Western

UNIVERSITY · CANADA

Midterm #2 Review
Sections 09/010

TA: Greydon Gilmore

Physiology 2130

Dec 16th, 2019

Western

Today

- Chapter 4: Endocrinology
- Chapter 5: Autonomic Nervous System
- Chapter 6: Muscle Physiology
- Chapter 7: Cardiovascular

Midterm Information

- When: December 19th from 9am-10am
- What: 35 multiple choice
  - Endocrinology (Beye) – 4 questions
  - Autonomic nervous system & Muscle (Stavraky) – 9-11 Questions
  - Cardiovascular (Stavraky) – 20-22 Questions
- Where:
  - ABBA-GANE: Alumni Hall 15
  - GHAB-POSA: Alumni Hall 201
  - PRIM-WOOD: Alumni Hall Stage
  - WU-ZIA: Somerville House 2316

Study Advice

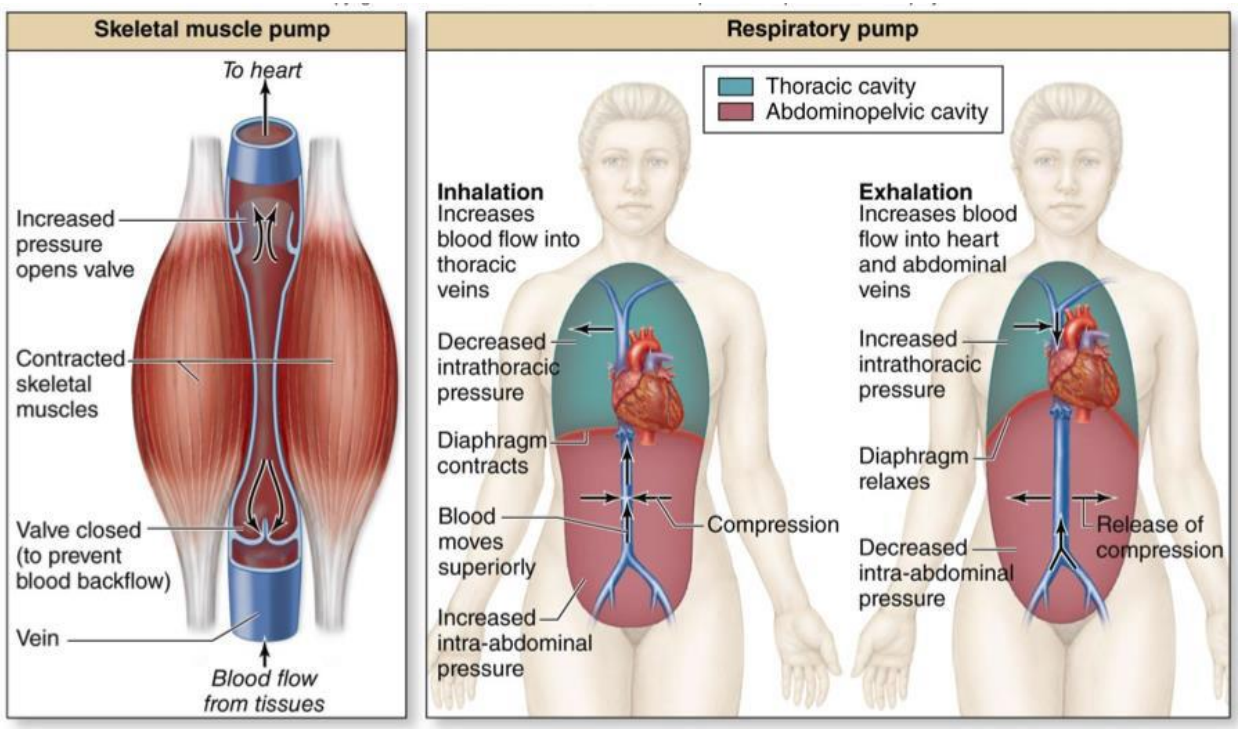
- Get a portable white board
  - White Board: http://a.co/bNSNYLM
  - Markers: http://a.co/aJp9YND
- Write out flash cards
- Study in a group
- Make a short PowerPoint presentation and teach your friends/family

Questions from online dropbox/Emails

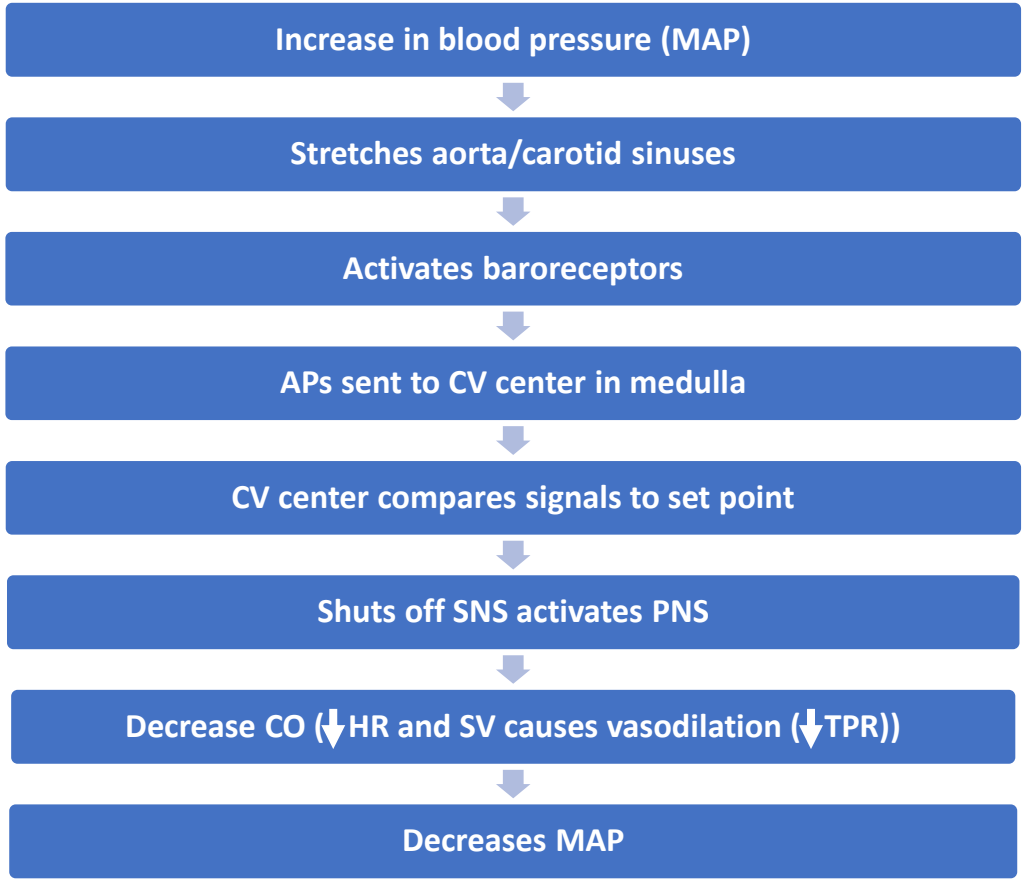
Western

Can you explain respiratory pump (which area is high pressure, which area is low pressure)

- During inhalation you have higher pressure in abdomen and lower in thorax
  - Blood will move from abdomen to thorax
- During exhalation you have higher pressure in thorax and lower in abdomen
  - Blood will move from thorax to abdomen



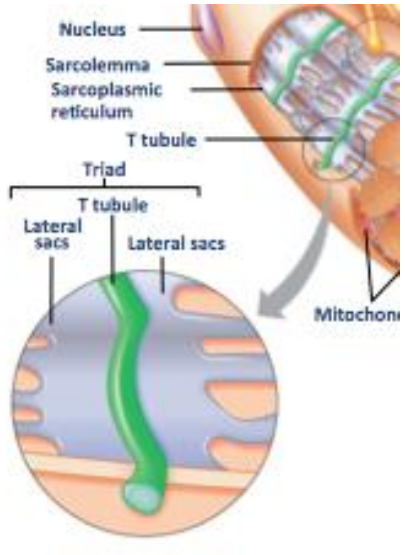
Can you explain the process of the baroreceptor reflex (in the perspective of decreased blood pressure/MAP)

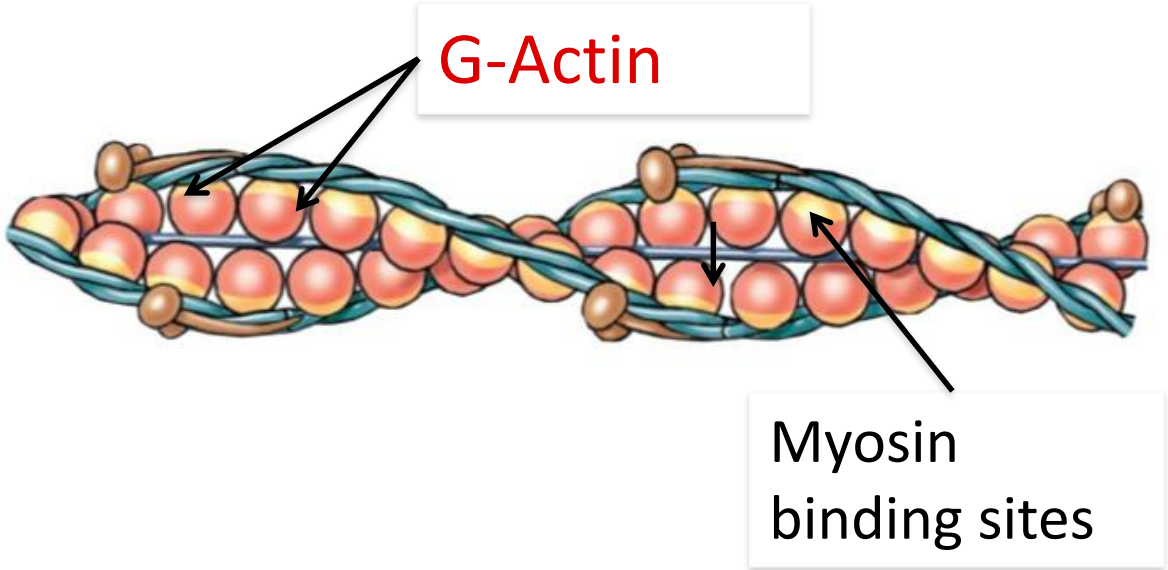


- In low pressure, baroreceptors reduce their firing
- This signals the CV center to shut off PNS and activate SNS

Why does the myosin head attach to the actin and not the myosin binding site? Is calcium released from the SR, or is it stored in the SR and released by the Lateral sac?

- The myosin binding site is part of the actin, they are the same structure
- Calcium is stored and released by the lateral sacs of the SR





Endocrinology
4 Questions on exam

Western

Which of the following is an incorrect statement concerning the adrenal glands?

a. an increase in ACTH release will result in increased mineralocorticoid production
b. the adrenal medulla produces compounds classified as catecholamines
c. production of cortisol is dependent on cholesterol as a precursor
d. all the compounds produced by the adrenal cortex will eventually bind intracellular receptors

Which of the following is a correct statement concerning the pancreas?

a. hyperglycemia causes the release of glucagon, which causes cells to take up glucose
b. hypoglycemia causes the release of glucagon, which causes cells to release glucose
c. hypoglycemia causes the release of insulin, which causes cells to take up glucose
d. hyperglycemia causes the release of insulin, which causes cells to release glucose

Types of Hormones

- Hormone: A chemical signal secreted into the bloodstream to act on a distant tissue
- The target cells of the hormone need the receptor

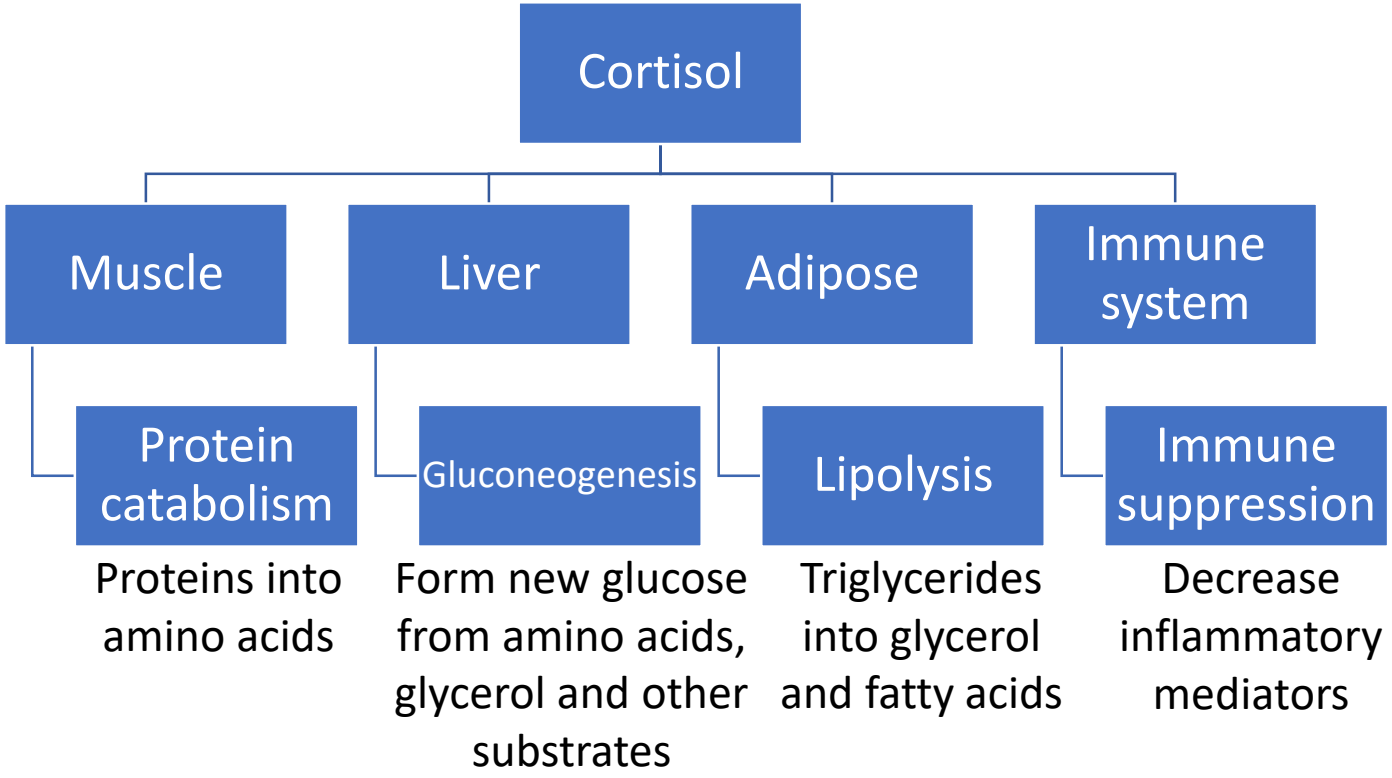
Parameter	Peptide/Protein	Steroid	Amine	
			Hydrophilic	Hydrophobic
Examples	Hormones that end in "-in"	Hormones that end in "-ol" or "-one"	Epinephrine	Thyroid Hormones
Precursor	Amino acids	Cholesterol	Tyrosine	Tyrosine
Solubility	Hydrophilic	Lipophilic	Hydrophilic	Hydrophobic
Blood transport	Dissolves	Bound to protein	Dissolves	Carrier protein
Receptor location	Cell surface	Intracellular	Extracellular	Intracellular
Speed of action	Fast	Slow	Fast	Slow
Goal	Alter existing proteins	Produce new proteins	Alter proteins	Produce new proteins

Adrenal Gland: Layers

	Layers	Categories of Hormones	Example	Stimulus	Effect
Cortex	Zona glomerulosa	Mineralocorticoids	Aldosterone	RAAS pathway (@ low BP)	Increase Na^+ reabsoprtion
Cortex	Zona fasciculata	Glucocorticoids	Cortisol	ACTH	-
Cortex	Zona reticularis	Androgens	DHEA	-	-
Medulla	Medulla	Catecholamines	Epinephrine	Sympathetic Nervous System	SNS response

Three classes of steroids: Mineralocorticoids, Glucocorticoids and Androgens

Cortisol



- Cortisol is catabolic: Break down larger molecules
- Cortisol levels peak early morning and decline throughout the day
- Cushing's disease (hypercortisolism): thinning skin, muscle wasting and weakness, stunted growth, increased infections, redistribution of fat tissue

Pancreas

	Glucagon	Insulin
Tissue	Islet of Langerhans	Islet of Langerhans
Made by	α- cells	β- cells
Stimulus	↓ Blood glucose Hypoglycemia	↑ Blood glucose Hyperglycemia
Effect	↑ Blood glucose (cells release glucose)	↓ Blood glucose (cells take up glucose)
Class	Peptide	Peptide

Antagonistic Effect

Autonomic Nervous System & Muscle

9-11 Questions on exam

Western

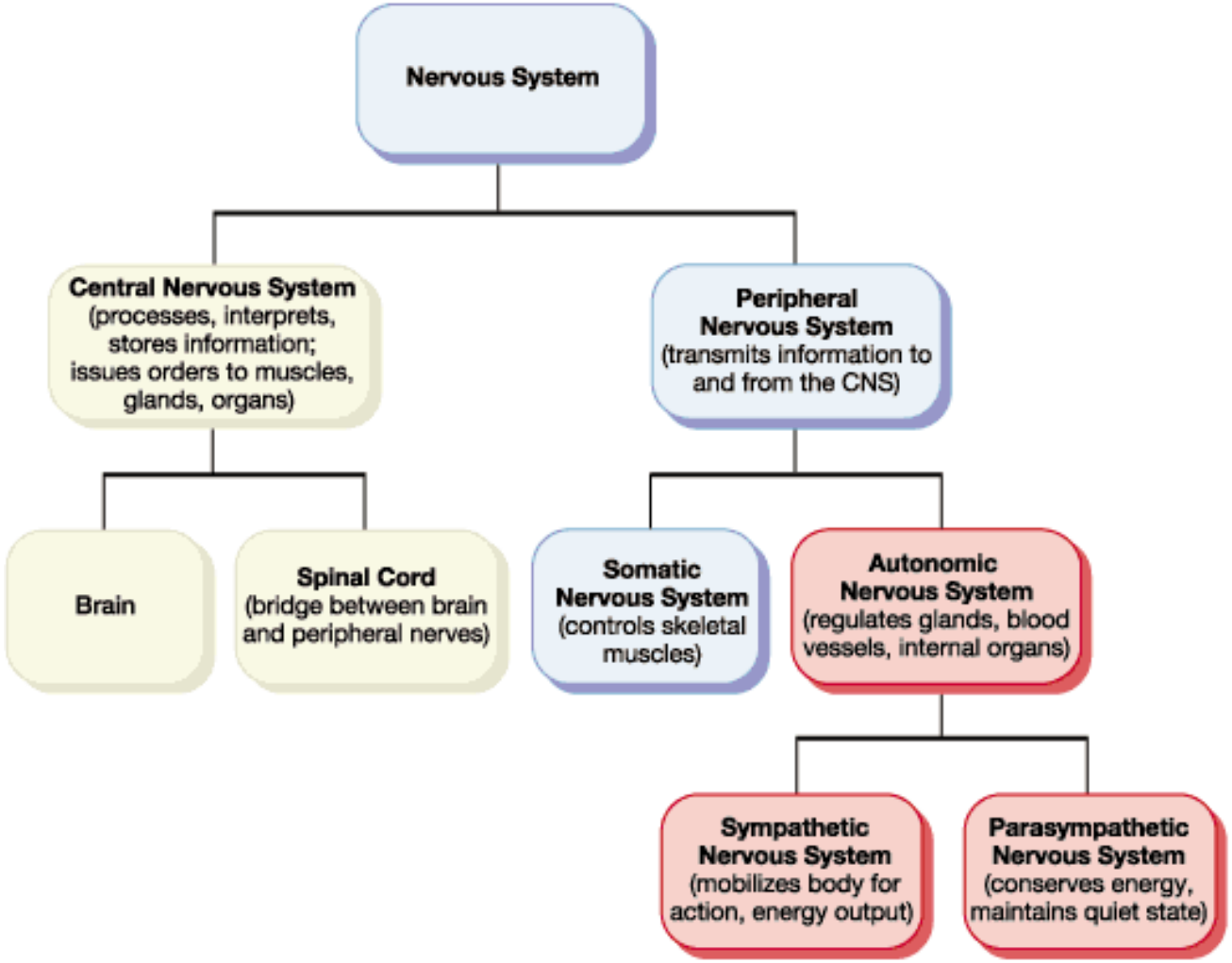
Which of the following physiological responses results from sympathetic action?

a. increase in heart rate
b. constriction of blood vessels
c. stimulation of gluconeogenesis/glycogenolysis
d. all the above

The two branches of the autonomic nervous system display which of the following properties?

a. up-down regulation by tonic control
b. antagonistic control
c. preservation of homeostasis
d. all of the above

Nervous System Divisions



Comparison of Autonomic and Somatic Motor Systems

Somatic

- Motor neuron releases Ach directly onto muscle cells/fibers → contraction

Parasympathetic

- Preganglionic long, postganglionic short
- Preganglionic release Ach
- Postganglionic release Ach

Sympathetic

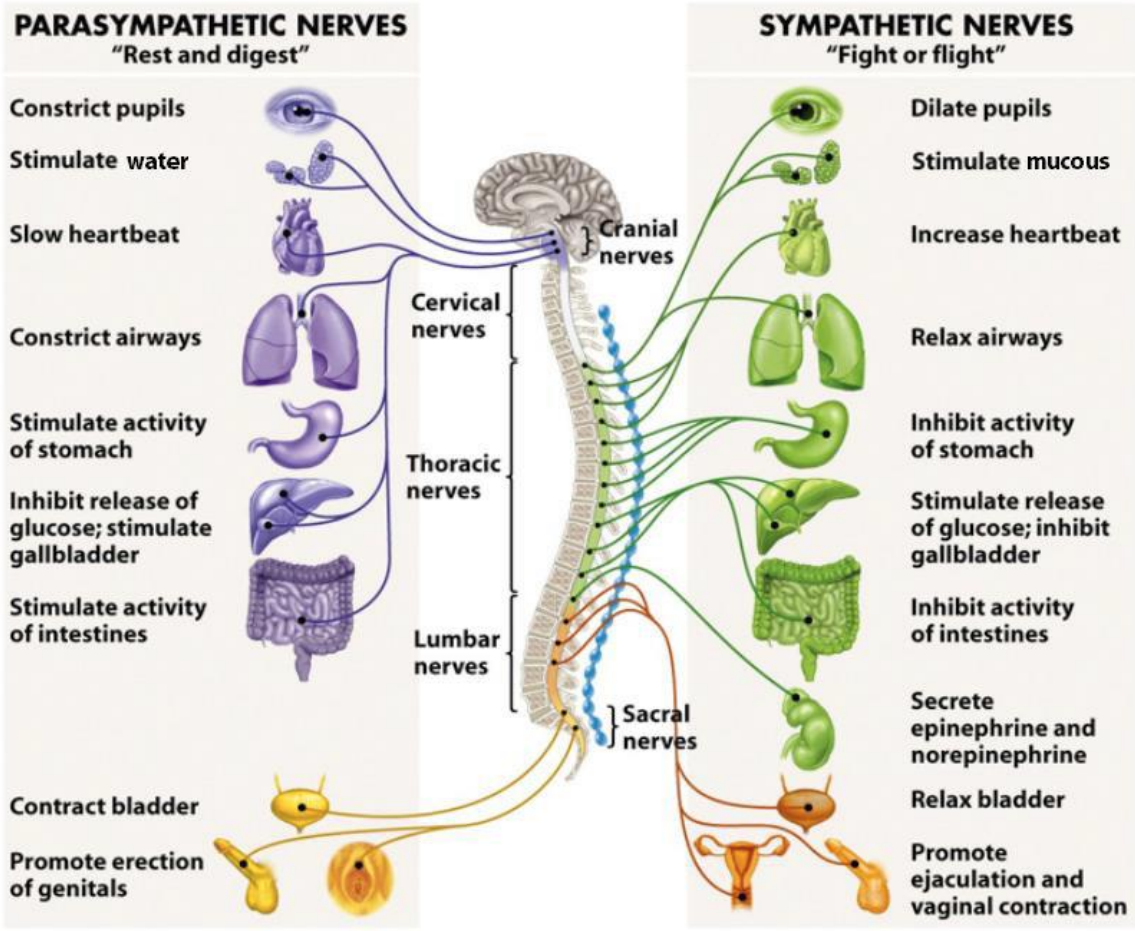
- Preganglionic short, postganglionic long
- Preganglionic release Ach
- Postganglionic release NE
- Adrenal gland only has preganglionic, which releases Ach → epinephrine

Sympathetic vs. Parasympathetic

Responses are usually antagonistic

But there are exceptions:

- Complimentary effect: saliva production
  - PNS → stimulate water and enzymes
  - SNS → stimulate thick mucous
- Cooperative effect: sexual function
  - PNS → induces erection, engorgement and secretions
  - SNS → Induces ejaculation, stimulates contraction



Autonomic vs. Somatic Motor Systems

	Autonomic	Somatic
Voluntary?	No	Yes
Myelination	Preganglionic – Myelinated Postganglionic – Non-myelinated	Myelinated
Number of neurons in path	Two	One
Efferent Transmitter	Acetylcholine and Noradrenaline	Acetylcholine
Target tissue	Smooth and cardiac muscle Adipose tissue, endocrine/exocrine glands	Skeletal muscle
Effect on target	Excitatory or inhibitory	Excitatory (Muscle contracts)

Parasympathetic vs. Sympathetic

	Parasympathetic (PSNS)	Sympathetic (SNS)
Preganglionic Neurotransmitter	Acetylcholine	Acetylcholine
Postganglionic Neurotransmitter	Acetylcholine	Norepinephrine
Location of autonomic ganglion?	Close to organ	Close to spinal cord
Innervates adrenal medulla?	No	Yes
When would you observe more activation?	Rest & Digest	Fight & Flight
If activated, what is the effect on heart rate?	Slows heart rate	Increases heart rate
If activated, what is the effect on breathing?	Constricts airways	Relaxes airways
Give an example of an organ/function with antagonistic effect.	- Constricts pupils - Increases digestion (ie. increases bile secretion, stomach motility increased - Increases secretions from pancreas	- Dilates pupils - Decreases digestion (reduces bile secretions, decreases stomach motility) - Decreases secretions from pancreas
Give an example of a cooperative effect.	- Genitalia - M/induces erection - F/engorgement and secretions	- Genitalia - M/induces ejaculation - F/stimulates contractions

Which is the correct structural organization of skeletal muscle?

a. whole muscle, muscle fiber, fascicle, myofibril, myofilaments
b. whole muscle, muscle fiber, myofibril, Fascicle, myofilaments
c. whole muscle, fascicle, muscle fiber, myofibril, myofilaments
d. whole muscle, fascicle, myofibril, muscle fiber, myofilaments

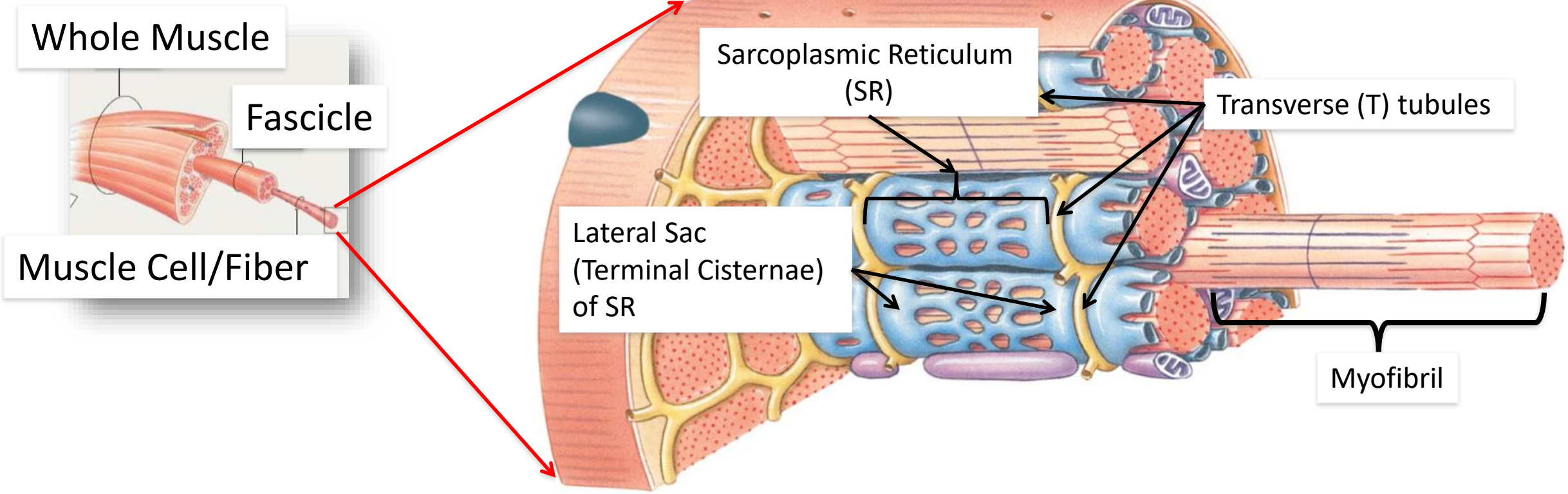
In the cross-bridge formation cycle between actin and myosin, what is the result of ATP hydrolysis?

a. power stroke
b. increase myosin head affinity for actin
c. cross bridge release (muscle relaxation)
d. cross bridge formation

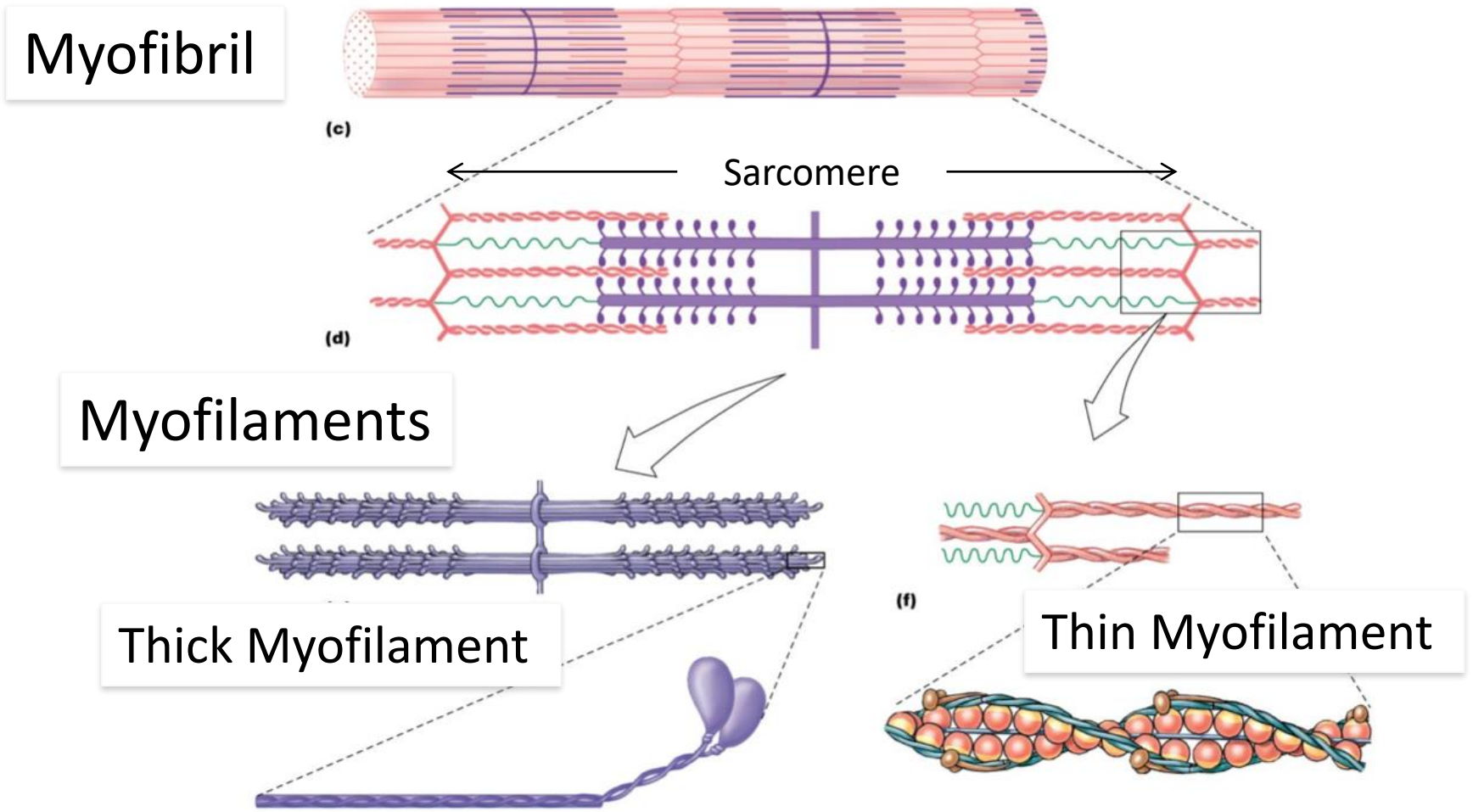
What is the difference between unfused and complete tetanus?

a. recruitment of more motor units
b. greater Ca^{2+} release from the SR
c. increased motor neuron stimulation
d. greater SNS stimulation

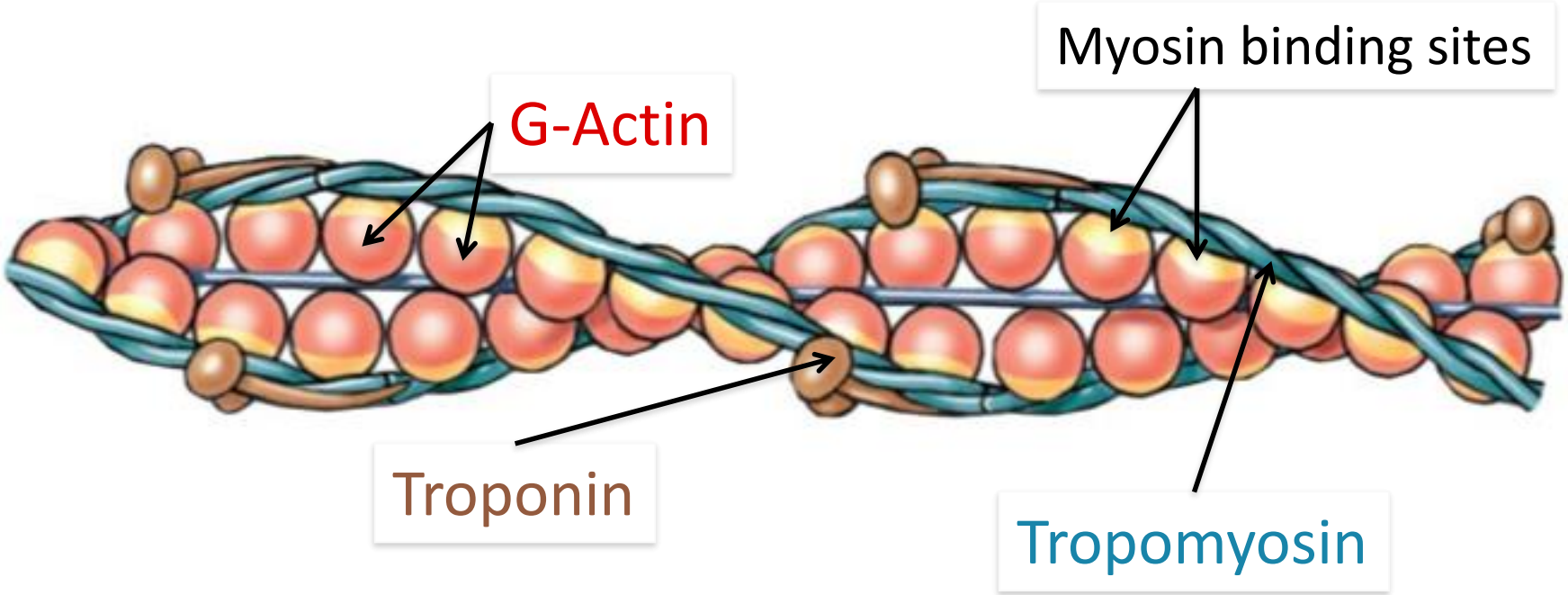
Muscle Structure



Muscle Structure

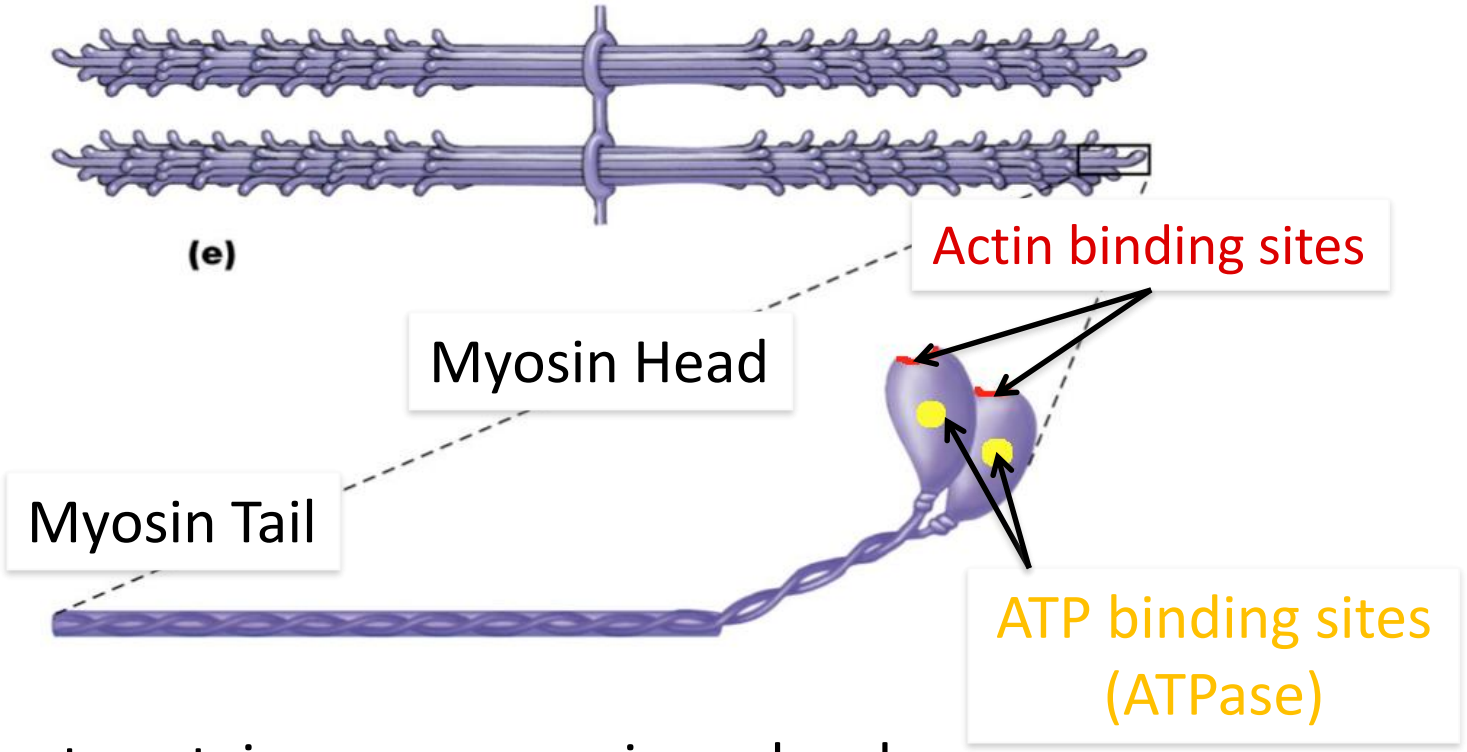


Thin Myofilament



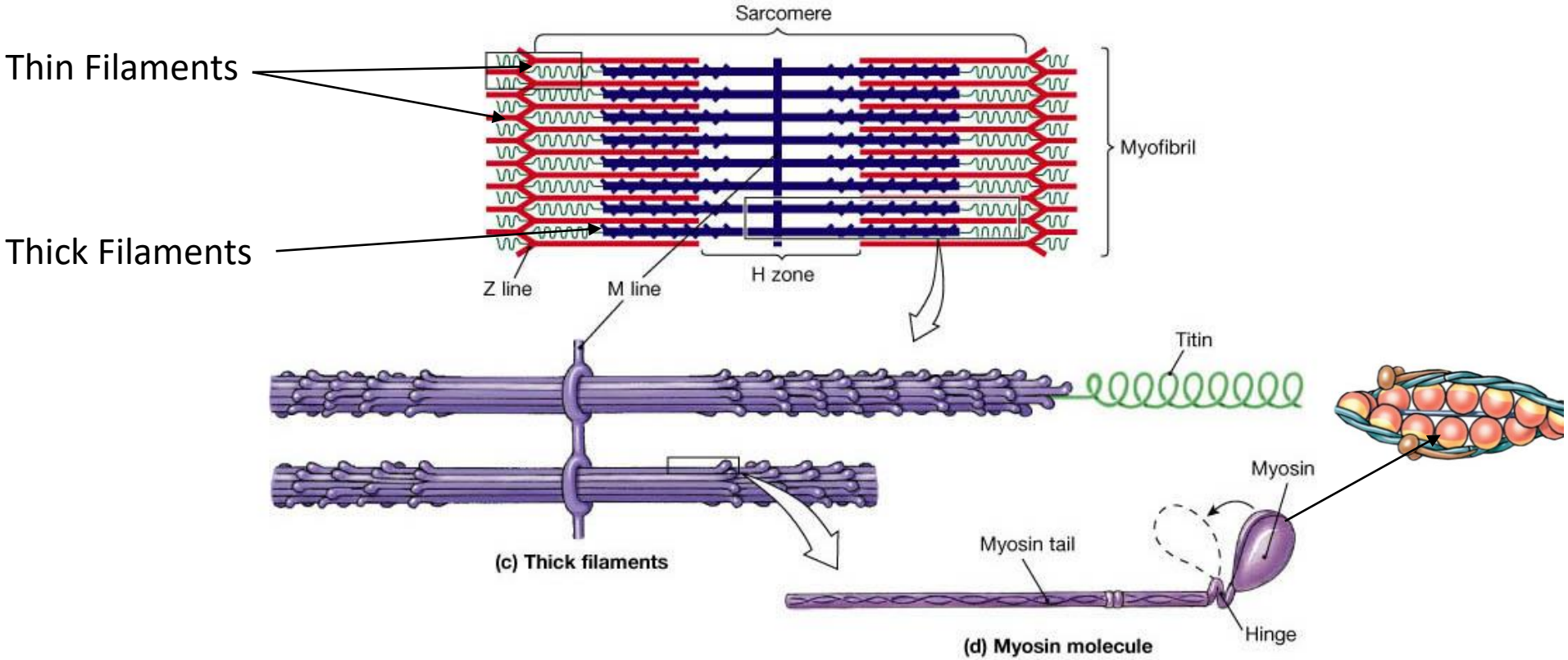
1. G-actin: forms alpha-helical chain with other G-actins and contains a myosin binding site
2. Tropomyosin: in relaxed state, tropomyosin works to cover the myosin binding sites on g-actin
3. Troponin: attached to tropomyosin and actin to hold tropomyosin over the myosin binding sites in relaxed state

Thick Myofilament



- Thick filament contains many myosin molecules
- Each head has a binding site for Actin and an ATPase
  - Breaks down ATP into ADP + P_i and releases energy for contraction

The Sarcomere

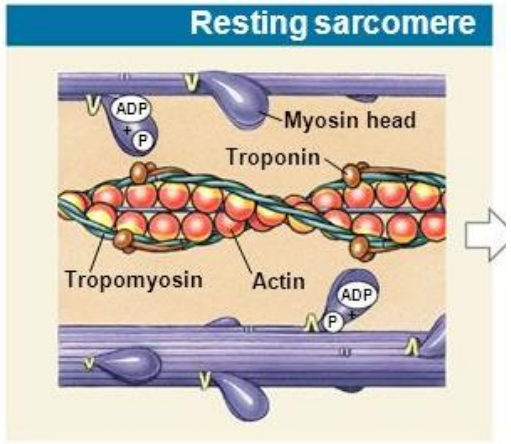


1. Z-line: where thin myofilaments are joined together
2. Sarcomere: area between the z-lines, which is smallest functional unit of muscle
3. Myosin head binds to G-actin on thin filament forming a cross-bridge. A Power Stroke is initiated an a muscle contraction will occur

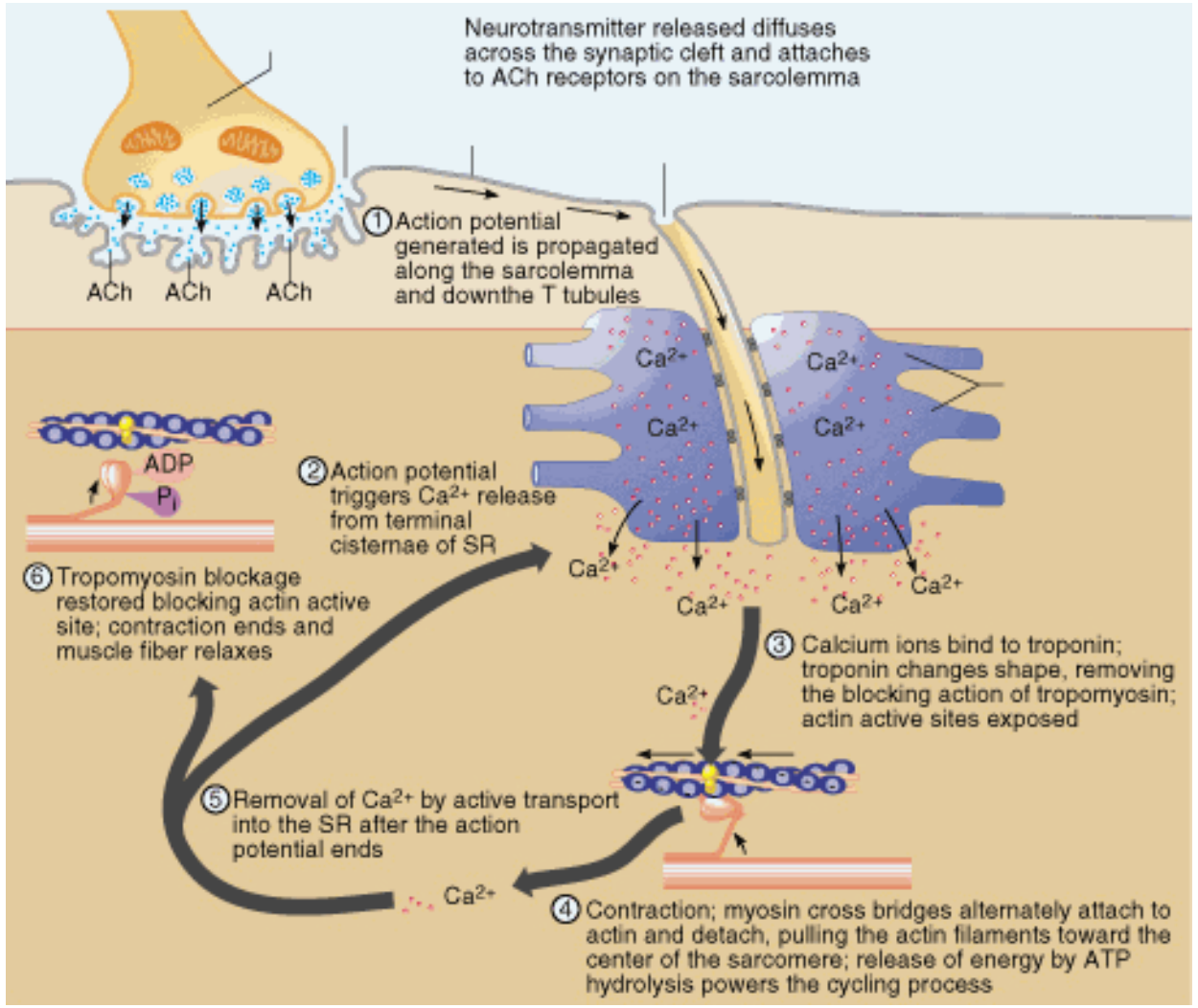
The Sliding-Filament Theory

1. Ca^{2+} binds troponin, causes tropomyosin to roll off and expose myosin binding site
2. Myosin head binds actin binding site, cross-bridge formation
3. P_i released from myosin head causing power stroke (muscle contraction)
4. ADP released, myosin still bound to actin until new ATP attached, myosin head detaches (cross-bridge broken)
5. ATP hydrolyzed to ADP + P_i

Cross-bridge is the connection of myosin head to actin (once binding site is clear). Cross-bridge will allow power stroke to occur (muscle contraction)

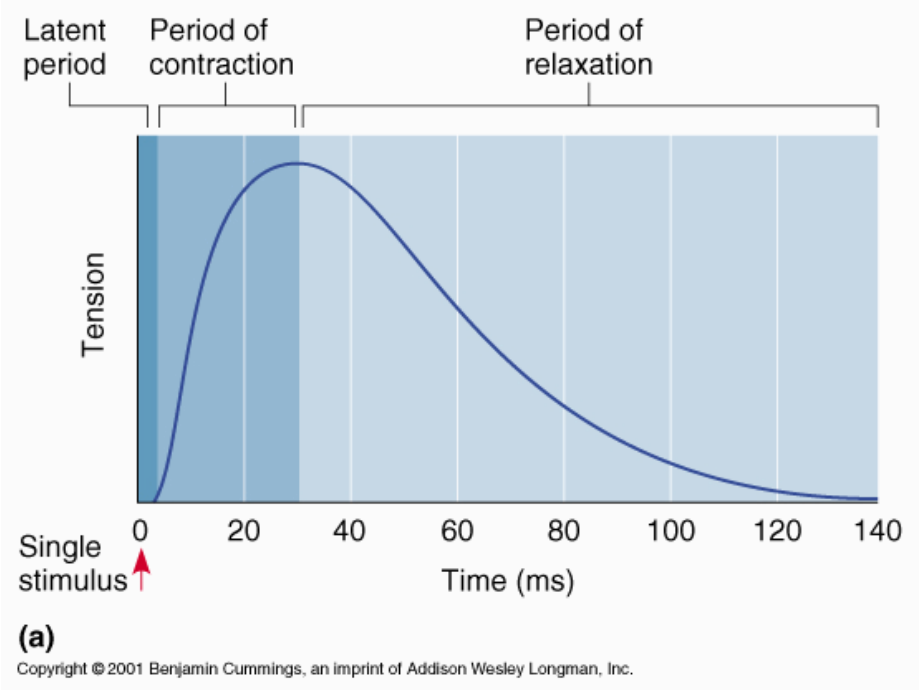


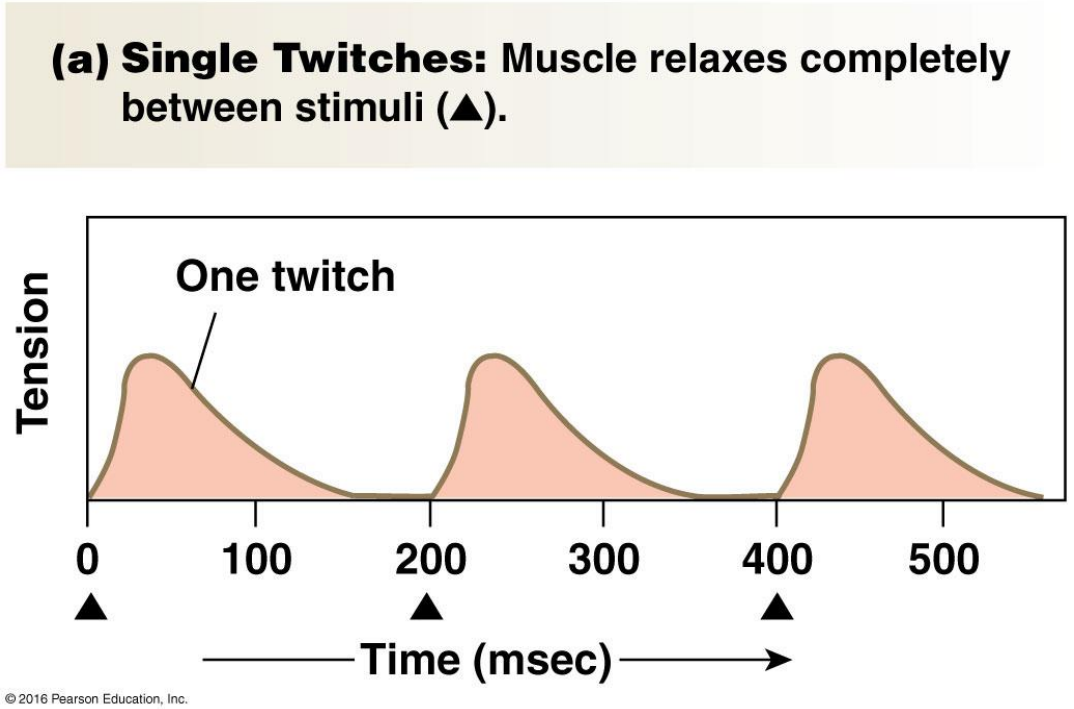
The Neuromuscular Junction and Sarcomere



Muscle twitch

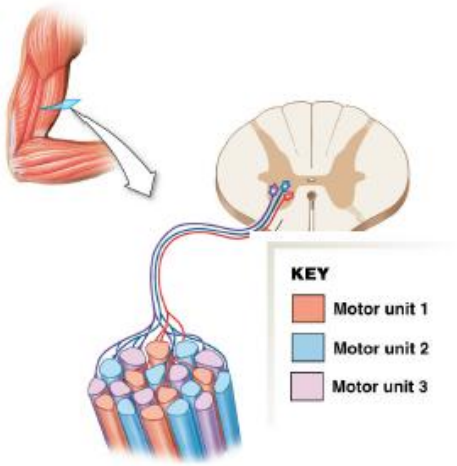
- Result of one AP from the motor neuron
- Difference in AP duration vs. twitch duration = allows summation

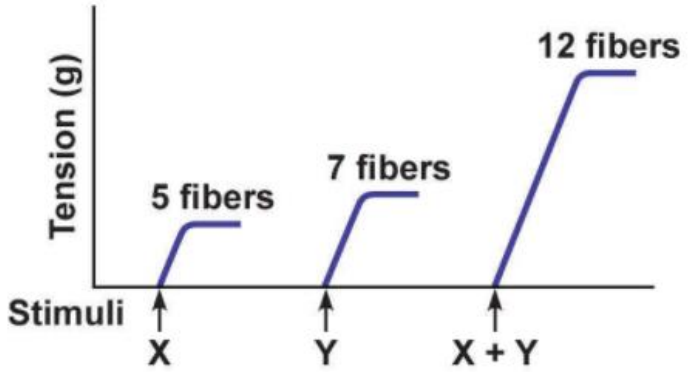




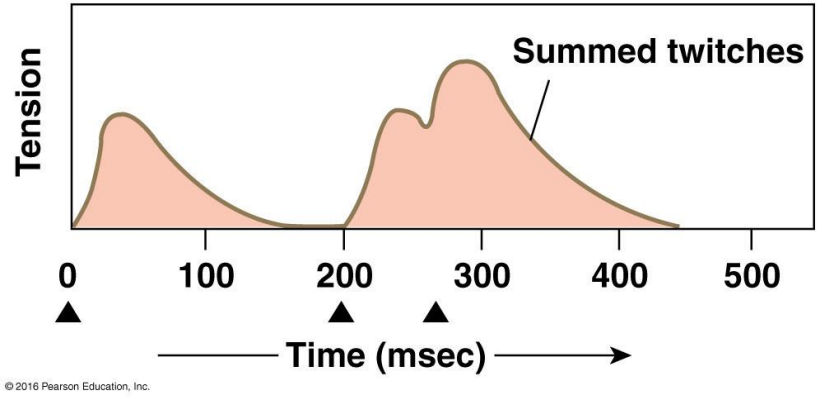
Grading of muscle contraction

1. Recruitment
   - Recruiting additional motor units
   - e.g. motor unit 1 innervates 6 fibers



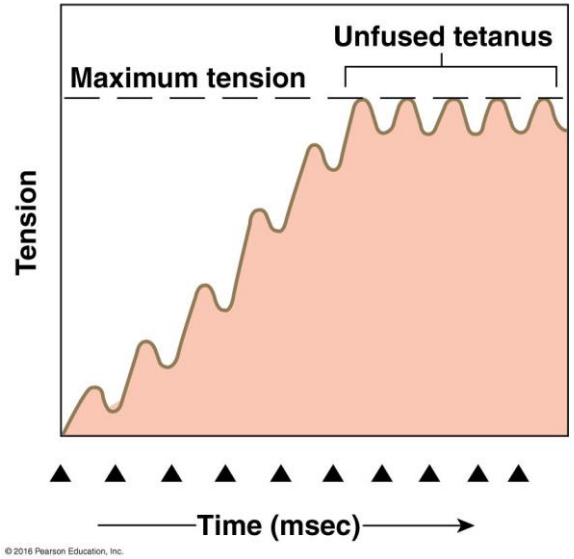


2. Summation
   - Increase the twitch frequency in the same motor unit (summation)

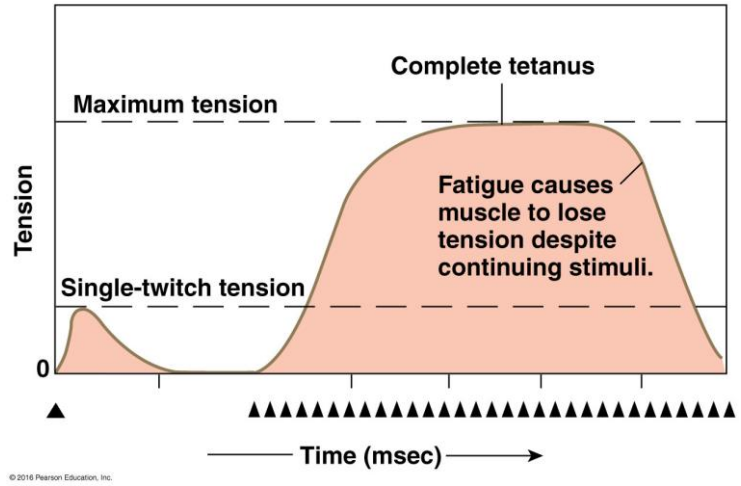


Tetanus

- Unfused tetanic contraction (or unfused tetanus)
  - Medium to high frequency APs will cause twitches to summate
  - Still has time to relax before next twitch



- Complete tetanus
  - At very high frequencies there is no relaxation between twitches
  - Twitches will summate to form smooth, sustained contraction



Cardiovascular
20-22 Questions on exam

Western

Which of the following pairings is NOT correct?

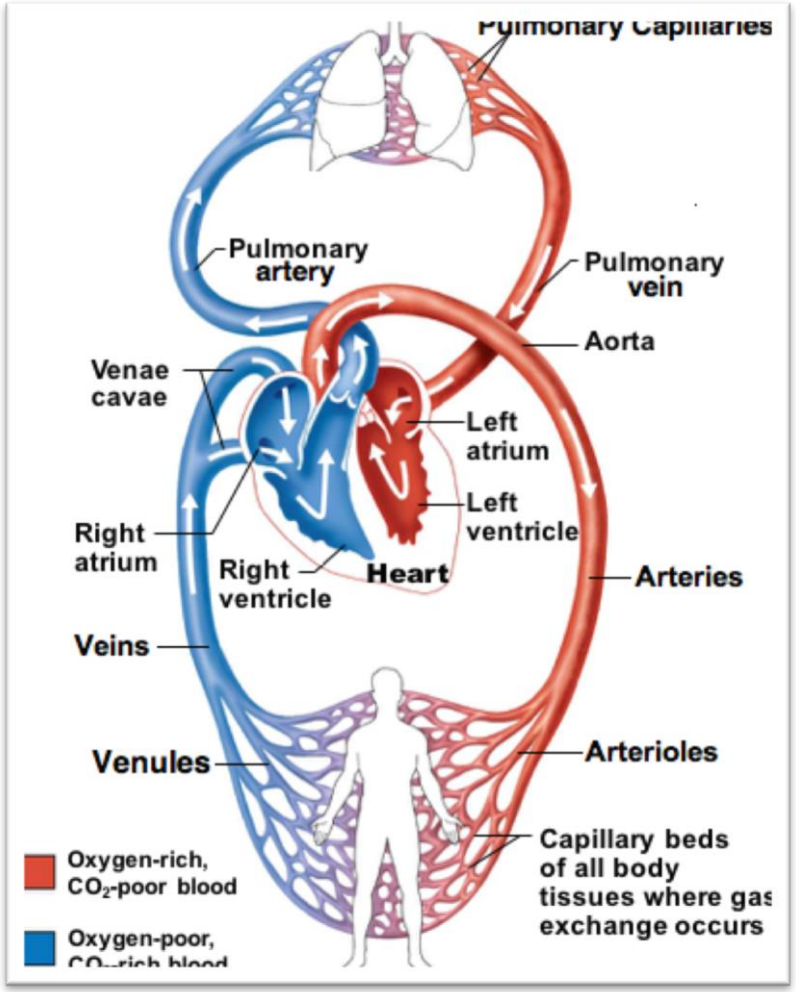
a. primary artery of the systemic circulation → aorta
b. narrow end of the heart that points downward → base
c. valve between ventricle and a main artery → semilunar
d. tough membranous sac that encases the heart → pericardium
e. all of the above are true.

The _____ supply blood to the heart muscle itself.

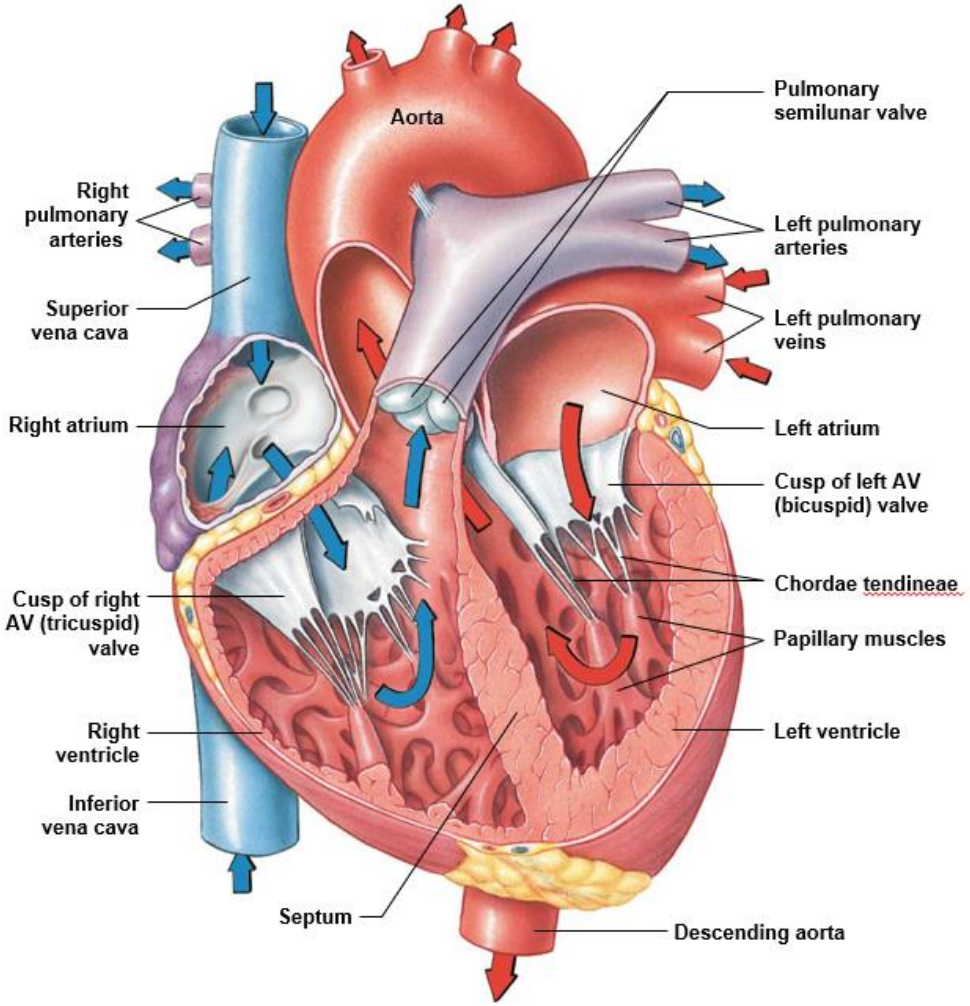
a. coronary arteries
b. coronary veins
c. pulmonary arteries
d. pulmonary veins

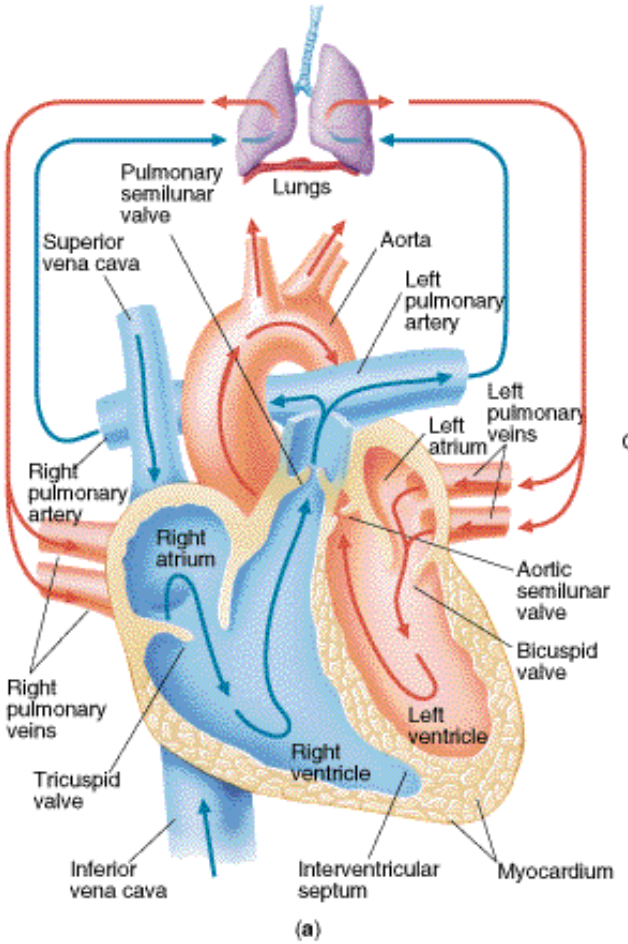
Blood volume distribution

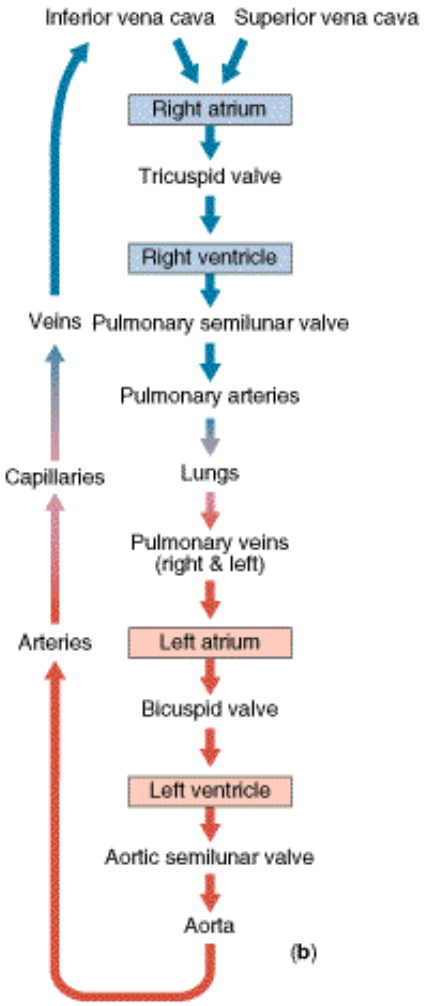
- Total Blood Volume (TBV) = 5 liters
- Heart and Pulmonary circ. = 15%
- Systemic arteries/arterioles = 10%
  - distribution vessels
- Systemic capillaries = 5%
  - exchange vessels
- Systemic veins/venules= 70%
  - capacitance vessels
  - low pressure, require valves to stop backflow



Heart Flow


Aorta
Pulmonary semilunar valve
Right pulmonary arteries
Left pulmonary arteries
Superior vena cava
Left pulmonary veins
Right atrium
Left atrium
Cusp of left AV (bicuspid) valve
Chordae tendineae
Cusp of right AV (tricuspid) valve
Papillary muscles
Right ventricle
Left ventricle
Inferior vena cava
Septum
Descending aorta


Pulmonary semilunar valve
Lungs
Superior vena cava
Aorta
Left pulmonary artery
Left pulmonary veins
Left atrium
Right pulmonary artery
Right atrium
Aortic semilunar valve
Bicuspid valve
Right pulmonary veins
Left ventricle
Tricuspid valve
Right ventricle
Inferior vena cava
Interventricular septum
Myocardium
(a)

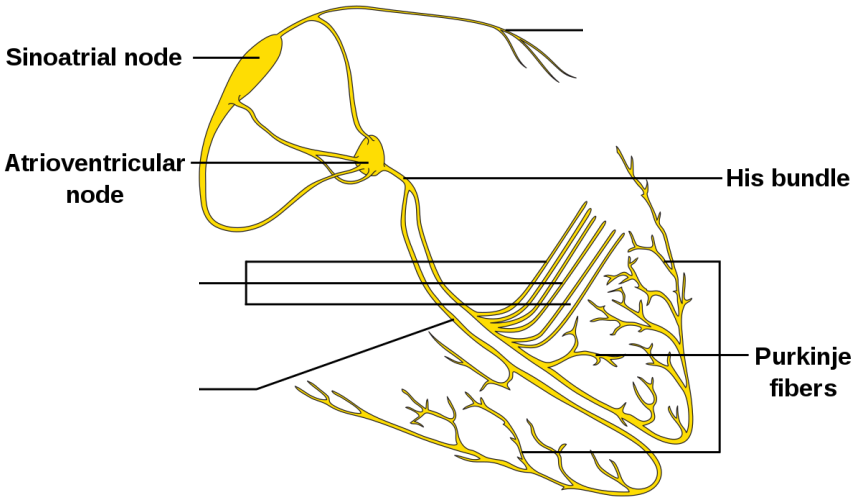

Inferior vena cava
Superior vena cava
Right atrium
Tricuspid valve
Right ventricle
Veins
Pulmonary semilunar valve
Pulmonary arteries
Capillaries
Lungs
Pulmonary veins (right & left)
Arteries
Left atrium
Bicuspid valve
Left ventricle
Aortic semilunar valve
Aorta
(b)

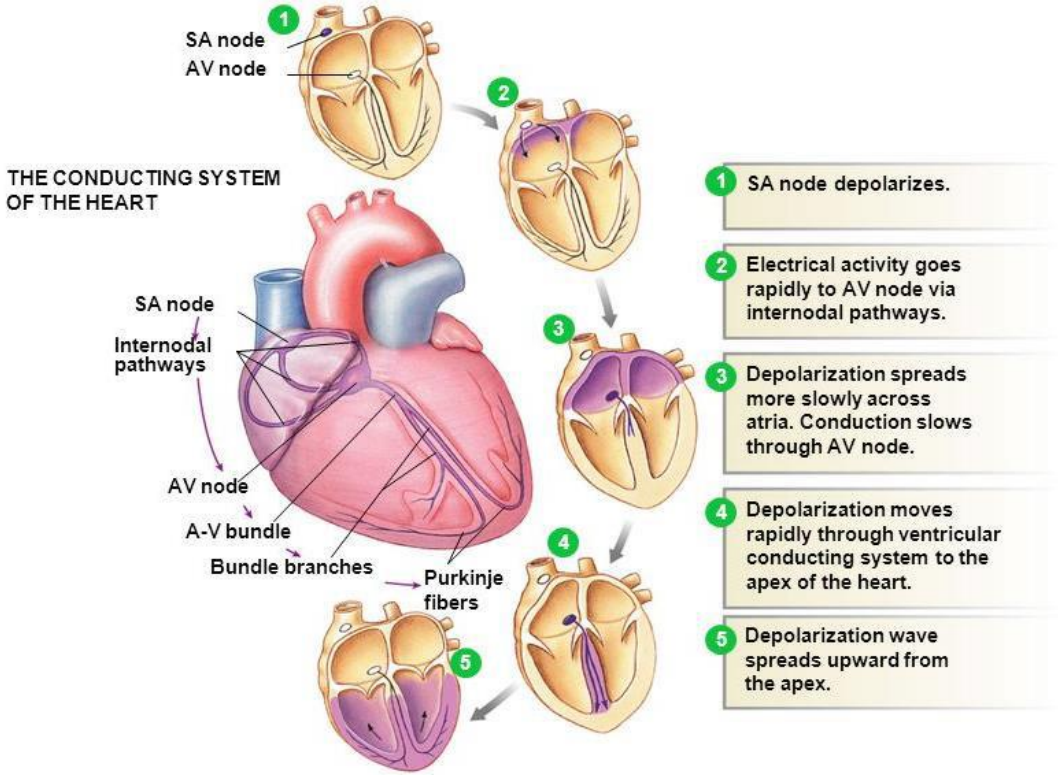
Which of the following is the correct sequence for the spread of cardiac action potentials?

a. SA node → internodal pathways → AV node → AV bundle → bundle branches → Purkinje fibers
b. SA node → AV node → internodal pathways → AV bundle → bundle branches → Purkinje fibers
c. SA node → internodal pathways → AV node → bundle branches → AV bundle → Purkinje fibers
d. SA node → internodal pathways → AV node → AV bundle → Purkinje fibers → bundle branches

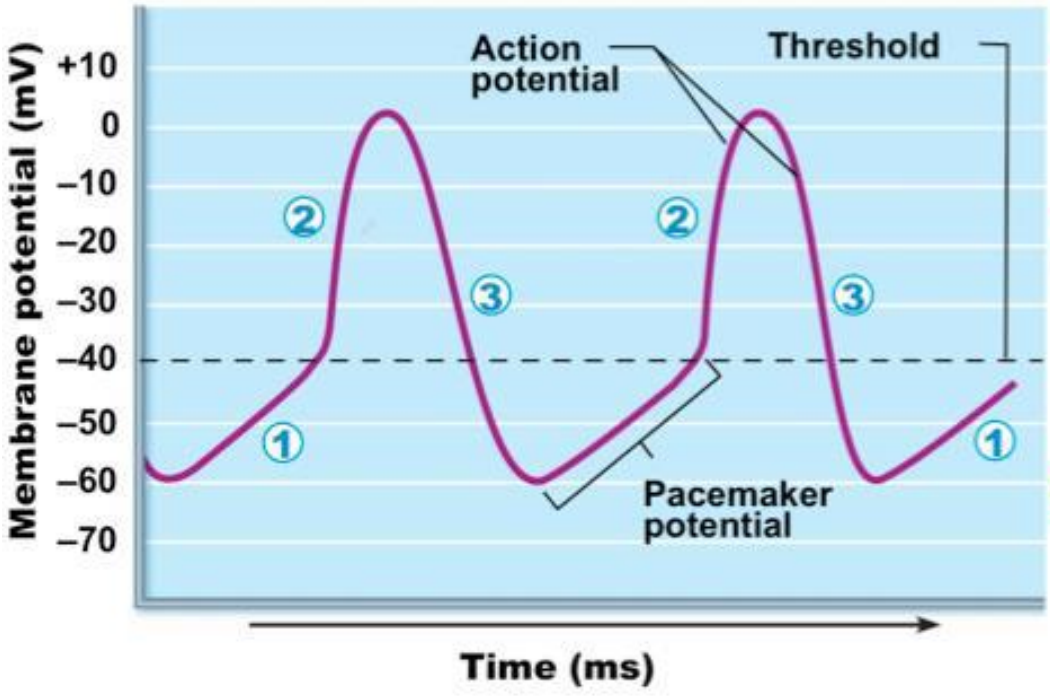
Signal Flow

- SA Node: Pacemaker of the heart
  - How does it generate fast spontaneous APs?
    - ➢ greater permeability to Na^+ and Ca^{2+}
    - ➢ decreased permeability to K^+
  - These properties naturally bring the cell to threshold





SA Node Action potential



1. **Pacemaker potential** This slow depolarization is due to both opening of Na^+ channels and closing of K^+ channels. Notice that the membrane potential is never a flat line.

2. **Depolarization** The action potential begins when the pacemaker potential reaches threshold. Depolarization is due to Ca^{2+} influx through Ca^{2+} channels.

3. **Repolarization** is due to Ca^{2+} channels inactivating and K^+ channels opening. This allows K^+ efflux, which brings the membrane potential back to its most negative voltage.

Compare/Contrast – Action Potentials

	Action Potential	Cardiac AP
RMP	RMP = -70 mV	RMP = ~-60 mV
Threshold	Threshold = -55 mV	Threshold = -40 mV
Stimulus	Graded Potential	Slow Leak (Na^+/Ca^{2+})
Depol. Channels	Depolarization = VG Na^+	Depolarization = VG Ca^{2+}
Repol. Channels	Repolarization = VG K^+	Repolarization = VG K^+
Hyperpol.	Hyperpolarization = Leak channels	N/A

Which of the following is INCORRECT regarding diastole (filling of the heart)?

a. atrioventricular valves are open
b. semilunar valves are closed
c. blood is flowing from the atria into the ventricles
d. pressure in the ventricles is greater than in the atria

Cardiac Cycle

Atrial Systole (Atrial Contraction)

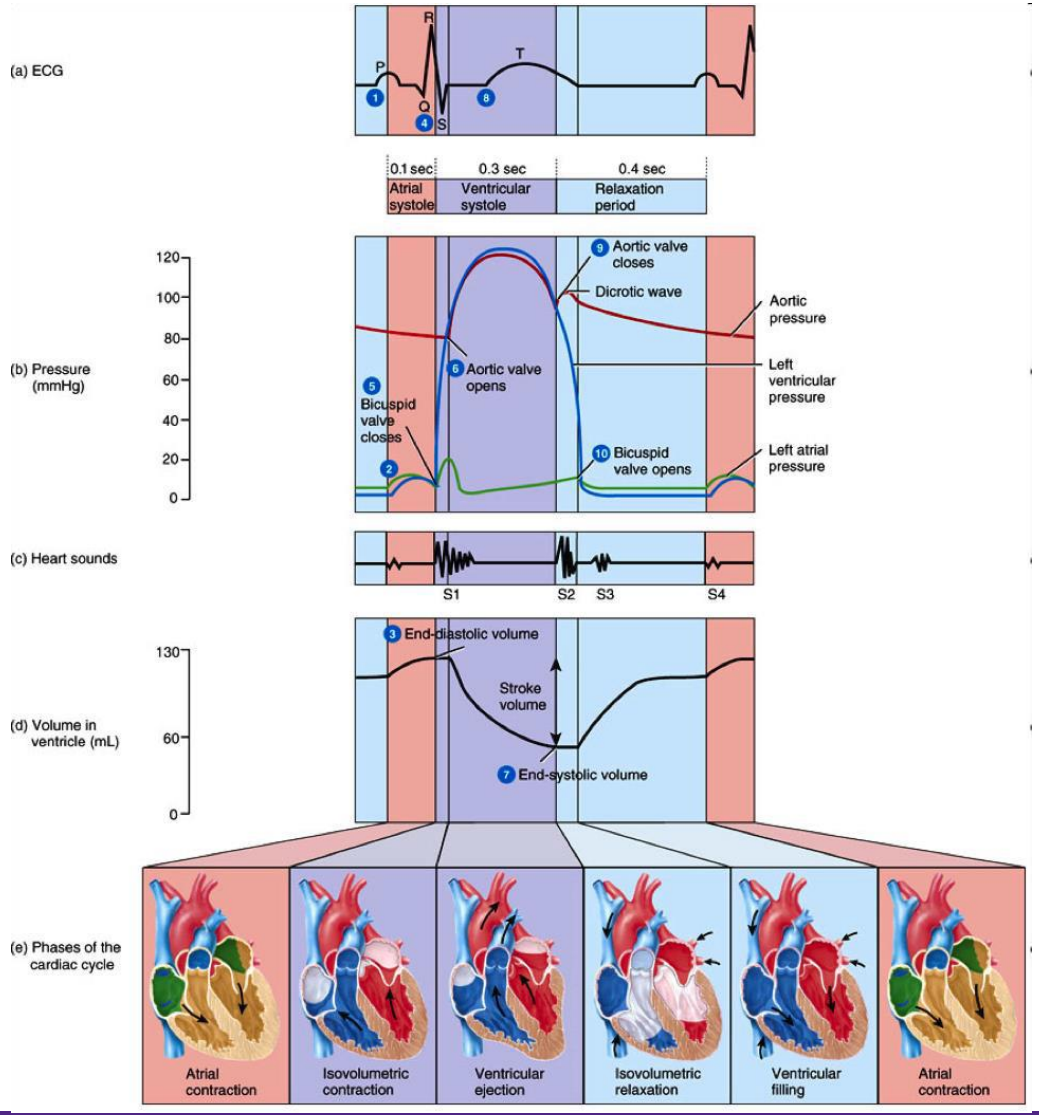
- ECG: P-wave from atria contraction (depolarization)
- Pressure: atria > ventricles
- Valves: AV valve already open
- Volume: blood (30%) fills ventricles to EDV

Early Ventricular Systole (Isovol. Contraction)

- ECG: QRS wave from ventricle contraction (depolarization)
- Pressure: aorta > ventricles > atria
- Valves: AV valve close
- Volume: no change in volume

Ventricular Systole (Ventricular Ejection)

- ECG: T-wave from ventricles finishing contraction
- Pressure: ventricles > aorta
- Valves: aortic valve open
- Volume: blood leaves ventricles to ESV



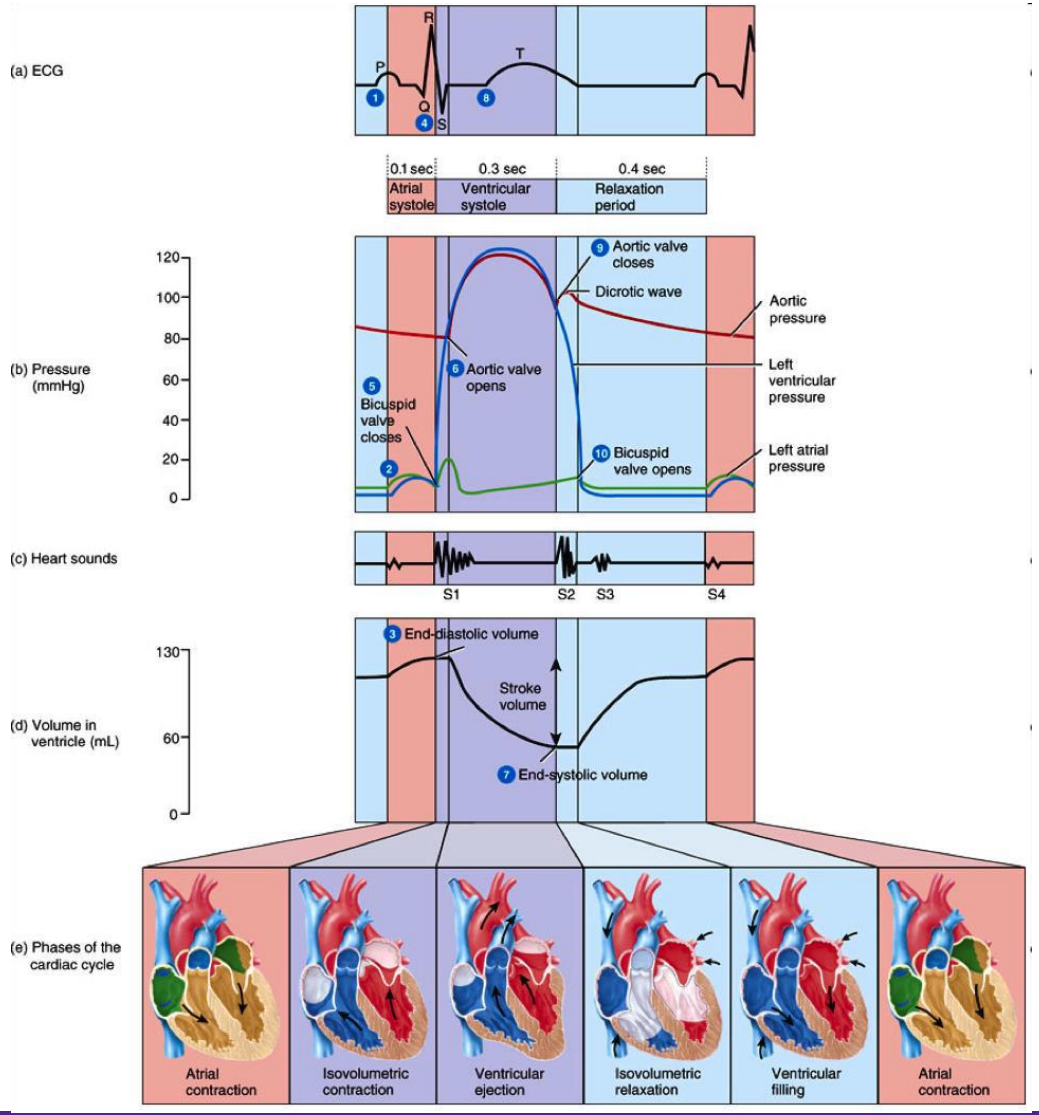
Cardiac Cycle

Early Ventricular Diastole (Isovol. Relaxation)

- ECG: no wave but ventricles relaxing
- Pressure: aorta > ventricles > atria
- Valves: aortic valve close
- Volume: no change in volume

Late Ventricular Diastole (Ventricular Filling)

- ECG: no wave but ventricles finish relaxing
- Pressure: atria > ventricles
- Valves: AV valve open
- Volume: blood (70%) fills ventricles



Heart Rate (HR)

- Average: 70 bpm
- Lower HR = “healthier” (i.e. athletes: 45 bpm)
- Max HR = 220 – age
- Controlled by autonomic nervous system
  - PS-NS: decreases HR
  - S-NS: increases HR

Stroke Volume = EDV - ESV

- End-Diastolic Volume (EDV): volume of blood in ventricles at end of ventricular diastole (just before they contract; end of Phase 1)
- End-Systolic Volume (ESV): volume of blood in ventricles at end of ventricular systole (just after contraction; end of Phase 3)
- Stroke volume = EDV – ESV
  = 160 ml – 90 ml
  = 70 ml
- Altering either EDV or ESV will change stroke volume

Cardiac output can be determined by which of the following formulas?

A. HR – SV
B. HR divided by SV
C. HR + SV
D. HR x SV

Cardiac Output (CO)

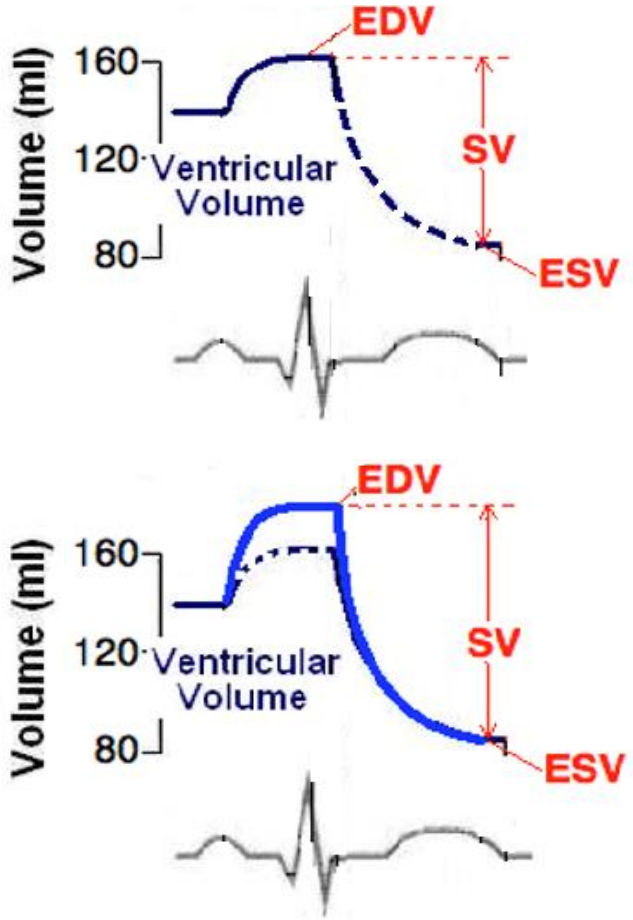
- Cardiac output: volume of blood pumped by each ventricle per minute
- CO = Heart Rate x Stroke Volume
  - Heart Rate = Beats per minute
  - Stroke Volume = Amount of blood pumped by each ventricle per beat
- At rest:
  - CO = 5 L/min
  - HR = 70 beat/min
  - SV = 70-80 mL/beat
  - CO = (70 beat/min)(0.07 L/beat) = 4.9 L/min
- During exercise:
  - CO can increase to 20-40 L/min
  - How? By changing HR and/or SV!

Overall Control of SV by ANS

- Stroke volume: the amount of blood pumped by each ventricle per beat
- Two factors that affect stroke volume:
  - ANS
  - Preload (end diastolic volume)
- PS-NS decreases SV
  - ↓ Ca^{2+} flow into cardiac cells
  - ↓ force of contraction
- S-NS increases SV
  - ↑ Ca^{2+} flow into cardiac cells
  - ↑ force of contraction

Stroke Volume

- During exercise, the S-NS is activated:
  - Heart contracts more forcefully and ejects more blood
  - Thus, ESV decreases
- Meanwhile, the heart is filling with more blood
  - Thus, EDV increases

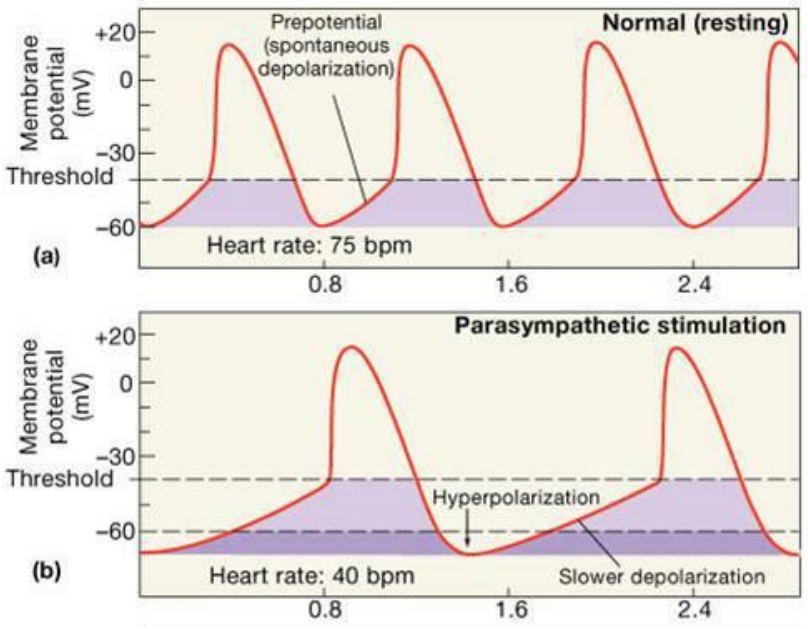


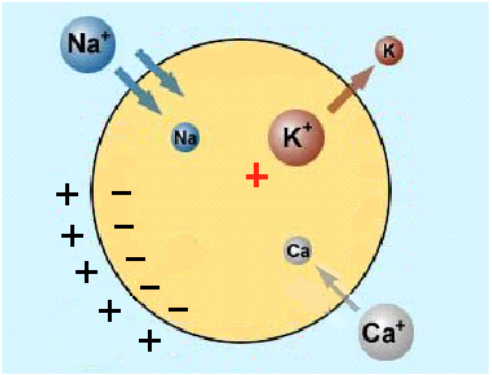
Stroke Volume and Preload

- Preload: the “load” on the cardiac muscle before contraction
- This “load” comes from the blood in the ventricles that stretches the ventricular muscle
  - Thus, higher EDV = greater preload

PNS Effect on HR

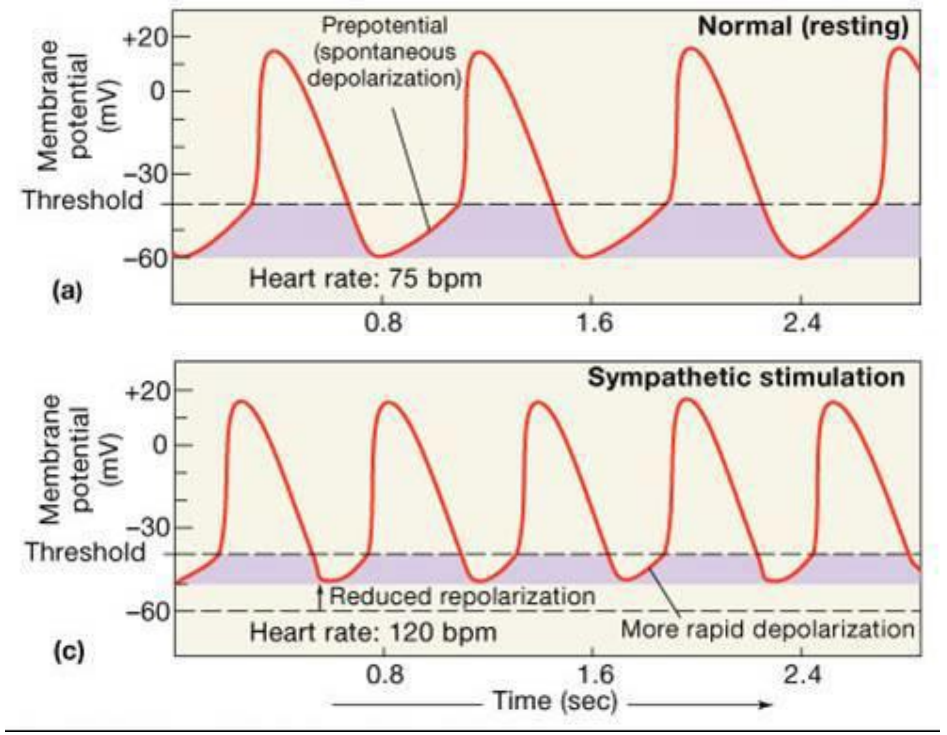
- PNS innervates SA and AV nodes through vagus nerve
  - PNS releases Ach, which binds to receptors on cells of SA and AV nodes
- ↑ K^+ permeability (i.e. more exits cell) and ↓ Ca^{2+} permeability (i.e. less enters cell)
- Net effect:
  - K^+ = HYPERPOLARIZATION
  - Ca^{2+} = Decreases slope of pacemaker potential

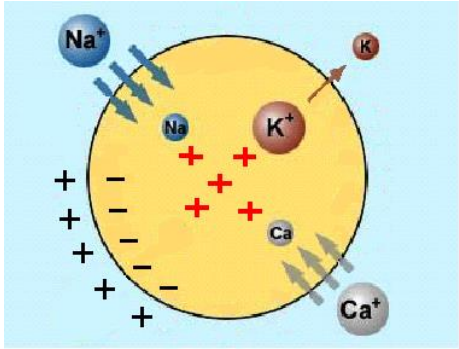




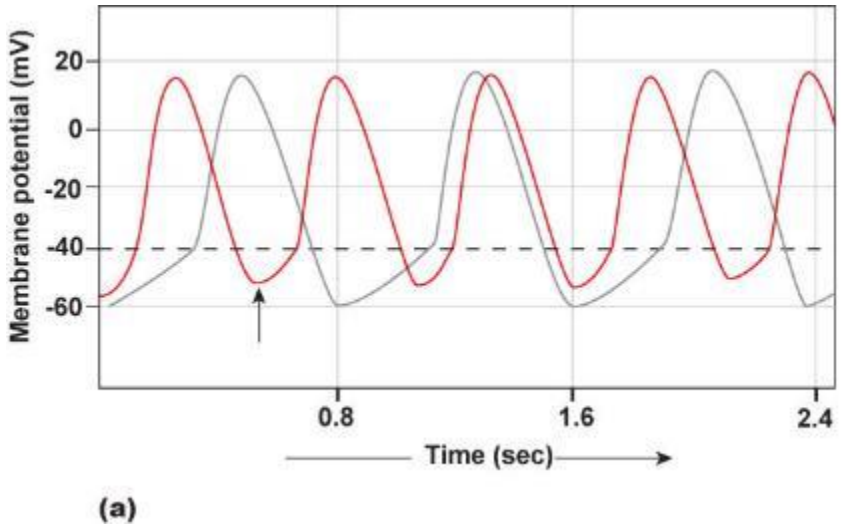
SNS Effect on HR

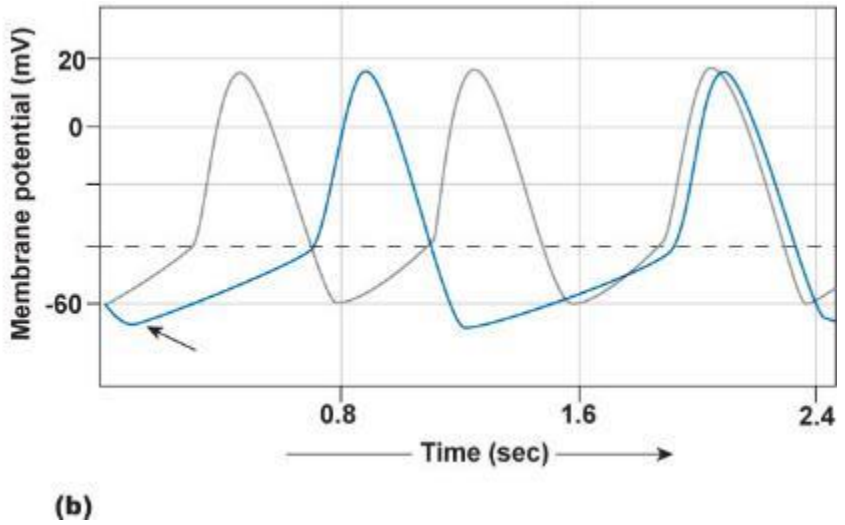
- SNS innervates SA, AV nodes and ventricular muscles
  - ➢ SNS releases NE, which binds to receptors on cells of nodes and muscle
- ↑ Na^+ and Ca^{2+} permeability (i.e. more enters cell)
- Net effect: DEPOLARIZATION and increased slope of pacemaker potential



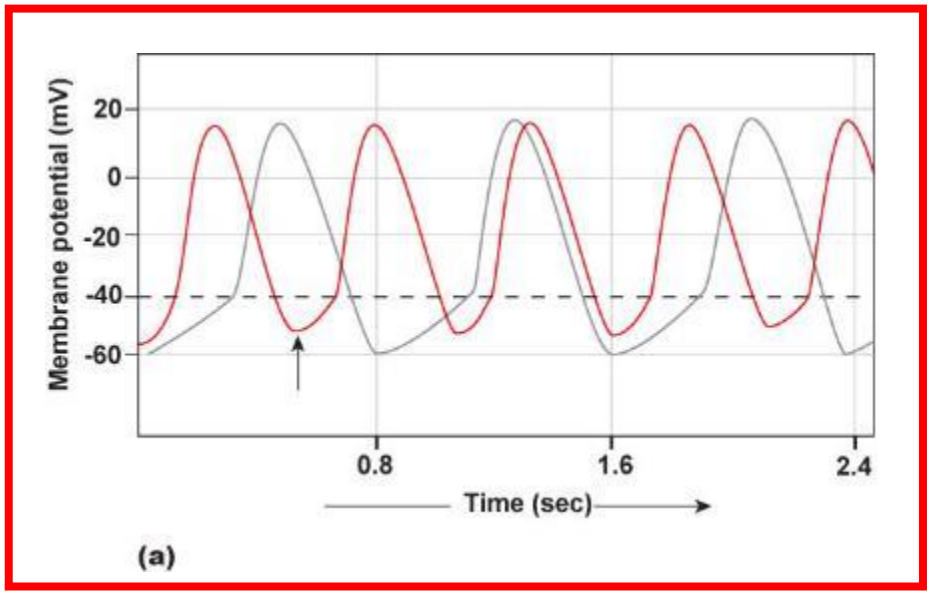


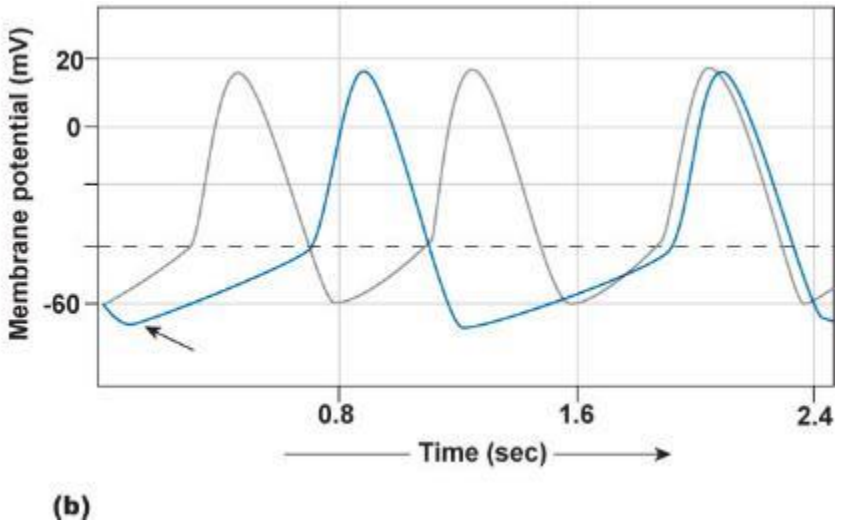
Which graph represents sympathetic influence on heart rate (in both cases the light grey line is under resting conditions)?





Which graph represents sympathetic influence on heart rate (in both cases the light grey line is under resting conditions)?





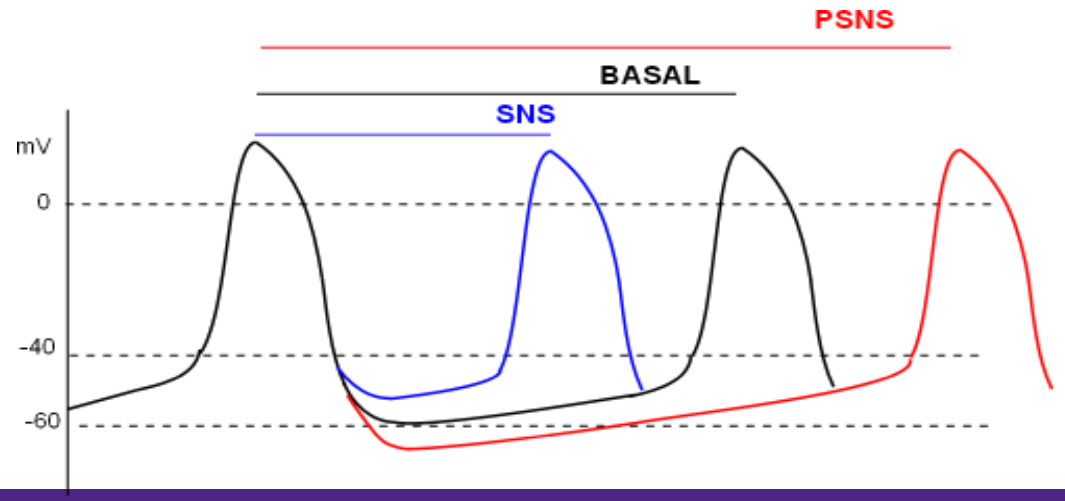
Summary of ANS Control of Heart Rate

PSNS

- Acetylcholine released onto SA/AV node
- Decreases heart rate and force of contraction
  - ➢ Increase K^+, decrease Ca^{2+} permeabilities
  - ➢ Decreases slope of pacemaker potential

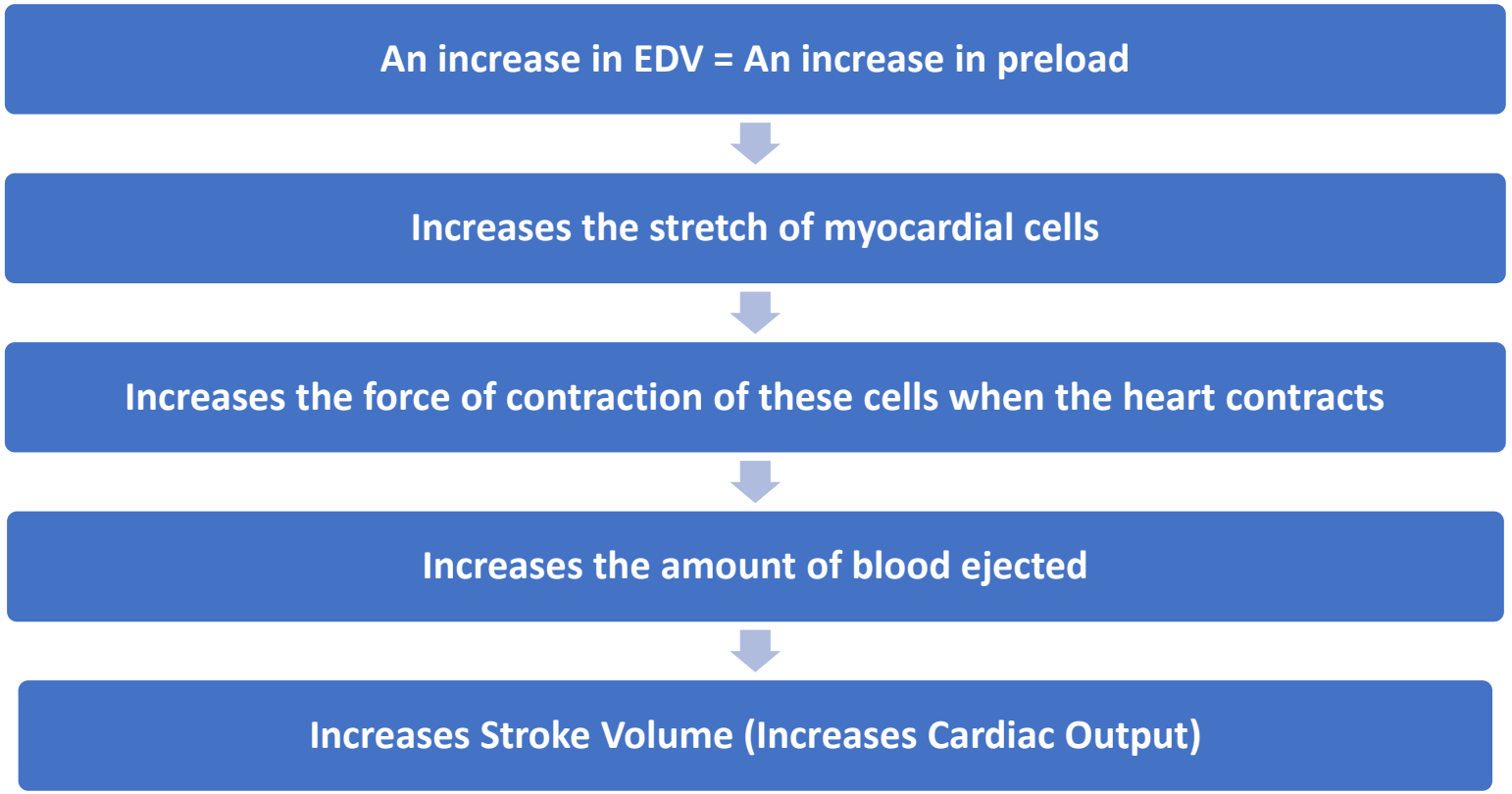
SNS

- Release norepinephrine onto SA/AV node
- Increases heart rate and force of contraction
  - ➢ Increase Na^+ and Ca^{2+} permeability
  - ➢ Increase slope of pacemaker potential



Frank-Starling Law

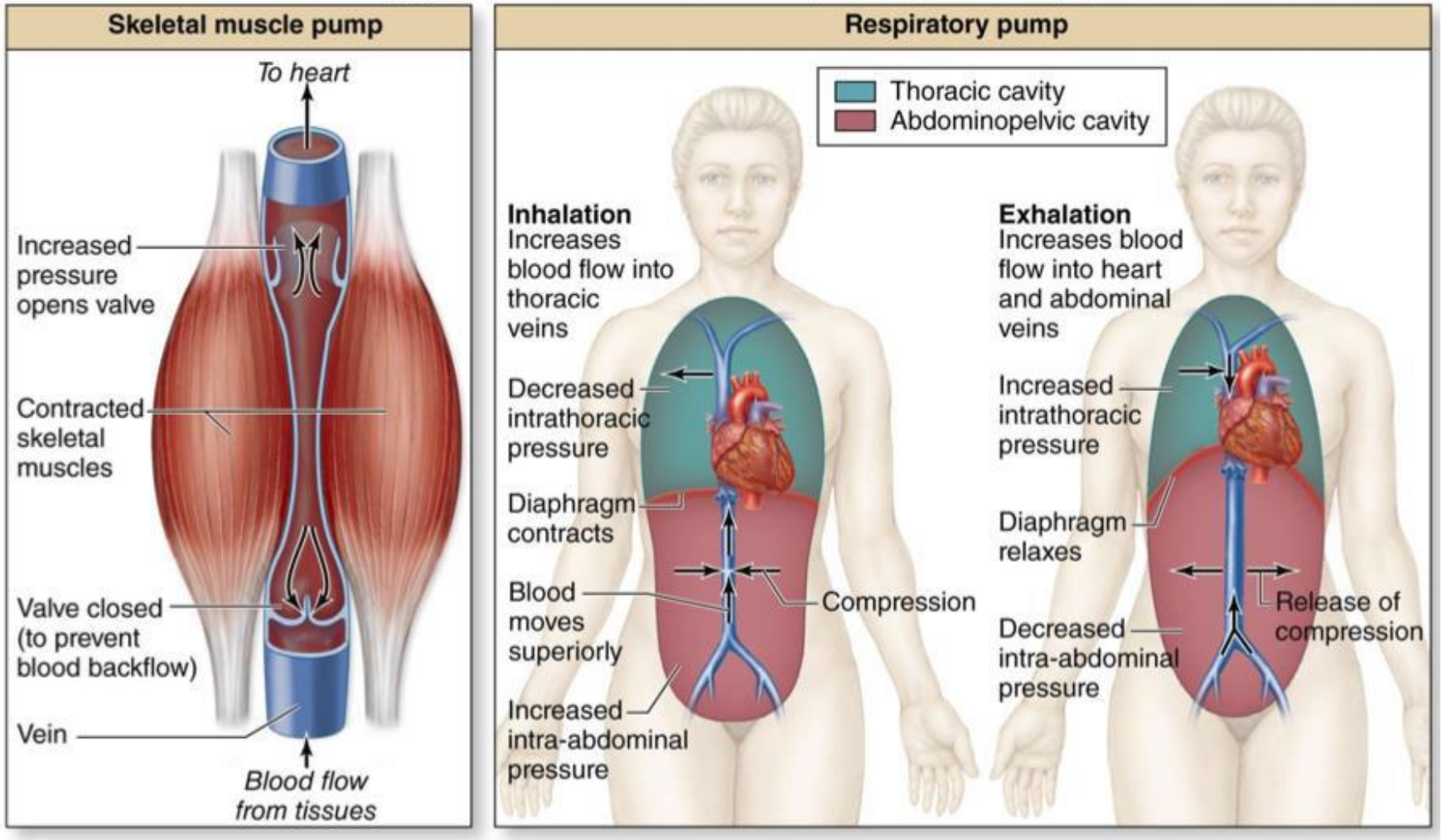
Frank-Starling Law states that “an increase in EDV will increase stroke volume”



Frank-Starling Law and Venous Return

- How to increase EDV? Increase venous return to the heart!
- During dynamic exercise:
  1. Muscle Pump: contracted skeletal muscle around veins pushes blood to heart
  2. Respiratory Pump: changes in pressure during breathing pushes blood towards the heart
  3. S-NS: constriction of veins squeezes blood to heart

Frank-Starling Law and Venous Return

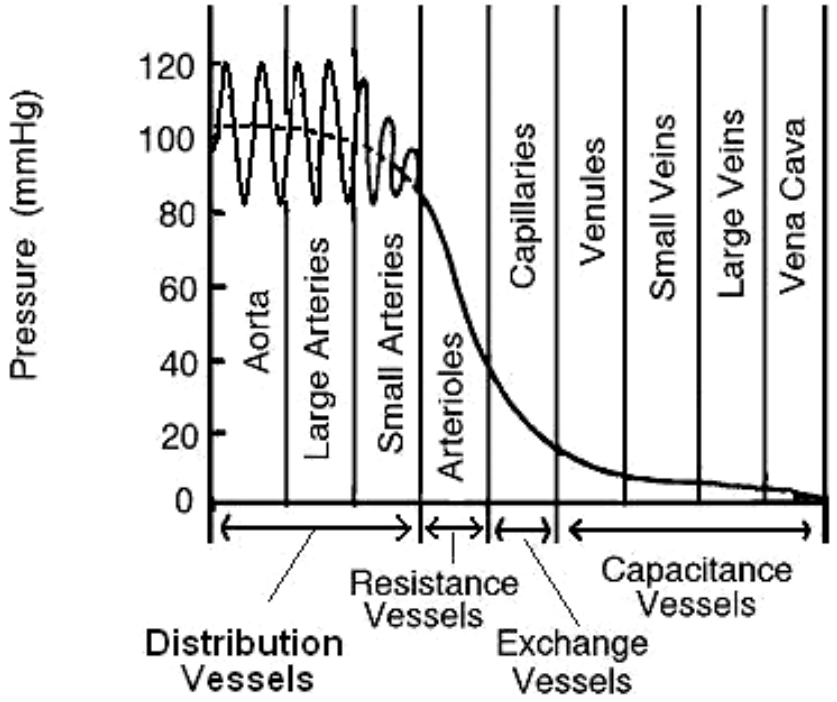


The aortic semilunar valve prevents blood from returning to the _____.

A. left ventricle
B. aorta
C. right ventricle
D. left atrium

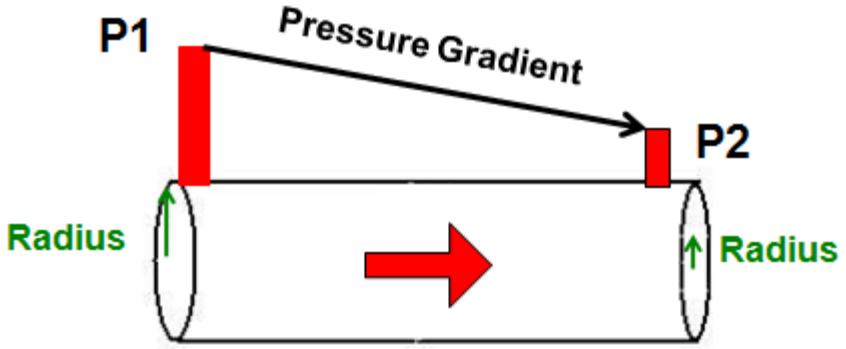
Blood Vessels

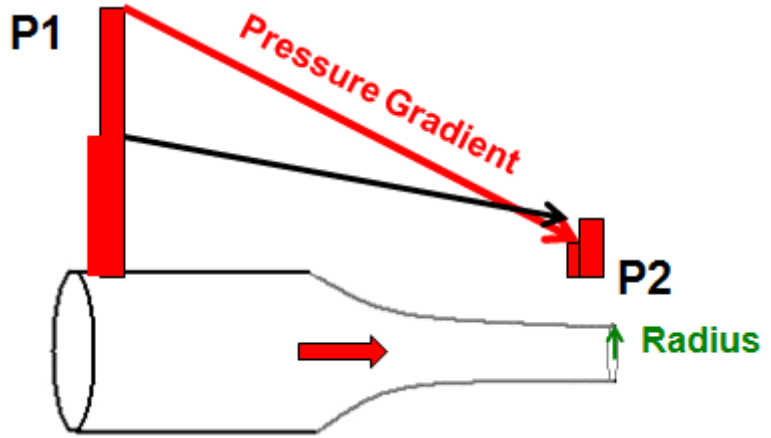
- Structural properties of vessels are what contribute to the blood pressure characteristics seen in circulation



Vessel Constriction and Blood Flow

- As the radius decreases the pressure gradient increases.





Relationship Between Pressure, Flow and Resistance (Page 232)

$$Resistance = \frac{L\,\eta}{r^4}$$

L = length of vessel
η = viscosity of the fluid
r = radius of the vessel

$$Resistance = \frac{1}{r^4}$$

$$Blood\ Flow = \frac{P_1 - P_2}{\frac{1}{r^4}} = \boxed{(P_1 - P_2) * r^4}$$

Just know this part of the equation

A small change in radius will have a LARGE effect on blood flow

Relationship Between Pressure, Flow and Resistance

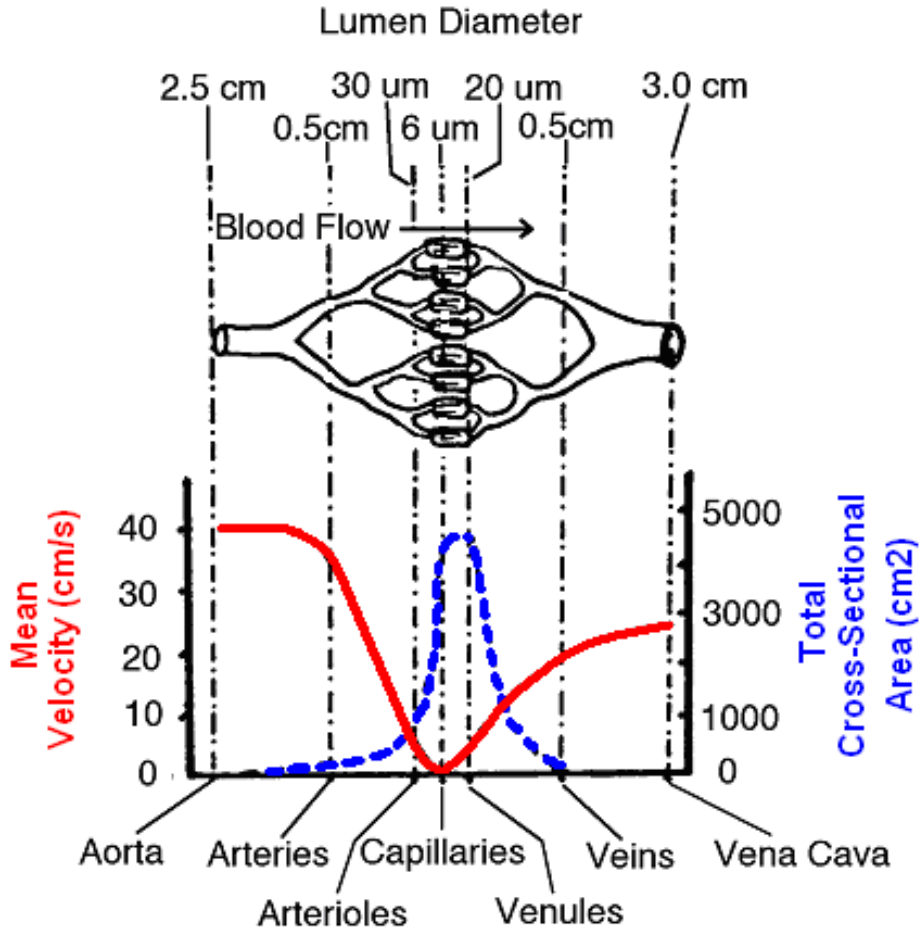
$$\boldsymbol{Blood\ Flow = (P_1 - P_2) * r^4}$$
$$\boldsymbol{Blood\ Flow = (4 - 2) * 1^4}$$
$$\boldsymbol{Blood\ Flow = 2\ \text{L/min}}$$

$$\boldsymbol{Blood\ Flow = (P_1 - P_2) * r^4}$$
$$\boldsymbol{Blood\ Flow = (10 - 2) * 0.5^4}$$
$$\boldsymbol{Blood\ Flow = 0.5\ \text{L/min}}$$

A small change in radius will have a LARGE effect on blood flow

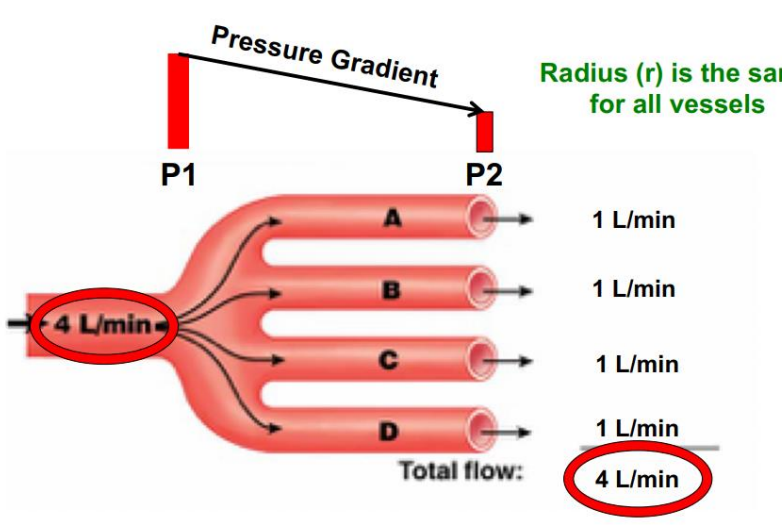
Defining terms

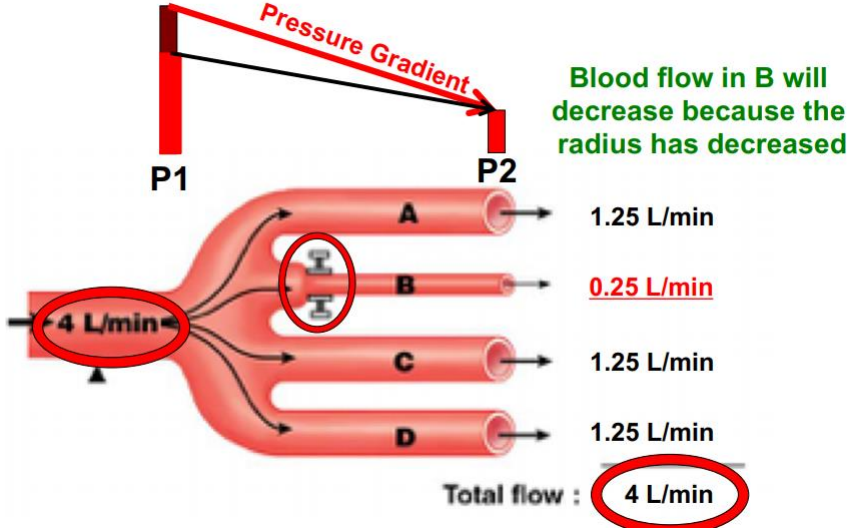
- Blood velocity (cm/sec): speed at which blood is moving through particular blood vessel
  - Fluid flows faster through a narrow tube than a larger tube
  - As cross sectional area increases mean velocity decreases
- Blood flow (L/min): volume of blood moving through set of vessels



Overall blood flow does not change

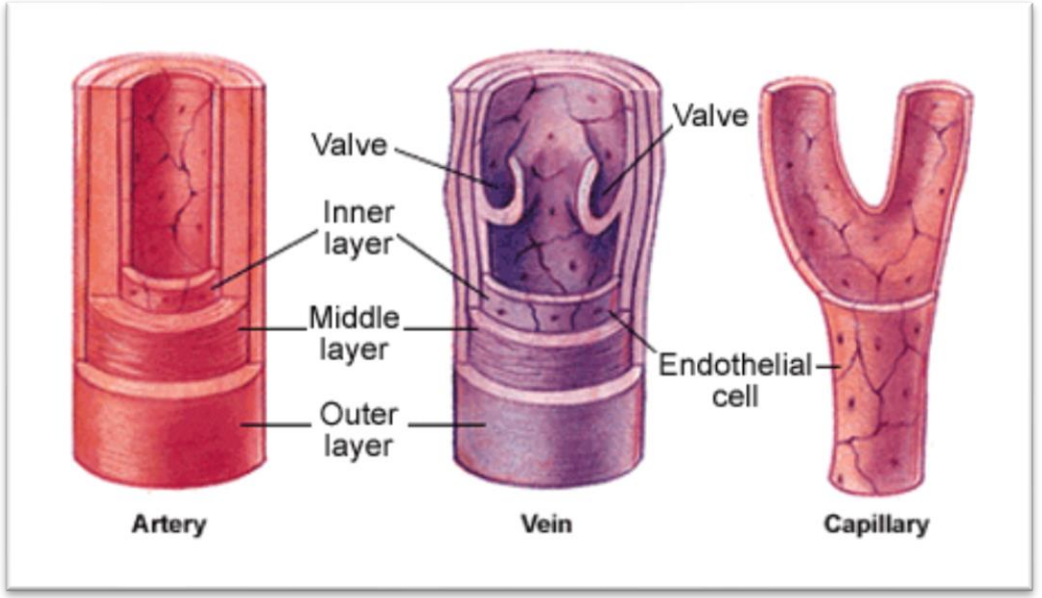
- Blood flow can change due to constriction in vessel





Arteries and Veins

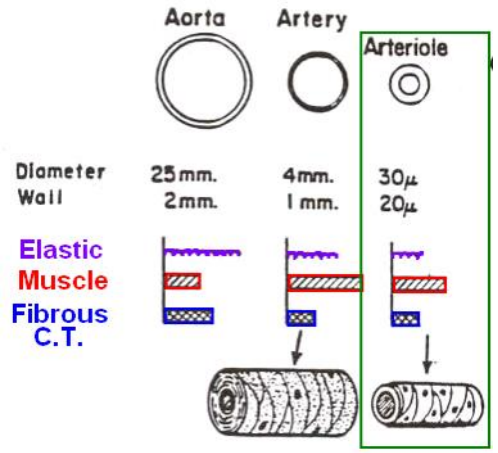
- Contain three layers:
  - Outer Layer – Tunica externa
    - Fibrous connective tissue
  - Middle Layer – Tunica media
    - Smooth muscle and elastic tissue
  - Inner Layer – Tunica interna
    - Endothelial cells
- Veins contain valves
- Capillaries have single layer of endothelial cells

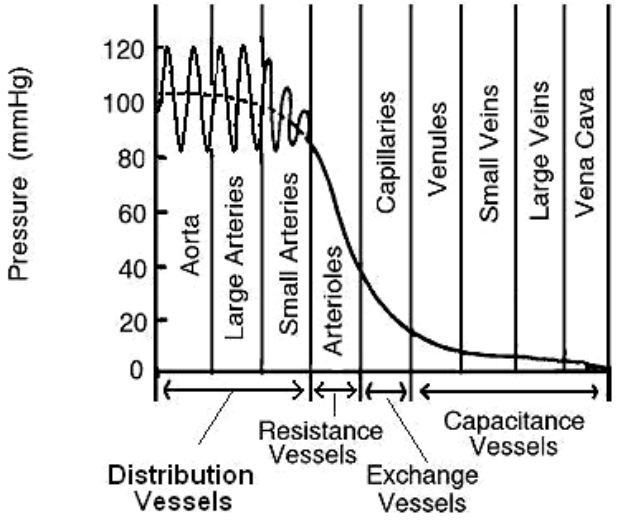


Aorta and Large Arteries

	Blood Characteristics	Structure	Purpose

Aorta/Large Arteries	- High blood pressure
	- 80-120 mmHg
	- High blood velocity
		- Large diameter
		- Elastic tissue
		- Thin walls
		  ▪ Easily distended
		  ▪ Low resistance to blood flow
		  ▪ Small drop in blood pressure
			- ‘Shock absorbers’
			- Distribute the blood

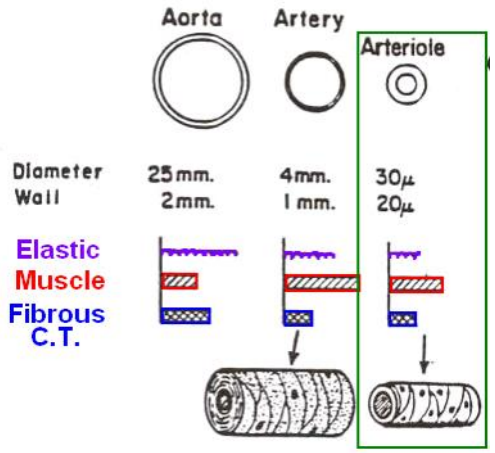


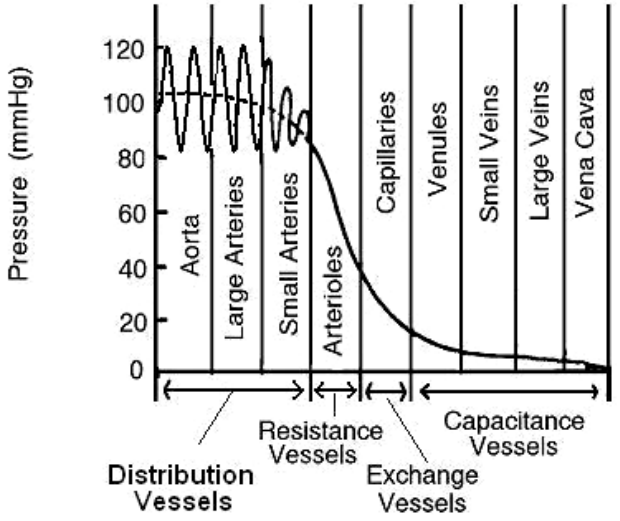


Arterioles

	Blood Characteristics	Structure	Purpose

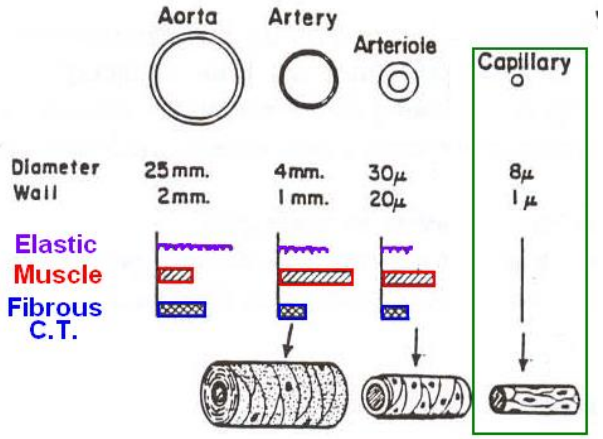
Arterioles	- Large drop in blood pressure
	- Lower blood velocity
		- Small diameter
		- Very thick walls
		- Smooth muscle of walls innervated by ANS
		  ▪ Causes vasoconstriction/dilation
		  ▪ Controls blood flow velocity
			- Resistance vessels
			- Control blood flow velocity to organs

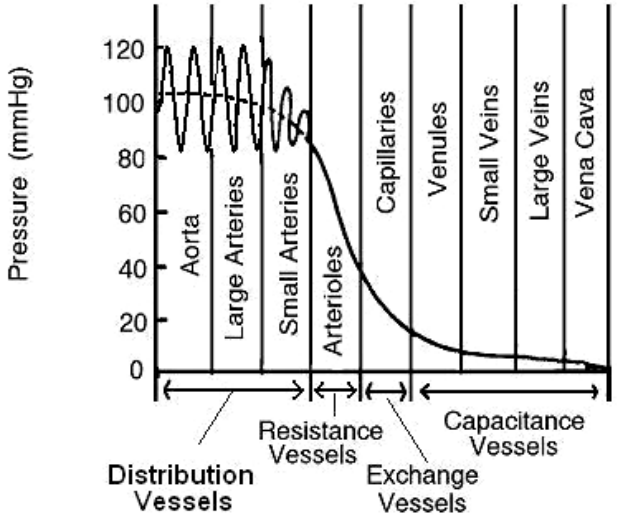




Capillaries

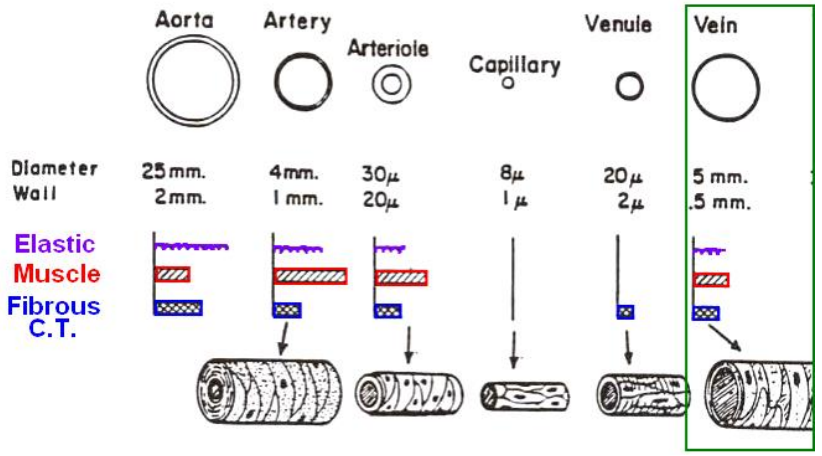
	Blood Characteristics	Structure	Purpose
Capillaries	- Low blood pressure - Small drop in blood pressure - Very low blood velocity (1-2 cm/sec)	- One endothelial cell thick - Large cross sectional area - Very large surface area ▪ Diffusion of gas, nutrients and waste	- Exchange vessels

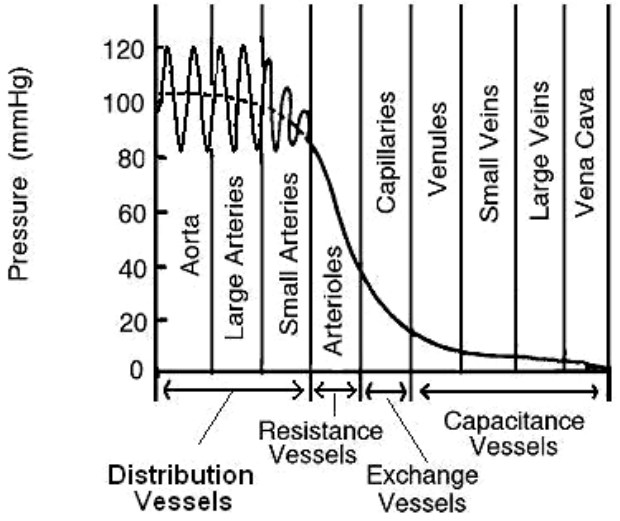




Veins

	Blood Characteristics	Structure	Purpose
Veins	- Low blood pressure - Low to medium blood velocity (5-10 cm/sec)	- Very thin walls with large diameter - Contain valves - Some elastic tissue - Smoth of smooth muscle innervated by ANS ▪ Vasoconstriction/dilation	- Capacitance vessels: 70% of TBV





At the arteriolar end of a capillary, _____ pushes fluid into the capillary:

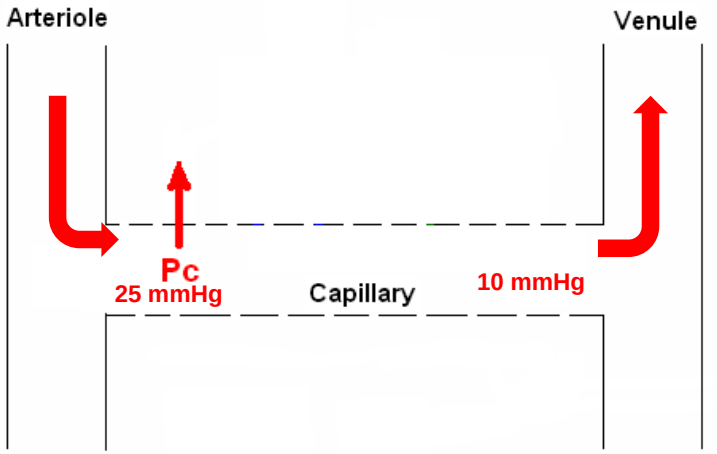
a. hydrostatic pressure in the interstitial fluid
b. osmotic pressure in the interstitial fluid
c. osmotic pressure in the capillary
d. hydrostatic pressure in the capillary

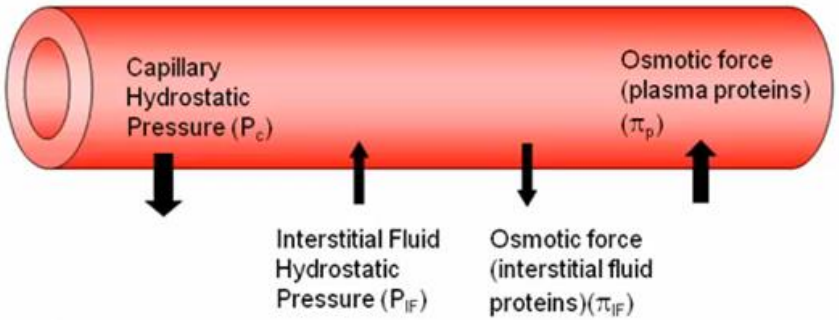
Starling Forces

- Two hydrostatic pressures
  - Capillary hydrostatic pressure
  - Interstitial fluid hydrostatic pressure
- Two osmotic pressures
  - Plasma osmotic pressure
  - Interstitial osmotic pressure

Capillary Hydrostatic Pressure (P_c)

- Pressure exerted by fluid in the capillary
- Pressure drives fluid OUT of capillary and is generated by ventricular systole (Filtration)

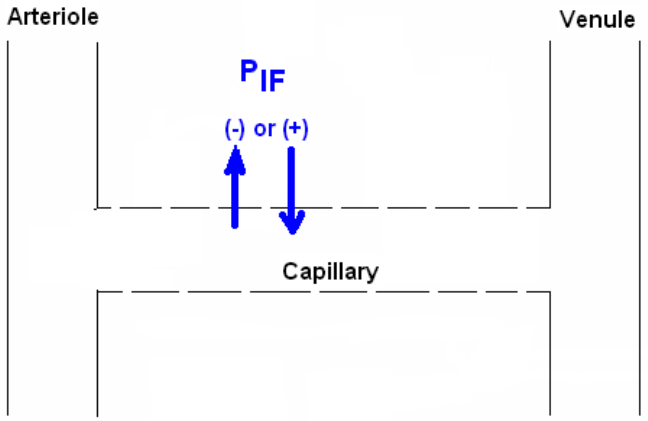


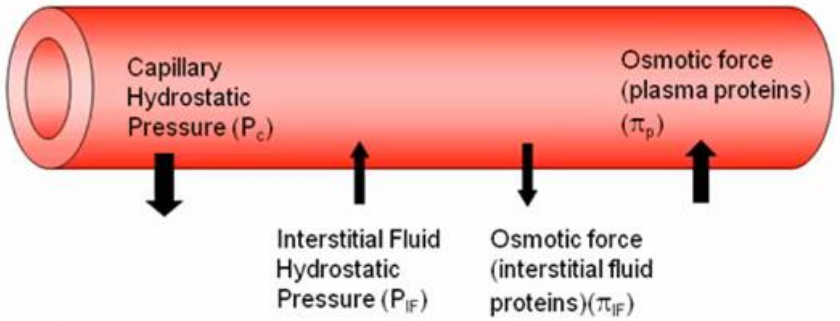


Interstitial Fluid Hydrostatic Pressure (P_{IF})

- Pressure exerted by fluid in the interstitial space between cells in the tissue
- Movement depends on pressure in the tissue
  - Can be negative → Filtration into tissue
  - Can be positive → Reabsorption into capillary

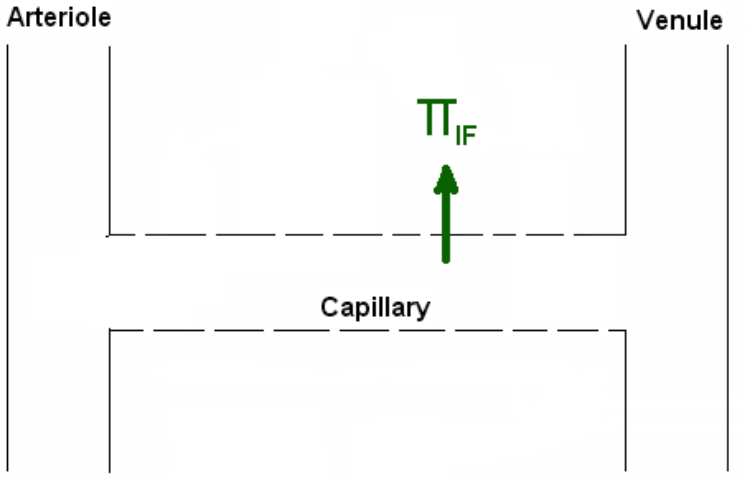
Subcutaneous tissues: -6mmHg
Interstitial fluid: +6mmHg

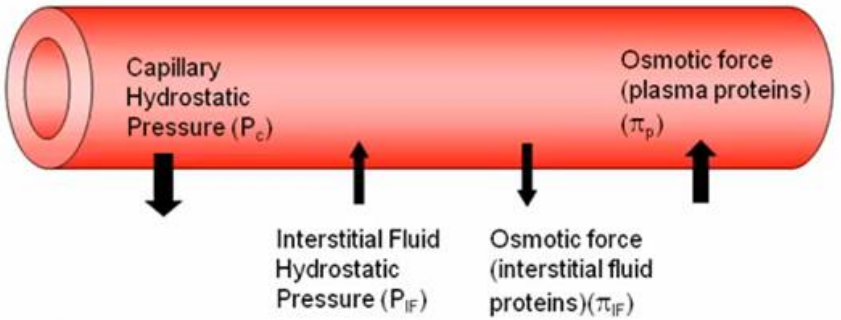




Interstitial Osmotic Pressure (π_{IF})

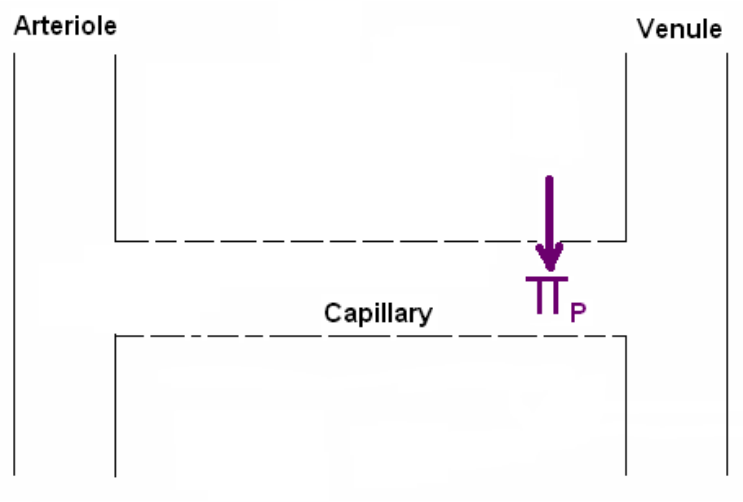
- Pressure caused by osmosis due to few proteins in interstitial fluid (5mmHg)
- Pressure drives fluid OUT of capillary and into tissue (Filtration)

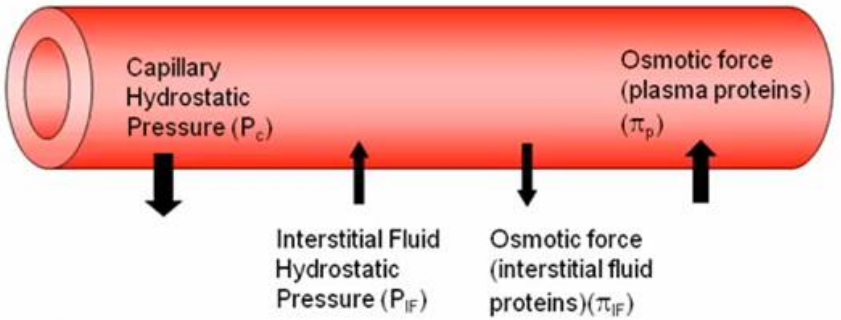




Plasma Osmotic Pressure (π_P)

- Pressure caused by osmosis due to proteins in plasma (28mmHg)
- Pressure drives fluid INTO capillary (Reabsorption)





Balance of Starling Forces

- Starling-Landis equation used to calculate net fluid movement (NFM) across capillary bed

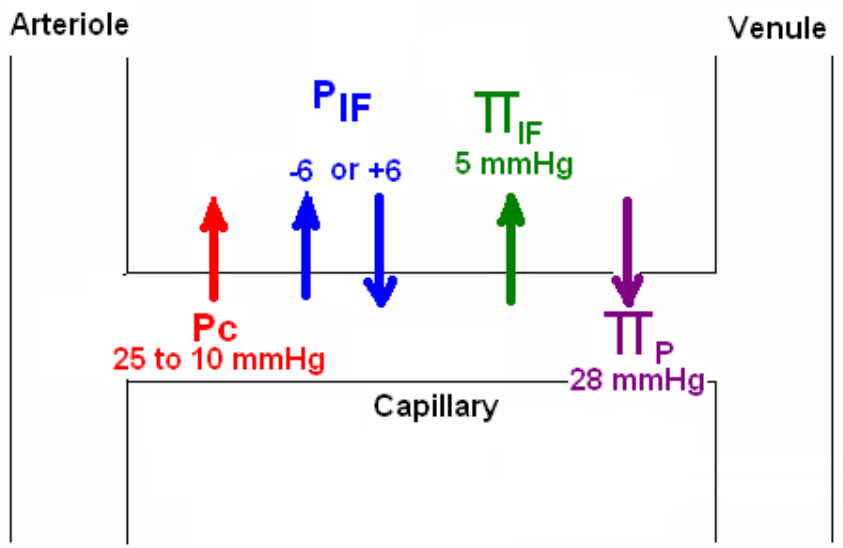
$$NFM = K_f[(P_c - P_{IF}) - (\pi_P - \pi_{IF})]$$

- K_f is filtration coefficient, which represents permeability of capillary (assume 1)

$$NFM = 1[(25 - (-6)) - (28 - (+5))]$$

$$NFM = +8\, mmHg$$

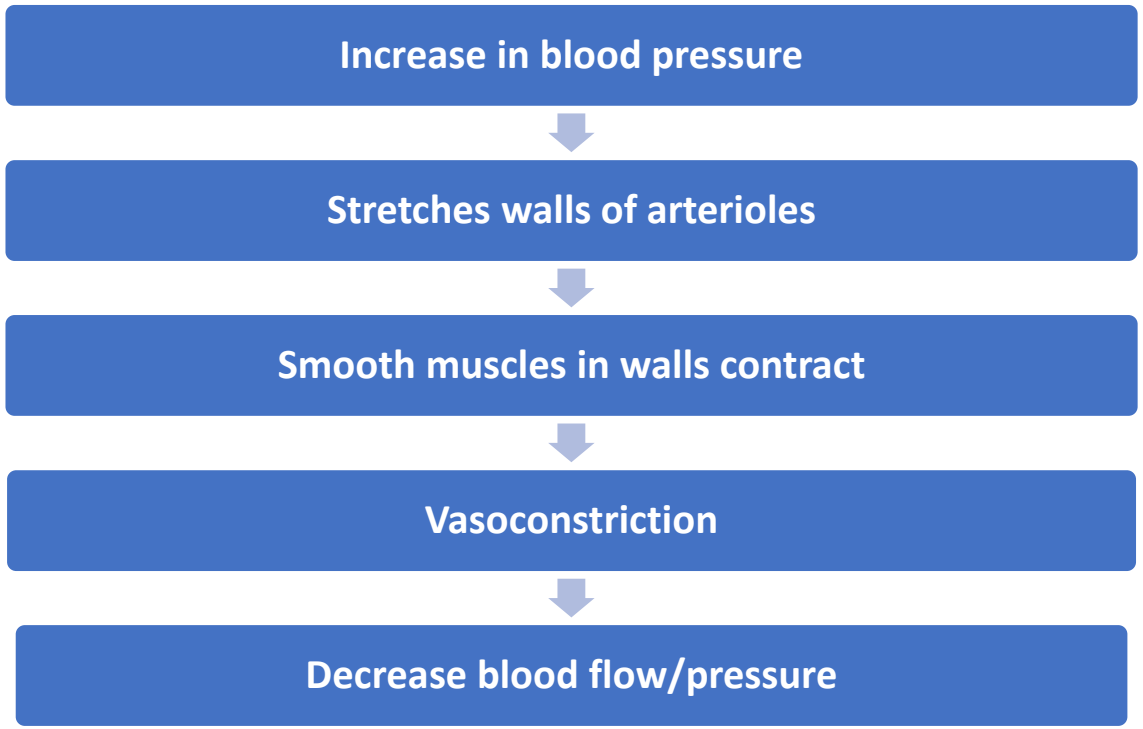
- If positive filtration OUT of capillary, if negative reabsorption INTO capillary



$$P_C = 10,\ P_{IF} = 1,\ \pi_{IF} = 5,\ \pi_C = 28$$

Local Intrinsic Mechanisms: Myogenic Theory

- Maintain blood flow when there is change in blood pressure
- Increase in blood pressure → contraction of smooth muscles in arterioles (Vasoconstriction)
- Decrease in blood pressure → relaxation of smooth muscles in arterioles (Vasodilation)



Local Intrinsic Mechanisms: Metabolic Theory

- Active tissue releases metabolic by-products called vasodilator metabolites (VDMs)
  - Increase in: CO^2, $[H^+]$, adenosine, temperature
  - Decrease in: O^2
- Increase in VDMs → relaxation of smooth muscles in vessels (Vasodilation)
- Leads to increase in blood flow rate

Humoral Mechanisms

- Regulation by substances present in blood
- Vasoconstrictor agents
  - Epinephrine: attaches to alpha receptors in blood vessels
    - Weak vasoconstriction
  - Angiotensin II: production stimulated by drop in blood pressure and drop in sodium levels
    - Very potent vasoconstrictor
  - Vasopressin: formed in the hypothalamus, promotes reabsorption of water in kidneys
    - High amounts will produce vasoconstriction

Humoral Mechanisms

- Regulation by substances present in blood
- Vasodilator agents:
  - Epinephrine: attaches to beta receptors in blood vessels of skeletal/cardiac muscle and the liver
    - Causes vasodilation
  - Kinins: blood protein involved in inflammation, blood pressure and blood coagulation
  - Histamine: released by damaged cells, involved in inflammatory response
  - Atrial natriuretic factor: released by atrial muscle cells
    - Very powerful vasodilator

Neural Control Mechanisms

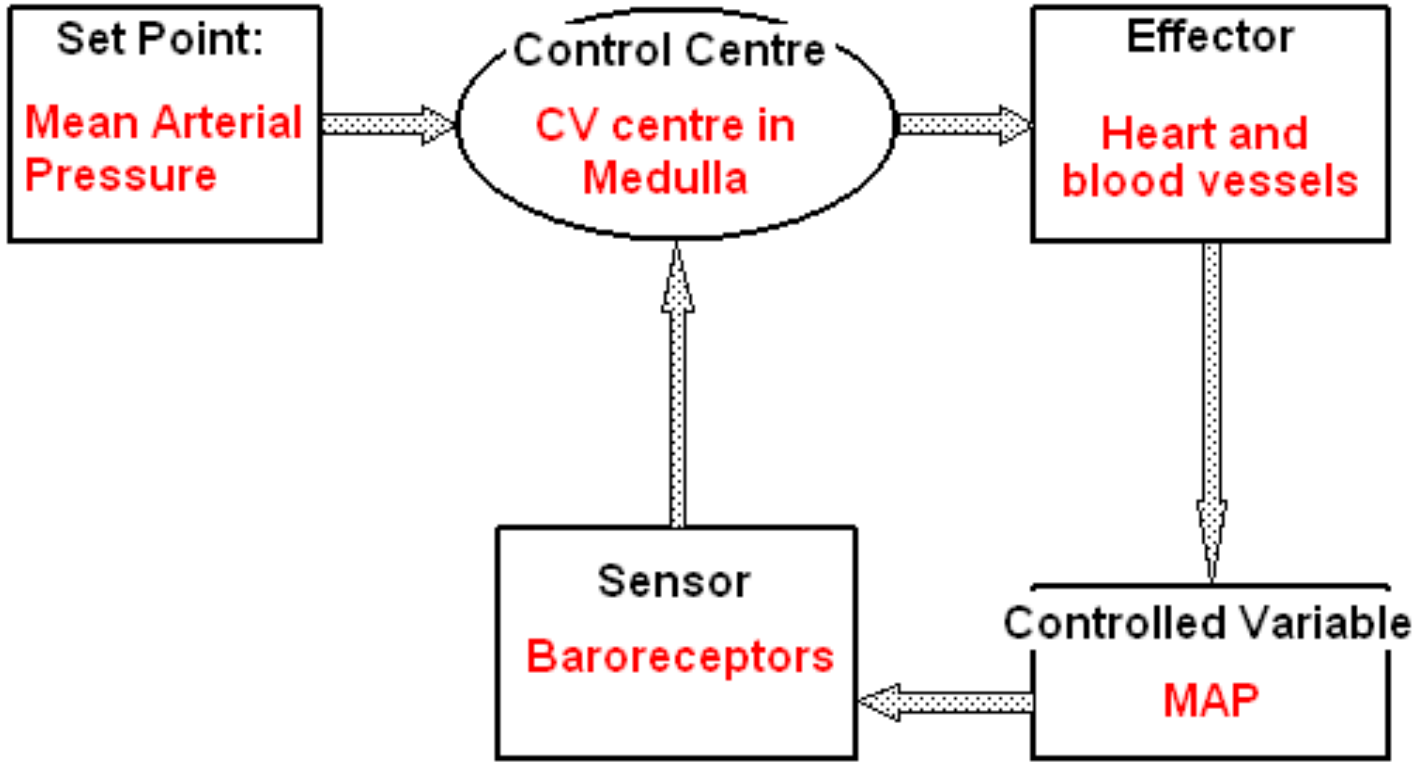
- ANS can rapidly induce changes in blood flow by vasodilation/constriction
- Sympathetic nervous system:
  - Norepinephrine released onto blood vessels → vasoconstriction
  - Acetylcholine causes release of epinephrine from adrenal glands
    - Alpha receptors constrict
    - Beta receptors dilate
- Parasympathetic nervous system:
  - Small indirect effect on blood vessels, causes weak vasodilation
    - Indirect because SNS is shut off

Baroreceptor Reflex

- Negative feedback system that maintains mean arterial pressure (MAP)
- Regulates cardiac output (CO) and total peripheral resistance (TPR)
  - TPR is all resistance encountered by blood in entire systemic circulation
- MAP is average pressure throughout the entire cardiac cycle

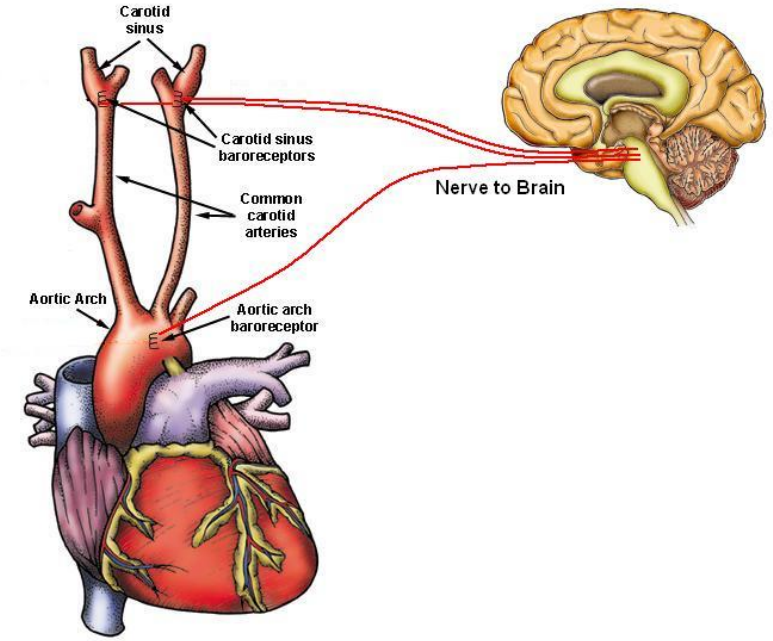
$$MAP = CO * TPR$$
$$CO = HR * SV$$

Baroreceptor Reflex

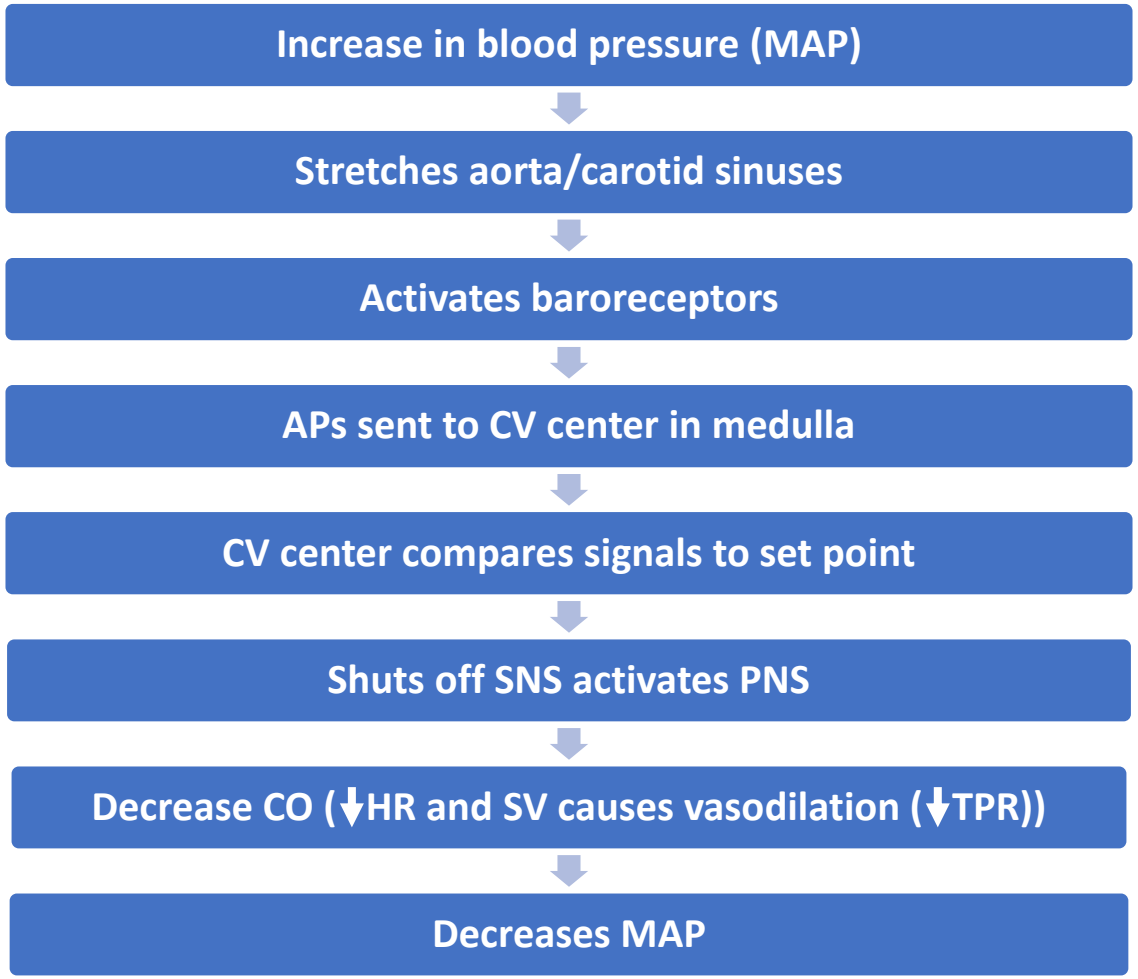


Baroreceptor Reflex

- Located in walls of aortic arch and carotid sinuses
- Stretch sensitive receptors that monitor blood pressure
- Send APs back to cardiovascular center in the medulla of brainstem



Baroreceptor Reflex



Equations to know

- CO = HR x SV
- Max HR = 220 – age
- Stroke volume = EDV –ESV
- $Blood\ Flow = (P_1 - P_2) * r^4$
- $NFM = K_f[(P_c - P_{IF}) - (\pi_P - \pi_{IF})]$
- $MAP = CO * TPR$

What Questions Do You Have?

You can ask in the **Owl forums** as well!

Also anonymously ask questions in the **online dropbox**!!

Western