.

Function : - reproduction, growth and development, regulation of cellular metabolism and energy balance, mobilization of body defenses.
Exocrine and endocrine: Pancreas, gonads, placenta.

Hormone

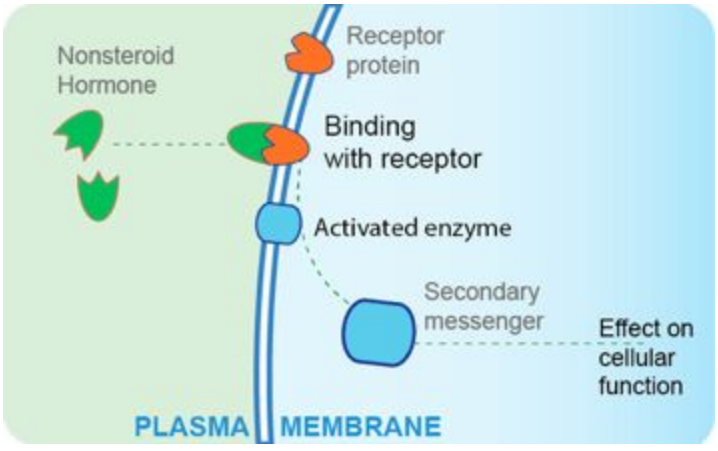
Def: - Long distance chemical signals, travel in blood or lymph.
Two main classes:

1) Amino acids : peptides and proteins
2) Steroids : synthesized from cholesterol (Gonadal and adrenocortical hormones)

Target and signal

- Target cell is affected like this:
  *) Alter plasma membrane permeability and membrane potiential\
  *) Stimulate gene transcription and protein translation
  *) Activate/ deactivate enzymes (via phosphorylation.)
  *) Induce secretory activity.
  *) Stimulate mitosis.

  2 types of hormone:
  Water soluble (aa- based except thyroid hormones)
  - Cannot enter cell

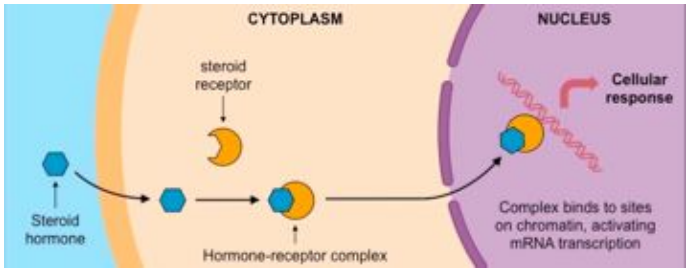


  - Act on PM receptors---> 2nd messenger
  - Eg: cAMP; PIP (4,5)- Ca2+, cGMP, etc (try to remember this.)

Lipid soluble

- Can entercell
- Act on intracellular receptors ----> directly activate genes.

Mechanism of Lipid soluble Hormone

- 

- Steroid hormone go through the plasmamembrane.
- Bind with the receptor inside the cell.
- Both of them go to the nucleus ===> DNA, alter the transcription process.

Blood level of hormones controlled by **negative feedback system feed back systems.**

- Increased hormone on target organ inhibit further hormone.
- Levels vary within narrow, desirable range.

Target cell specificity.

- Target cells must have specific receptoprs to which hormone binds.
- 3 factors affect cell activation : - blood levels of hormone
  - Relative nO of receptors
  - Affinity (sternghth) fo binding between receptor and hormone

Level of hormone affects number of receptors a cell have

Up reglation : - cells form more hormone cuz of low hormone level

Down reguaition : - cells lose receptors cuz of high hormone level .

Lipid soluble vs Water soluble

Table 16.2 Comparison between Lipid- and Water-Soluble Hormones

	LIPID-SOLUBLE HORMONES	WATER-SOLUBLE HORMONES
Consist of	All steroid hormones and thyroid hormone	All amino acid–based hormones except thyroid hormone
Sources	Adrenal cortex, gonads, and thyroid gland	All other endocrine glands
Stored in secretory vesicles	No	Yes
Transport in blood	Bound to plasma proteins	Usually free in plasma
Half-life in blood	Long (most need to be metabolized by liver)	Short (most can be removed by kidneys)
Location of receptors	Usually inside cell	On plasma membrane
Mechanism of action at target cell	Activate genes, causing synthesis of new proteins	Usually act through second-messenger systems

Anterior pituitary hormone produce 6 types of hormones

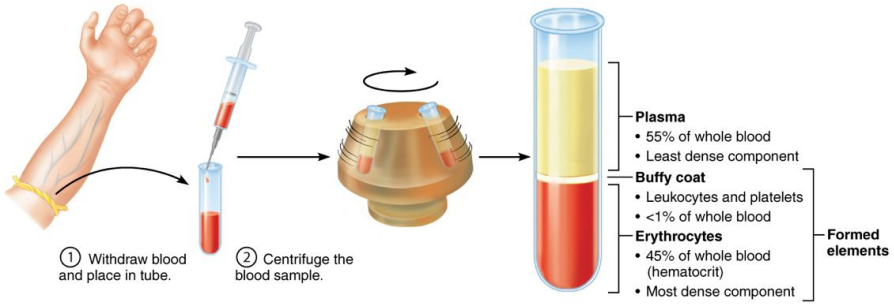
- **Growth hormone** (**GH**)
- **Prolactin** (**PRL**)
- **Thyroid-stimulating hormone** (**TSH**)
- **Adrenocorticotropic hormone** (**ACTH)**
- **Follicle-stimulating hormone** (**FSH**)
- **Luteinizing hormone** (**LH**)

Lecture 11: Blood 1.

- Funtion of blood : - transport (O2, nutrients, waste, Co2), Regulation (temp, pH, fluid volume), Protection (prevents blood loss, infection - antibody) .
- Plasma : non living fluid matrix
- Formed elements.
  ===> enclosed in plamsa, have definite structure and shape.
  ===> Cells are suspended in plasma.
  Formed elements : - enrythrocytes (RBCs)
  - Leukocytes (WBCs)
  - Platelets

Composition of blood

- Hematocrit (about 45%) - red color (most dense component
- Buffy coat 1% (Leukocytes and platelets)
- Plasma (55%- white thing)



Physical properties

Sticky , opaque fluid with metallic taste
High in o2: scarlet red
Low in O2 : Dark red
pH: 7.35-7.45
More **dense than water** and **5x viscosity** (due mainly to RBCs)
Makes **8% of body weigh**t.
Average volume : 5-6 L
4-5 L

Blood plasma

- Straw colored, sticky fluid
- 100 dissolved solutes
- Nutrients, gases, hormones, wastes, proteins, inorganic ions
- Plasma proteins are most abundant solutes
  Remain in blood, not taken up by cells
  Proteins produced mostly by liver
  Albumin: makes up 60% of plasma proteins
    - Function as **carrier** of other molecules, **buffer, contributes to plasma osmotic pressure, which helps to keep water in the blood stream.**

Formed elements

- Enthrocytes, WBCs and plateltes
- **Only WBCs are complete cells**
- RBCs have no nuclei or other organelles
- Platelets are **cell fragments**
- **Most formed elements survive** in the bloodstream **for only few days.**
- **Most blood cells originate in bone marrow** and do not divide.

Enthrocytes

- Diameter (7.5um) , contribute to gas transport
- Biconcave disc shape, is anucleate, no organelles
- Contains hemoglobin (Hb) for gas transport
- RBC diameters are larger than some capillaries
- Spectrin and other plasma membrane protein provides flexibility to change shape.
- RBcs have no mitochondria,
- ATP production is anaerobic, do not consume O2 they transport.
  Function
- Gas transtot
- M ; 13-18 g /100 ml of blood, F: 12-16g/100 ml

-

  F :12-16 g/100ml
- Each Hb mole transport 4 O2
- Each RB contains 250 million Hb molecules
- O2 loading in lungs produce **oxyhemoglobin**
- **O2 unloading in tissues produce deoxyhemolglobin**
- **Carbaminohemoglobin.**

- Hematopoiesis : - formation of all blood cells.

Erythropoiesis: Process of formation of RBCs-15 days

- >2 mil RBCs are made every second

Enrythrocyte Disorders

Lecture 14:

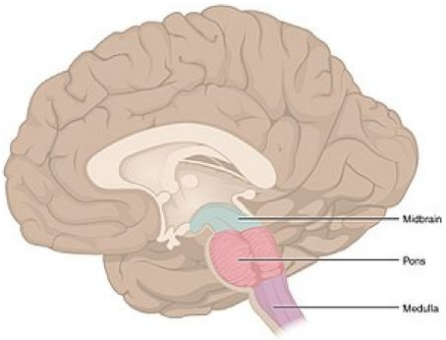
Need to remember: - Extrinsic Innervation

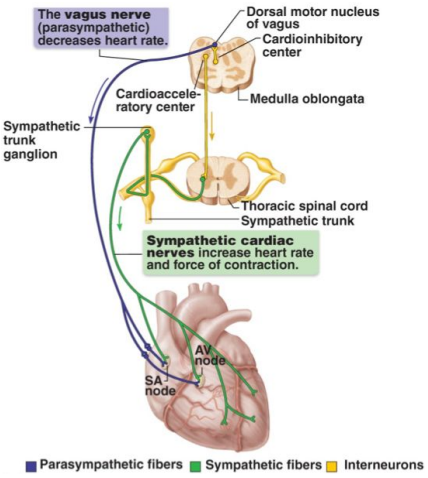
- ECG tracing and the nature of the information it is providing
- Events occurring during each phase of the cardiac cycle.
- Mechanism for the regulation of heart rate and stroke volume.

Start the review

- Extrinsic innervation

- Modifying the basic Rhythm of the heart
- Heartbeat is modified by ANS via cardiac centers in the medulla oblongata.
- Medulla oblongata :Inferiormost part of the brain stem.



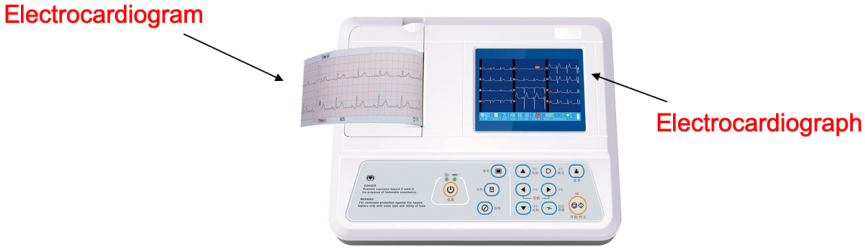


- Cardioacceleratory center: - Send signals through sympathetic trunk to increase both rate and force. Stimulates SA and AV nodes, heart muscle, and coronary arteries
- Cardio inhibitory center: - Parasympathetic send signals via vagus nerve to decrease rate

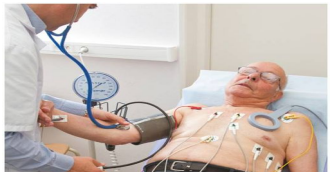
Electrocardiography

- Electrocardiograph: - Can detect electrical currents generated by heart.
- Electrocardiogram: - (ECG or EKG) is a graphic recording electrical activity.
    - A composite of all action potientials at given time (not a tracing of a single AP.)

How it work : - Electrodes are placed at various points on body to measure voltage differences , 12 lead ECG is the most typical electrode.

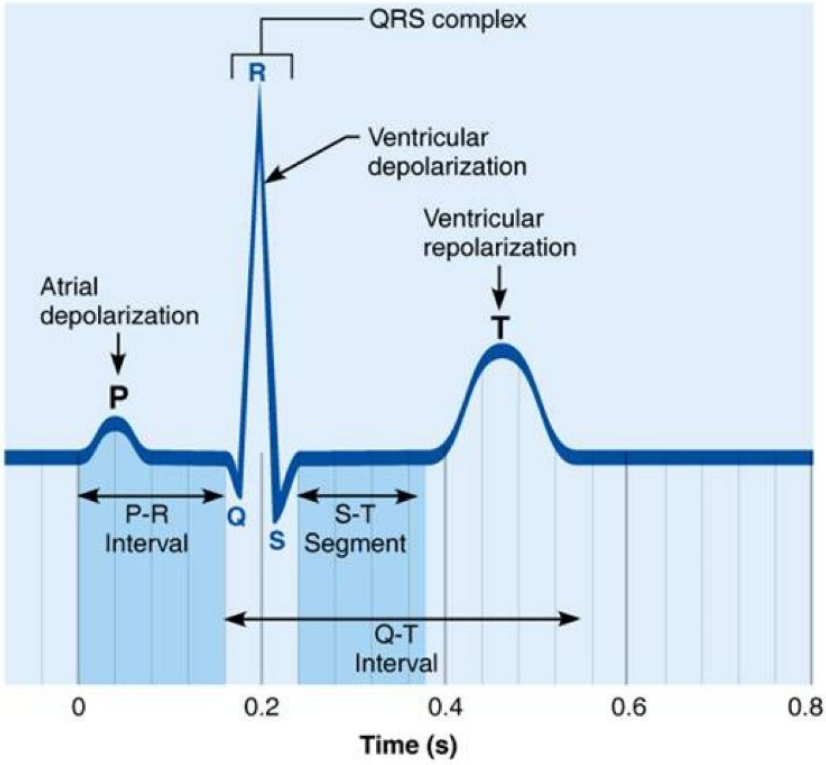


- Electrocardio graph : (not just a lead clip onto the finger.)



- Features of the Electrocardiograph.

-P wave: Depolarization of SA node and atria.
-QRS complex: ventricular depolarization and atrial repolarization.
-T wave: - ventricular repolarization.
- P-R interval : beginning of excitation
- S-T segment : - entire ventricular myocardium depolarized.
- Q-T interval: beginning of ventricular depolarization through ventricular repolarization.



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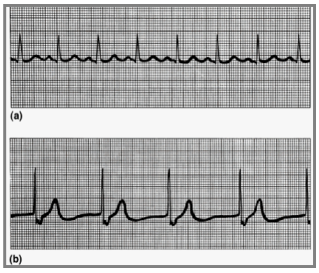
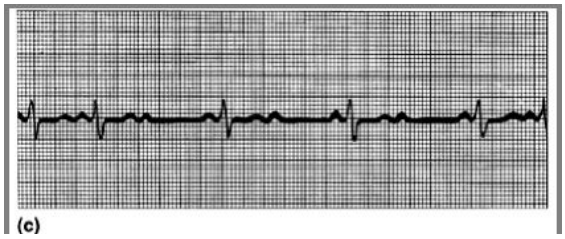
Electrocardiogram issues:

Normal/healthy

Junctional Rhythm



- Junctional Rhythm : SA node non functional. AV node takes over and paces heart at 40-60 beats per minute. (P waves absent)



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- Second degree heart block : - AV nodes fails to conduct some SA node impulses. More P values than QRS waves. In this tracing there are usually two P waves for each QRS.
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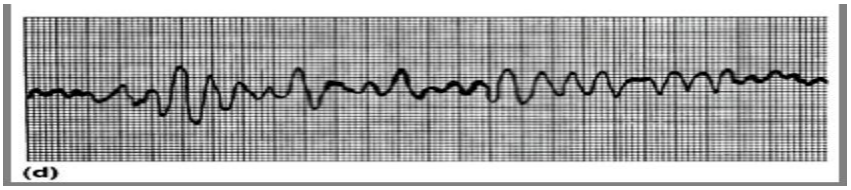


- Ventricular fibrillation: electrical activity is disorganized. Action potentials occur randomly throughout ventricles.
- =>>> results in chaotic grossly abnormal ECG deflections, seen in acute heart attack and after electrical shocks.

Mechanic of heart function

- Systole: period of heart contraction
- Diastole : period of relaxation
- Cardiac cycle : blood flow through heart during one complete heartbeat.

Phase of cardiac cycle

1. Ventricular filling: mid-to-late diastole

   Pressure is low; 80% of blood passively flows from atria through open AV valves into ventricles from atria (SL valves closed)

Atrial depolarization triggers atrial systole (P wave), atria contract, pushing remaining 20% of blood into ventricle

End diastolic volume (EDV): volume of blood in each ventricle at end of ventricular diastole

Depolarization spreads to ventricles (QRS wave)

Atria finish contracting and return to diastole while ventricles begin systole

2. Isovolumetric contraction

Atria relax; ventricles begin to contract

Rising ventricular pressure causes closing of AV valves

Isovolumetric contraction phase is split-second period when ventricles are completely closed (all valves closed), volume remains constant, ventricles continue to contract

When ventricular pressure exceeds pressure in large arteries, SL valves are forced open

Pressure in aorta reaches about 120 mm Hg

-

3. Isovolumetric relaxation: early diastole

Following ventricular repolarization (T wave), ventricles relax

End systolic volume (ESV): volume of blood remaining in each ventricle after systole

Ventricular pressure drops causing backflow of blood from aorta and pulmonary trunk that triggers closing of SL valves

Ventricles are completely closed chambers momentarily

Referred to as isovolumetric relaxation phase

4. Closure of aortic valve raises aortic pressure as backflow rebounds off closed valve cusps

Referred to as dicrotic notch

Atria continue to fill during ventricular systole and when atrial pressure exceeds ventricular pressure, AV valves open; cycle begins again

Heart beats around 75 times per minute

Cardiac cycle lasts about 0.8 seconds

Atrial systole lasts about 0.1 seconds

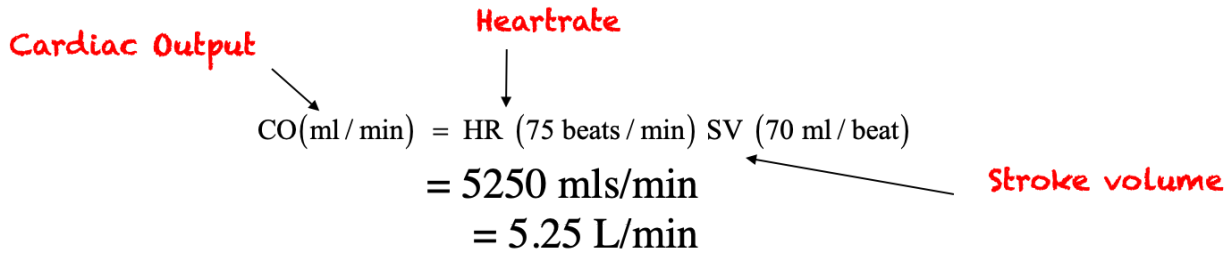
Ventricular systole lasts about 0.3 seconds

Quiescent period is total heart relaxation that lasts about 0.4 seconds à BACK TO TOP

Regulation of heart pumping.

- Cardiac output: amount of blood pumped out by a single ventricle in 1 minute. Equals heart rate (HR) times stroke volume (SV)
- Stroke Volume : - volume of blood pumped out by one ventricle with each beat,

Calculation formula : - -



- Maximal CO is 4-5 times resting and reserve cardiac output.
- Maximal CO may reach 35L/min in trained athletes.
- Cardiac reserve : - difference between resting and maximal CO.

STROKE VOLUME THINGY

- EDV (End diastolic volume) : - amount of blood flowing into the ventricles when relaxed.
- ESV (End systolic volume.): - the volume of blood in a ventricle at the end of contraction, or systole, and the beginning of filling, or diastole. ESV is the lowest volume of blood in the ventricle at any point in the cardiac cycle.
- SV: Systolic volume

$$SV = EDV - ESV$$

EXP:

- EDV is affected by the length of ventricular diastole and venous pressure at 120ml/beat
- ESV is affected by arterial BP and force of ventricular contraction at 50ml /beat
- SO normal SV = EDV-ESV= 120ml-50ml = 70ml/beat

Regulation of stroke volume by preload

- Preload: - degree to which cardiac muscle cells are stretched just before they contract.
- Changes in preload causes changes in SV because : - Affects EDV
- Cardiac muscle exhibits a length-tension relationship.
- At rest, cardiac muscle cells are shorter than optimal length; leads to dramatic increase in contractile force.

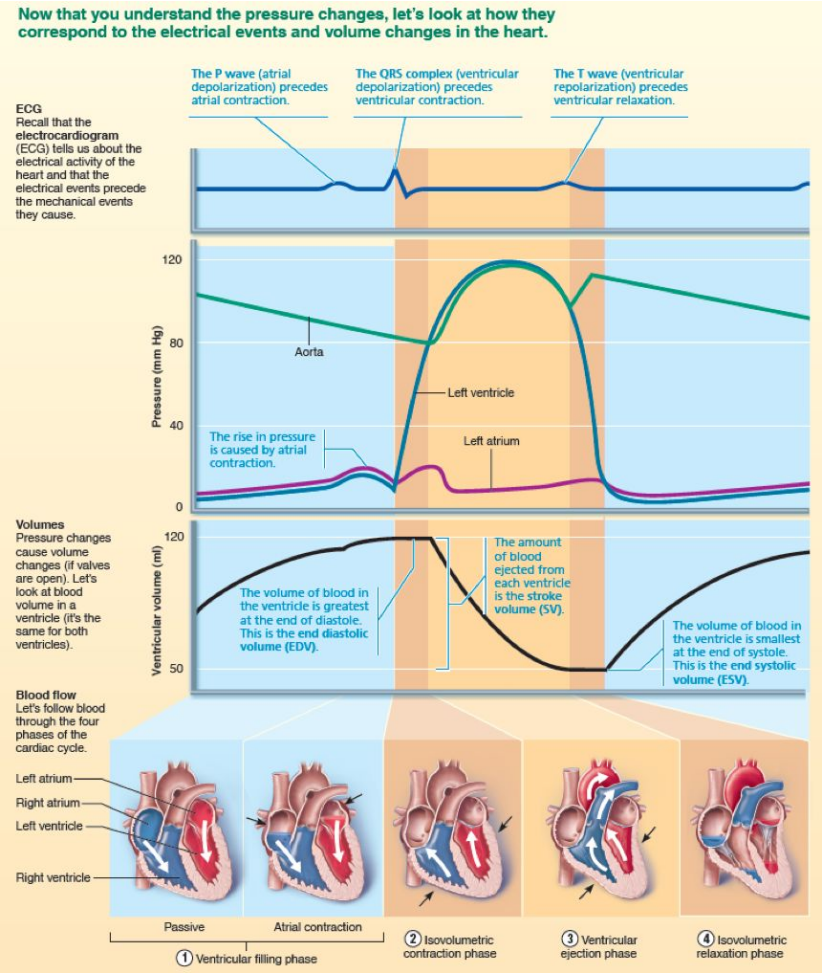
- Frank- Starling law of the heart :

- DEF: - Relationship between preload and SV.
- Rules : - Within defined limits, the heart will pump whatever volume of blood it receives.
    - Over a fairly wide range, there is a proportional relationship between EDV and stroke volume.

- Factors that regulates preload : - Venous turn : amount of blood returning to heart.
  ⇒ - SLow heartbeat and exercise increase venous return.
- Increased venous return distends (stretches) ventricles and increases contraction force.

Regulation of stroke volume by contractility (Contractile strength at given muscle length)

- Independent of muscle strength and EDV
  Mechanism;
    - Increased contractility lowers ESV, caused by : - Sympatric Epinephrine release stimulates ⇒ Increased Ca2+ influx => more cross brigde formations
    - Positive intropic agents increase contractility- •- Thyroxine, glucagon, epinephrine, digitalis, high extracellular Ca2+



- Decreased by negative inotropic agents: - Acidosis (excess of H+), increased extracellular K+, calcium channel blockers.

- First phase: - Passive
- Second phase : - Atrial contraction
- Isovolumetric contraction phase.
- Ventricular ejection phase
- Isovolumetric contraction phase.

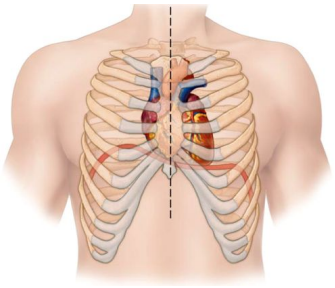
LECTURE 13

4.2.1. internal and external anatomy of the heart

4.2.2. Trace the pathway followed by blood in both the pulmonary and systemic circuits
4.2.3. organization of the coronary circulation
4.2.4. Compare the physiological properties of cardiac muscle cells vs those of skeletal muscle cells
4.2.5. Compare the electrical properties of contractile cardiac muscle cells vs those of autorhythmic cardiac muscle cells
4.2.6. Explain how the intrinsic conduction system of the heart allows it to function as a pump.

- Electrical mechanism of the heart.

- Size, location and Orientation of Heart
- Size: Approximately the size of a fist.
- Location:
- In mediastisnum (e*mbranous partition between two body cavities or two parts of an organ, especially that between the lungs*) between second rib and fifth intercosal space

- On superior surface of the diaphragm
- Two-thirds of heart ot left of midsternal line
- 

- Anterior to vertebral column, posterior to sternum

Layers of the heart

- Pericardium : - fibrous pericardium :- protects heart, anchor heart, prevents overfilling
    - Serous pericardium: - parietal and visceral (epicardium) layers

- Myocardium : - the bulk of the heart : branching cardiac mucle cells arranged into bundles and the connective tissue wrappings of these bundles.
- Endocaridum : Inner layer of the heart wall
- Layer of endothelium + connective tissue layer on inner myocardial surface.
- Continousous with endothelum of vessels leaving and entering the heart.

Electrical mechanism of the heart
- Automaticity : - bv
- SA node : Sinoatral node
- Pace makerc cells : they can delpolarize by themselves, send sigals through Bachman's Bundle.
- All neighborign cells also delpolarize at well.
- Both RA in LA in a cordinated way.
- AV node : atrial ventricular node : only connection between atria and ventrical.
- Internodal tracts connect SA node and AV node.
-From AV node : - Watch the atria and see how they contract.
There is a tiny delay between atria contration and ventricle contraction (due ot AV node) .(a tenth of the second.)
    - Bundle of HIs (HISSSS).
    - Lots of high way.

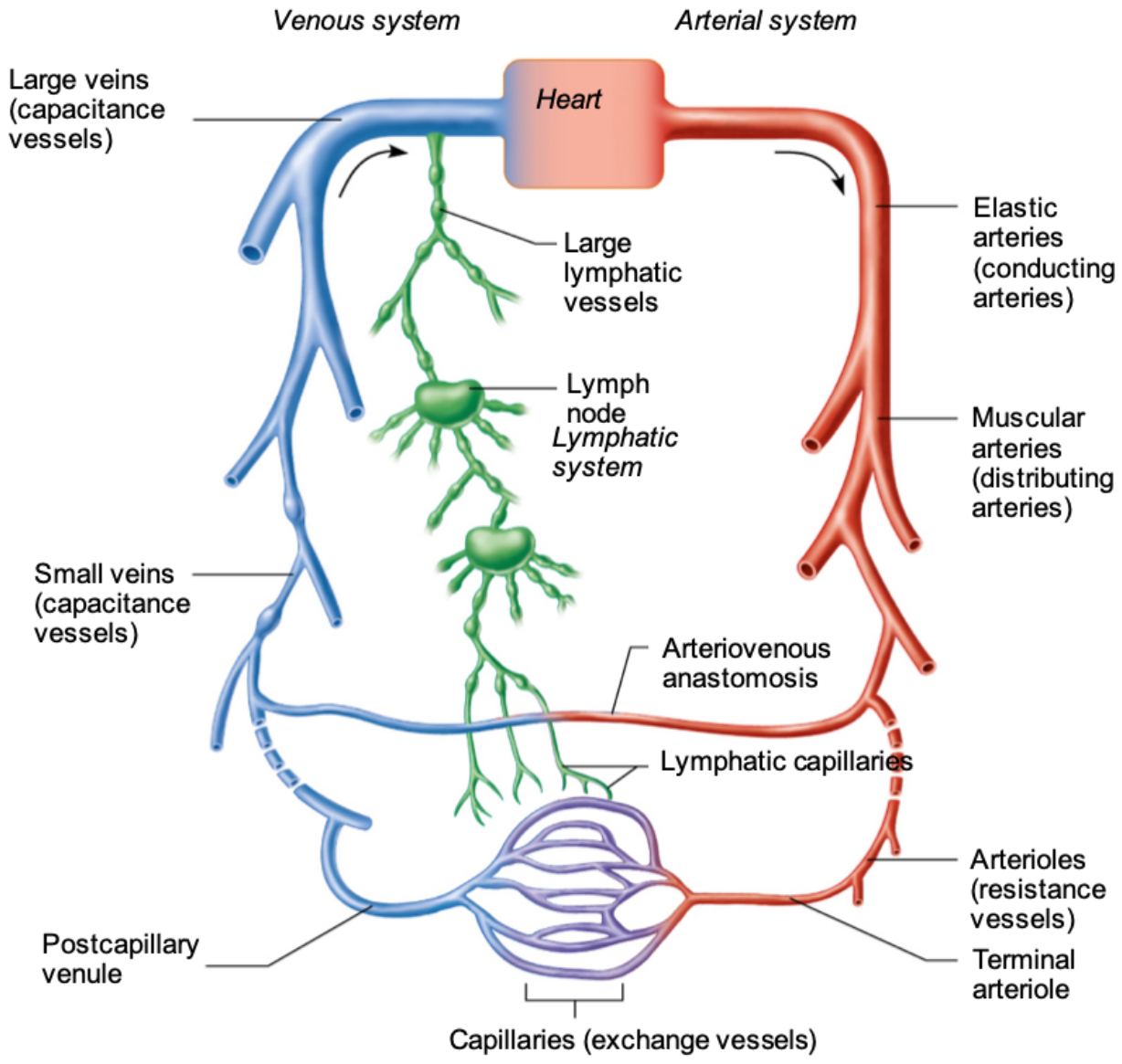
Lecture 15: blood vessels and human dynamic (blood flow.)

- 4.3. Blood vessels and hemodynamics:
- 4.3.1. Compare and contrast the structure of the walls of arteries, capillaries and veins
- 4.3.2. Compare the 3 types of arterial vessels
- 4.3.3. Define microcirculation and compare the 3 types of capillaries
- 4.3.4. Describe the structure and functions of the venules and veins

- 4.3.5. Define blood flow, blood pressure, resistance, peripheral resistance

BLood vessels : - Delivery sustem of dynamic structures tha tbegins and ends at heart
Arteries : carry blood away from the heart

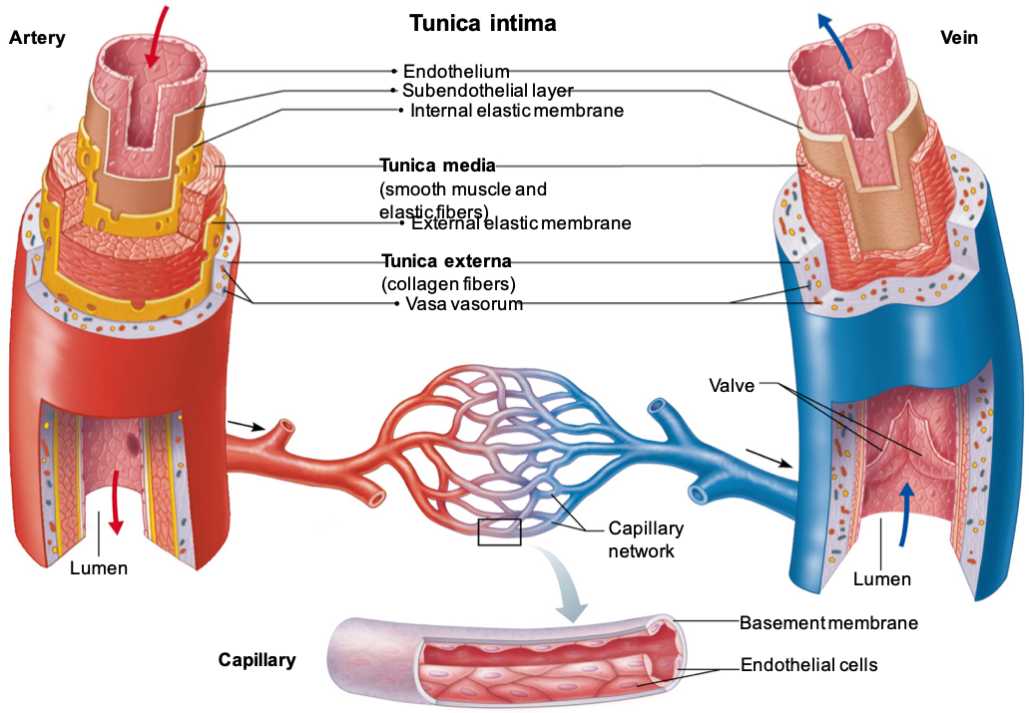
Capillaries : direct contact with tissues cells, directly serve cellular needs.
Veins : - carry blood toward the heart (small, grow from capillaries.) , bood high in CO2



- Proportion of blood in the body.

Types of blood vessels- 1) Tunica intima: intimately associadte with blood

2) Tunica media : In the middle



3) Tunica externa : on the external of blood vessels.

- Capillaries is very small : even a single cell can fit in.

    - The dots call vasa vasorum : system of tiny blood vessels found in larger vessels

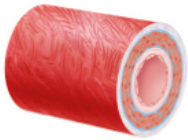
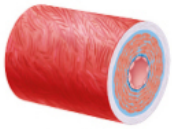
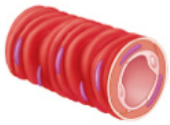
    - Details: (same as on previous slide – you may find it easier to study looking at the diagrams, so I included it twice)
    - 1. Tunica intima
    - Innermost layer that is in “intimate” contact with blood
    - Endothelium: simple squamous epithelium that lines lumen of all vessels
    - Continuous with endocardium
    - \
    - Slick surface reduces friction
    - *Subendothelial layer*: connective tissue basement membrane
    - Found only in vessels larger than 1 mm
    - 2. Tunica media

- Middle layer composed mostly of smooth muscle and sheets of elastin
- Sympathetic vasomotor nerve fibers innervate this layer, controlling:
- Vasoconstriction: decreased lumen diameter
- Vasodilation: increased lumen diameter
- Bulkiest layer responsible for maintaining blood flow and blood pressure
- 3. Tunica externa
- Outermost layer of wall
- Also called *tunica adventitia*
- Composed mostly of loose collagen fibers that protect and reinforce wall and anchor it to surrounding structures
- Infiltrated with nerve fibers, lymphatic vessels
- Large veins also contain elastic fibers in this layer
- Vasa vasorum: system of tiny blood vessels found in larger vessels
- Function: to nourish outermost external layer

Different types of arteries and veins

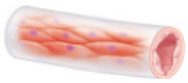
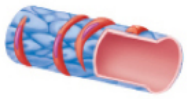
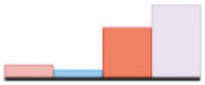
There are different types of arteries and veins

Table 19.1 Summary of Blood Vessel Anatomy

VESSEL TYPE/ ILLUSTRATION*	AVERAGE LUMEN DIAMETER (D) AND WALL THICKNESS (T)	RELATIVE TISSUE MAKEUP			
		Endothelium	*Elastic Tissues*	*Smooth Muscles*	*Fibrous (Collagenous) Tissues*
ARTERIES					
 Elastic artery	D: 1.5 cm T: 1.0 mm				
 Muscular artery	D: 6.0 mm T: 1.0 mm				
 Arteriole	D: 37.0 µm T: 6.0 µm				

*Size relationships are not proportional. Smaller vessels are drawn relatively larger so detail can be seen. See column 2 for actual dimensions.

Table 19.1 Summary of Blood Vessel Anatomy

VESSEL TYPE/ ILLUSTRATION*	AVERAGE LUMEN DIAMETER (D) AND WALL THICKNESS (T)	RELATIVE TISSUE MAKEUP			
		Endothelium	*Elastic Tissues*	*Smooth Muscles*	*Fibrous (Collagenous) Tissues*
CAPILLARIES					
	D: 9.0 µm T: 0.5 µm				
VEINS					
 Venule	D: 20.0 µm T: 1.0 µm				
 Vein	D: 5.0 mm T: 0.5 mm				

*Size relationships are not proportional. Smaller vessels are drawn relatively larger so detail can be seen. See column 2 for actual dimensions.

- The interior of the vein is a biiger space, the opening is larger, u can fit the blood in.
- Cross section : - Thick wall for artery, thin wall fro vein. (because arterie have to pumb blood)
- Arteries has thicker wall (under high pressure) ; Veins can be a bit thinner without losing its function.

Arteries:

- Elastic arteries : - largest vessels in the body, highly elastic
    - have smooth muscle, push out the beating of the heart itself.
    - Take the blood right out of the heart.

- Muscle arteries : - Have more smooth muscle, less elastic tissue.
-Can contract to regulate blood flow around the body.

- Arterioles : - Can change dimateres change resistance to blood flow.
- Lead to capillary beds. (smaller smaller smaller to capillary beds.)
-

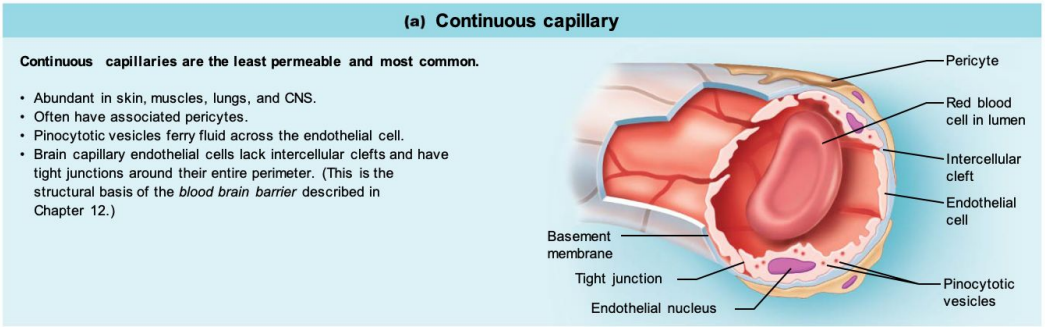
Elastic Arteries → Muscular Arteries → Arterioles

Capillaries

- Dumping off waste, pick up o2 and vice versa
- Walls just thin tunica intima: in smallest vessels, one cell forms entire cicumference.
- Pericytes : spider shaped stems cells help stabilize capialry wall, control permeabiltty a , and play a role in vessel pair

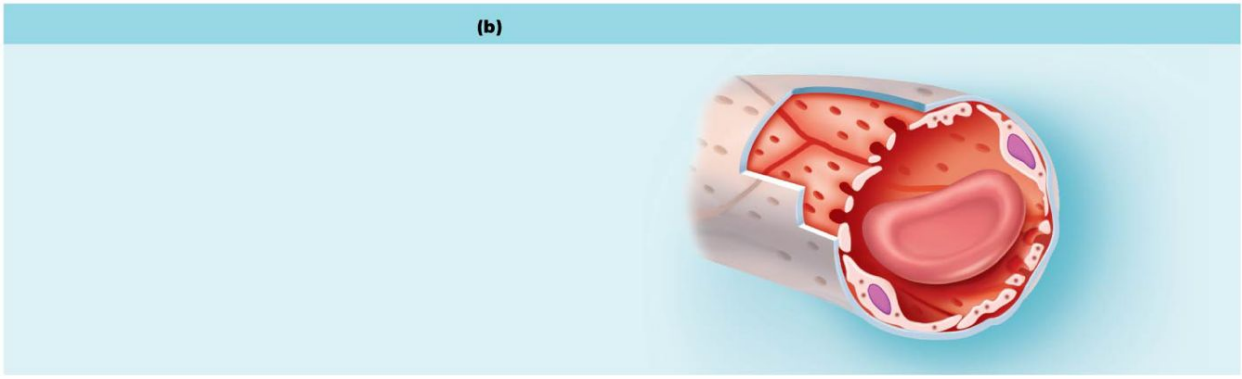
3 types

1) Continuous capillaries



- Most capillaries in the body.
- Very permeable

2) Fenestrated capillary :



- Allow even larger thing to pass through. (kidney, guts)

3) Sinusoidal.
   - Same size. Larger hole. Found in specialized region of the body. Allow bigger cells to go in and out of the

Permeability : 1 < 2 < 3.

Capillaries bed (where aterioles and venules meet)

- Individual cells.
- Exchange of gas
- The color represent what happening.

Issues with capillaries

- 1) Vascular shunt : channel that directly connects ateriole with venule (bypasses true capillaries) - consists of metarteriole and through fare channel
2) Precapillary Sphincter : cuff of smooth muscle surrounding each true c
These are reponsive chemiclas stimulation

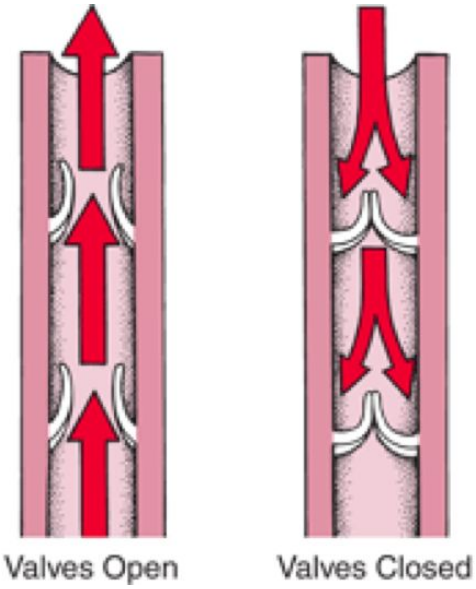
Veins : carry blood toward the heart.

- Have all tunics, but thinner walls, with large lumes compared with corresponding arteries
- Tunica media is thin, but externa is thick.

Venules
- •Capillaries unite to form *postcapillary venules*

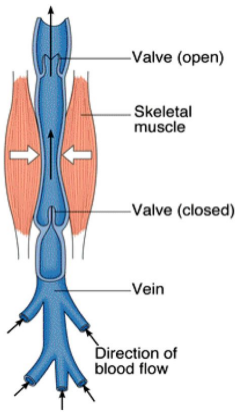
- –Consist of endothelium and a few pericytes
- –Very porous; allow fluids and WBCs into tissues
- •Larger venules have one or two layers of smooth muscle cells
- formed when venules converge

Regulation of flow through arteries and veins



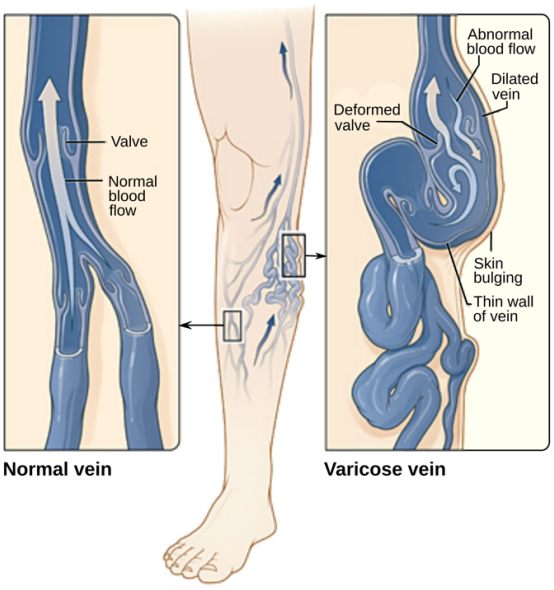
- Venous valves system : - Prevent backflow of blood
  - Most abundant in veins of limbs

- Muscle helps blood flow through veins



ISsues with blood vessels

- Varicose veins : - Blood cooling in the legs and arms\



Anastomoses

- Vascular anastomoses : - interconnections of blood vessels.
- Arterial anastomoses : - Provide alternate pathways

LECTURE 12:

- 17.6 Hemostasis prevents blood loss
- 17.7 Transfusion can replace lost blood

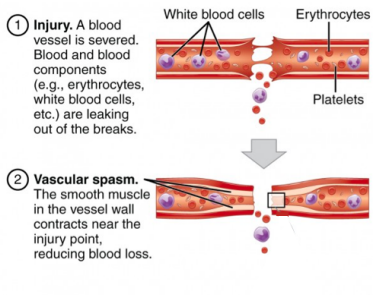
- Homeostasis: - Stopping of bleeding. - If a blood vessel breaks, a whole series of reactions is set in motion to accomplish this thing.
- Response is fast, localized and highly regulated.
- Requires clotting factors and substances released by platelets and injured tissues.

1) Vascular spasm
2) Platelet plug formation
3) Coagulation (blood clotting)

Clot retraction occurs after hemostasis.

3 steps :

1) Vascular spasm (trigger by direct injury to vascular smooth muscle, chemicals released by endothelial cells and platelets and pain reflexes)
    - Vasoconstriction: Smooth muscle in small blood vessels wall contracts, reduce the amount of blood flow.
    - Most effective in small blood vessels
    - Significantly reduce blood flow until other mechanisms kick in.



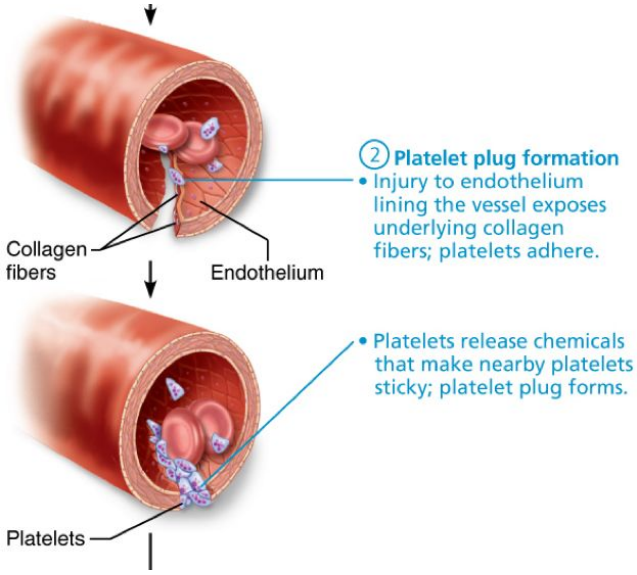
2) Platelet Plug formation.

Mechanism: - Platelet tick to exposed collagen fibers of damage vessels.

- Von Willebrand Factor (VWF): helps to stabilize platelet- collagen adhesion.
- Platelet activation : - Swell become spiked and sticky, release chemical messengers
- ADP: - Cause more platelets to stick and release their contents
- Serotonin, thromboxane A2: enhance vascular spasm and platelet aggregation

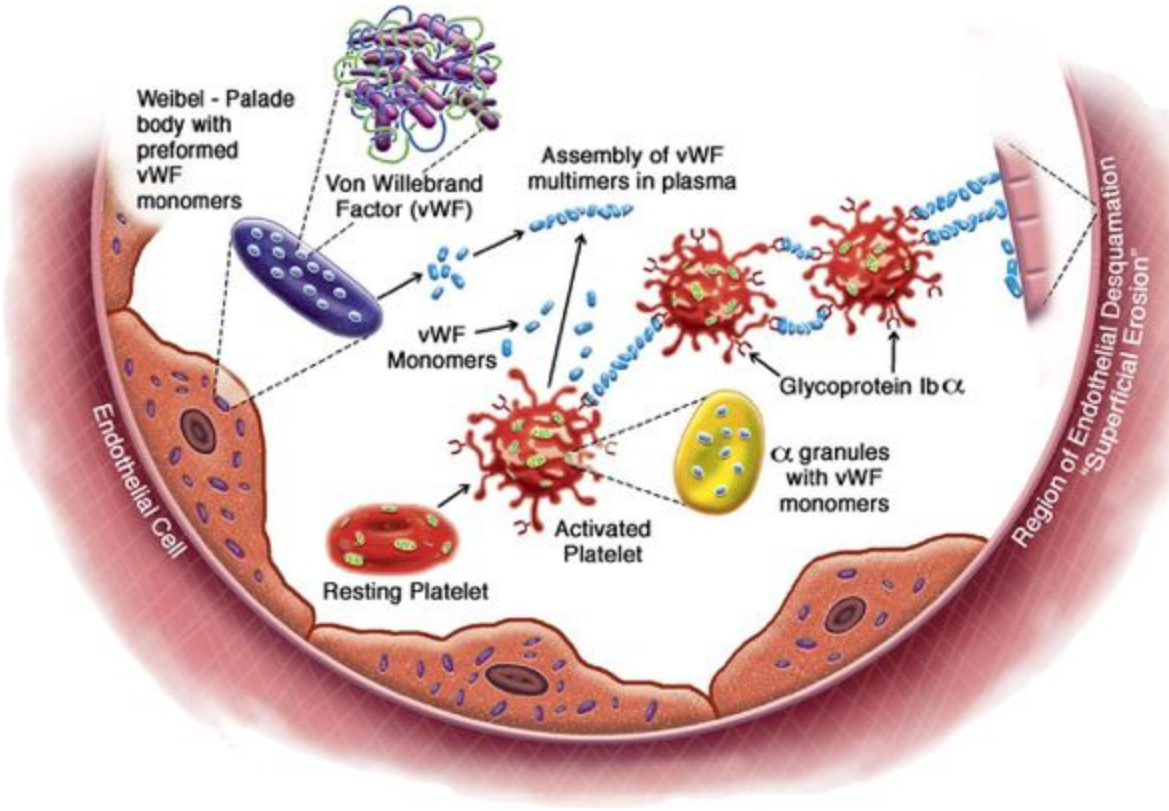
This steps follow positive feedback

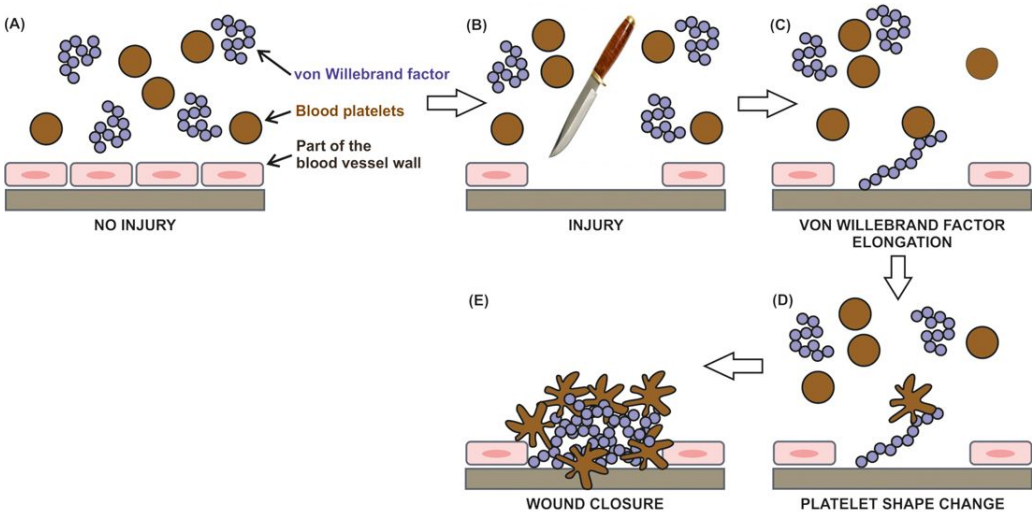
- As more platelets stick, they release more chemicals
- Platelet plugs are fine for small vessel tears, larger breaks in vessels need additional help.



Von Willebrand Factor:

- made my megakaryocytes and endothelial cells.
- Forms globular protein
- Lengthen and shorten base on the injury. (Unfolds and extends in response to injury)
- Surface protein on activated platelets binds to VWF.


Weibel - Palade body with preformed vWF monomers
Von Willebrand Factor (vWF)
Assembly of vWF multimers in plasma
vWF Monomers
Glycoprotein Ib α
α granules with vWF monomers
Activated Platelet
Resting Platelet
Endothelial Cell
Region of Endothelial Desquamation "Superficial Erosion"

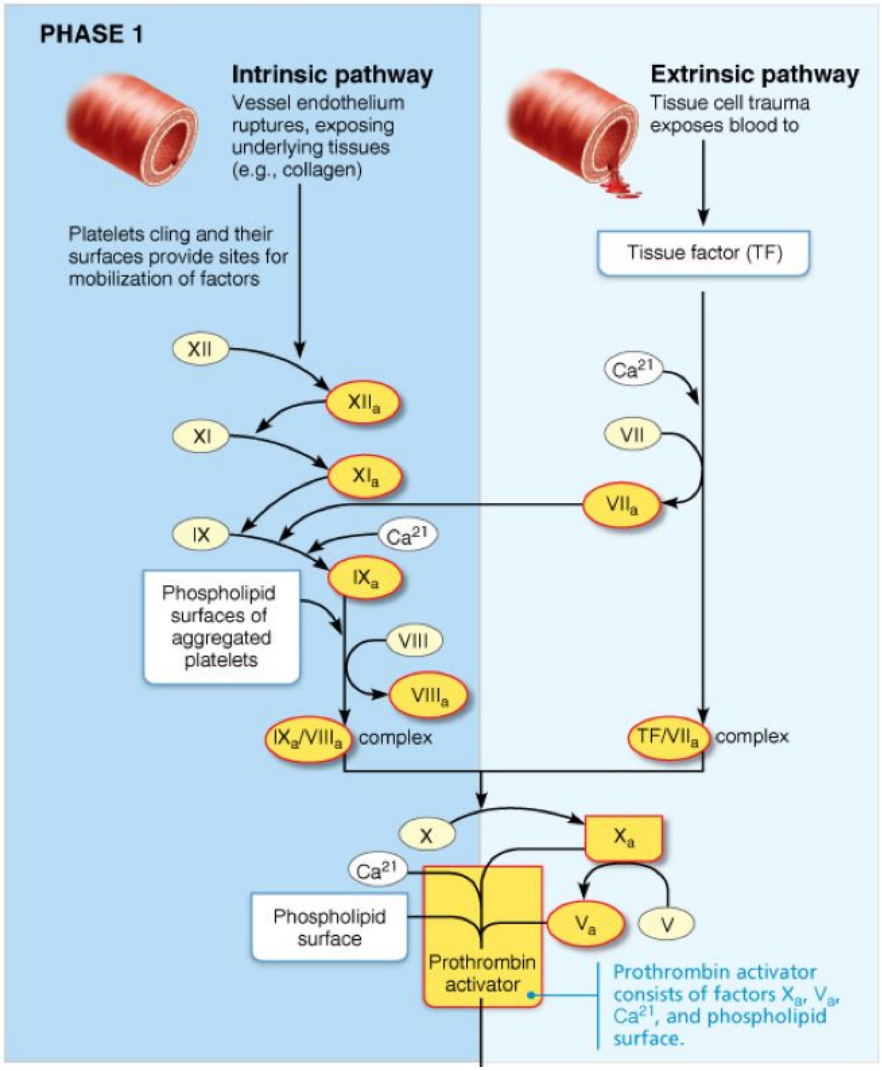

(A)
von Willebrand factor
Blood platelets
Part of the blood vessel wall
NO INJURY
(B)
INJURY
(C)
VON WILLEBRAND FACTOR ELONGATION
(D)
PLATELET SHAPE CHANGE
(E)
WOUND CLOSURE

Step 3: Coagulation : Reinforces platelet plug with fibrin threads.

- Blood clots are effective in sealing larger vessel breaks.
- Blood is (locally) transformed from liquid to gel in a multistep process.
- Series of reactions use clotting factors (procoagulants), mostly plasma proteins synthesized by the liver.
- Number I to XIII in order of discovery.
- Vitamin K needed ot synthesize four factors
- Most circulate in blood in active form.
- Happen in 3 phases : - —-> Prothrombin activator———> thrombin ———> fibrin mesh

Phase 1: 2 pathways to Prothrombin Activation.

- Crucial components in both pathways are negatively charged membranes. In particular, activated platelets display negatively charged phosphatidylserine, once known as *PF3* (platelet factor 3). Many intermediates of both pathways can be activated only when on such phospholipid surfaces. The intermediate steps of each pathway *cascade* toward a common intermediate, factor X (Figure 17.14). Once factor X has been activated, it complexes with calcium ions and factor V on a phospholipid surface to form prothrombin activator. This is usually the slowest step of the blood clotting process, but once prothrombin activator is present, the clot forms in 10 to 15 seconds.



- Factor X then complexes with Ca2+ and PF3 and factor V to form prothrombin activator.

Intrinsic pathway

- Clotting factors present within the blood
- Triggered by negatively charged surfaces (activated platelets, collagen, glass or test tube.)

Extrinsic pathway.

- Clotting factors are located outside blood
- Triggered by exposure to TF (tissue factor) or factor III
- Faster pathway.
- Prothrombin:a complex of a dozen blood coagulation factors that functions in catalyzing prothrombin into thrombin. Prothrombin activator is released in the body by a cascade of chemical reactions in response to damage in a blood vessel.

- Phase 2 : Prothrombin activator: Catalyzes transformation of prothrombin to active enzyme thrombin.

Table 17.3 Blood Clotting Factors (Procoagulants)

FACTOR NUMBER	FACTOR NAME	NATURE	SOURCE	PATHWAY; FUNCTION
I	Fibrinogen	Plasma protein	Liver	Common pathway; converted to fibrin (insoluble weblike substance of clot)
II	Prothrombin	Plasma protein	Liver*	Common pathway; converted to thrombin (converts fibrinogen to fibrin)
III	Tissue factor (TF)	Plasma membrane glycoprotein	Tissue cells	Activates extrinsic pathway
IV	Calcium ions (Ca^{2+})	Inorganic ion	Plasma	Needed for essentially all stages of coagulation process; always present
V	Proaccelerin	Plasma protein	Liver, platelets	Common pathway
VI†				
VII	Proconvertin	Plasma protein	Liver*	Both extrinsic and intrinsic pathways
VIII	Antihemophilic factor (AHF)	Plasma protein	Liver, lung capillaries	Intrinsic pathway; deficiency results in hemophilia A
IX	Plasma thromboplastin component (PTC)	Plasma protein	Liver*	Intrinsic pathway; deficiency results in hemophilia B
X	Stuart factor	Plasma protein	Liver*	Common pathway
XI	Plasma thromboplastin antecedent (PTA)	Plasma protein	Liver	Intrinsic pathway; deficiency results in hemophilia C
XII	Hageman factor	Plasma protein; activated by negatively charged surfaces (e.g., glass)	Liver	Intrinsic pathway; activates plasmin; initiates clotting *in vitro*; activation initiates inflammation
XIII	Fibrin stabilizing factor (FSF)	Plasma protein	Liver, bone marrow	Cross-links fibrin, forming a strong, stable clot

*Synthesis requires vitamin K

†Number no longer used; substance now believed to be same as factor V

Phase 3 : —-> fibrin mesh.

- Thrombin converts soluble fibrinogen to fibrin. (take the cap off the pen, let it stick).

  Fibrin - forms basic structure of clot
  - Causes plasma to become a gel-like trap, catching formed elements.

- Thrombin + Ca2+ activate factor III ——> cross- links fibrin and strengthens and stabiliezes clot
- Anticoagulants: - factors that normally dominate in blood to inhibit coagulation.

Bleeding disorders.

-•Abnormalities that prevent normal clot formation

•

•Thrombocytopenia: Deficient number of circulating platelets

–Severe cases: Petechiae appear as a result of spontaneous, widespread hemorrhage

–Due to suppression or destruction of red bone marrow (e.g. malignancy, radiation, or drugs)

–Platelet count <150,000/ml is diagnostic (normal is 150-400,000/ml)

–

–Treatment: transfusion of concentrated platelets

Clot retraction and Fibrinolysis

Retraction :

- Fibrinolysis : Process that removes unneeded blood clots when healing has occurred. (begins after 2 days and continue in several days.)
- Plasminogen ——> plasmin (fibrin digesting enzyme)
- tPA, factor III, thrombin also play a role

Lecture 13:

- Ways to study : - study groups
    - Review sessions
    - Reading
    - Explaining to others.
    - Explaining to yourself.
    - Write out key concepts without looking a book/ slides.

Structure of the heart.

1. Pericardium
    - Fibous perrcariduum : provide protection for the heart.
    - Parietal layer of serous pericardium: prevent friction.
      THick myocaridum :, not much around the atria. Because it is contract a lot harder in the ventricle. Ventricle sends everything thtought the pulmonary circuit

Gross anatomy of the heart

- Seperate ventricles
- Coulbe atria, could be oracele
- Need to learn the diagram by heart.
- Why triscupid and biscupid (number of flaps.)

Define the artery and veins

- Artery : go away from the heart
- Veins : go into the heart.
  ====> oxygen in blood does not define whether it is artery or vein.

2 sources of blood go in the right atrial : - superior venacava
- Inferior vena cava

Veins and artery is feeding the heart tissues itself. Artery feed oxygen and nutrients to the heart, vein pick up waste.
===> Provide the heart muscle itself with oxygen and remove carbon dioxide.

2 circuits of the heart.

1) Pulmonary
   : - blood go from heart to the lung. (right atrial and right ventricle) : passive filling of the ventricle.
- .BLood sitting for 0.5 sec in atrial, and drop to the right ventricle. (V of blood flow in atrial determined stroke volume)
- Blood get pupmed by Rright, go through SL valve, go out of the pulmonary trunk.

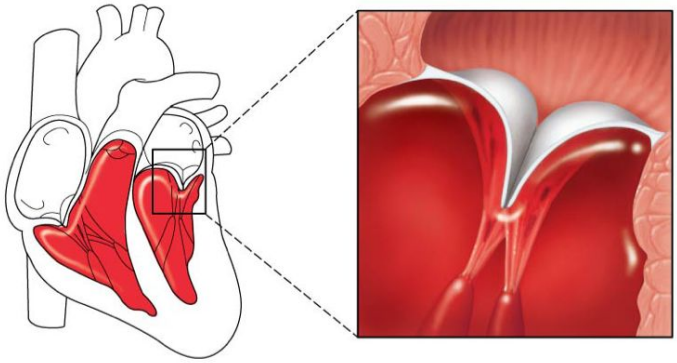
2) Systemic

- BLood passive fling in the left atrial (work the same)
- Blah blah smae
- Then blood get pupmed (left ventricle contraacted) ——-> aorta.
  Duo pumps ====> functioning at the same time.

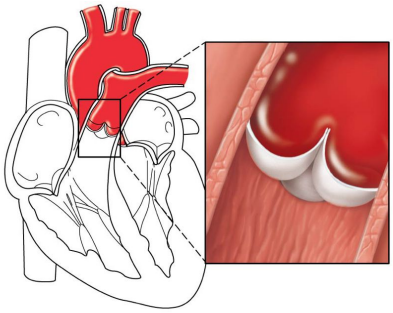
Heart sounds

- Associated with closing of the heart valve
  Tricuspid valve, bicuspidval, mitral valeves.
  - First, getting closing of bicuspid and triscuspid (lub.) Then the mitral valves closing (dub)

Event 1: Av valves closed, atrial presseue less than ventricular pressure.



- The valve mechanism (study that) . Semilunar valve.
- Nguoc lai voi Av valve (interms of mechainis.)



- **(b)**

- As ventricles relax and intraventricular pressure falls, blood flow back from artereis, fliiing the cusps of semilunar vlavles and forcing them to close.

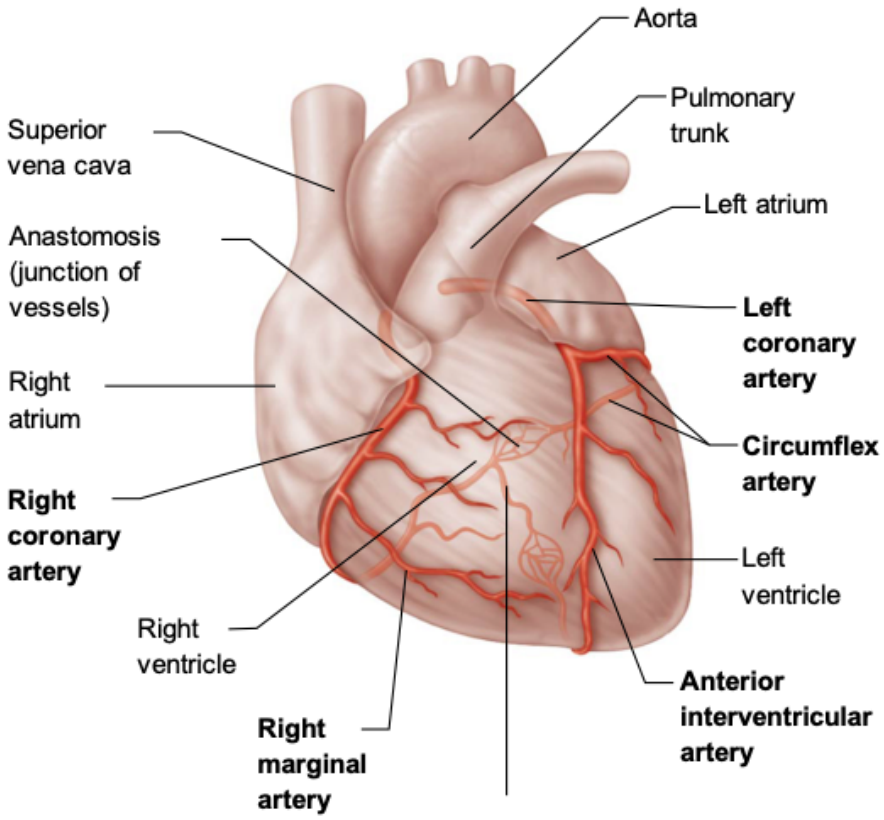
Problems with valve integrity.

- Incompetent valve: valve gonna flow back throught the alve, so the heart has to repump it agian and ag ia ===> rushing sounds.
- Valvular setenosis = vales i stoo hard blood cannot flow through . Clikcing ousnd, or whislting sound

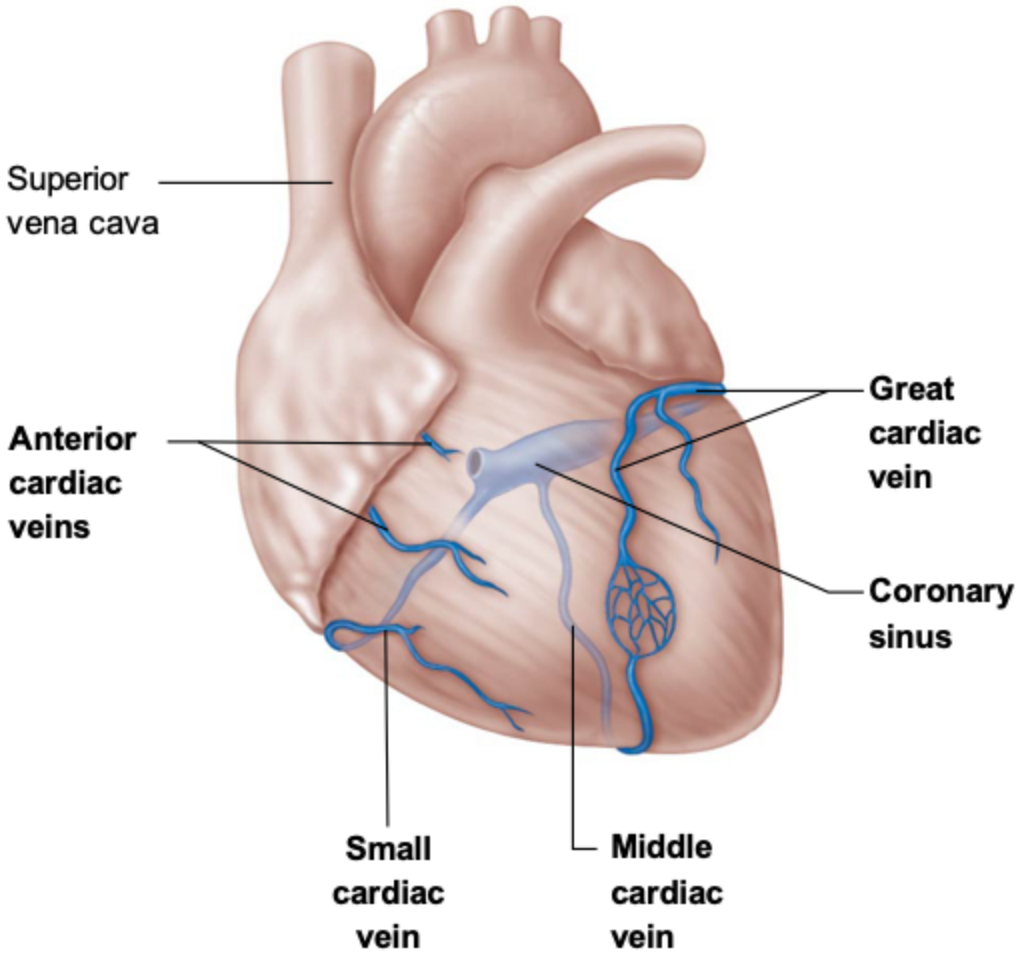
—

- Coronary artery : - gettign the blood to the muscle (systemic blood). It feedings it own myocaridum with its own blood. The blood come out of the heart and go back with oxygen.

-



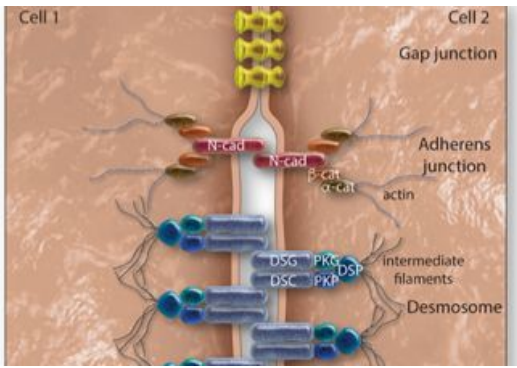
- Anastomosis : - Lots of differnt path that the blood can take. If you can only have 1 path. If blood clot happen, sth block a path, it provides option (redundancy) -
- Coronary veins : collect waste from myocardium

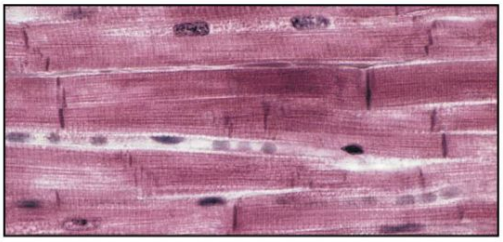


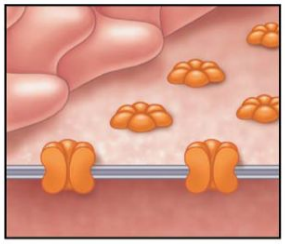
- Coronary sinus.

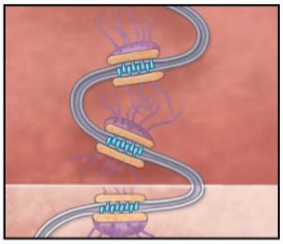
Myocaridum (cariduc musct as a group
cle)

- Intercalted disc (some striation) . there are gap juncitons , allow AP to across the whole heart —-> work together, contract
- Also whole the heart cell together.



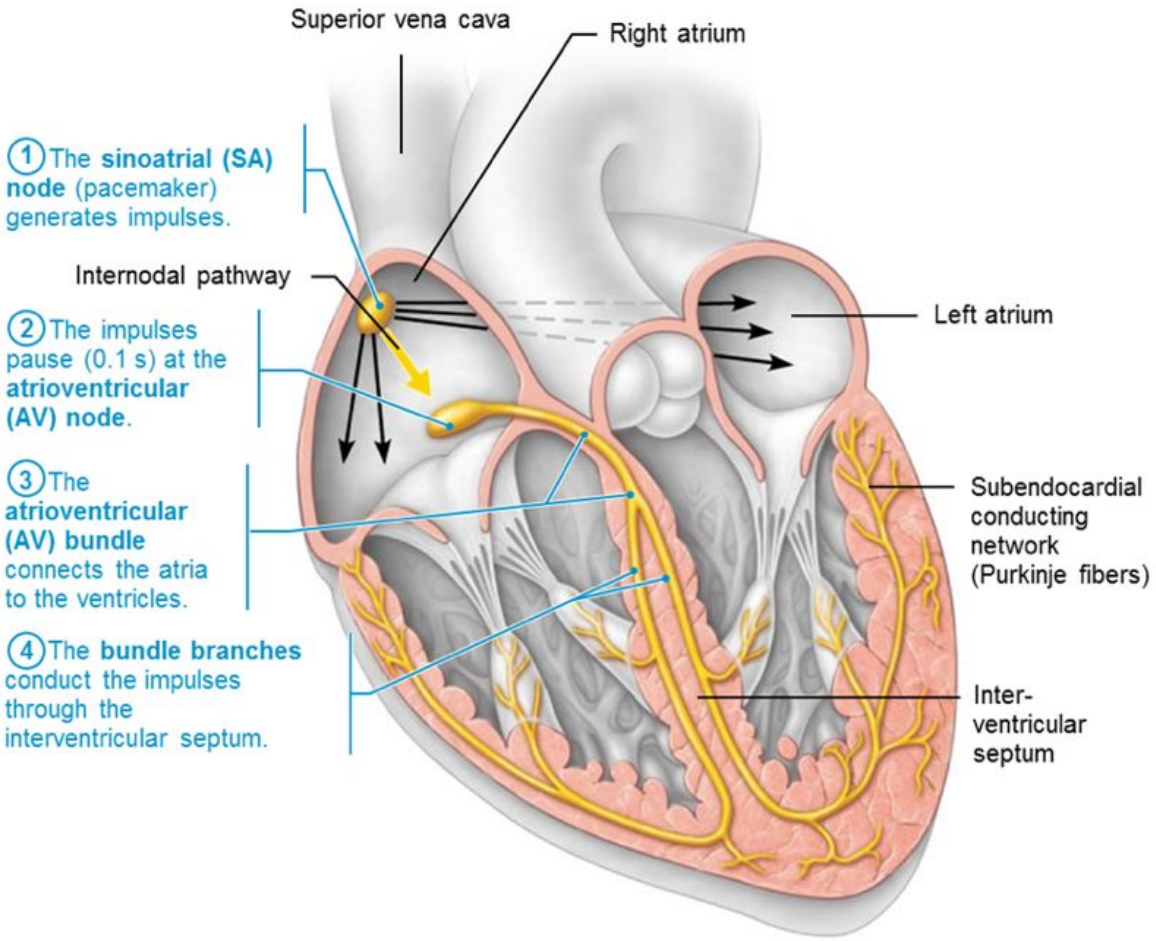






Pace maker cells

- They can go thorught slow depolarizadaiton : (modified muscle cells) . Most of them are found int he Sa NOde===> generate signal ===>
- Intrinsic conductign system is getiting more brancing, smaller , eventually get the signal go throught the entire heart.
- Split second 0.1 sec between contraction between top part and lower part
  If there is a question :- it contracts at a single unit.



- If u take the heart out of the body, it will still beat, cuz of the SA node.

Extrinsic Innervation of the Heart Modifying the Basic Rhythm

- Modified heart beats (lower it) . Negative feedback signals.
- The brain sends the signal down the SA node to tell the heart slow down. (75 beats per minute).
- Heart beat modified ANS cardiac centers in medulla oblongata.

Pr segment : a fracition of th second
QRS: Ventricular depolarization
ST segment:

T: Ventricular repolarization.
U wave

Juntional rthy : sa node is not workign, AV node takeover. Only able to do 45 beats per minute.
Dont have the p wave
Second dgree heart block : AV node is not workign properly. Whole bunch of p waves.
Fibrilation : all over place : heart attack.

Cardiac cycle.
Ventricular voume phase: blood flow in atria ===> fill in ventricular
Isovolumetric : begin of ventriclear contraction, just enought to close th evlave. (blood want to go ghere but the valves are closed.)
Ventricular ejection phase: - The ventricle contrat
Isovolumetric

OVer first phase, the volume contine to increase. (the rate o fgeting blood)
The ventricular ejection : the volume decrease rapidly, the system get reset.

The diagram explain

SO the diagram

- If u see p wave, try to relate about pressure, volume, heart sound, valves (gonna be on the exam.)

Cardiac output :

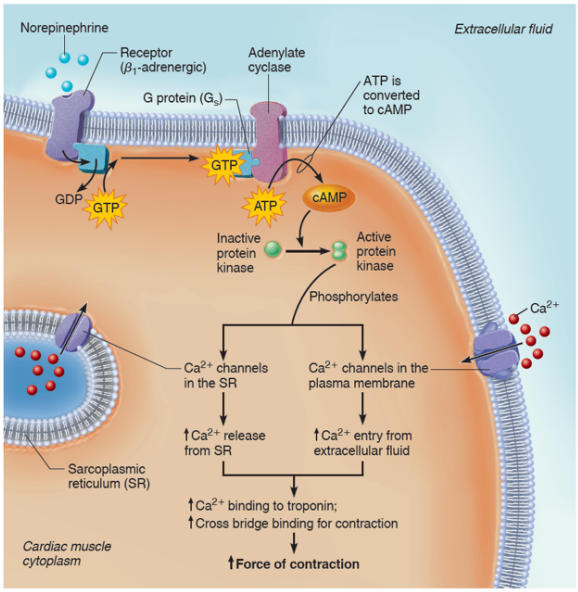
Preload : - Stretch of ventricle- impacted by amount of blood coming into the heart from veins
Contractility L strenghth of muscle contraction
After load = back pressure.

Norepinephrine increases heart contractility.

Norepinephrine Increases Heart Contractility Via a Cyclic AMP Second Messenger System

cAMP is the second messenger in this figure
The purpose is to activate a kinase, which in turn activates the calcium channels

more calcium = stronger contraction



- More Ca2+ = stronger contraction.
- (norepinephrine.)
- Calcium receptors.

Heart rate

- Autonomic nervous system : bring down from 100 to 75.
- Chemicals
- Other

Lecture 11:

17.1 The functions of blood are transport, regulation and protection

17.2 Blood consists of plasma and formed elements

17.3 Erythrocytes play a crucial role in oxygen and carbon dioxide transport

17.5 Platelets are cell fragments that help stop bleeding

17.6 Hemostasis prevents blood loss

17.7 Transfusion can replace lost blood

Internal transport system of they body.