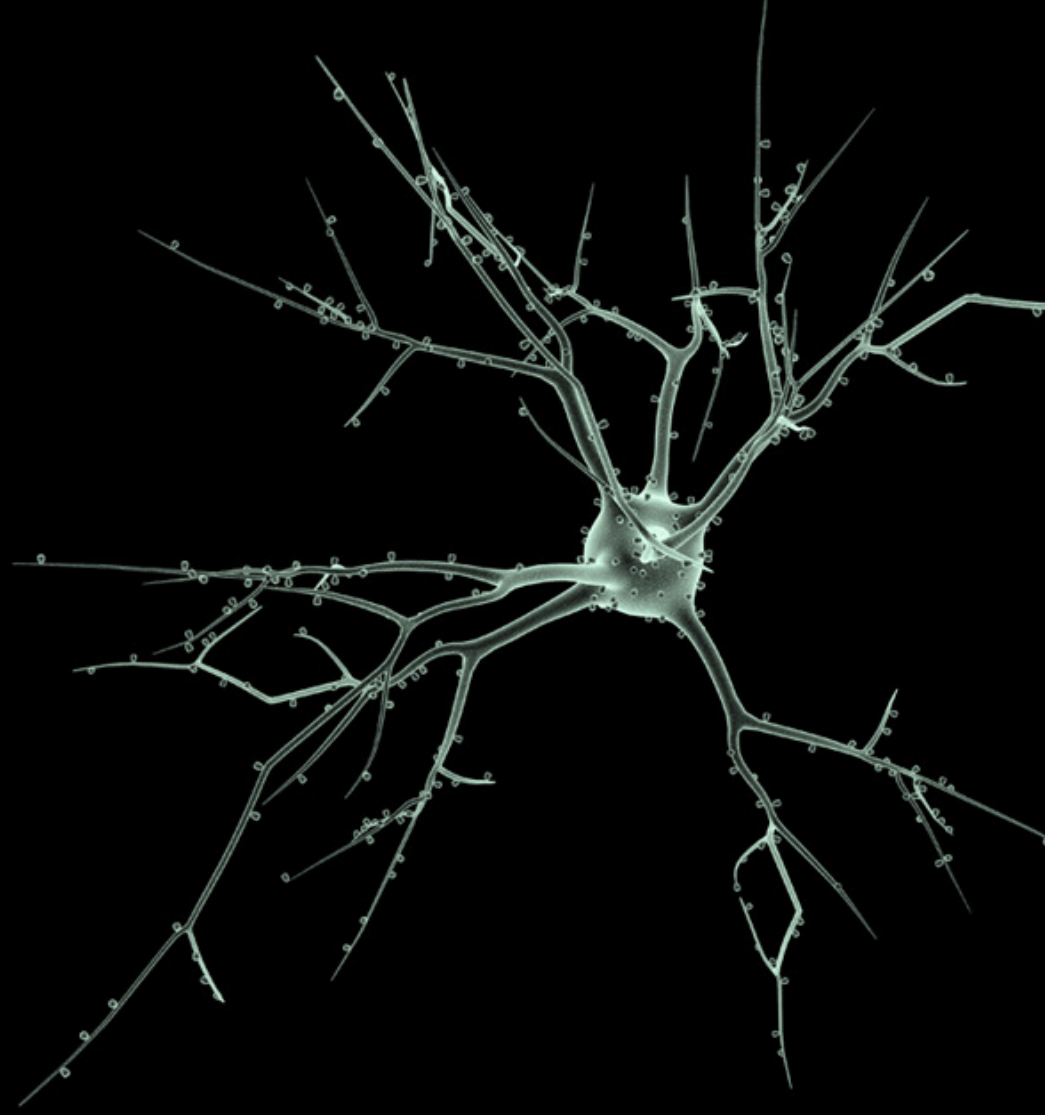
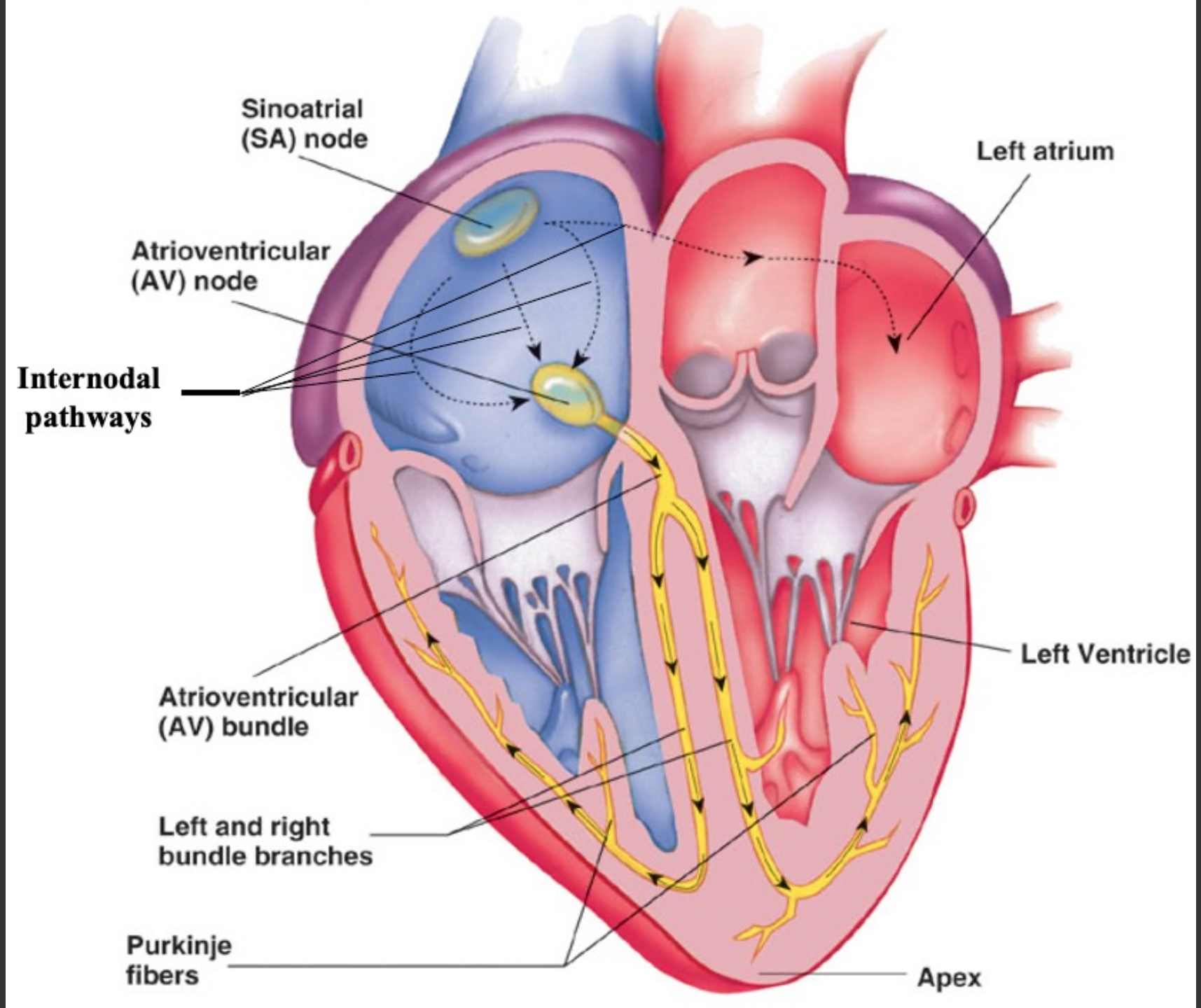
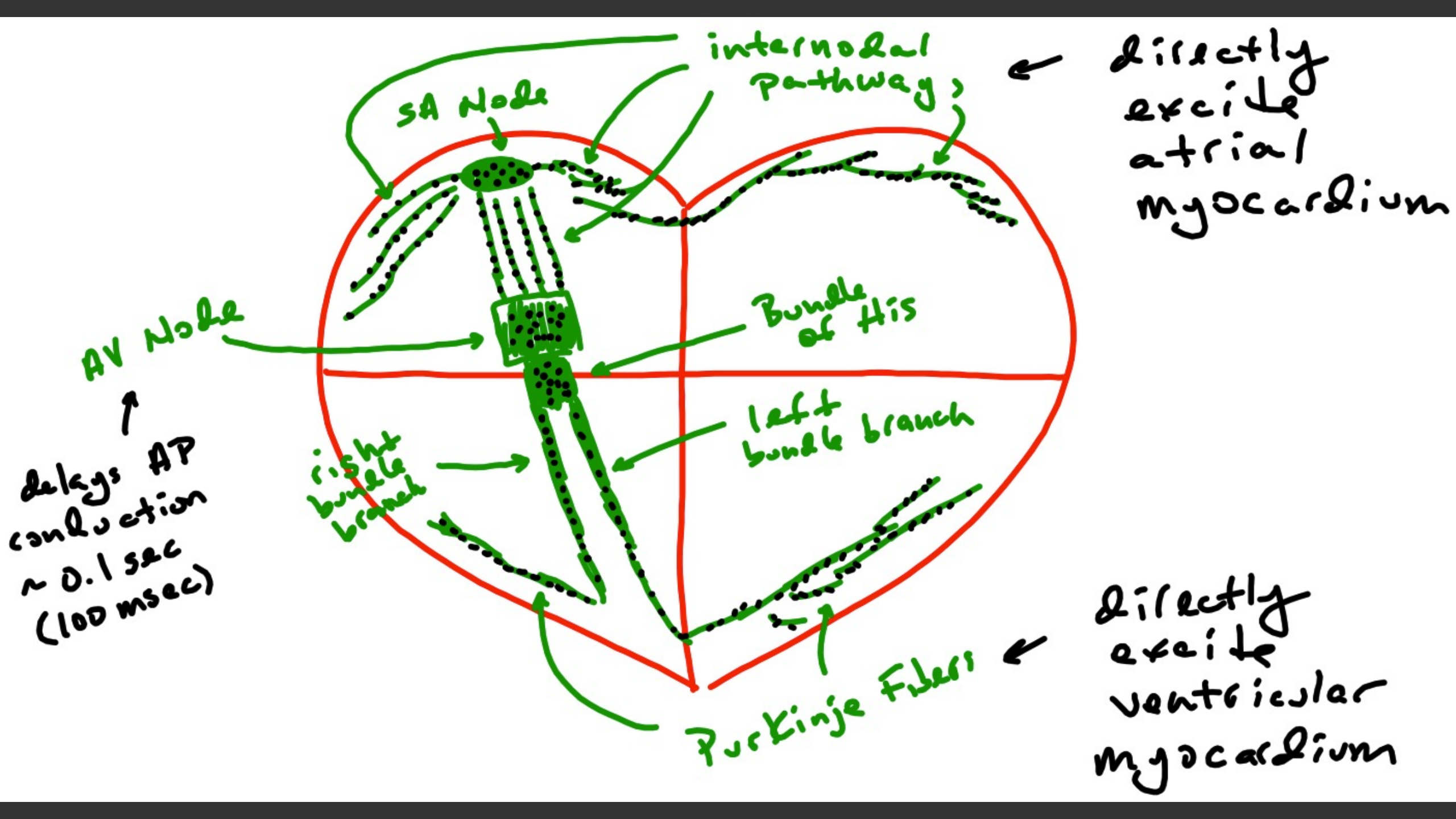
ANT 3120
Physiology – Exam 3



Cardiac Conduction System

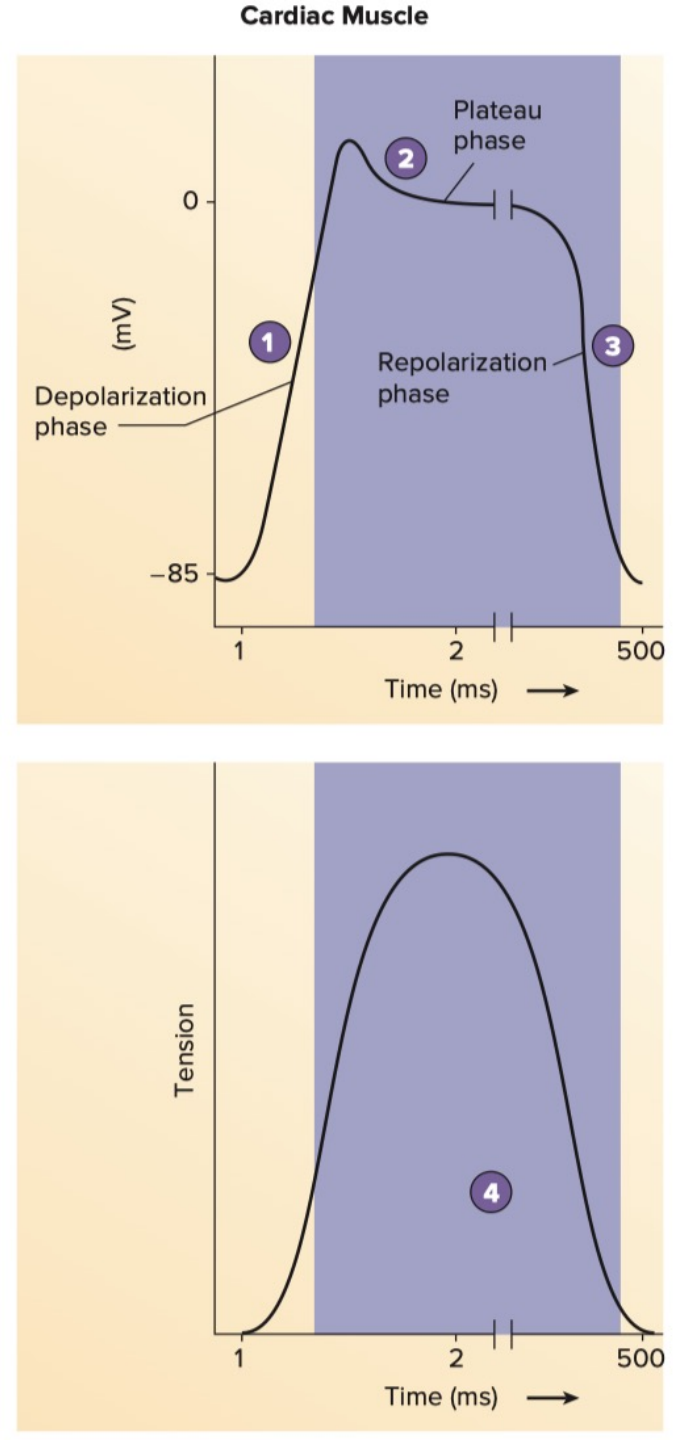
- Group of specialized , highly excitable cells that control heart rate
- Spontaneously generates and conducts action potentials
  - Sinoatrial node (SA node)
  - Atrioventricular node (AV node)
  - AV bundle / Bundle of His
  - Purkinje fibers


Sinoatrial
(SA) node
Left atrium
Atrioventricular
(AV) node
Internodal
pathways
Atrioventricular
(AV) bundle
Left Ventricle
Left and right
bundle branches
Purkinje
fibers
Apex


internodal pathways
directly excite atrial myocardium
SA Node
AV Node
delays AP conduction ~ 0.1 sec (100 msec)
Bundle of His
left bundle branch
right bundle branch
Purkinje Fibers
directly excite ventricular myocardium

Cardiac Conduction System – Action Potential

- Pacemaker potential initiated
  - Slow depolarization from resting V_m towards threshold
    - Via the opening of HCN channels and Ca^{2+} channels
      - HCN channels transport Na^{+} inward
      - Ca^{2+} channels transport Ca^{2+} inward
- Fast depolarization occurs once threshold is reached
  - Opening of voltage-gated Ca^{2+} channels
    - Ca^{2+} transported inward
- Fast repolarization
  - Opening of voltage-gated K^{+} channels causes fast repolarization
    - K^{+} transported outward
- Action potentials conduct through the cardiac conduction system
  - These action potentials then elicit action potentials in cardiac muscle



1 Depolarization phase

- Voltage-gated Na^+ channels open.
- Voltage-gated K^+ channels close.
- Voltage-gated Ca^{2+} channels begin to open.

2 Early repolarization and plateau phases

- Voltage-gated Na^+ channels close.
- Some voltage-gated K^+ channels open, causing early repolarization.
- Voltage-gated Ca^{2+} channels are open, producing the plateau by slowing further repolarization.

3 Final repolarization phase

- Voltage-gated Ca^{2+} channels close.
- Many voltage-gated K^+ channels open.

4 Refractory period effect on tension

- Cardiac muscle contracts and relaxes almost completely during the refractory period (purple shaded area).

Permeability changes in pacemaker cells

1 Pacemaker potential

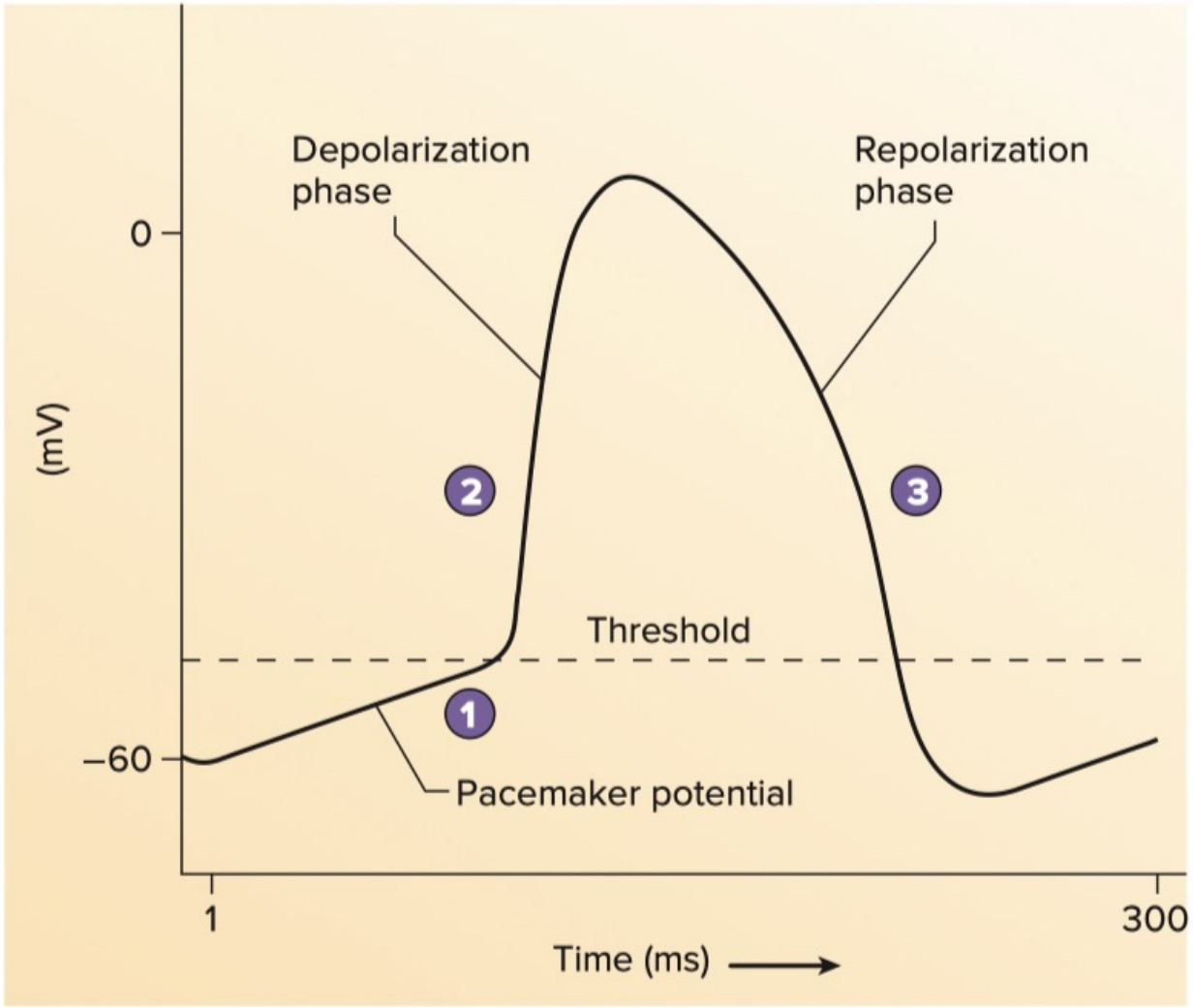
- A small number of Na^+ channels are open.
- Voltage-gated K^+ channels that opened in the repolarization phase of the previous action potential are closing.
- Voltage-gated Ca^{2+} channels begin to open.

2 Depolarization phase

- Voltage-gated Ca^{2+} channels are open.
- Voltage-gated K^+ channels are closed.

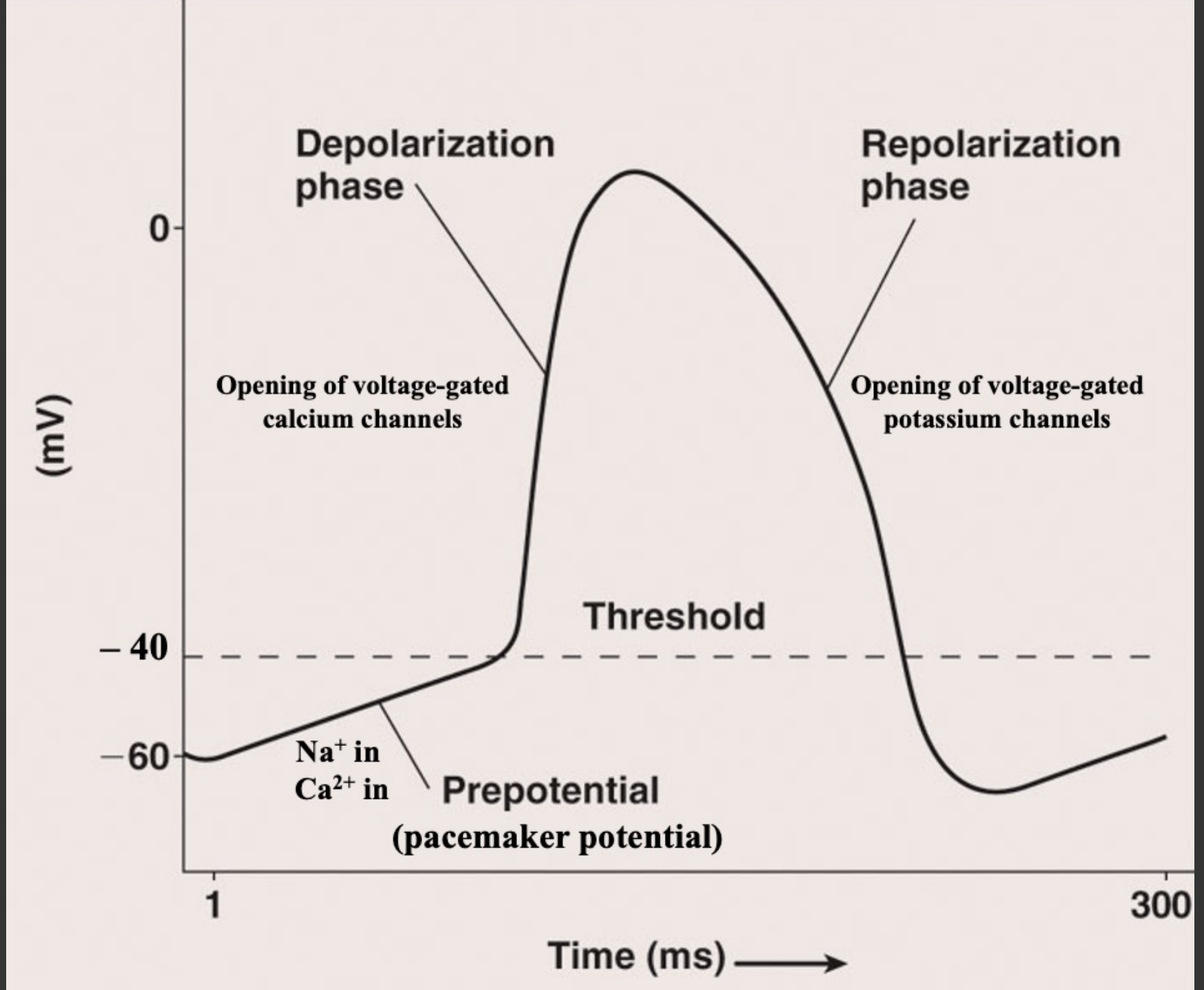
3 Repolarization phase

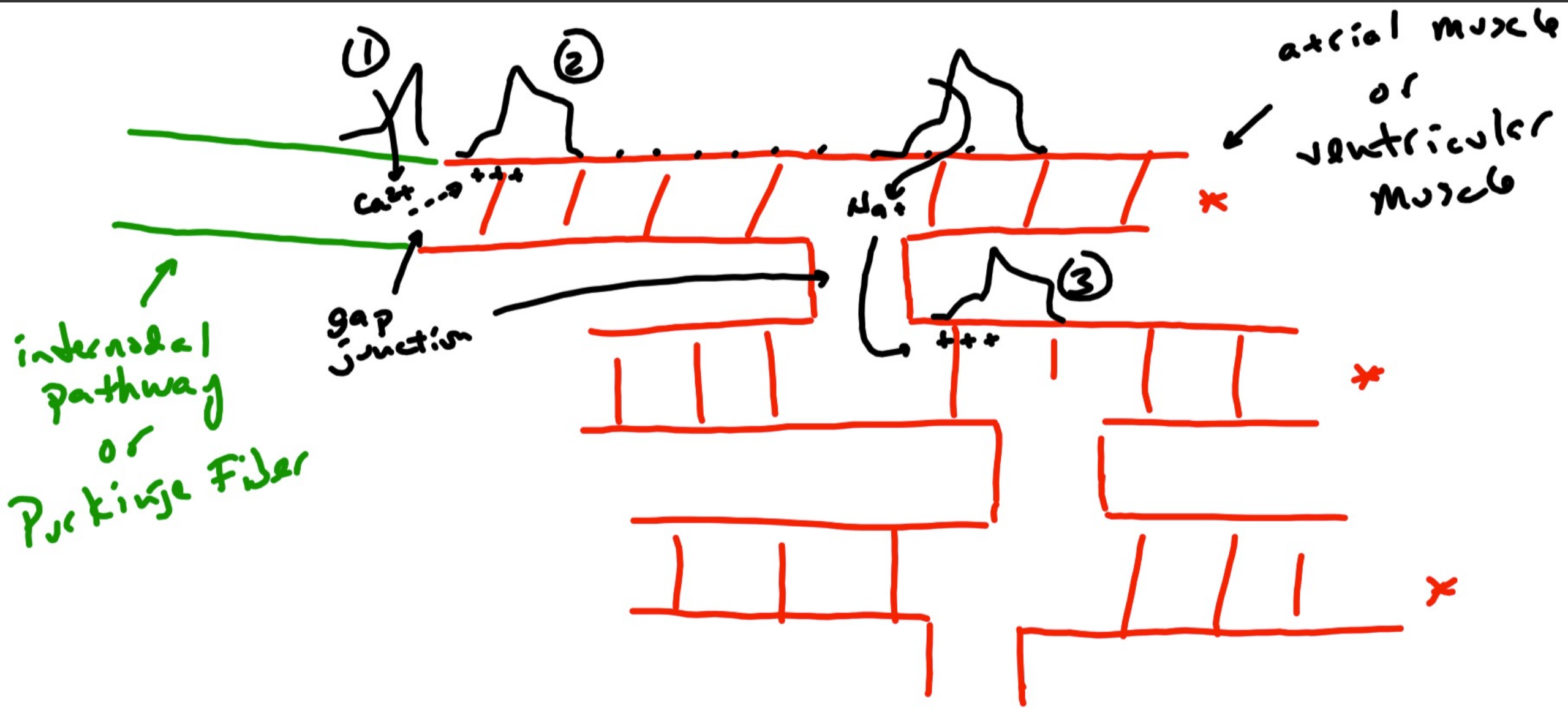
- Voltage-gated Ca^{2+} channels close.
- Voltage-gated K^+ channels open.

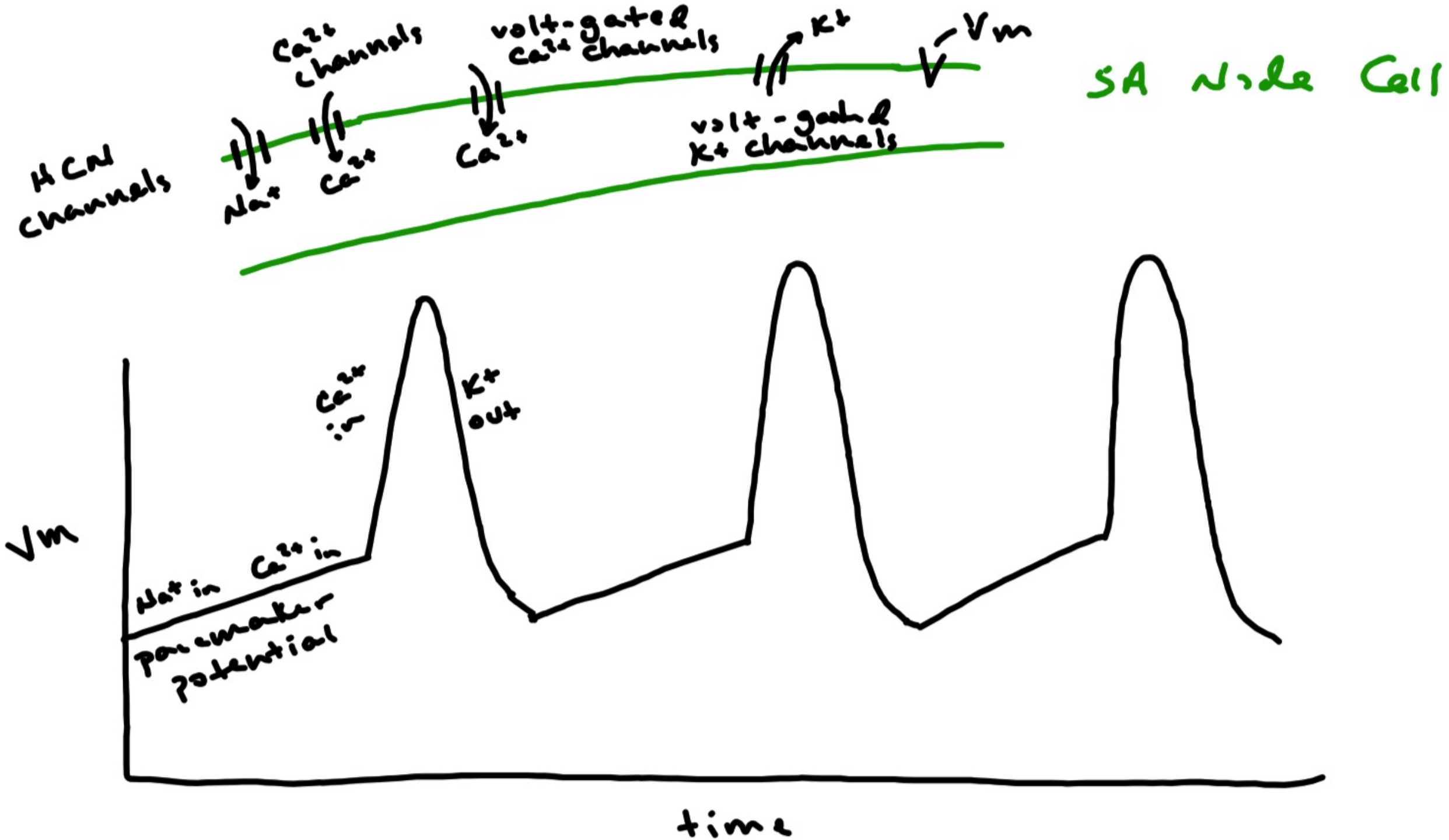


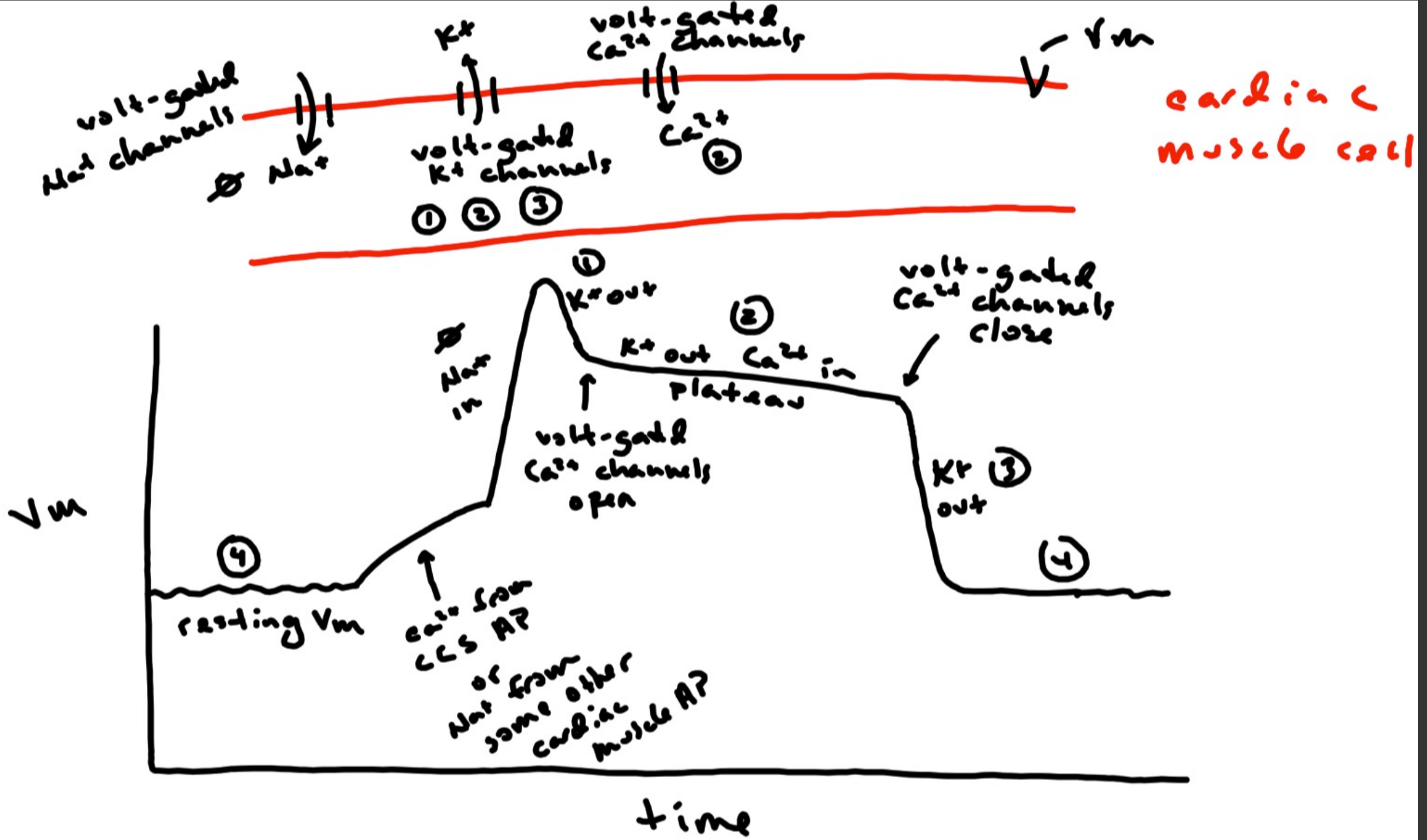
PROCESS FIGURE 20.15 Pacemaker Potential

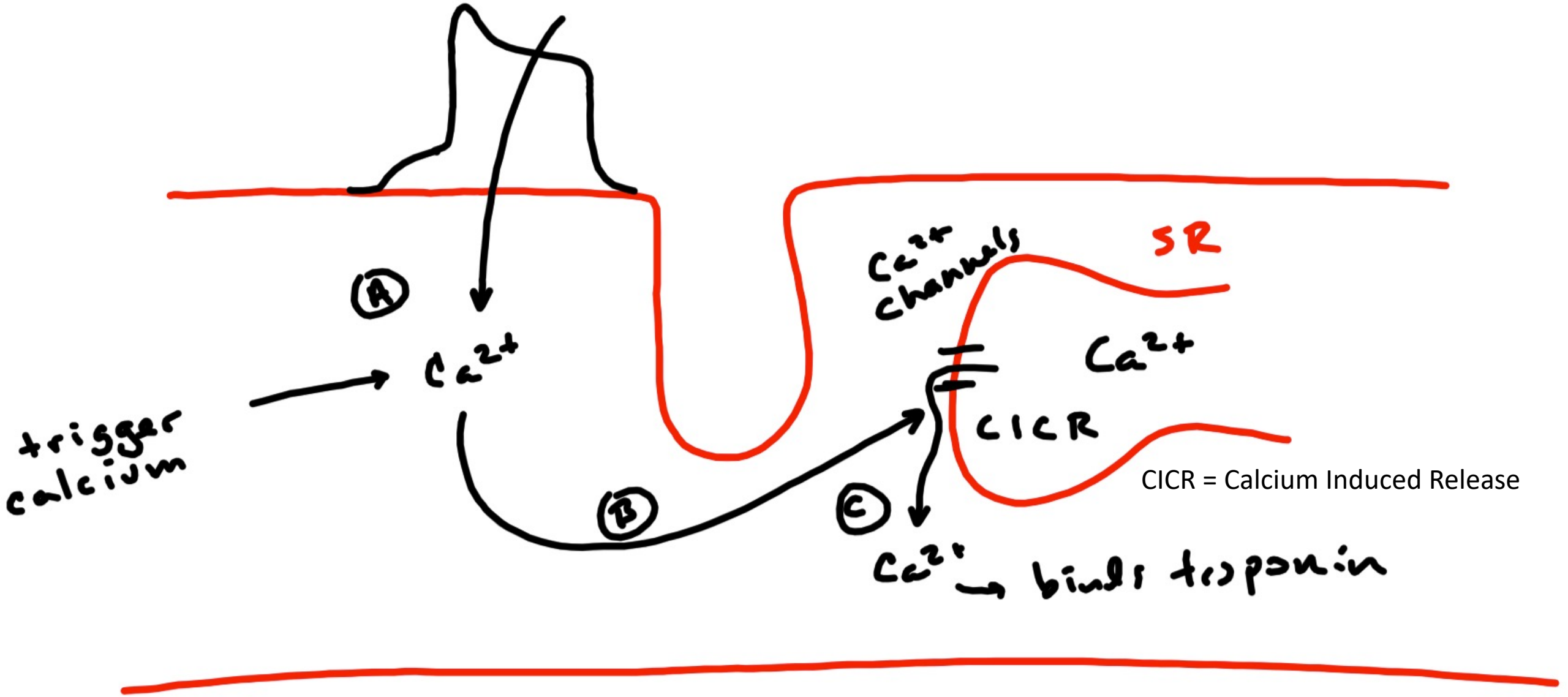
The production of action potentials by the pacemaker cells of the SA node is responsible for the autorhythmicity of the heart.


Depolarization phase
Repolarization phase
0
(mV)
Opening of voltage-gated calcium channels
Opening of voltage-gated potassium channels
Threshold
– 40
–60
Na+ in
Ca2+ in
Prepotential
(pacemaker potential)
1
300
Time (ms)


1
2
3
Ca++
+++
Na+
+++
atrial muscle
or
ventricular
muscle
gap
junction
internodal
pathway
or
Purkinje Fiber


SA Node Cell
HCN channels
Ca^{2+} channels
volt-gated Ca^{2+} channels
K^+
$-V_m$
Na^+
Ca^{2+}
Ca^{2+}
Ca^{2+}
volt-gated K^+ channels
Ca^{2+} in
K^+ out
V_m
Na^+ in
Ca^{2+} in
pacemaker potential
time


volt-gated Na+ channels
Na+
K+
volt-gated K+ channels
volt-gated Ca2+ channels
Ca2+
- Vm
cardiac muscle cell
1 2 3
Vm
time
4
resting Vm
Na+ in
1
K+ out
2
K+ out Ca2+ in
plateau
volt-gated Ca2+ channels open
volt-gated Ca2+ channels close
K+ 3
out
4
Na+ from CCS AP
or from some other cardiac muscle AP

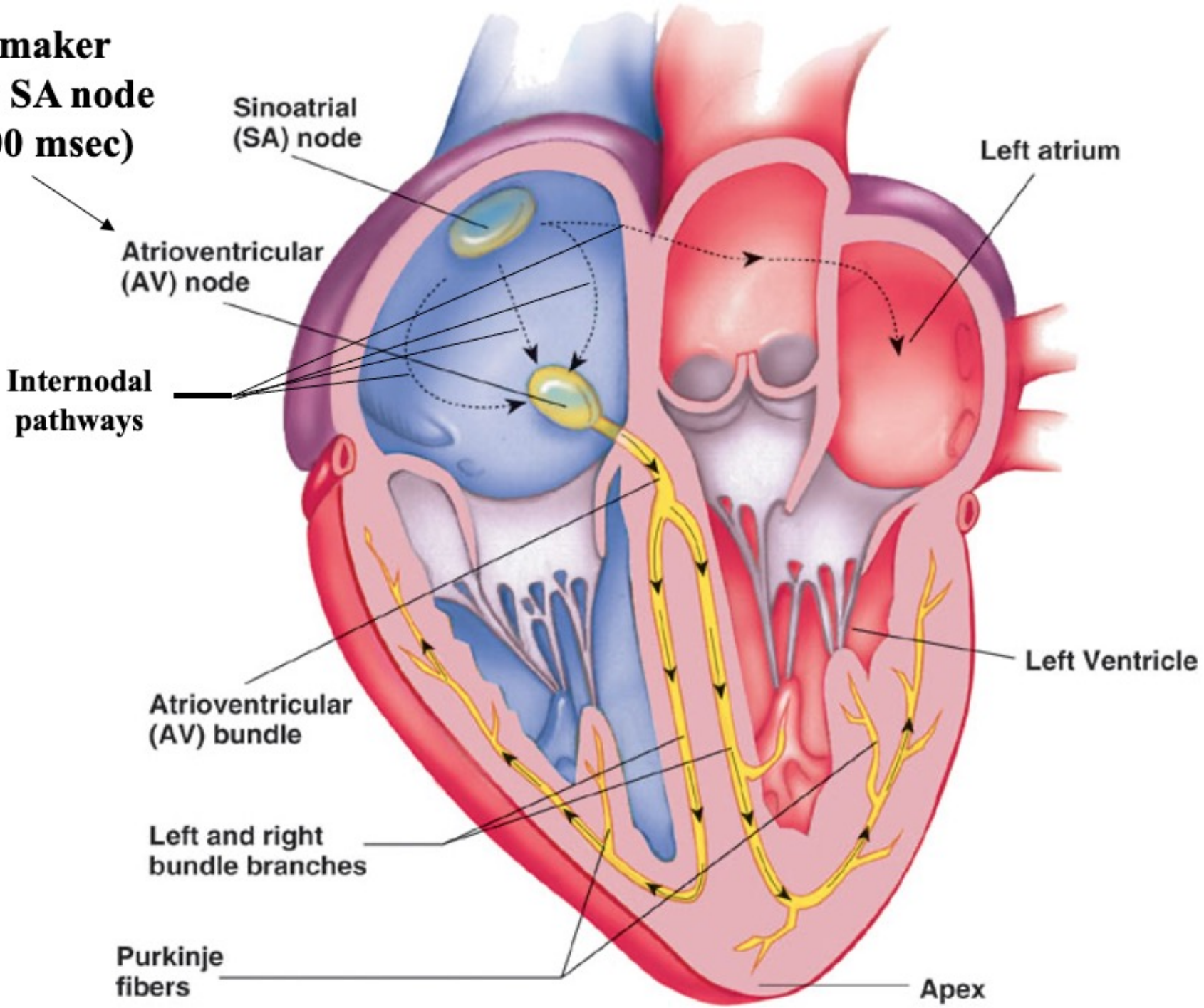


CICR = Calcium Induced Release

Cardiac Conduction System

- **Sinoatrial node (SA node)**
  - "Pacemaker" of the heart
  - Spontaneously generates action potentials at a rate of about 70 to 80 per minute
  - Conducts action potentials to the cardiac muscle of the atria via internodal pathways
  - Conducts action potentials to atrioventricular node via internodal pathways
- **Atrioventricular node (AV node)**
  - Receives action potentials from the SA node
  - Delays conduction of action potentials approximately 100 msec (i.e. 0.1 seconds)
    - Prevents action potentials from spreading to Purkinje fibers too soon
      - Ultimately allows atria to fully contract before ventricles contract
  - Conducts action potentials to AV bundle / Bundle of His after the 100 msec delay
- **AV bundle / Bundle of His**
  - Receives action potentials from the AV node
  - Conducts action potentials to the right and left bundle branches
- **Right bundle branch and Left bundle branch**
  - Receive action potentials from the Bundle of His
  - Conduct action potentials to the Purkinje fibers
- **Purkinje fibers**
  - Receive action potentials from the AV bundle / Bundle of His
  - Conduct action potentials to the cardiac muscle of the ventricles

Delays pacemaker potential from SA node (delay of ~ 100 msec)



Ectopic Pacemaker

- Ectopic Pacemaker = Any part of the heart other than the SA node that generates a rhythm
- Most common ectopic pacemaker is AV node
  - Would slow heart rate to approximately 40 to 60 beats / min
- Purkinje fibers could be an ectopic pacemaker
  - Would slow heart rate to approximately 25 to 45 beats / min
- Ectopic pacemakers can either slow down or speed up heart rate

Cardiac Muscle Fiber / Cell

- Cardiac muscle cells (myocardium) are arranged in a functional syncitium
  - Cardiac muscle makes a bulk of the atrial and ventricular walls

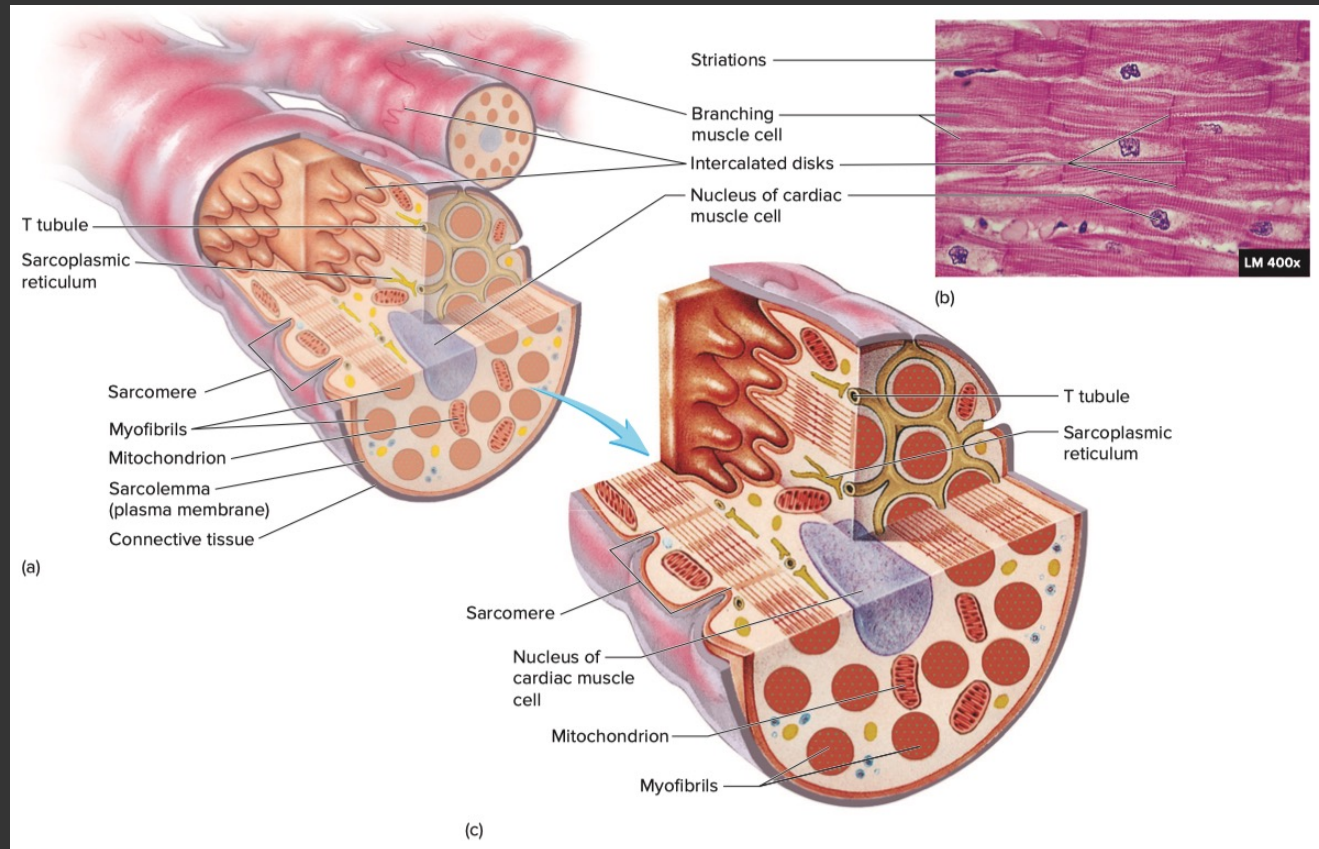
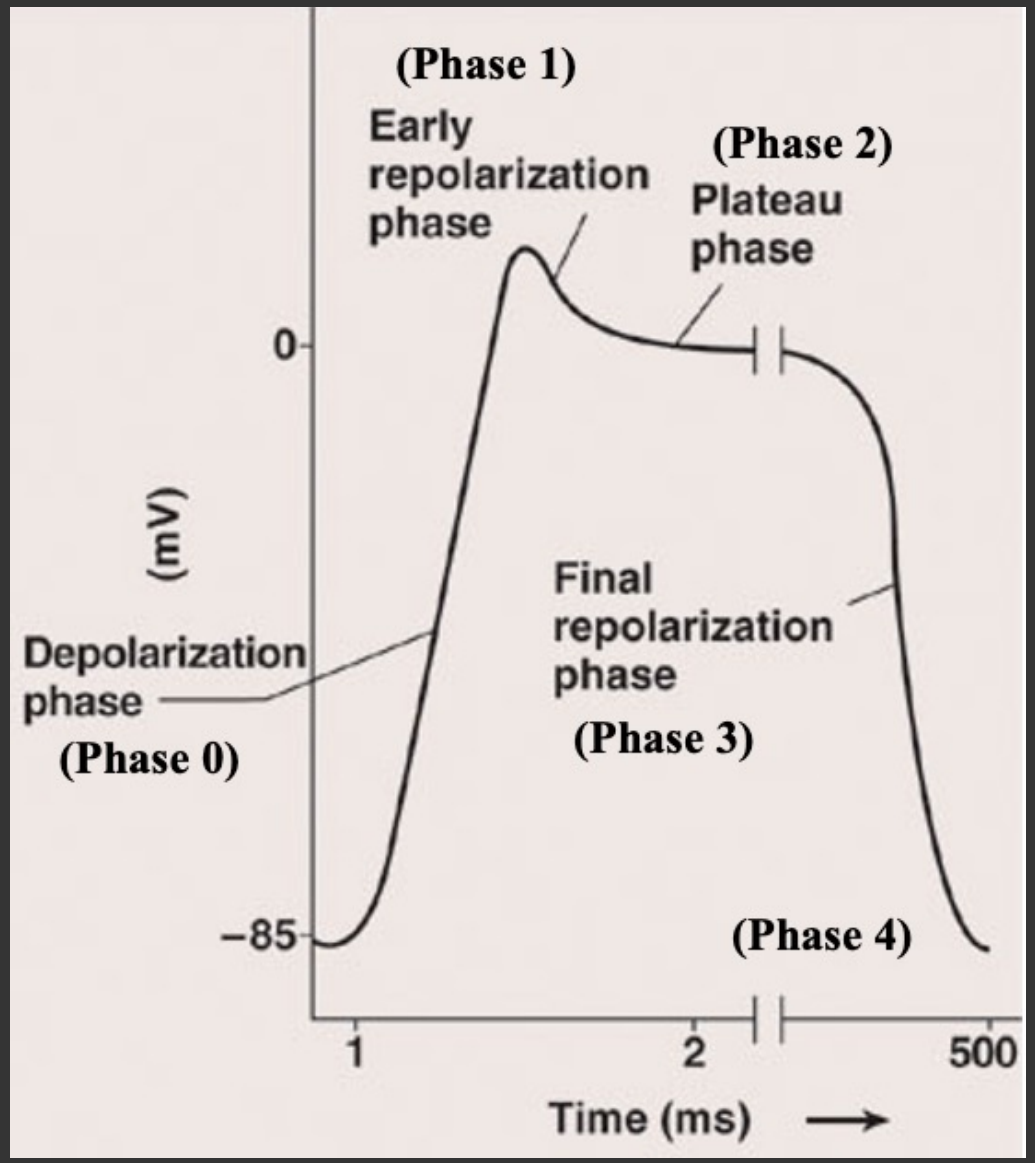


FIGURE 20.12 Histology of the Heart

(*a*) Cardiac muscle cells are branching cells with centrally located nuclei. The cells are joined to one another by intercalated disks. Gap junctions in the intercalated disks allow action potentials to pass from one cardiac muscle cell to the next. (*b*) A light micrograph of cardiac muscle tissue. The cardiac muscle cells appear striated because of the arrangement of the individual myofilaments. (c) As in skeletal muscle, sarcomeres join end-to-end to form myofibrils, and mitochondria provide ATP for contraction. Sarcoplasmic reticulum and T tubules are visible but are not as numerous as they are in skeletal muscle. (b) ©Ed Reschke AP|R

Cardiac Muscle Fiber / Cell – Action Potential

- Phase 4 (resting V_m)
  - Resting V_m established primarily via K^+ channels
- Phase 0 (fast depolarization phase)
  - Occurs if threshold is reached
    - Threshold reached via:
      - Action potential from cardiac conduction system
      - Action potential from adjacent cardiac muscle cell
  - Voltage-gated Na^+ channels open
    - Rapid transport of Na^+ into the cell
      - Causes fast depolarization
- Phase 1 (early fast repolarization)
  - Voltage-gated K^+ channels open with threshold
    - Rapid transport of K^+ out of cell
      - Causes initial , fast repolarization
- Phase 2 (plateau phase)
  - Voltage-gated Ca^{2+} channels open
    - Rapid transport of Ca^{2+} into the cell
  - Voltage-gated K^+ channels are still open at this time
  - Therefore , Ca^{2+} enters the cell while K^+ exits the cell
    - However , transport of K^+ predominates
  - Repolarization occurs slowly at this time
- Phase 3 (final fast repolarization phase)
  - Outward transport of K^+ continues while voltage-gated Ca^{2+} channels close
    - Causes fast repolarization



Cardiac Muscle Fiber / Cell – Contraction

- Action potential of cardiac muscle cell causes contraction of cardiac muscle cell
  - Excitation-Contraction Coupling
    - Sliding filament model (thin filament slides over thick filament)
  - Calcium from phase 2 (trigger calcium)
    - Causes calcium to be released from sarcoplasmic reticulum
      - Calcium-induced calcium release
      - Calcium binds to troponin
        - Initiates sliding filament model
          - Thin filament slides over thick filament
            - Causes shortening of sarcomere
- Contraction stops when calcium is pumped back into the sarcoplasmic reticulum
- Contraction of the atrial and ventricular myocardium
  - Allows ventricles to pump blood
    - Right ventricle pumps blood into the pulmonary trunk
    - Left ventricle pumps blood into the aorta

Electrocardiogram (ECG) / Elektrokardiogram (EKG)

- Recording of the electrical activity (i.e. action potentials) of cardiac muscle
  - Electrical activity can be recorded on the surface of the body
    - Amplitude of electrical activity is large , which is why it can be measured
      - Amplitude is large because the heart is a functional syncitium
- NOT a recording of the action potentials of the cardiac conduction system
- NOT a recording of mechanical activity
- Recorded with an **electrocardiograph**

Electrocardiogram (ECG) / Elektrokardiogram (EKG)

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- NOT a recording of the action potentials of the cardiac conduction system
- NOT a recording of mechanical activity
- Recorded with an **electrocardiograph**

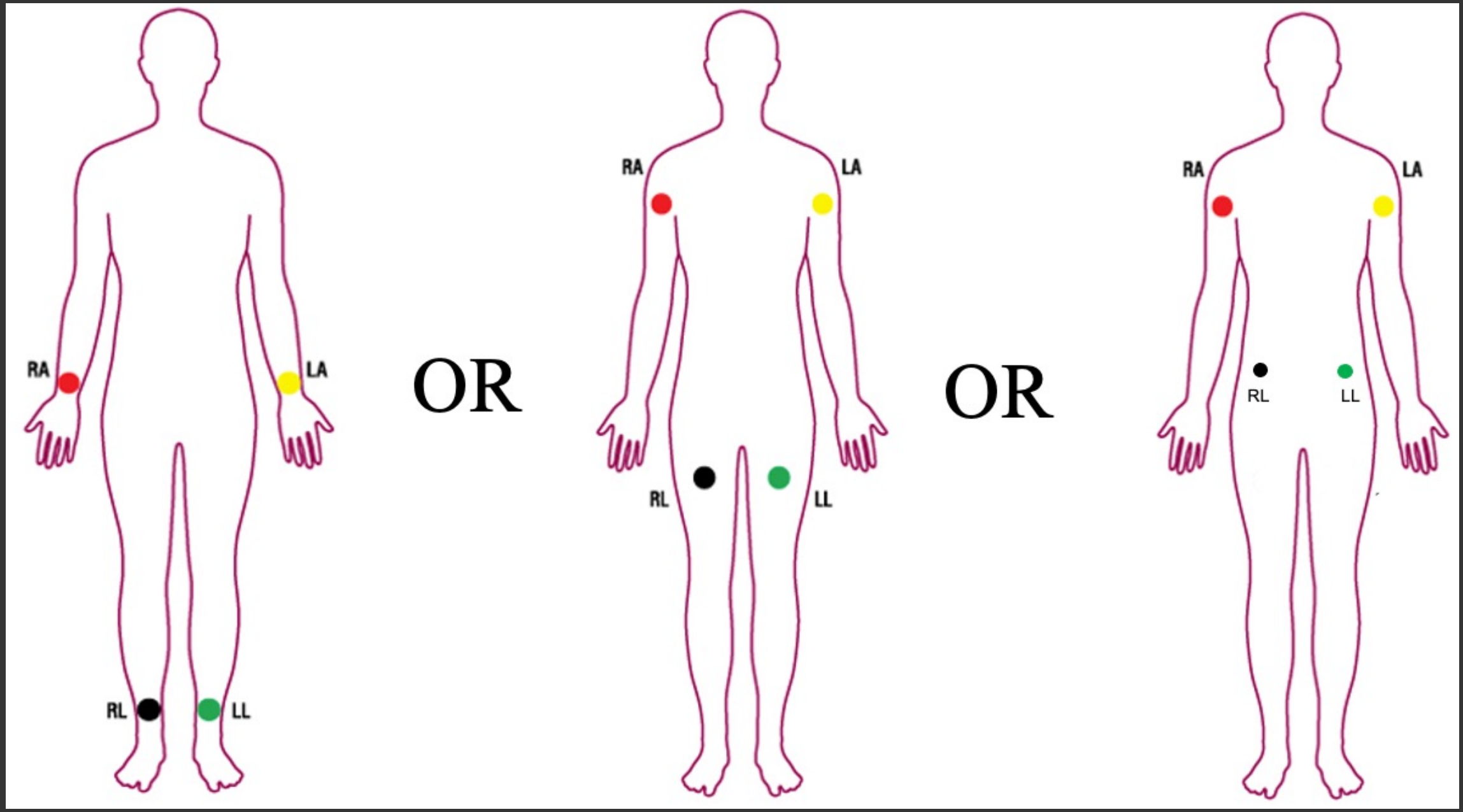
ECG – Leads

- **Standard bipolar limb leads**
  - Measure the electrical activity of the heart in a frontal plane
  - Lead I: right arm (–) to left arm (+)
    - Measures electrical activity across the heart at a 0° angle
  - Lead II: right arm (–) to left leg (+)
    - Measures electrical activity across the heart at a +60° angle
  - Lead III: left arm (–) to left leg (+)
    - Measures electrical activity across the heart at a +120° angle

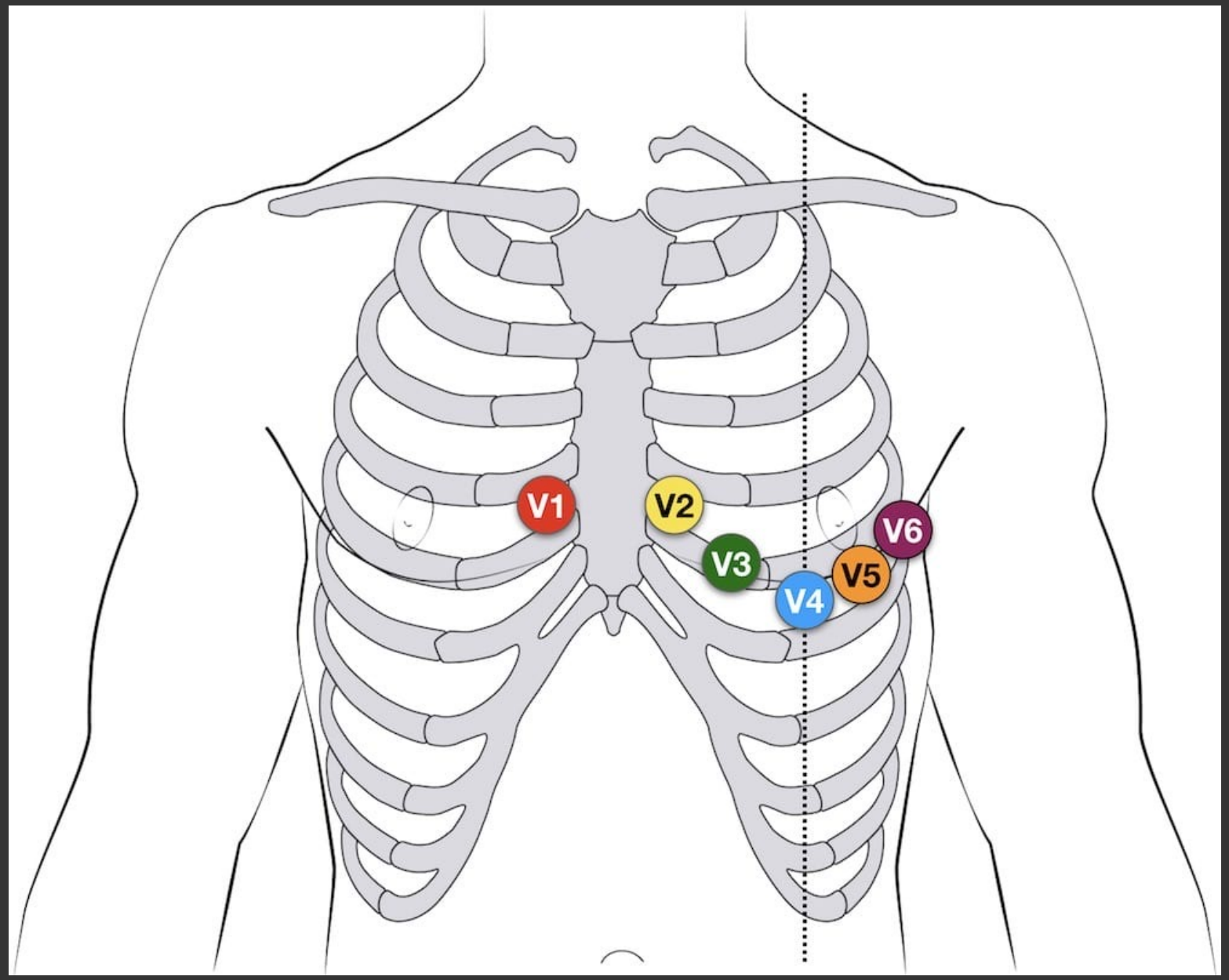
- **Augmented unipolar limb leads**
  - Measure the electrical activity of the heart in a frontal plane
  - aVR: right arm (+) to central terminal ground lead (joining of left arm and left leg)
    - Measures electrical activity across the heart at a –150° angle
  - aVL: left arm (+) to central terminal ground lead (joining of left leg and right arm)
    - Measures electrical activity across the heart at a –30° angle
  - aVF: left leg (+) to central terminal ground lead (joining of right arm and left arm)
    - Measures electrical activity across the heart at a +90° angle

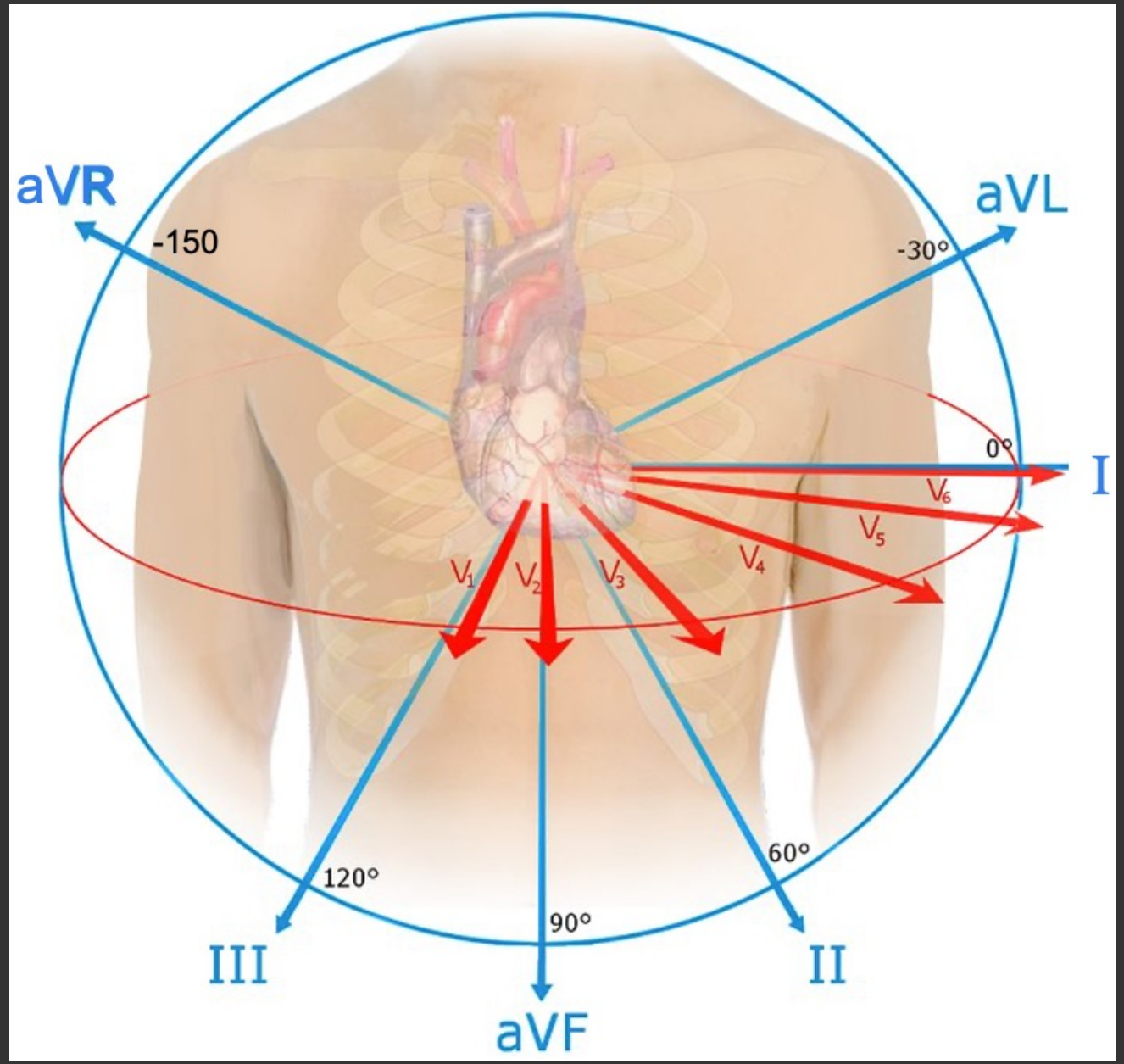
- **Chest leads / Precordial leads**
  - Measure the electrical activity of the heart in a transverse plane
  - V1 , V2 , V3 , V4 , V5 , V6 leads arranged across the chest

ECG – Limb Lead Electrode Placement

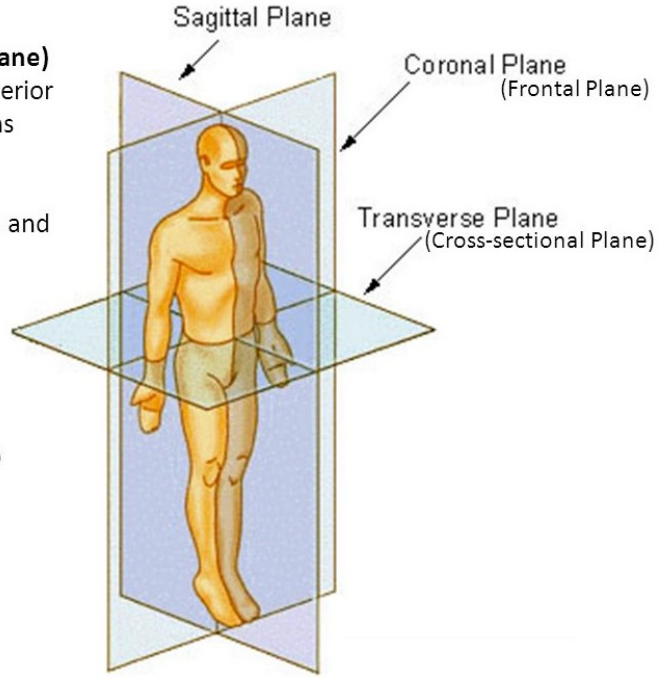


ECG – Chest / Precordial Lead Electrode Placement





- **Coronal Plane (Frontal Plane)**
  - Divides body into anterior and posterior portions
- **Sagittal Plane**
  - Divides body into left and right portions
  - Midsagittal Plane
  - Parasagittal Plane
- **Transverse Plane (Cross-sectional Plane)**
  - Divides the body into superior and inferior portions



Limb Leads: measure the electrical activity of the heart in a frontal plane

Chest Leads: measure the electrical activity of the heart in a transverse plane

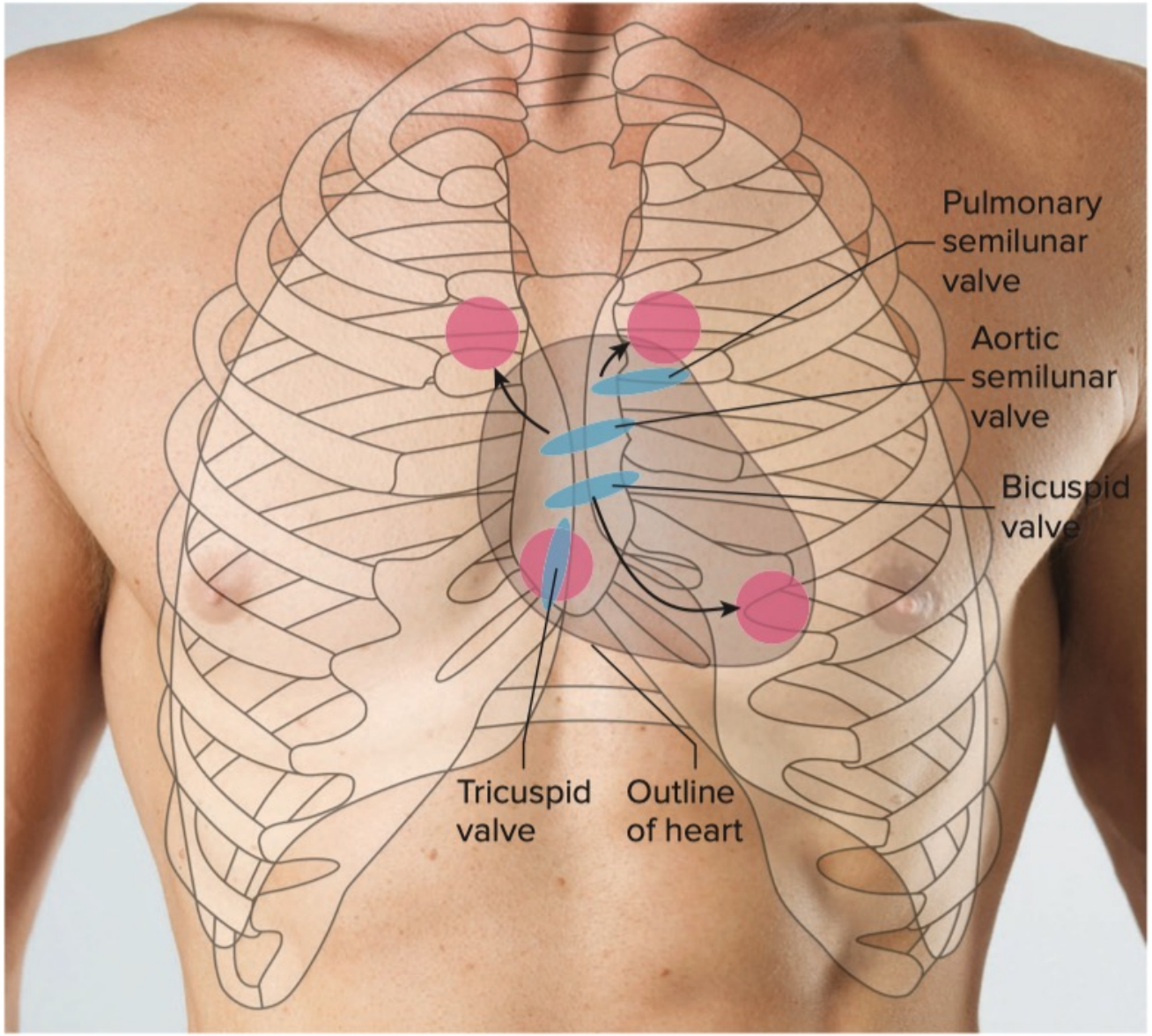


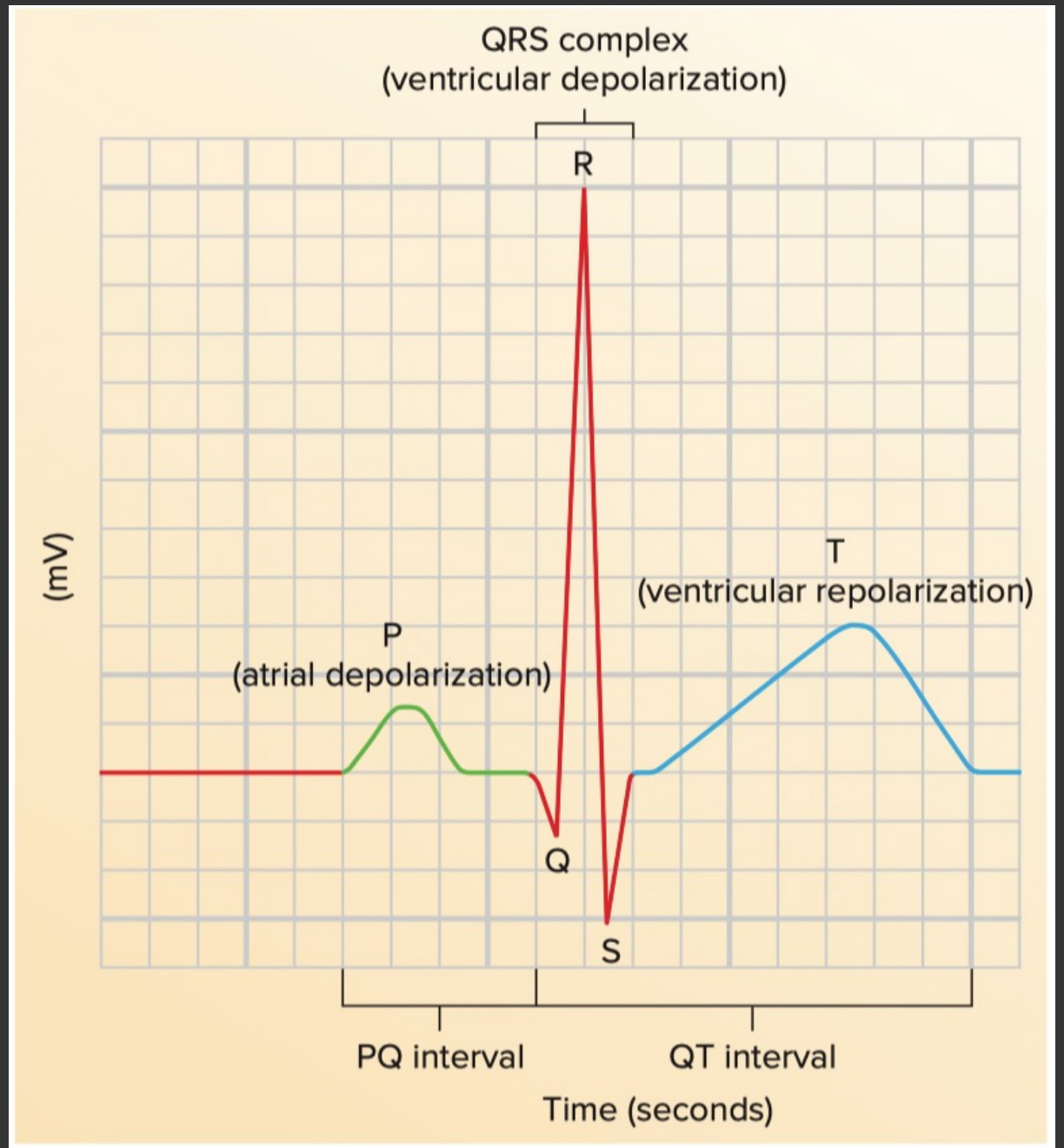
FIGURE 20.20 Location of the Heart Valves in the Thorax

Surface markings of the heart in the male. The positions of the four heart valves are indicated by *blue ellipses*, and the sites where the sounds of the valves are best heard with the stethoscope are indicated by *pink circles*.

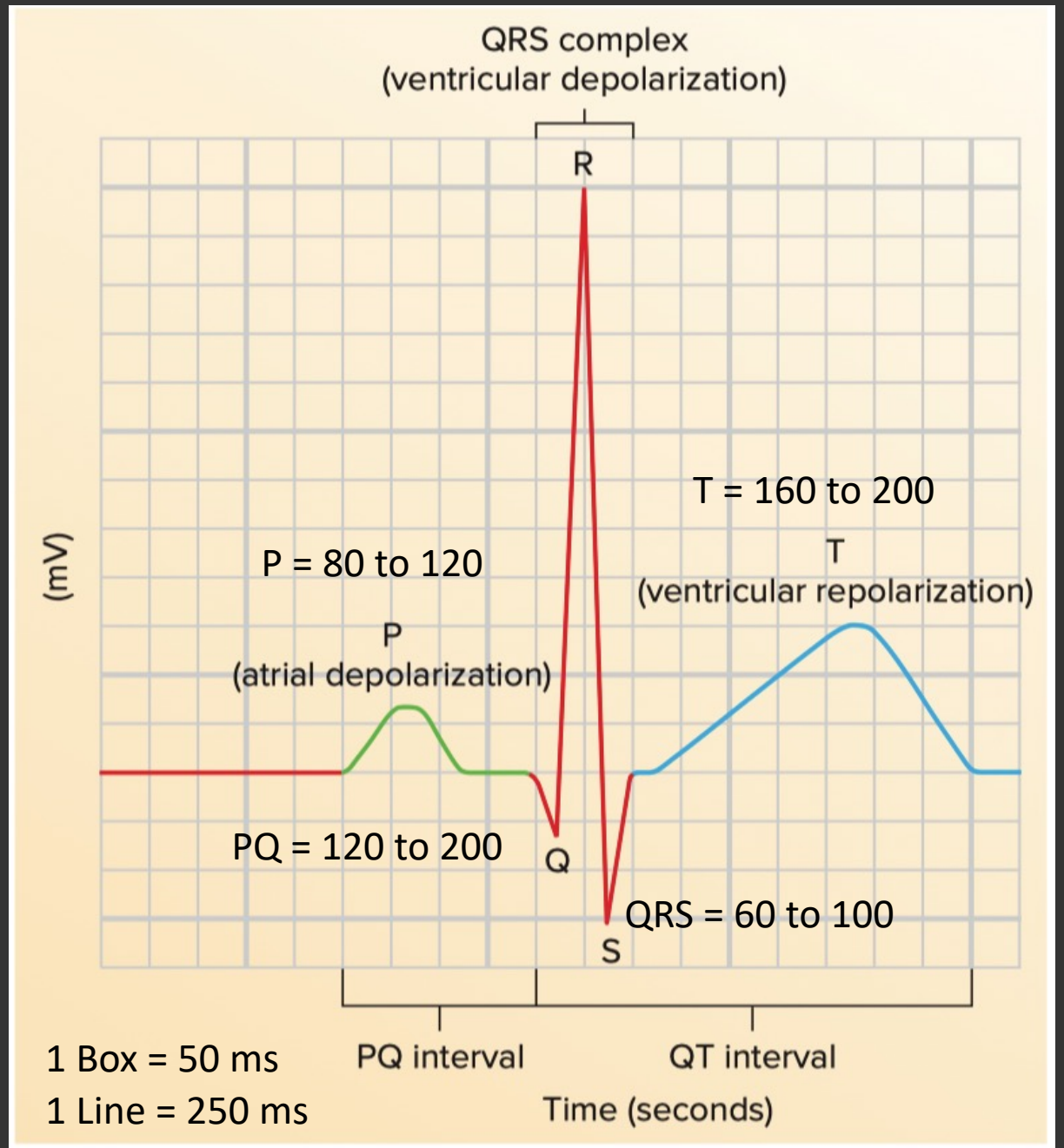
ECG – Trace

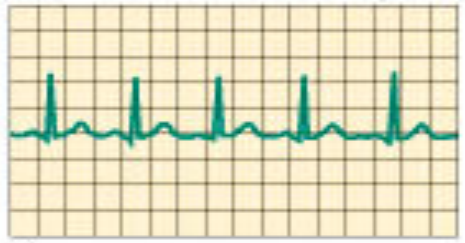
- P wave
  - Measure of the depolarization of atrial muscle
  - Normally 80 to 120 msec in duration
- QRS complex
  - Measure of the depolarization of ventricular muscle
  - Normally 60 to 100 msec in duration
- T wave
  - Measure of the repolarization of ventricular muscle
  - Normally 160 to 200 msec in duration
- PQ interval / PR interval
  - From the beginning of the P wave to the beginning of the Q wave
  - Time from onset of atrial depolarization to onset of ventricular depolarization
    - Time it takes action potentials to conduct through cardiac conduction system
  - Normally 120 to 200 msec in duration

ECG – Trace

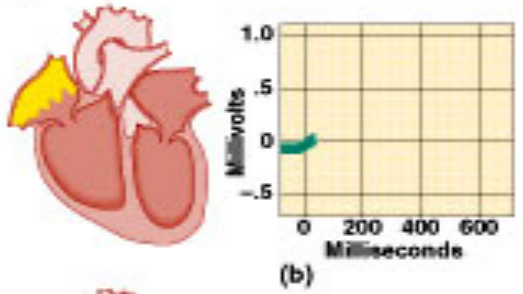

QRS complex
(ventricular depolarization)
R
(mV)
T
(ventricular repolarization)
P
(atrial depolarization)
Q
S
PQ interval
QT interval
Time (seconds)

ECG – Trace


QRS complex
(ventricular depolarization)
R
T = 160 to 200
T
(ventricular repolarization)
P = 80 to 120
P
(atrial depolarization)
(mV)
PQ = 120 to 200
Q
QRS = 60 to 100
S
1 Box = 50 ms
1 Line = 250 ms
PQ interval
QT interval
Time (seconds)



(a)



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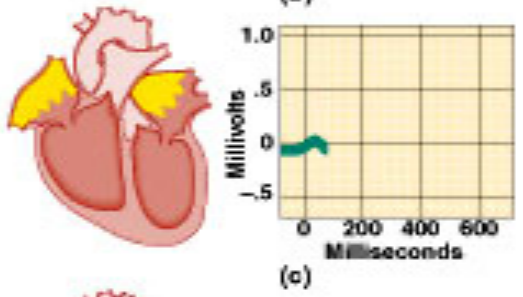

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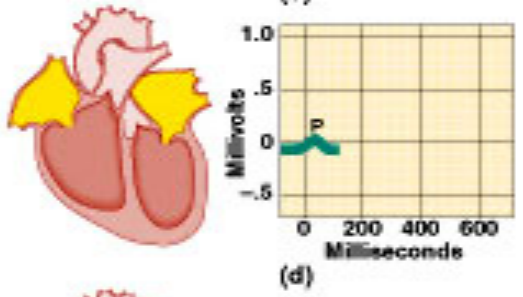

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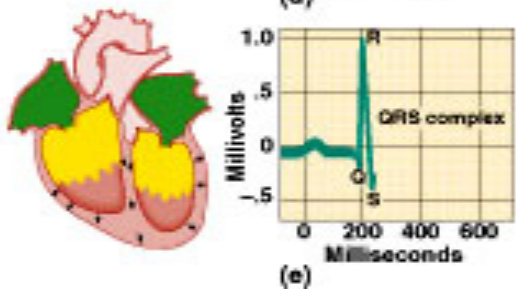

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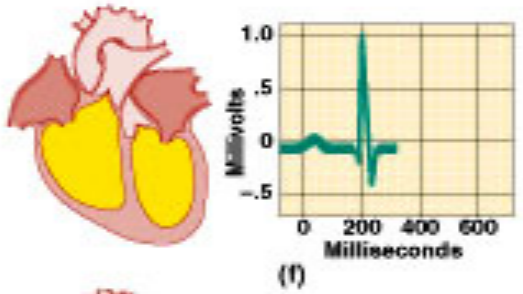

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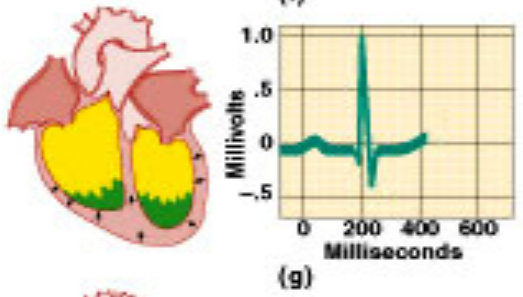
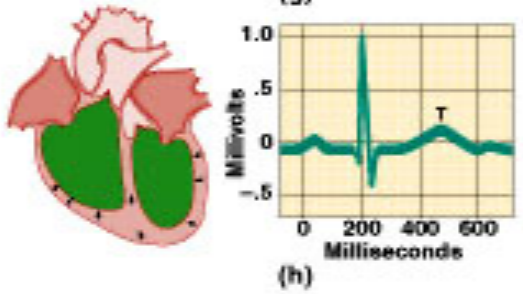
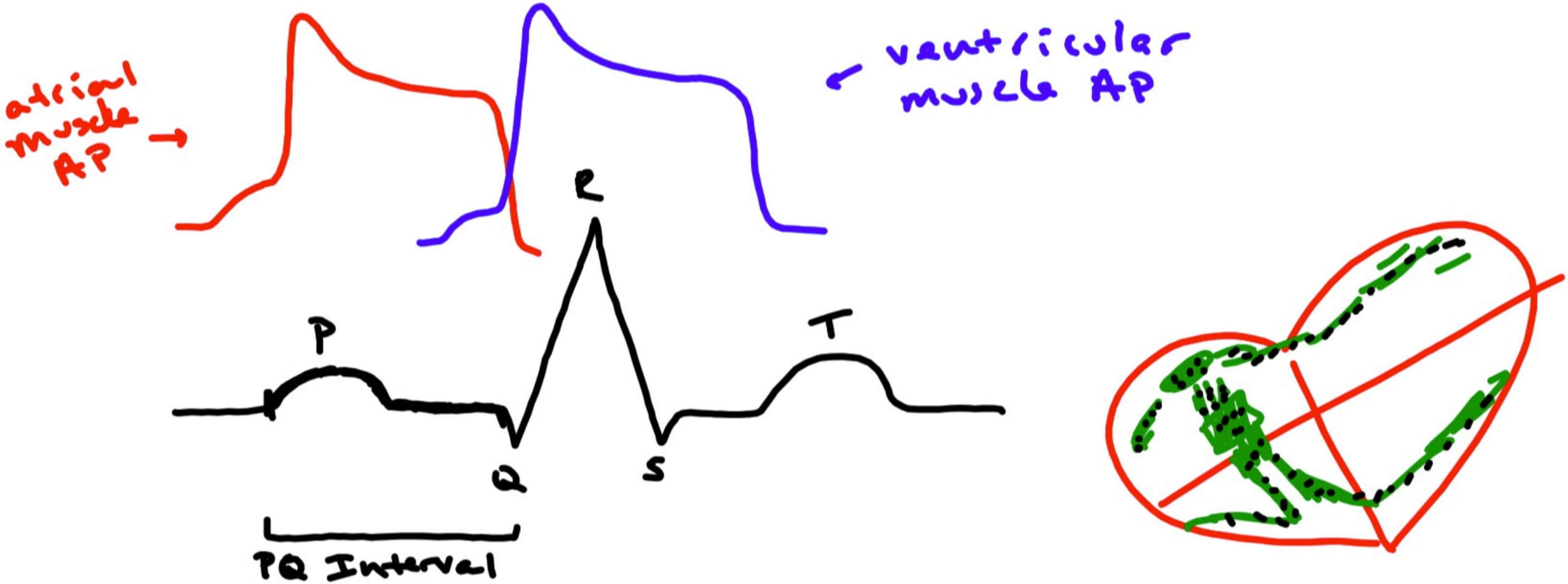


(g)



(h)



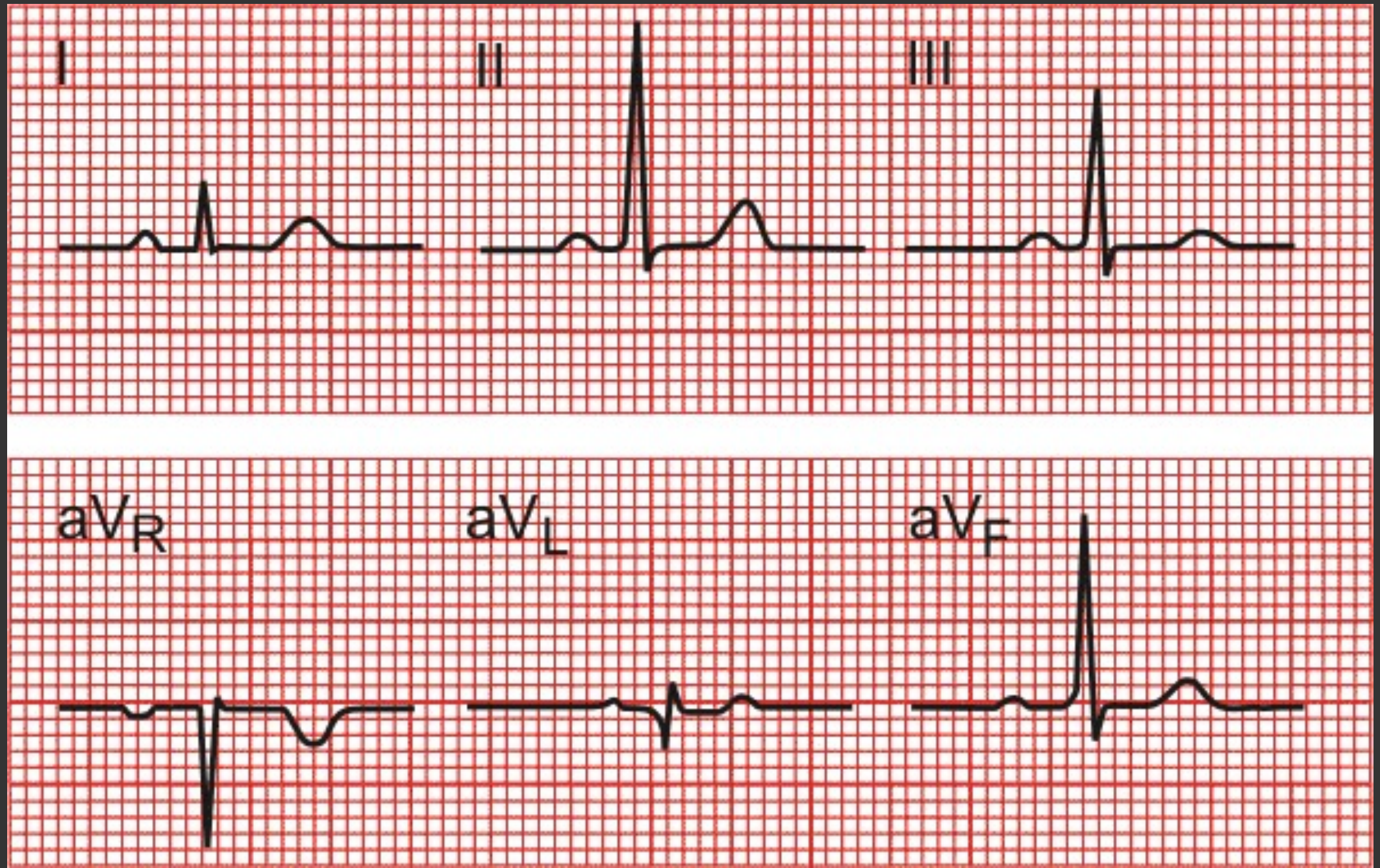
P wave: dep of atria

QRS : dep of ventricles

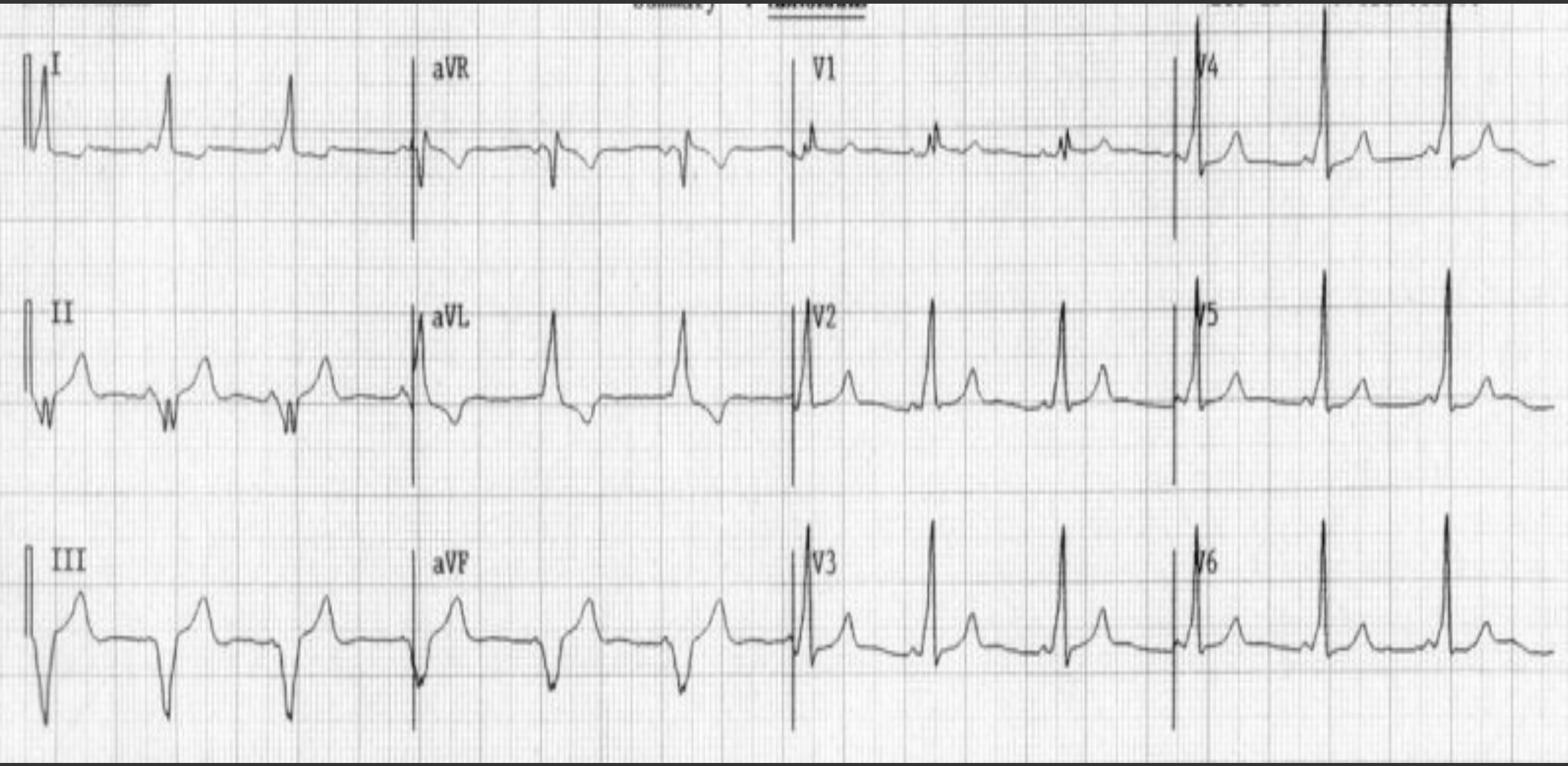
T wave : rep of ventricles

PQ interval: time it takes AP's to conduct through the CCS

ECG – Trace of Limb Leads

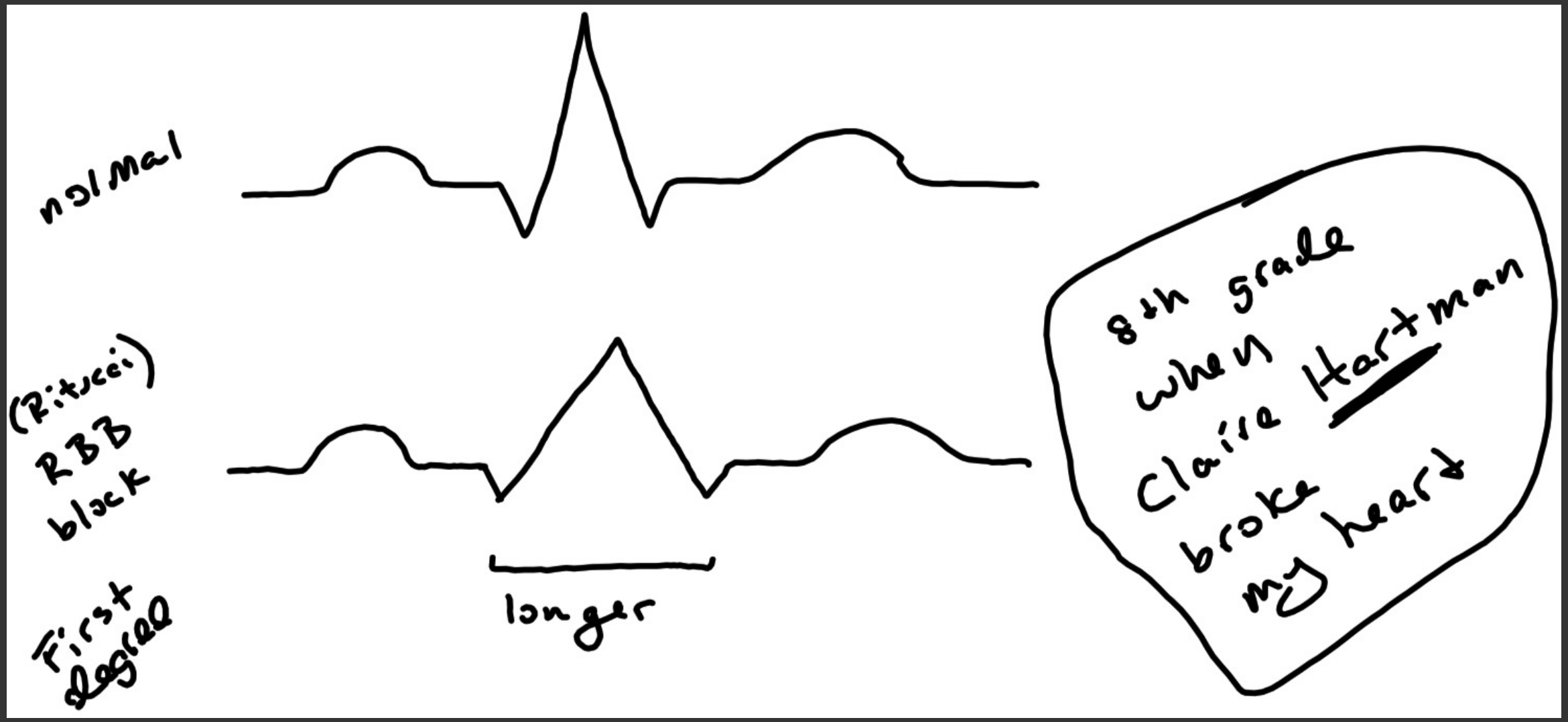


ECG – 12 Lead Trace



Arrhythmia / Dysrhythmia

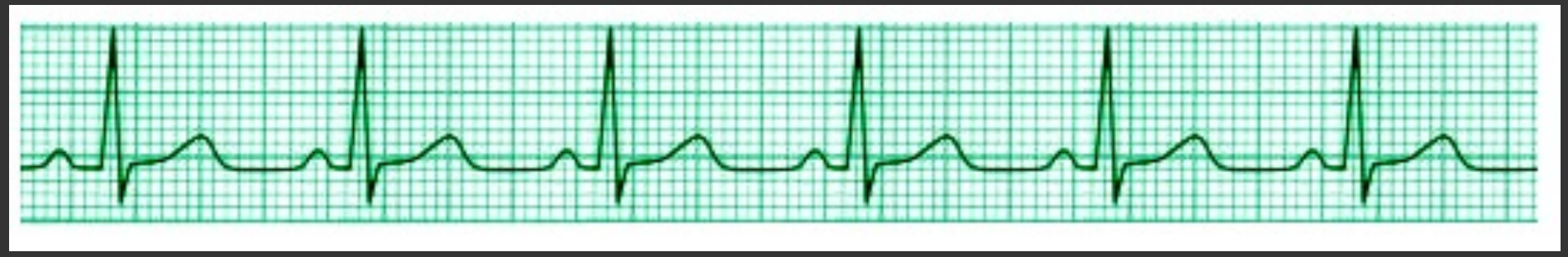
- Condition where the electrical activity is irregular



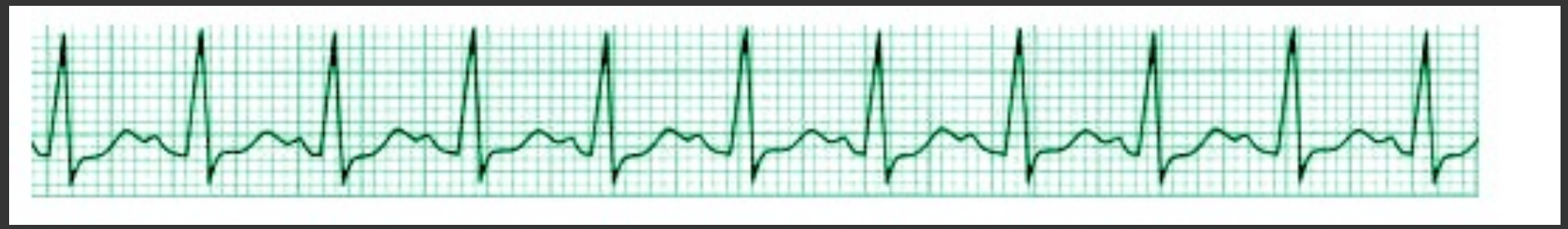
Tachycardia

- Tachycardia – heart rate above 100 beats per minute
  - e.g . high sympathetic tone; hyperthyroidism , ectopic pacemaker

normal

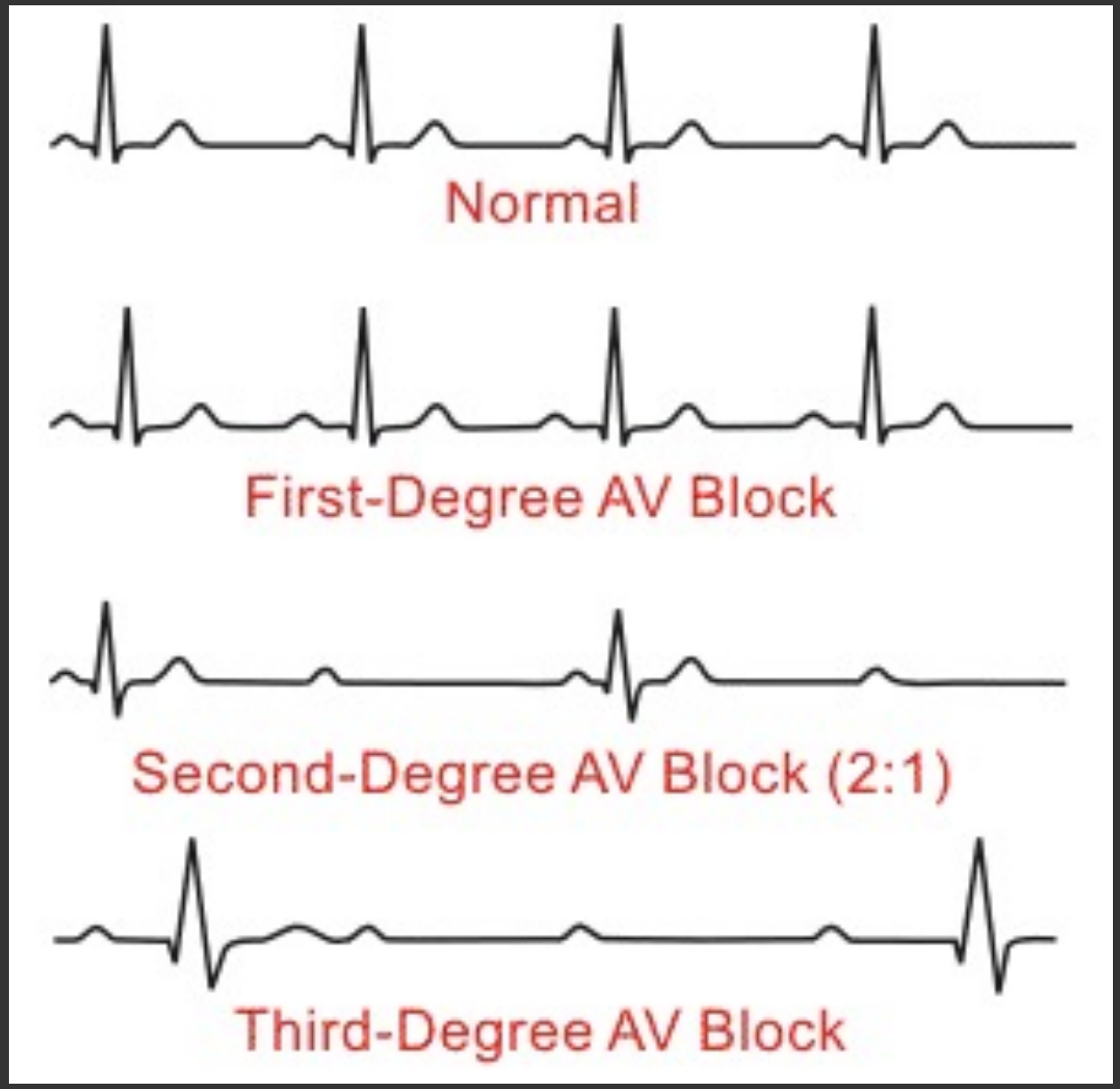


tachycardia



Heart Block

- ***Normal:***
  - *Conduction of action potentials is slowed*
  - *Not serious . . . needs no medical attention*
- ***First-Degree:*** *Type I / Wenckebach*
  - Conduction of action potentials is slowed more
  - Not too serious but dizziness can occur
- ***Second-Degree:*** *Type II / Mobitz*
  - Conduction of action potentials is slowed more and blocked
  - Most will require an **artificial pacemaker**
- **Third-Degree:**
  - Complete block of action potential conduction
  - No relationship between P wave and QRS complex
  - Most often due to heart disease or congenital
  - Artificial pacemaker will be required



Normal

2nd degree Type I

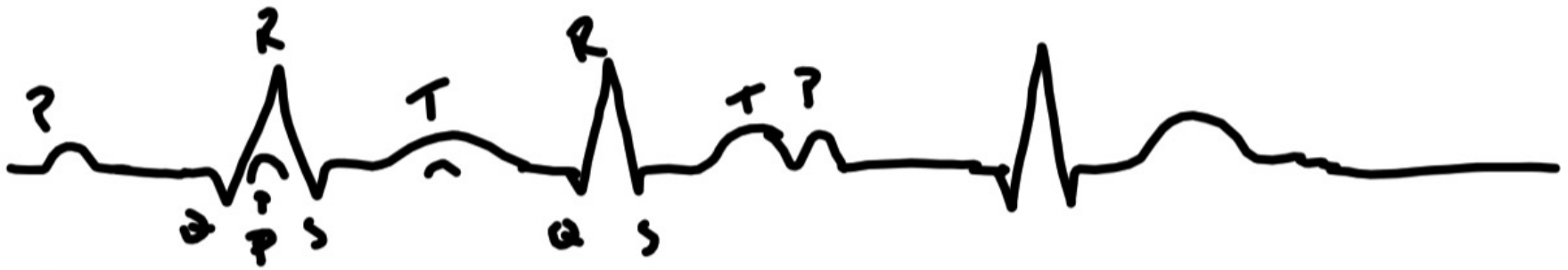
missed QRS

2nd degree Type II

missed

probably going to need an artificial pacemaker

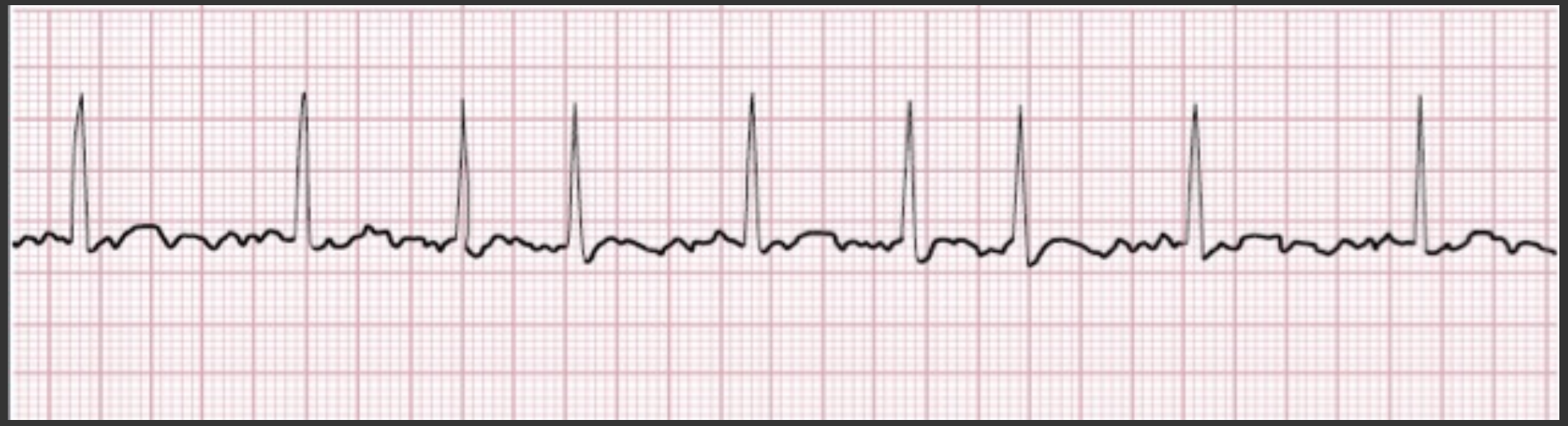
3rd degree



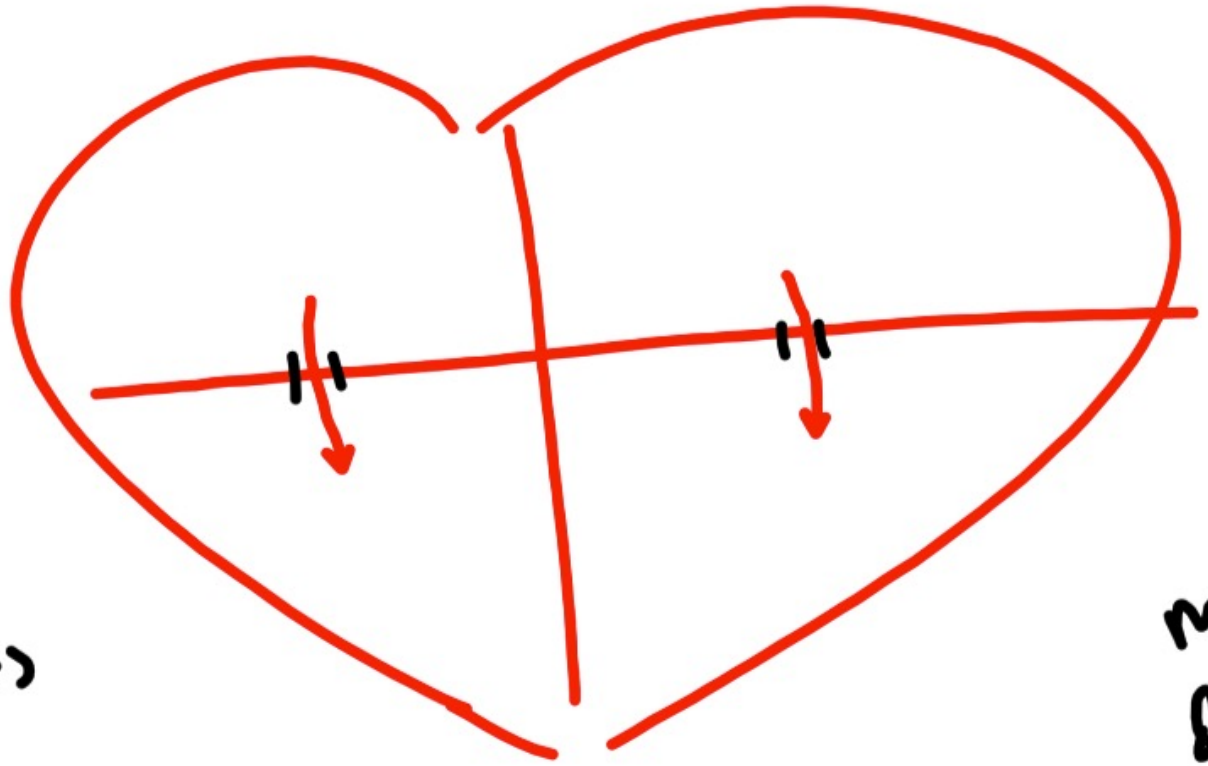
will require a pacemaker

Atrial Fibrillation

- *Block of action potential in atria; AV node activated sporadically*
- *No observable P waves and no constant QRS spacing*
- *Atria do not fully contract*
  - *Certain percentage of blood fails to be delivered to the ventricles*
- *Often presents with no symptoms*
  - *Reason: sufficient amount of blood is delivered to the ventricles*
  - *However , increases risk of blood clots due to stasis in atria*



A F:b



* might not present with symptoms, so why?

most blood is delivered from atria to ventricles w/o atria contracting

can lead to blood clots in atria

excess blood sits in atria (stasis)

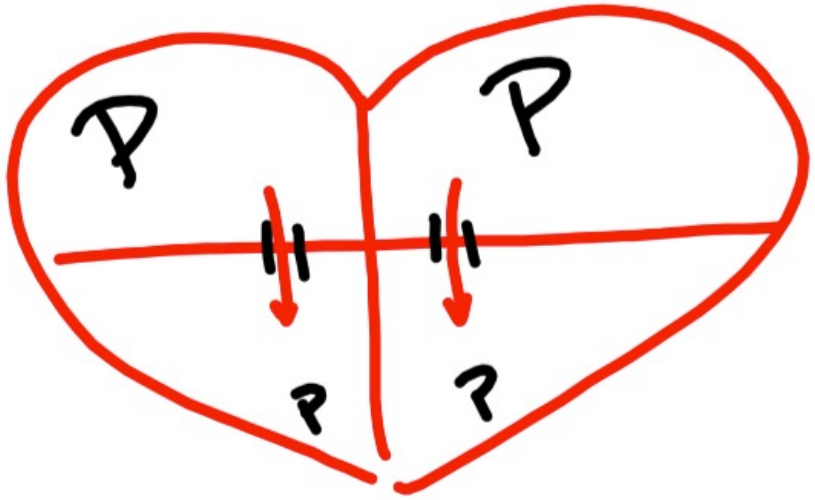
Opening and Closing of Heart Valves

- *Valves function to allow the flow of blood through the heart in only one direction*
  - *Blood from atria to ventricles*
  - *Blood from ventricles to great arteries*

- *Opening and closing of valves is passive*
  - *Dictated by pressure changes in the chambers of the heart and great arteries*
  - *Atrioventricular (AV) valves (tricuspid valve and mitral valve)*
    - *Open when atrial pressure exceeds ventricular pressure*
    - *Close when ventricular pressure exceeds atrial pressure*
  - *Semilunar valves (pulmonary valve and aortic valve)*
    - *Open when ventricular pressure exceeds pulmonary trunk and aortic pressure*
    - *Close when pulmonary trunk and aortic pressures exceed ventricular pressure*

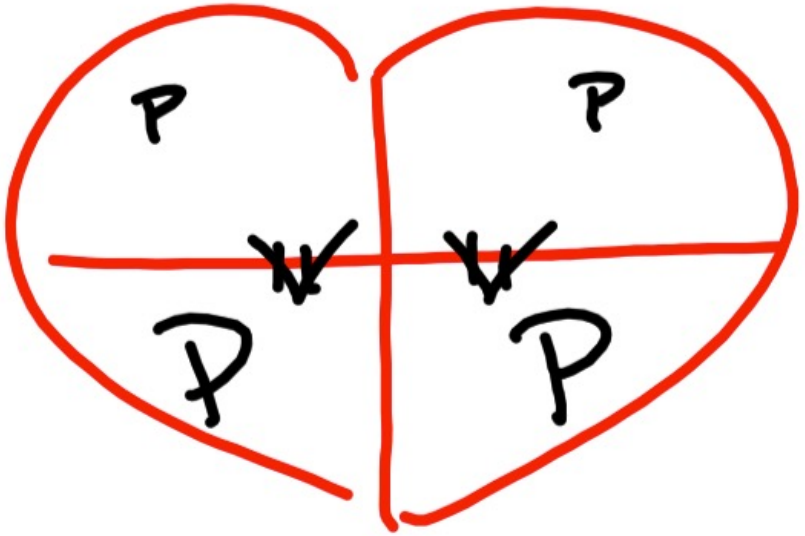
- *Closing of valves and the collision of blood against them produce heart sounds*
  - *First heart sound ("lub") – S_1*
    - *Closing of AV valves*
  - *Second heart sound ("dub") – S_2*
    - *Closing of pulmonary and aortic semilunar valves*

Opening of AV Valves



atrial P > vent P

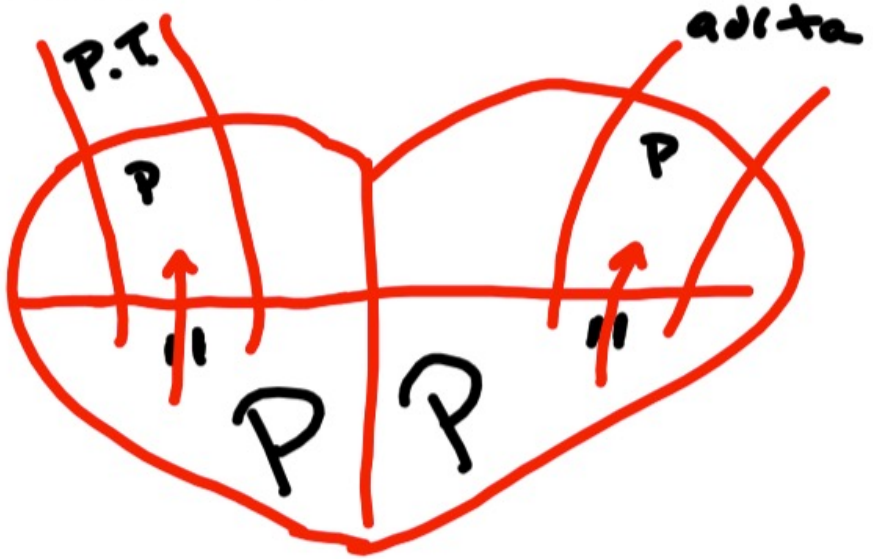
Closing of AV values



vent P > atria P

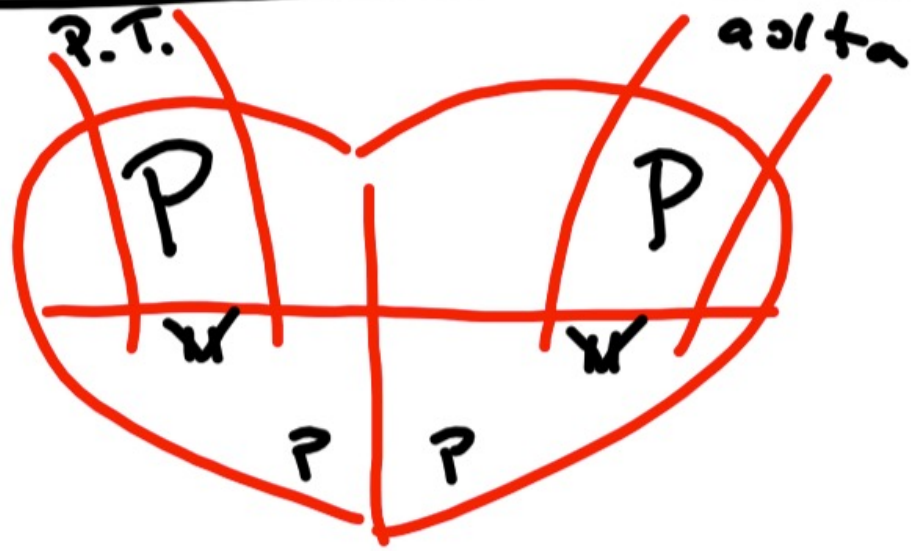
first heart sound (S_1)

Opening of Semilunar Valves



vent P > Great Artery P

Closing of Semilunar Valves



Great Artery P > vent P

second heart sound (S_2)

Normal

closed

opened

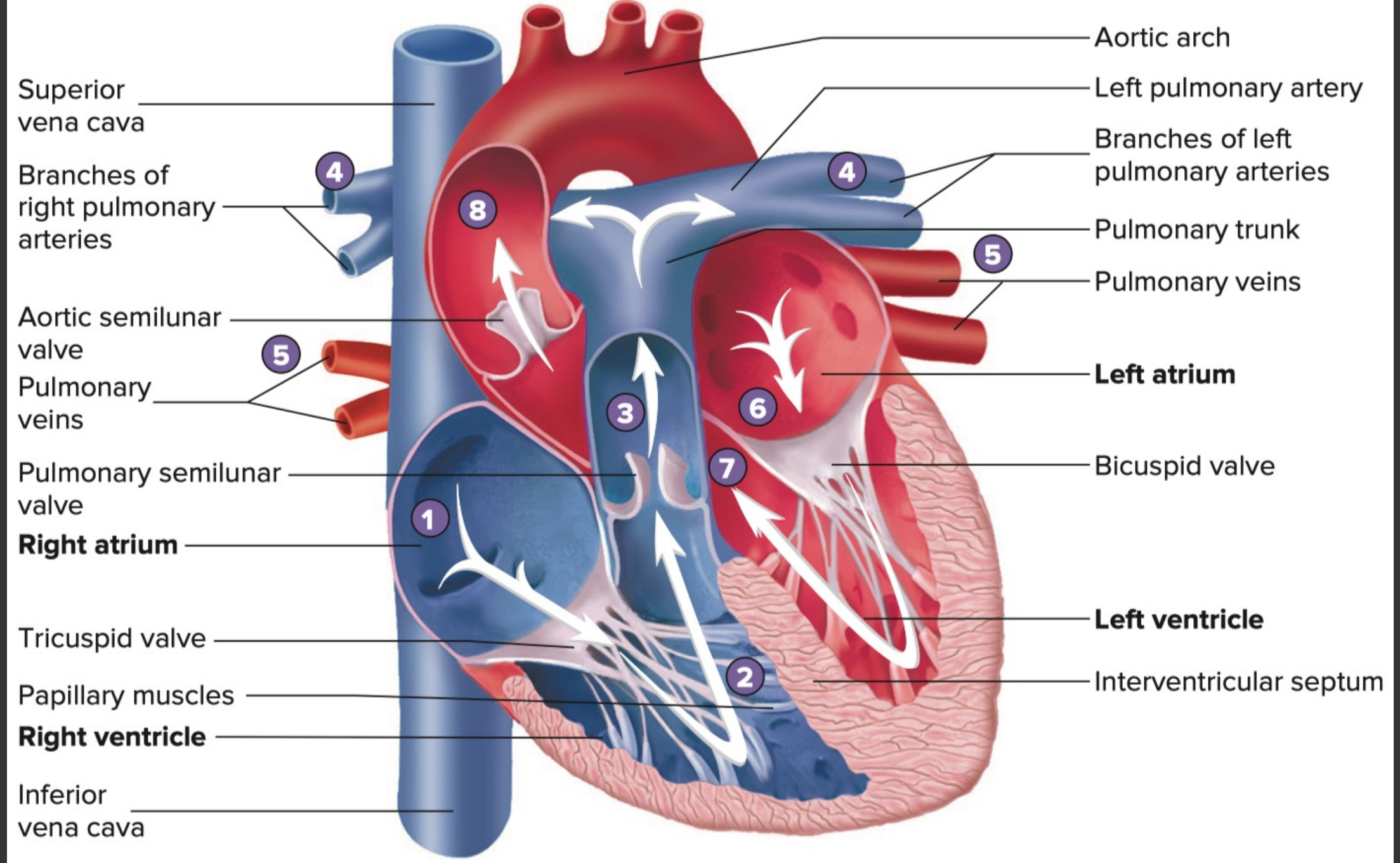
Stenosis (narrowed)

closed

or

heart works harder

Normal	Ins / Reg (improper closing)
 opened	 opened
 closed	 closed
	heart works harder


Superior vena cava
Branches of right pulmonary arteries
Aortic semilunar valve
Pulmonary veins
Pulmonary semilunar valve
Right atrium
Tricuspid valve
Papillary muscles
Right ventricle
Inferior vena cava
Aortic arch
Left pulmonary artery
Branches of left pulmonary arteries
Pulmonary trunk
Pulmonary veins
Left atrium
Bicuspid valve
Left ventricle
Interventricular septum
1
2
3
4
4
5
5
6
7
8

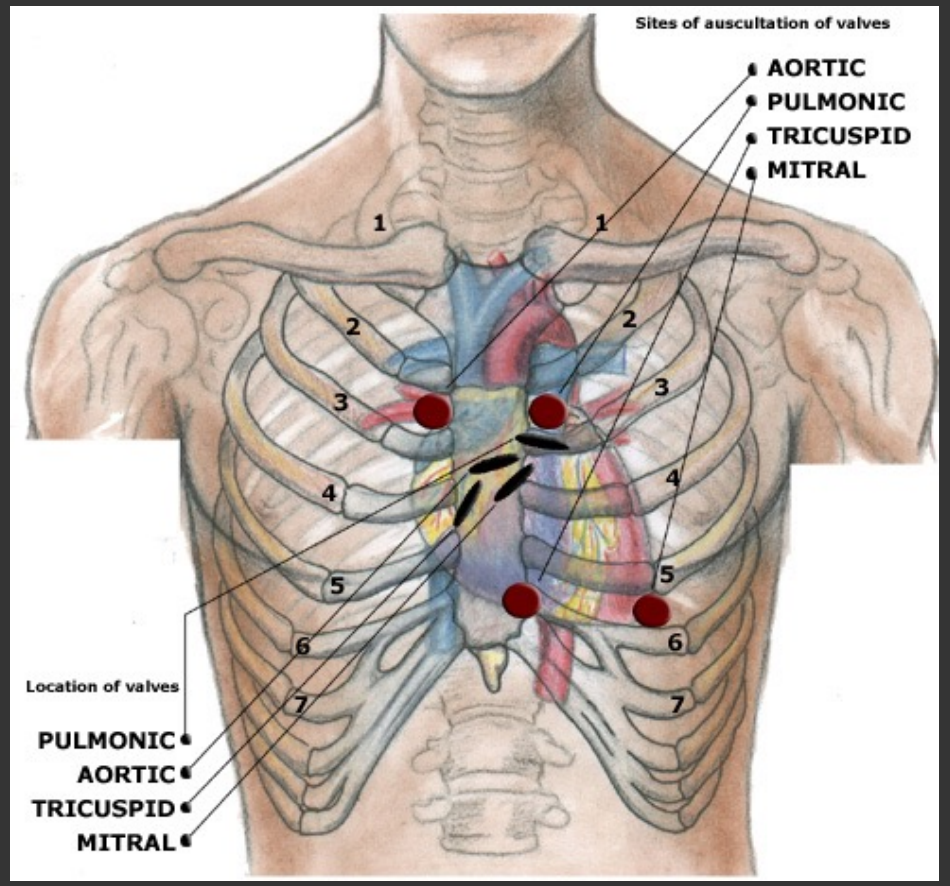
(a)

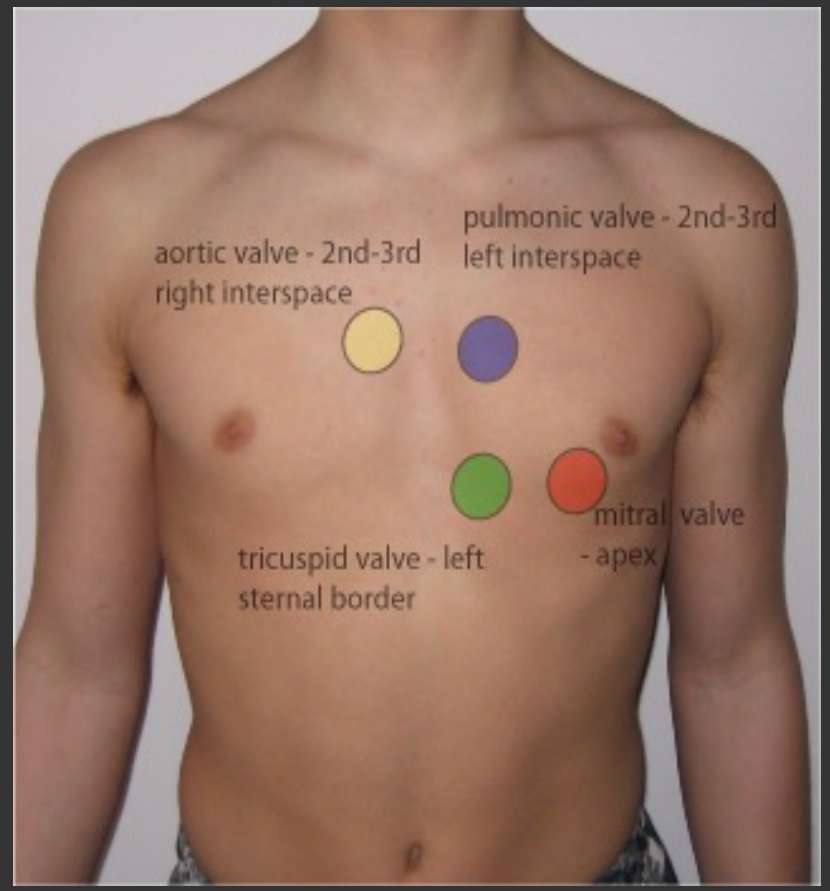
Heart Murmurs

- Indicates turbulent blood flow
- Septal defects (hole in the septum) and increased blood flow through the heart
- Valve disorders
  - Stenosis
    - Narrowing of valves
      - Creates resistance to flow
      - Murmur heard when blood flows through valve
  - Insufficiency / Regurgitation
    - Improper closing of valves
      - Back flow of blood through valve
      - Murmur heard when valves close
  - Aortic valve disorders are the most common
- Systolic murmur (between S_1 and S_2)
  - AV regurgitation
  - Semilunar stenosis
- Diastolic murmur (between S_2 and S_1)
  - Semilunar regurgitation
  - AV stenosis

Auscultations of the Heart

- Second intercostal space
  - Left side slightly away from midline: pulmonary semilunar valve
  - Right side slightly away from midline: aortic semilunar valve
- Fifth intercostal space
  - Left side slightly away from midline: tricuspid valve
  - Left side close to the nipple line: mitral valve





Cardiac Cycle

- Events that make up one heart beat

- Systole
  - Heart spends approximately ⅓ of its time in systole
  - Atrial systole
    - Time the atria are contracting
  - Ventricular systole
    - Time the ventricles are contracting
  - If systole is used without specifying a chamber , ventricular systole is implied

- Diastole
  - Heart spends approximately ⅔ of its time in diastole
  - Atrial diastole
    - Time the atria are relaxing
  - Ventricular diastole
    - Time the ventricles are relaxing
  - If diastole is used without specifying a chamber , ventricular diastole is implied

- Divided into five periods
  - Isovolumic contraction
  - Period of ejection
  - Isovolumic relaxation
  - Passive ventricular filling
  - Active ventricular filling

* Cardiac Cycle

- Isovolumic Contraction
- Period of Eject

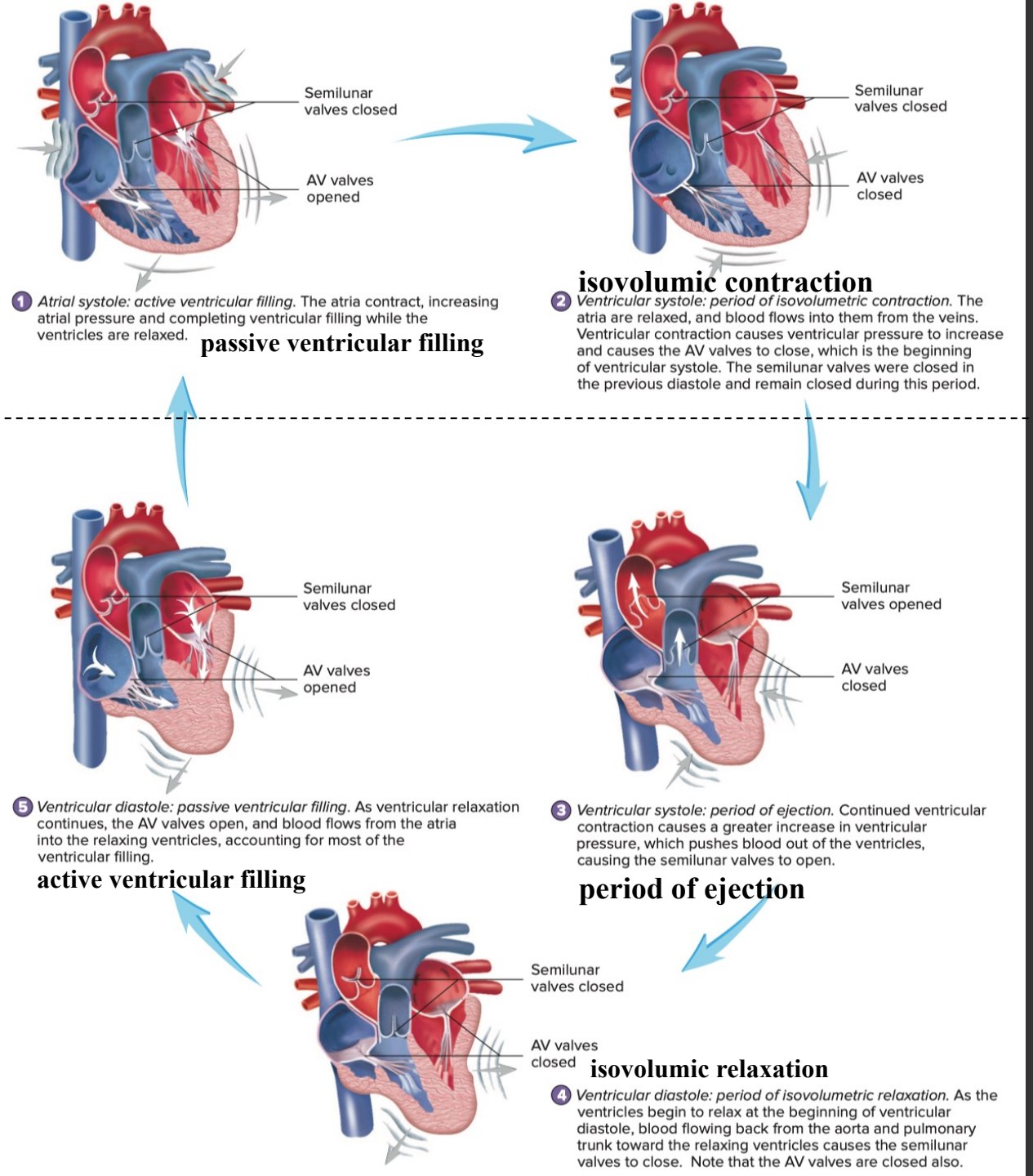
} Systolic

- Isovolumic Relaxation
- Passive Ventricular Filling
- Active Ventricular Filling

} Diastolic

Systole

Diastole

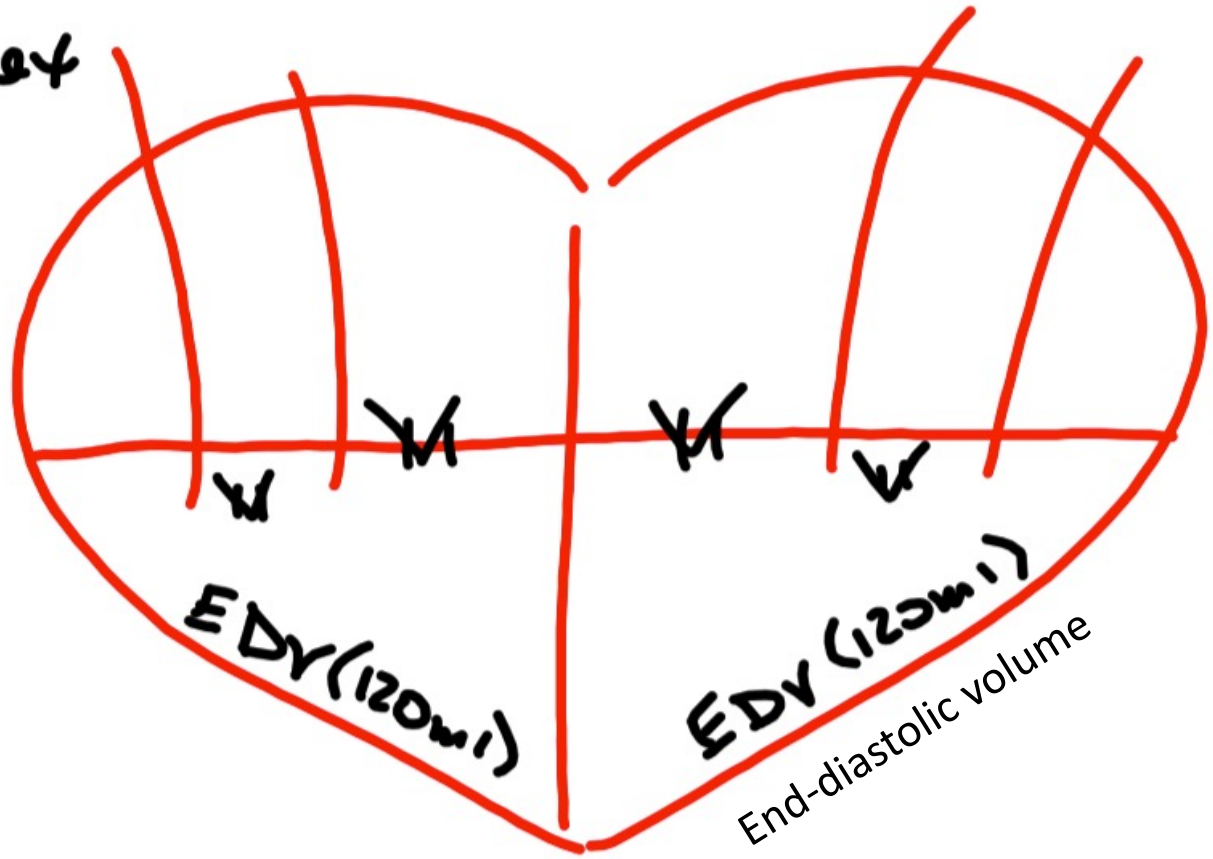


Cardiac Cycle – Systole – Isovolumic Contraction

- Isovolumic contraction (part of systole)
  - Ventricles begin to contract
    - Ventricular pressure increases and eventually exceeds atrial pressure
      - AV valves snap shut (all heart valves are now closed)
        - Therefore, no blood flow into or out of ventricles
          - Ventricular blood volume does not change
        - First heart sound (S_1)
        - AV insufficiency first heard at this time

Isovolumic Contraction ← systole

initiated by QRS complex



End-diastolic volume

murmur for an AV ins/reg could be heard

ventricles contract

↑ vent P → vent P > atrial P → AV valves closer (S1)

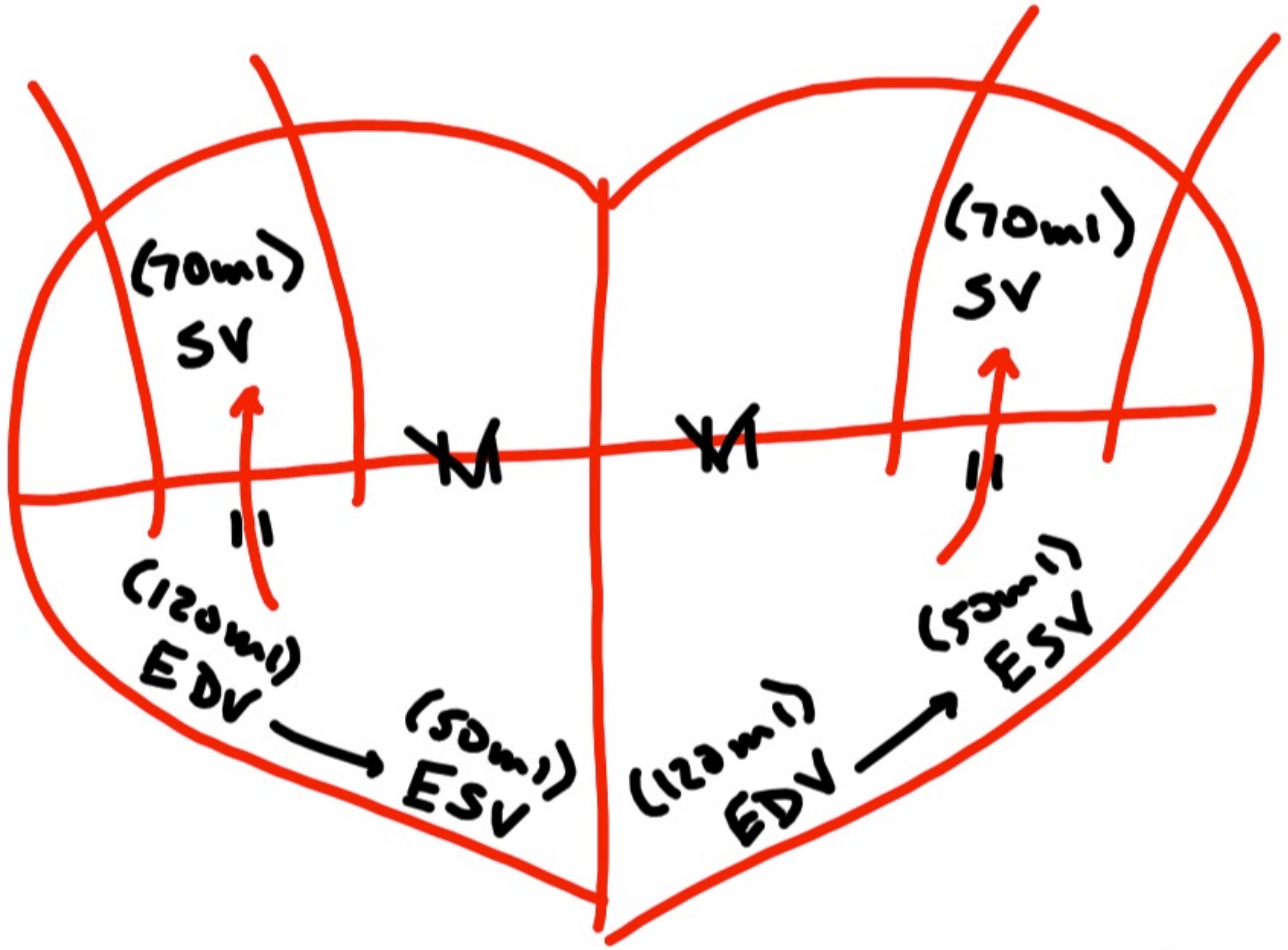
semilunar valves were already closed

all valves closed

Cardiac Cycle – Systole – Period of Ejection

- S_1 is still evident at the onset of ejection
- Ventricular pressure increases further and exceeds pressure of great arteries
  - Forces aortic and pulmonary semilunar valves to open
    - Blood ejected from ventricles into pulmonary trunk and aorta
      - Approximately same volume of blood ejected to each
        - This despite the great difference in pressure
- Stroke volume (SV)
  - Volume of blood ejected to each great artery (~ 70 ml)
- End-diastolic volume (EDV)
  - Volume of blood in ventricles prior to ejection of blood (~ 120 ml)
- Ejection fraction (EF)
  - Percentage of blood ejected from each ventricle (55 to 70% is normal)
  - EF = (SV) / (EDV) x 100
- End-systolic volume (ESV)
  - Volume of blood in the ventricles after the stroke volume is ejected
  - ESV = (EDV) – (SV)
- Semilunar stenosis first heard at this time

Period of Ejection ← systole



murmur due to a semilunar stenosis

ventricles continue to contract

↑↑ Vent P → Vent P > Great Artery P → open semilunar valves

Cardiac Cycle – Diastole

- Isovolumic relaxation (part of diastole)
  - Relaxation of ventricles
    - Ventricular pressure decreases below pressure of great arteries
      - Semilunar valves snap shut (all heart valves are now closed)
        - Ventricular blood volume does not change
        - Second heart sound (S2)
        - Semilunar insufficiency first heard at this time

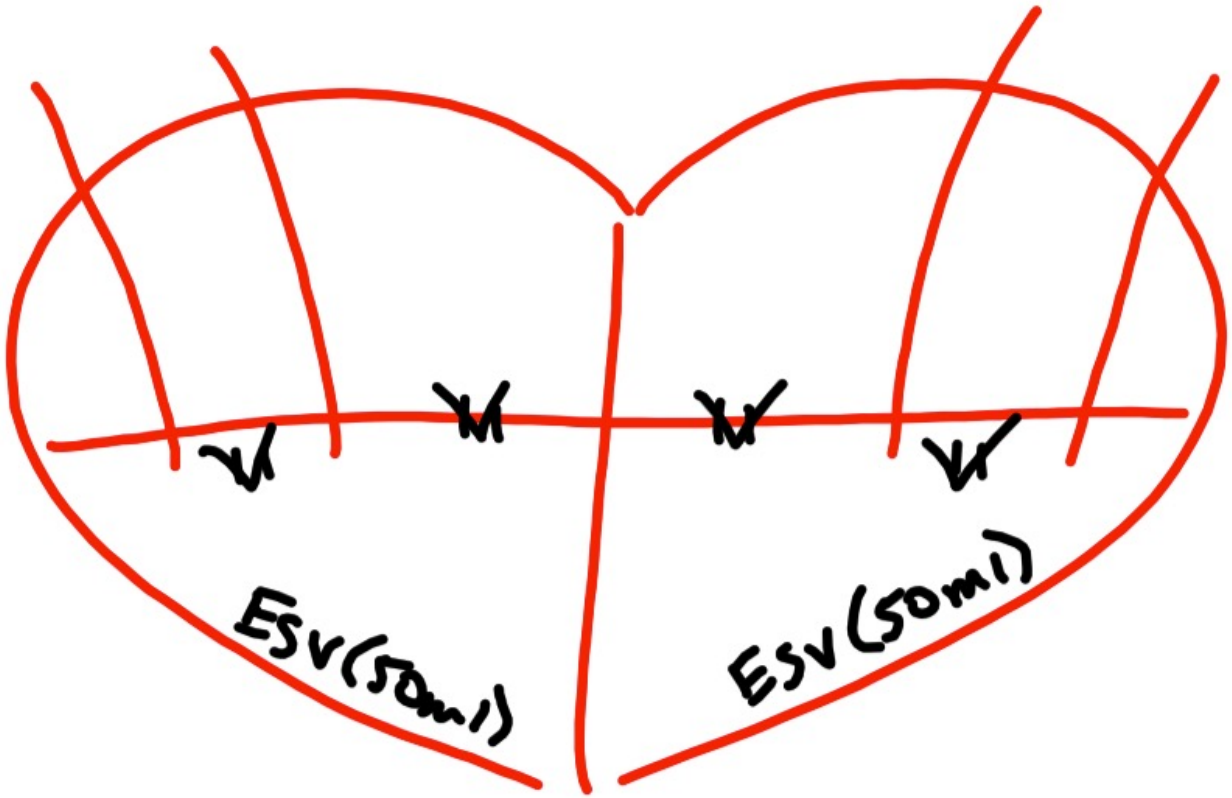
- Passive ventricular filling (part of diastole)
  - Ventricular pressure decreases further and is now lower than atrial pressure
    - Forces AV valves to open
      - Blood flows passively from atria into ventricles
      - AV stenosis first heard at this time

- Active ventricular filling (part of diastole)
  - Atria contract and actively fill ventricles with more blood
  - End-diastolic volume (EDV)
- Volume of blood in ventricles when filled (~ 120 ml)

Isovolumic Relaxation ← diastole

murmurs due to a semilunar insufficiency

↑Great Artery P because of SV



ventricles are relaxed ∴ ↓ventricular blood volume

↓Vent P → Great Artery P > vent P → (S2) close semilunar valves

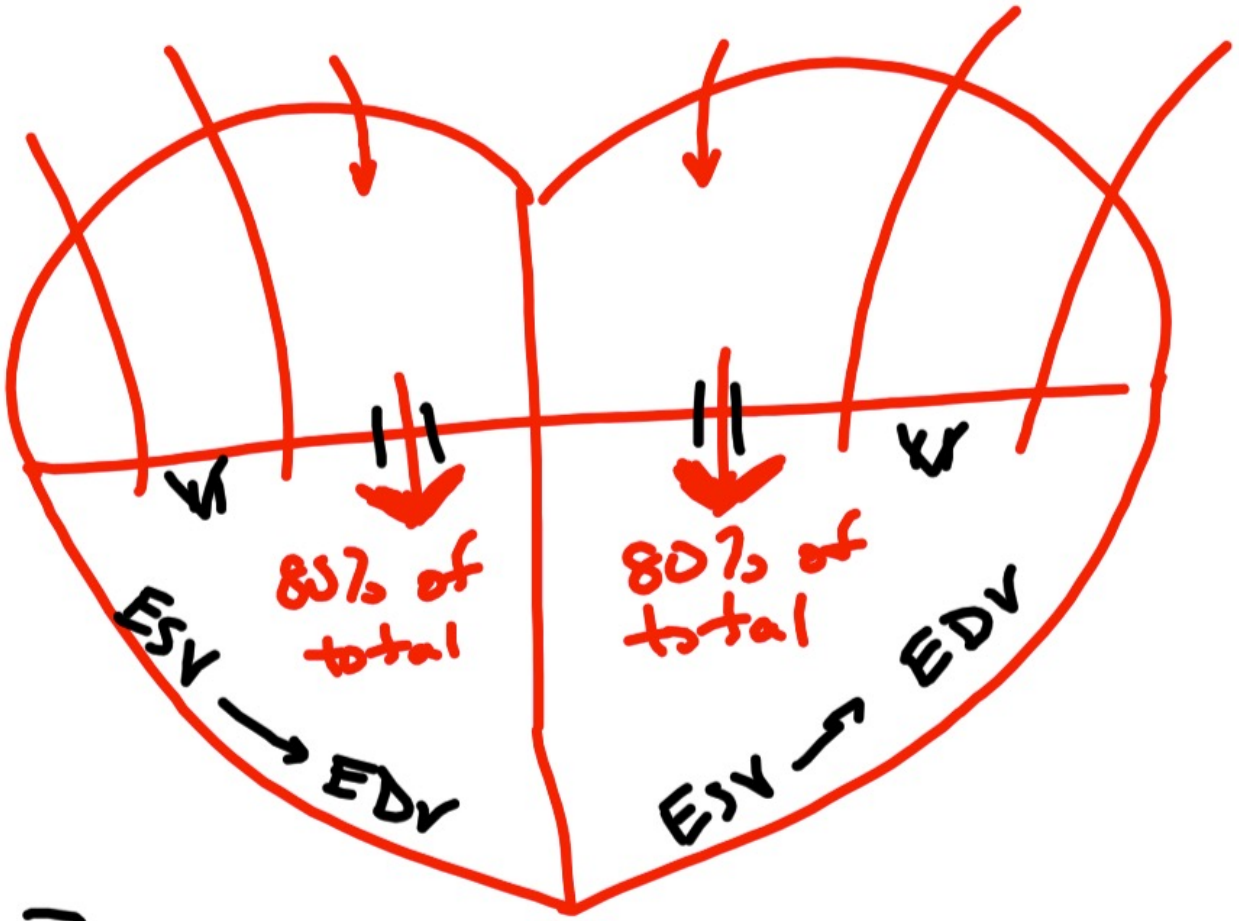
AV valves already closed

all valves closed

Passive Ventricular Filling ← diastole

atria are relaxed →

murmur due to an AV stenosis

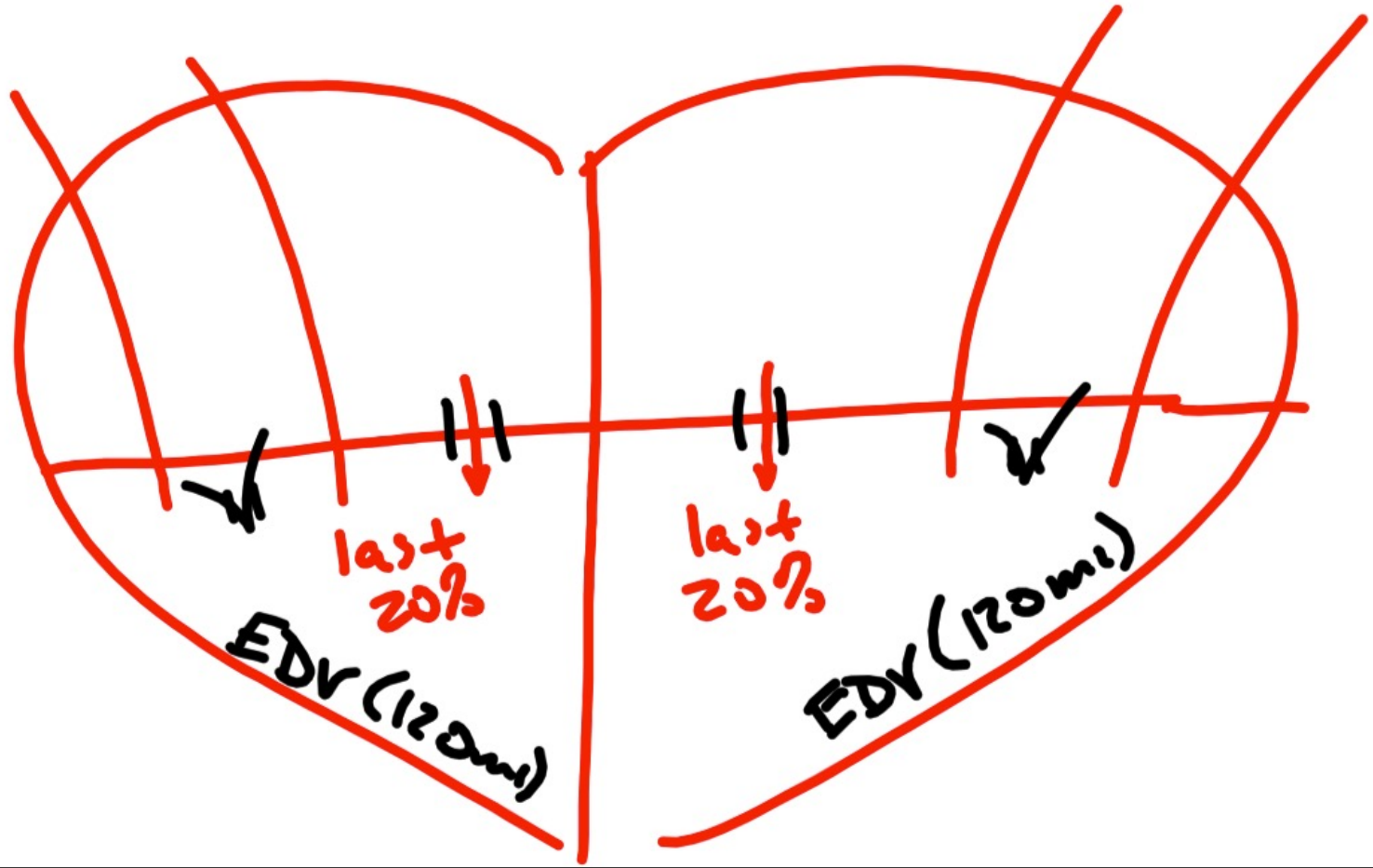


with veins return to the atria ↓

↑ atrial P ⟶ atrial P > vent P → AV valves open

Active Ventricular Filling ← Diastole

atria are contracting



Cardiac Output (CO)

- Volume of blood pumped by either the left or right ventricle per minute
- CO = (SV) x (HR)
- = (70 ml blood / heart beat) x (70 heart beats / minute)
- = 4,900 ml blood / minute or 4.9 liters blood / minute
- Heart is most efficient when heart rate is low and stroke volume is high
- Inotropic – something that affects heart contractility and therefore stroke volume
- Chronotropic – something that affects heart rate

$$ESV = EDV - SV$$

$$SV = EDV - ESV$$

$$EDV = ESV + SV$$

$$E.F. = \frac{SV}{EDV} \cdot 100$$

an E.F. greater than 50% is normal

* E.F. is an indicator of how healthy the heart is

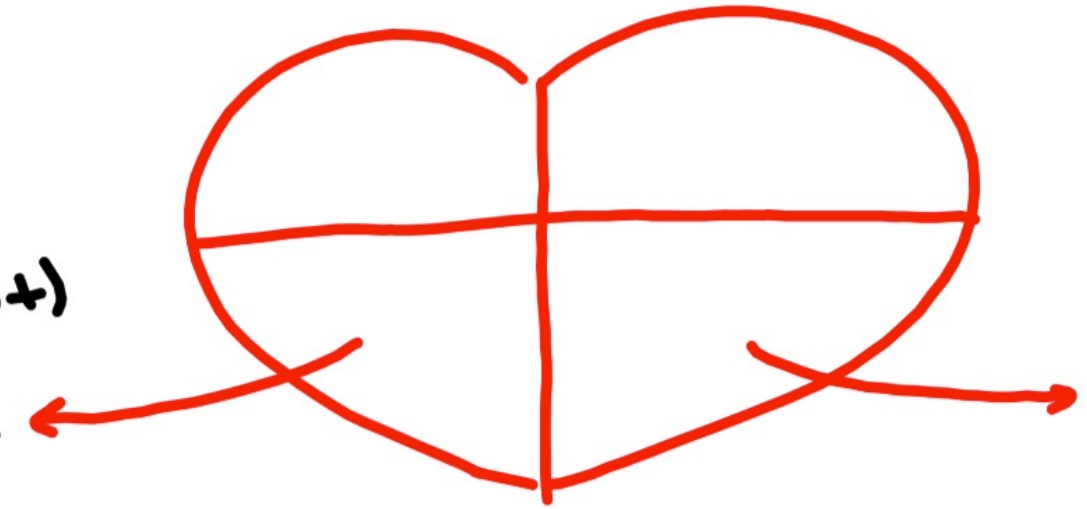
* Unhealthy heart (i.e. heart failure)
  ∴ weak heart muscle

$$E.F. = \frac{30ml}{120ml} \cdot 100$$

$$= 25\%$$

(cardiac output)

~ 5 l blood /min



(cardiac output)

~ 5 l blood /min

$$CO = HR \times SV$$

$$= \frac{70 \text{ beats}}{\text{min}} \times \frac{70 \text{ ml blood}}{\text{beat}}$$

$$= 4,900 \text{ ml blood/min} \rightarrow \sim 5 \text{ l blood/min}$$

* Efficient Heart:

↓ HR ∴ TSV

↗

$$CO = \frac{50 \text{ beats}}{\text{min}} \times \frac{100 \text{ ml blood}}{\text{beat}}$$

$$= 5 \text{ l/min}$$

Regulation of Cardiac Output

- Intrinsic Regulation
- Extrinsic Regulation

Intrinsic Factors

- Health of the myocardium
  - Healthy: normal contractility
  - Unhealthy: decreased contractility (negative inotropic effect)

- Preload
  - Volume of blood in the ventricle immediately prior to systole (i.e. EDV)
  - Frank – Starling law of the heart
    - Relationship between preload and heart contractility
      - Increased preload causes increased contractility (to a point)
        - Increases SV
        - Positive inotropic effect
      - Decreased preload causes decreased contractility
        - Decreases SV
        - Negative inotropic effect
- Small changes in preload cause changes in stroke volume

Intrinsic Factors

- Afterload
  - Resistance in the great arteries that ventricles must overcome to pump blood
    - i.e. factors that affect blood pressure
  - Stroke volume remains constant with afterloads up to 180 mm Hg
    - However , heart must increase contractility to maintain SV

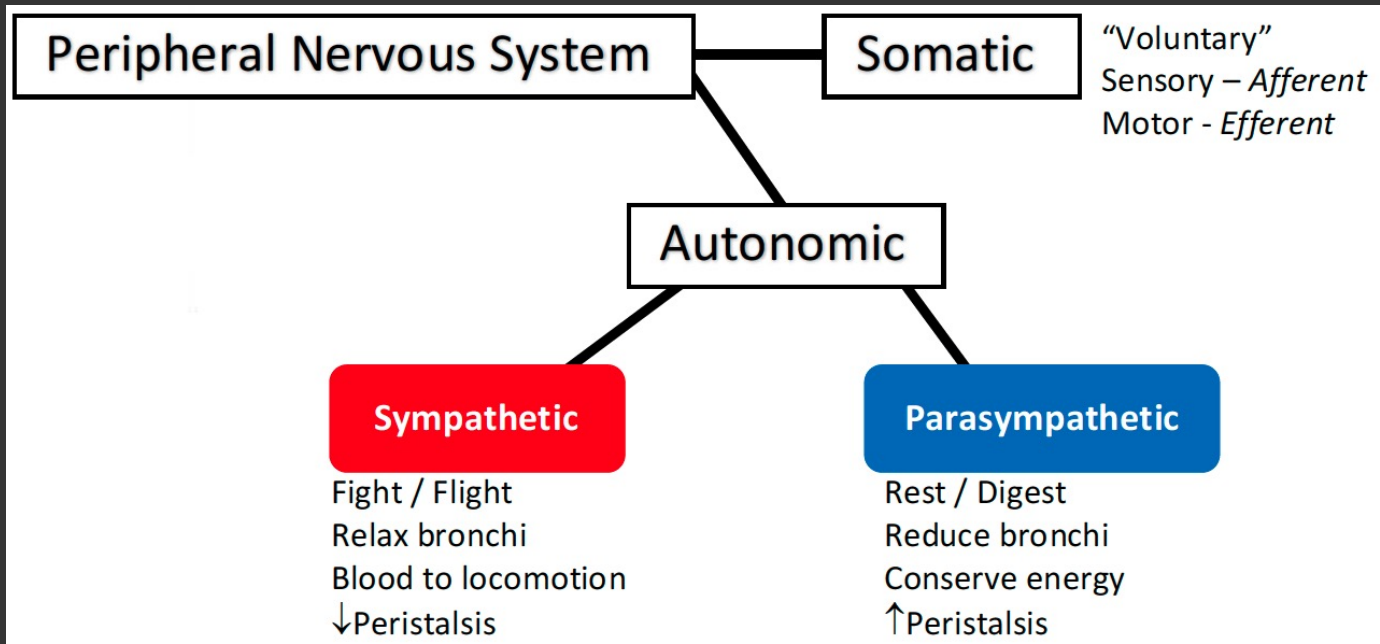
Preload	Stroke Volume	Afterload	Stroke Volume
90 ml	50 ml	100 mmHg	70 ml
120 ml	70 ml	120 mmHg	70 ml
150 ml	90 ml	150 mmHg	70 ml
180 ml	110 ml	180 mmHg	70 ml
200 ml	125 ml	200 mmHg	60 ml
220 ml	140 ml	220 mmHg	50 ml

Extrinsic Regulation of Cardiac Output

- Cardiac Control Centers of the Medulla
- Hormones
- Body Temperature

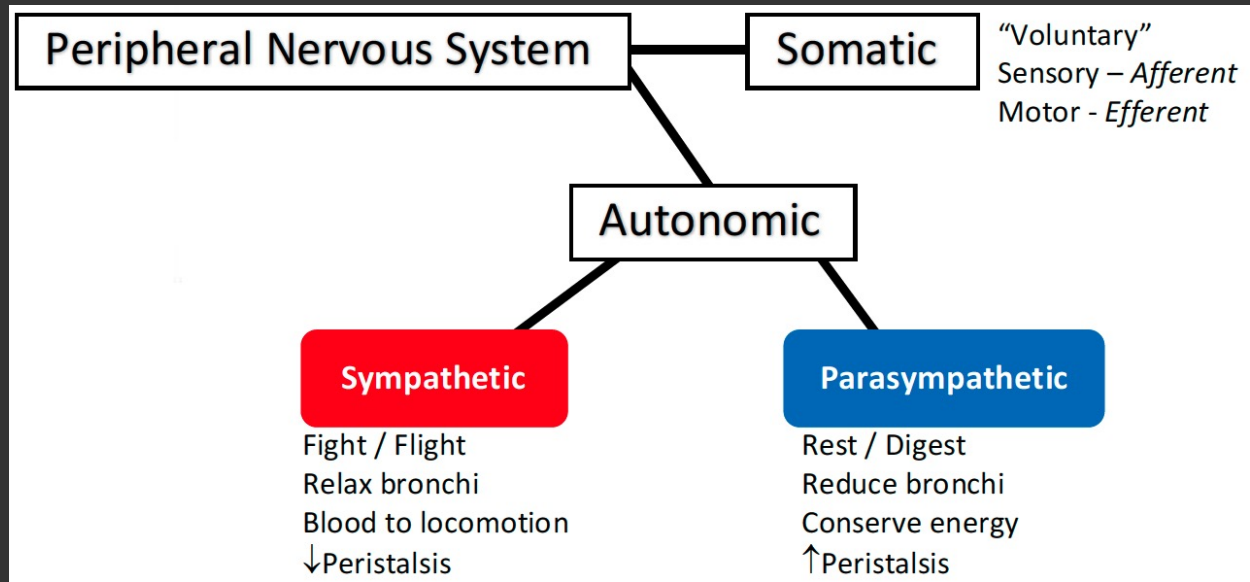
Extrinsic Factors – Cardiac Control Center

- Control via :
  - Medulla :
    - Autonomic Nervous System
  - Vagus Nerve :
    - Parasympathetic
      - Primary controller of resting heart rate
      - Innervates SA node , AV node , atrial myocardium
      - Has an inhibitory influence on the heart
        - Decreases heart rate
          - Negative chronotropic effect



Extrinsic Factors – Cardiac Control Center

- Control via :
  - Cardiac Nerve
    - Autonomic Nervous System
      - Sympathetic
    - Primary controller of heart contractility
    - Contributes very little to resting heart rate
    - Can cause large changes in heart rate contractility
    - Innervates SA node , AV node , atrial and ventricular myocardium
    - Has an excitatory influence on the heart
      - Increases heart rate
        - Positive chronotropic effect
      - Increases heat contractility
        - Positive ionotropic effect

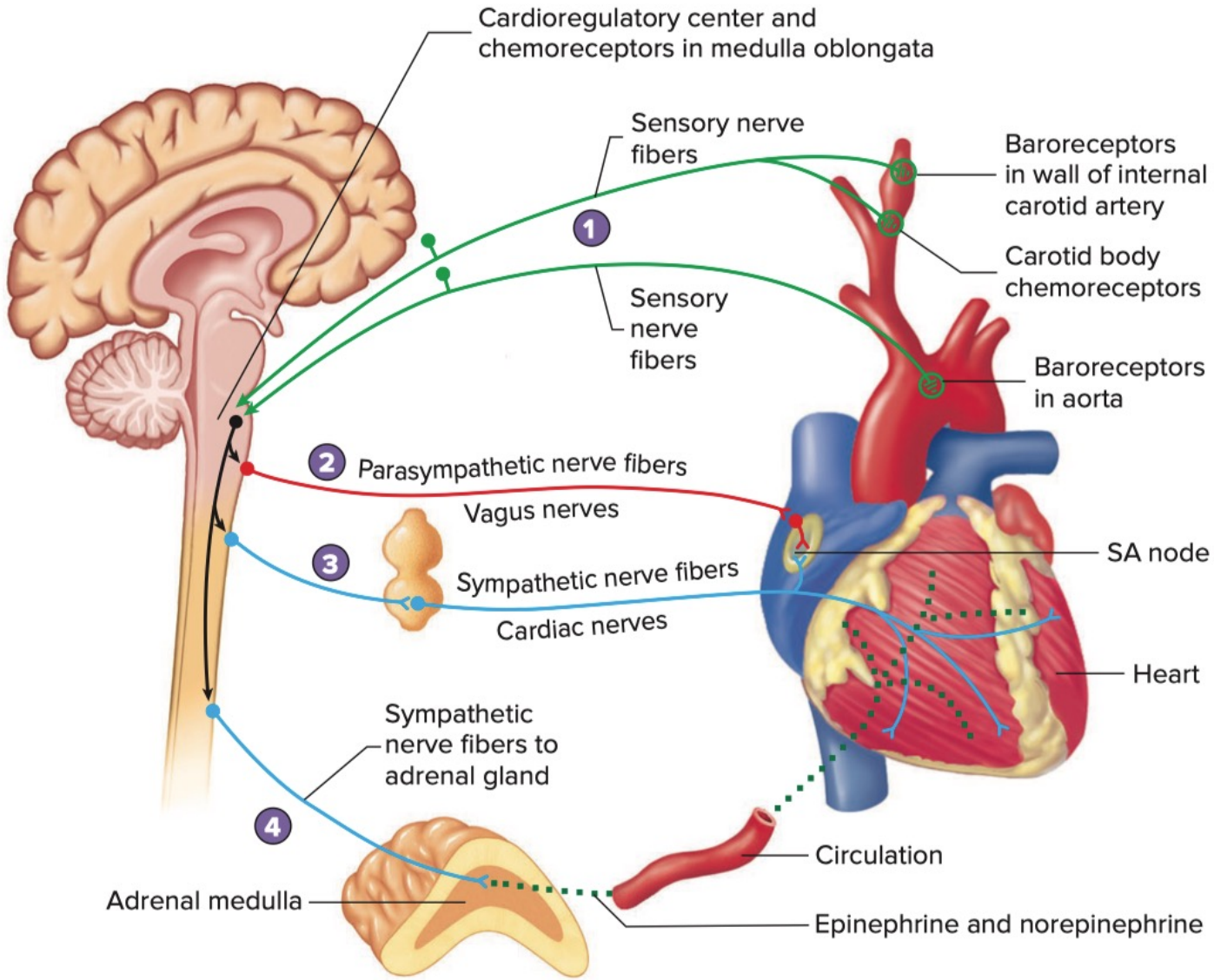


1. Sensory neurons (*green*) carry action potentials from baroreceptors and carotid body chemoreceptors to the cardioregulatory center. Chemoreceptors in the medulla oblongata also influence the cardioregulatory center.

2. The cardioregulatory center controls the frequency of action potentials in the parasympathetic neurons (*red*) extending to the heart through the vagus nerves. The parasympathetic neurons decrease the heart rate.

3. The cardioregulatory center controls the frequency of action potentials in the sympathetic neurons (*blue*). The sympathetic neurons extend through the cardiac nerves and increase the heart rate and the stroke volume.

4. The cardioregulatory center influences the frequency of action potentials in the sympathetic neurons (*blue*) extending to the adrenal medulla. The sympathetic neurons increase the secretion of epinephrine and some norepinephrine into the systemic circulation. Epinephrine and norepinephrine (*dotted green line*) increase the heart rate and stroke volume.



PROCESS FIGURE 20.22 Baroreceptor and Chemoreceptor Reflexes

Reflexes in response to changes in blood pressure, pH, blood O_2, and blood CO_2 levels help regulate the activity of the heart to maintain homeostasis. Sensory neurons (*green*) carry action potentials from sensory receptors to the medulla oblongata. Sympathetic (*blue*) and parasympathetic (*red*) neurons exit the spinal cord or medulla oblongata and extend to the heart to regulate its function. Epinephrine and norepinephrine (*dotted green line*) from the adrenal gland also help regulate the heart's action (*SA* = sinoatrial). AP|R

* Positive inotrope is something that causes an increase in heart contractility (↑SV)

* Negative inotrope is something that causes a decrease in heart contractility (↓SV)

* Positive chronotrope is something that causes an increase in heart rate

* Negative chronotrope is something that cause a decrease in heart rate

* Unhealthy heart (i.e. heart failure)

↳ ↓ contractility

↳ ↓ SV

↑

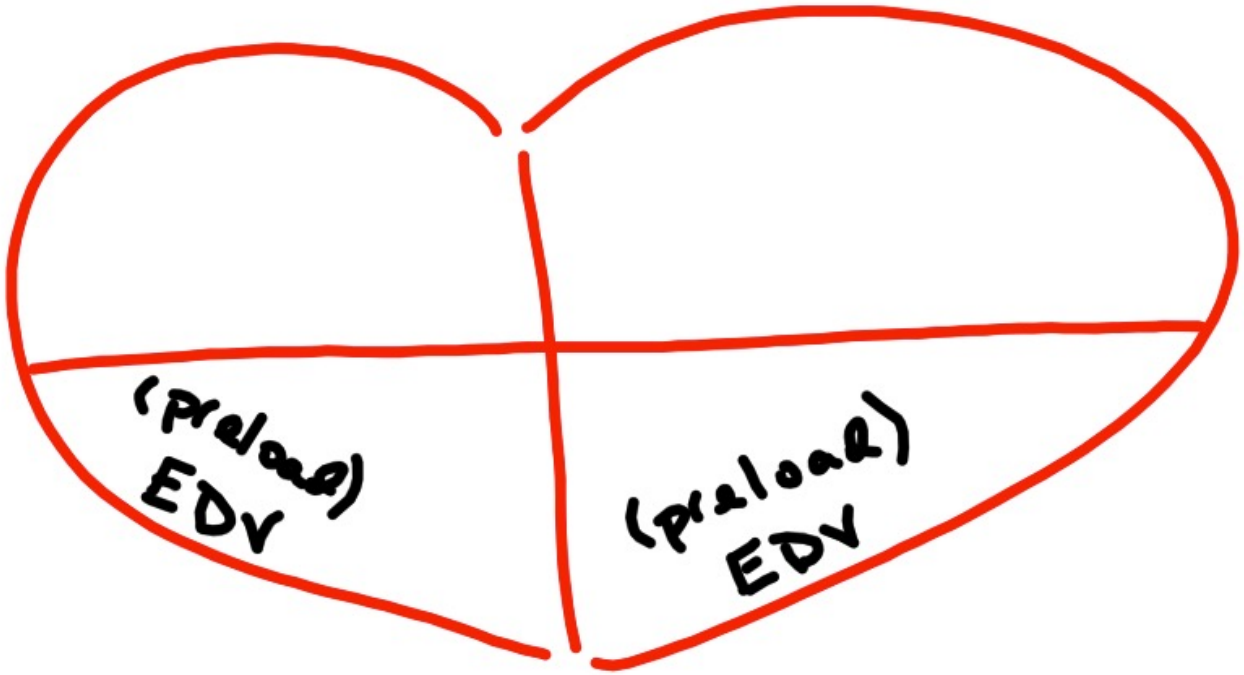
body responds by: ↑ HR

inefficient hard

↑

weak

heart muscle



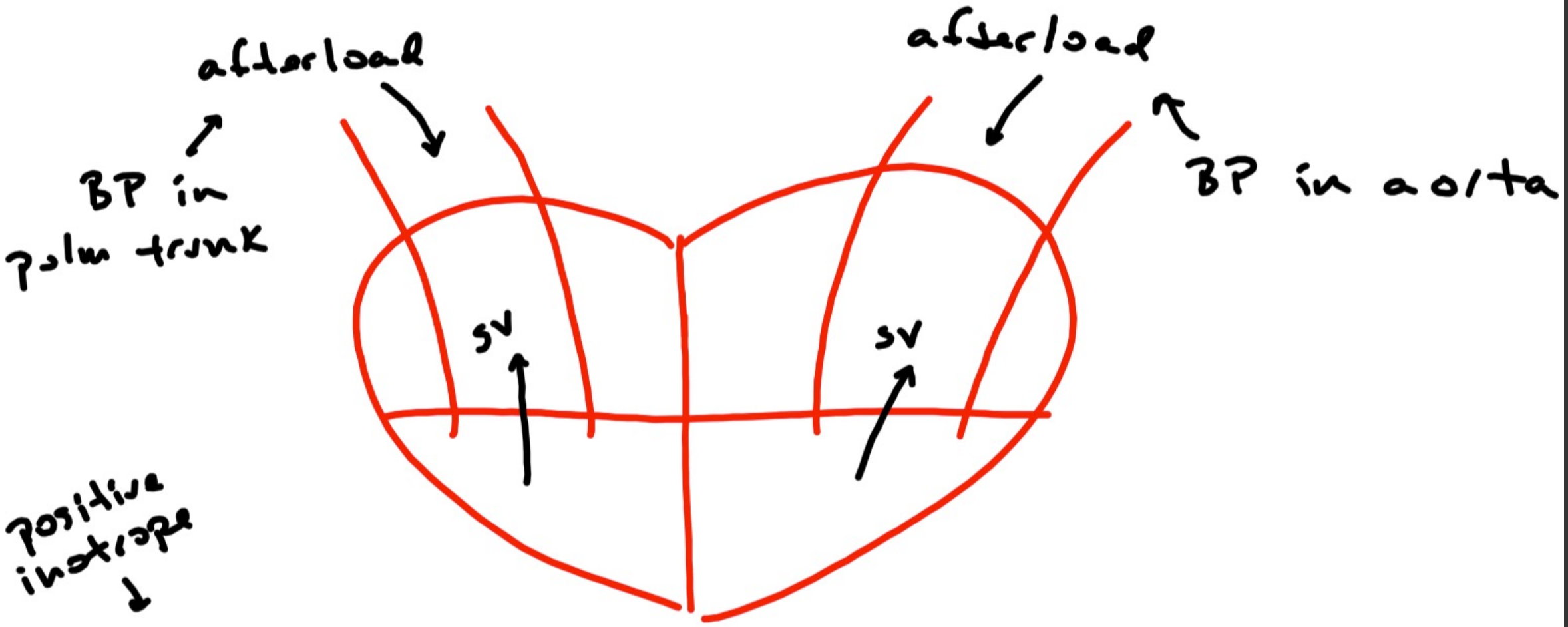
positive inotrope

↓

↑ preload → ↑ contractility → ↑ SV

↓ preload → ↓ contractility → ↓ SV

negative inotrope

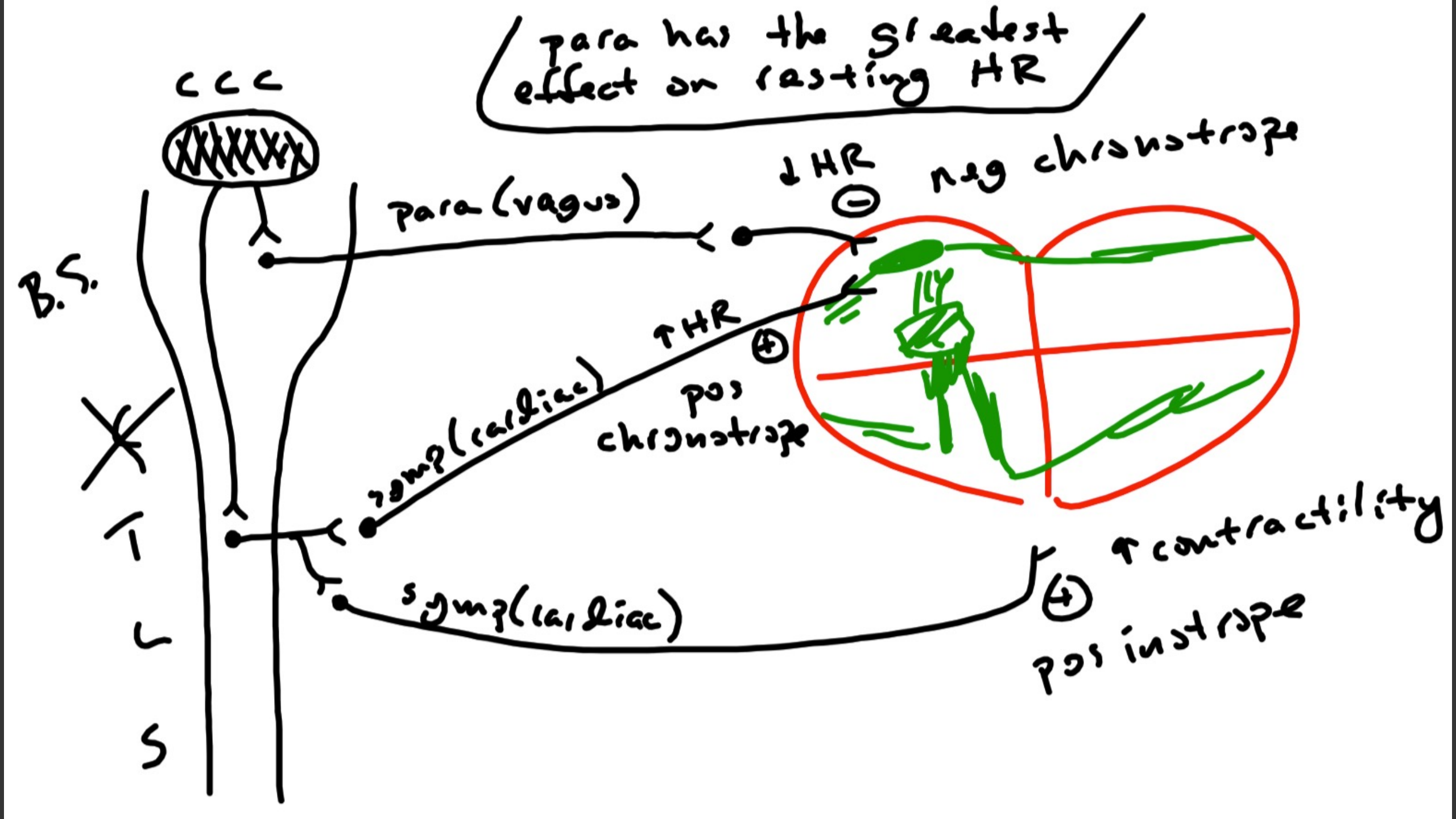


positive inotrope ↓

↑ afterload → ↑ heart contractility

↓ afterload → ↓ heart contractility

↑ negative inotrope

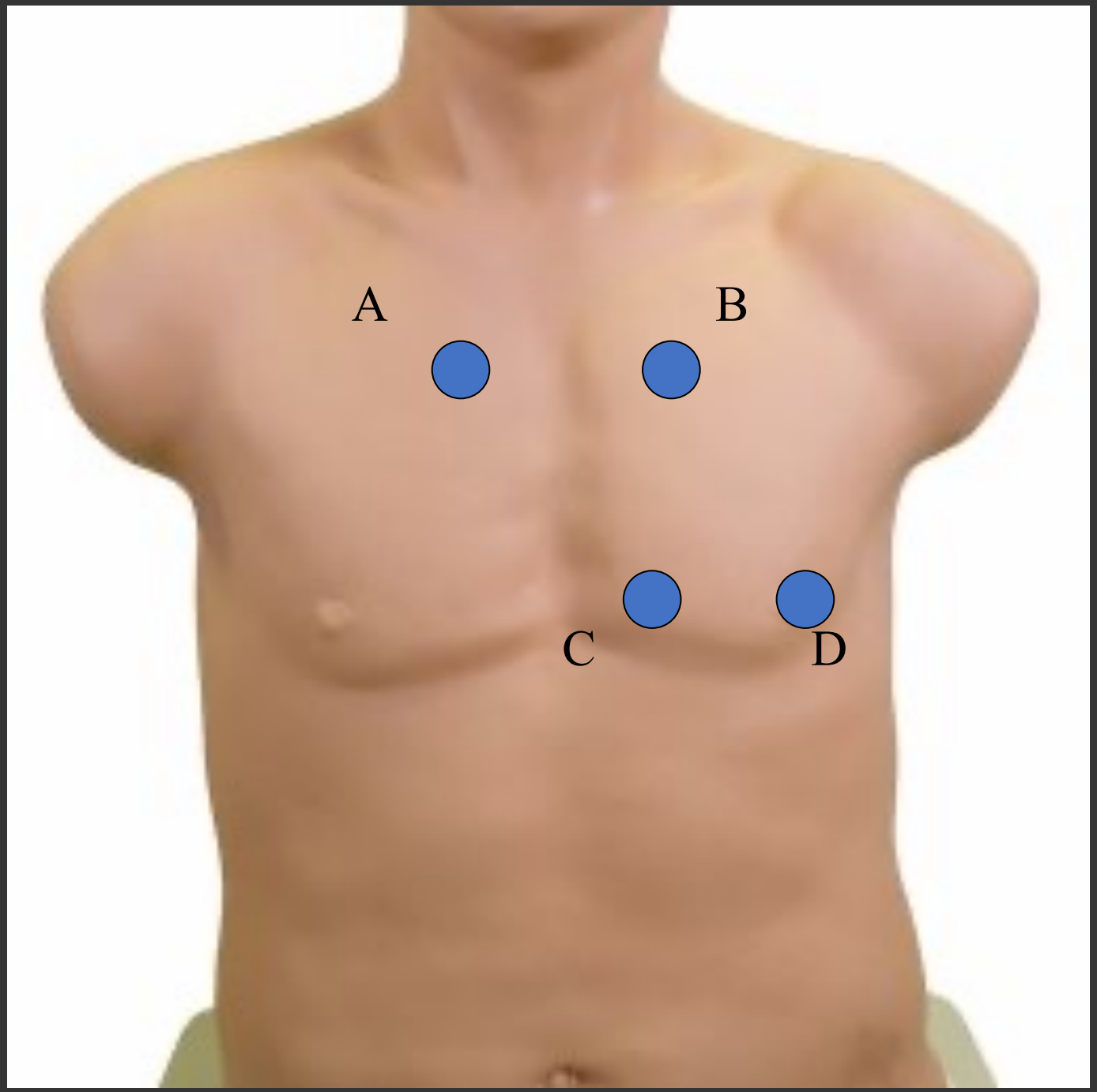

para has the greatest effect on resting HR
CCC
B.S.
para (vagus)
↓HR
neg chronotrope
symp (cardiac)
↑HR
pos chronotrope
symp (cardiac)
↑ contractility
pos inotrope
T
L
S

Extrinsic Factors – Hormones

- Epinephrine and norepinephrine released from adrenal gland
  - Increases heart rate
    - Positive chronotropic effect
  - Increases heart contractility
    - Positive inotropic effect
- Thyroid hormones (T_3 and T_4) released from thyroid
  - Increase heart rate
    - Positive chronotropic effect
  - Increase heart contractility
    - Positive inotropic effect

Extrinsic Factors – Body Temperature

- A change of 1°C changes heart rate approximately 10 beats per minute
  - Affects action potential generation and conduction
- Increase temperature
  - Increase heart rate
    - Positive chronotropic effect
  - Decrease temperature
    - Decrease heart rate
      - Negative chronotropic effect


A
B
C
D

Blood Vessels

- Arteries
  - Strong , thick , muscular elastic vessels
    - Adapted to carry blood under high pressure
      - Highest normal pressure is approximately 110 mm Hg
    - Contain approximately 10% of total blood volume
    - Function to distribute blood to the body
      - Carry oxygen , nutrients , hormones , enzymes , etc. to cells
    - Continually branch down to arterioles (smallest branches of an artery)
- Arterioles
  - Site of largest blood pressure drop
  - Contain approximately 5% of total blood volume
  - Contain large amounts of smooth muscle in their walls
  - Function to regulate blood pressure and blood flow to organs
  - Greatest resistance to blood flow
  - Blood flow and blood pressure influenced by sympathetic and local factors

Factors Contributing to Venous Return

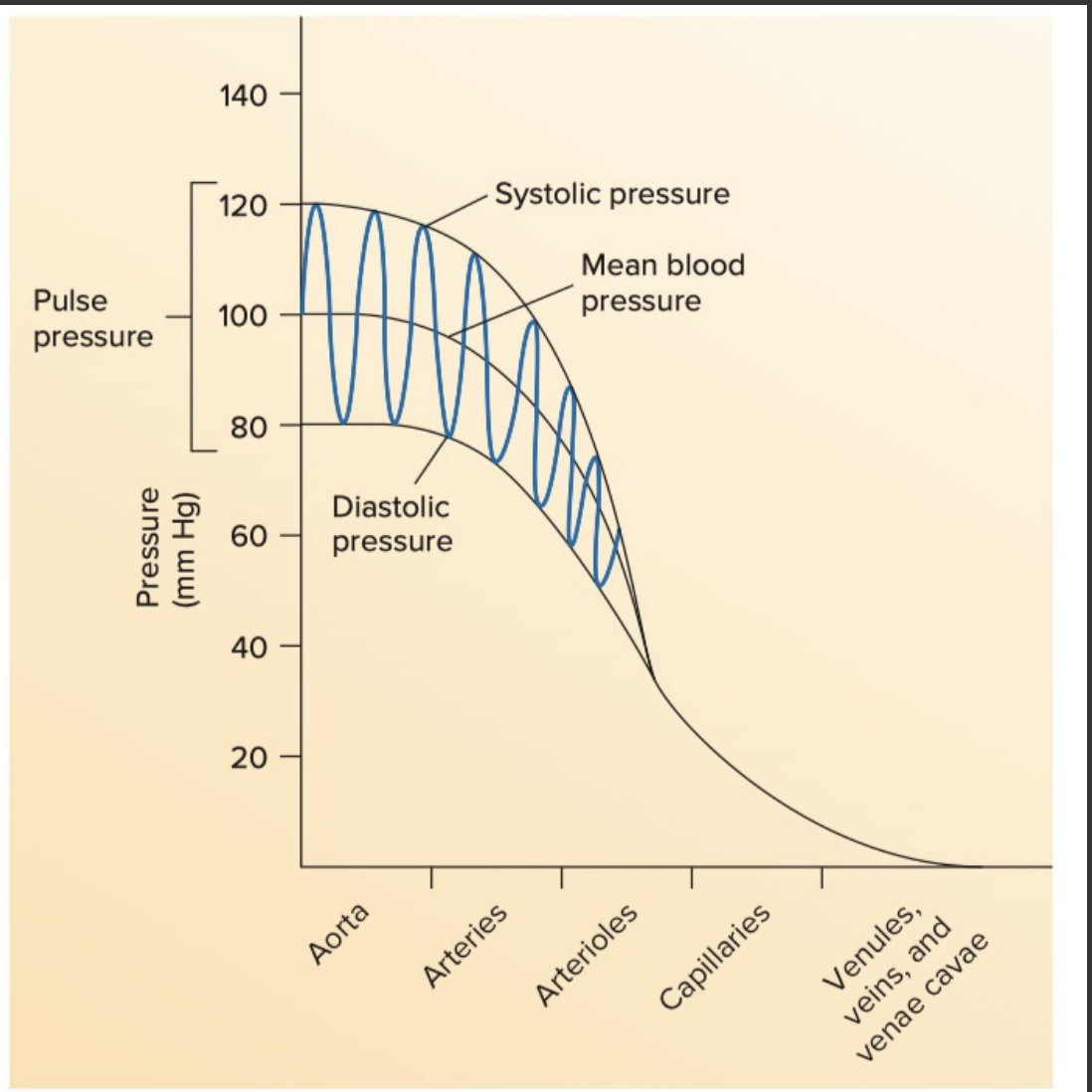
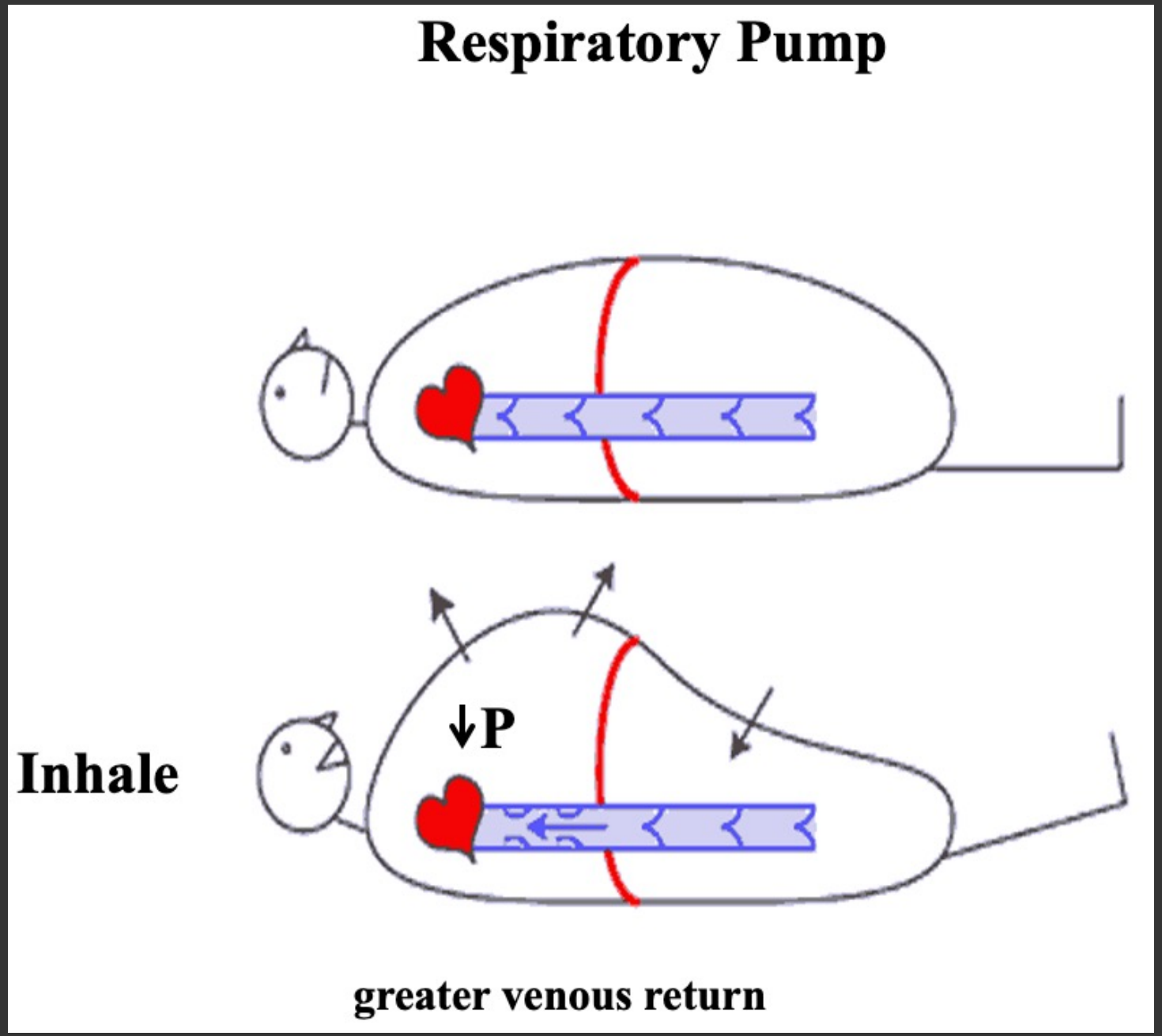
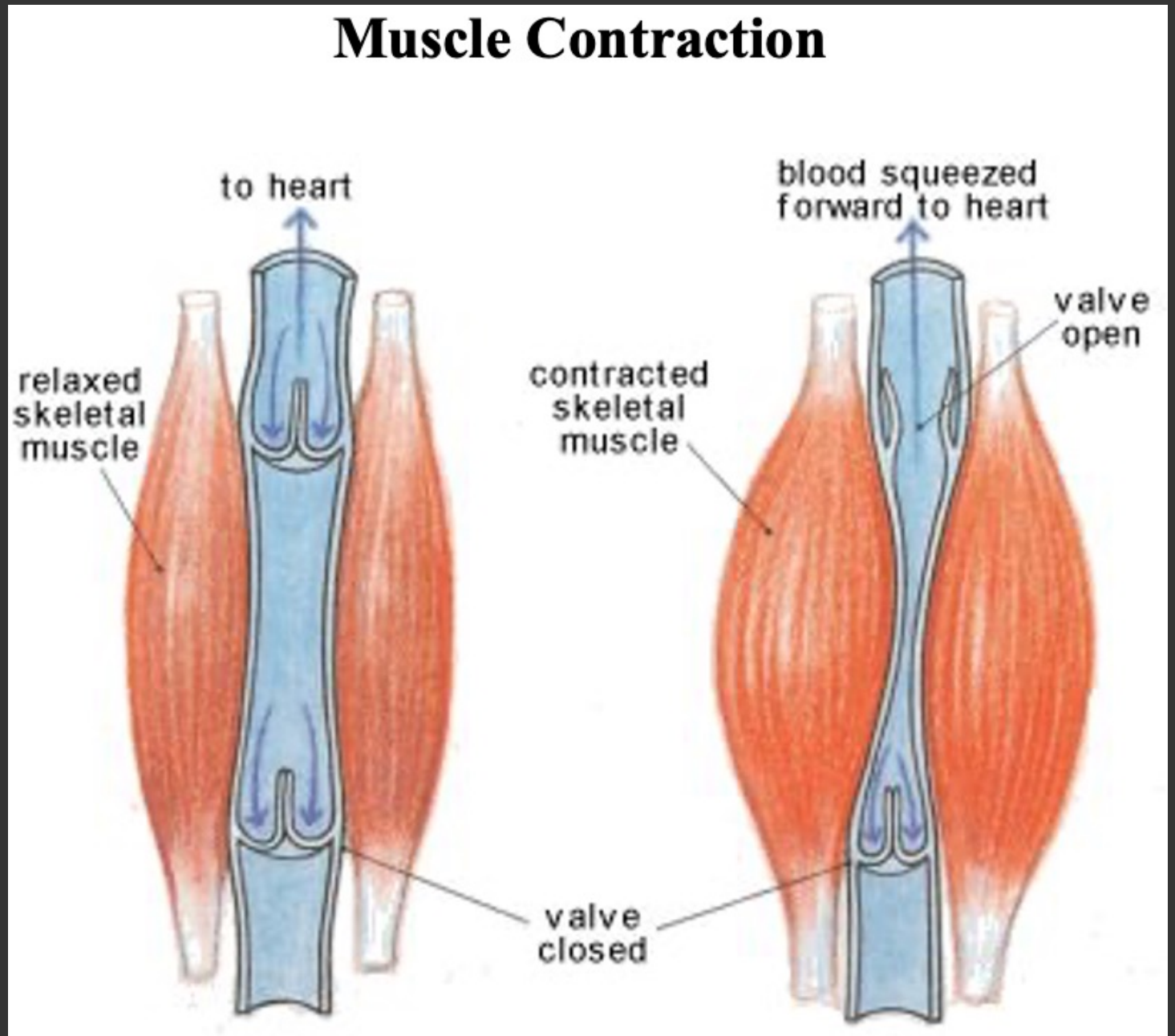


FIGURE 21.35 Blood Pressure in the Major Blood Vessel Types
In small arteries and arterioles, blood pressure fluctuations between systole and diastole are reduced. No fluctuations in blood pressure occur in capillaries and veins.

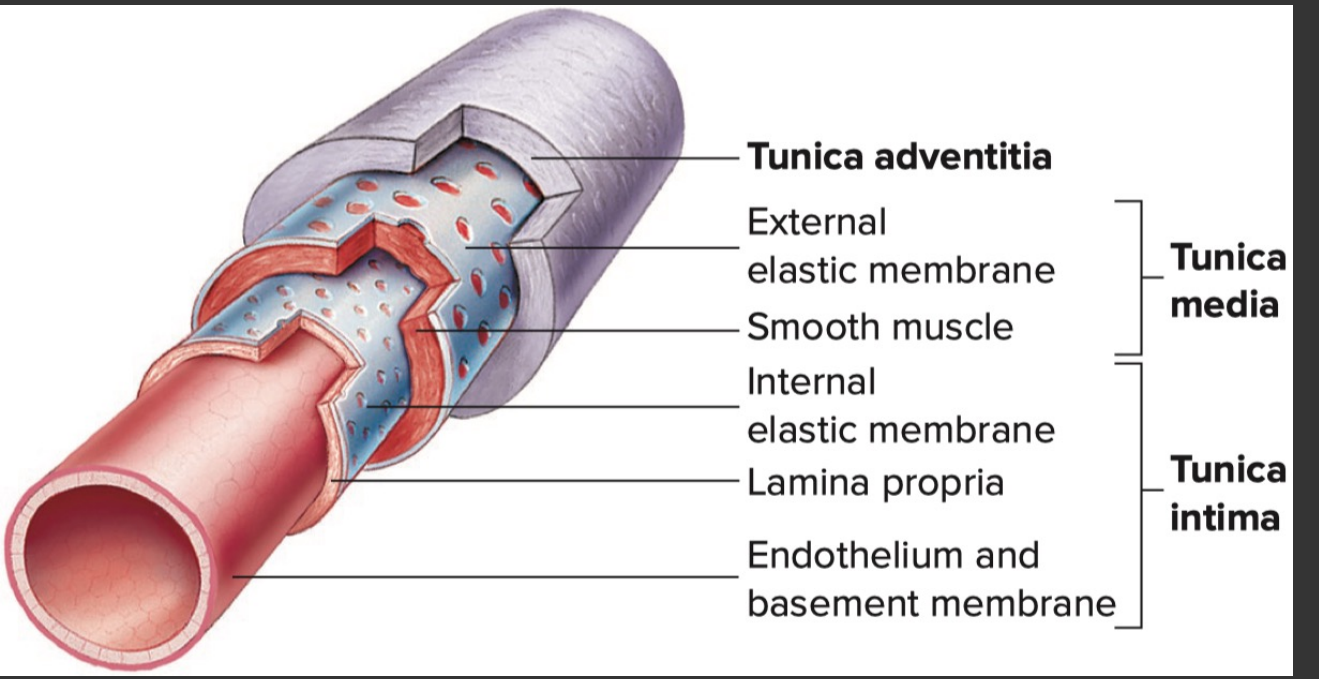
Factors Contributing to Venous Return



Factors Contributing to Venous Return



Veins and Venules



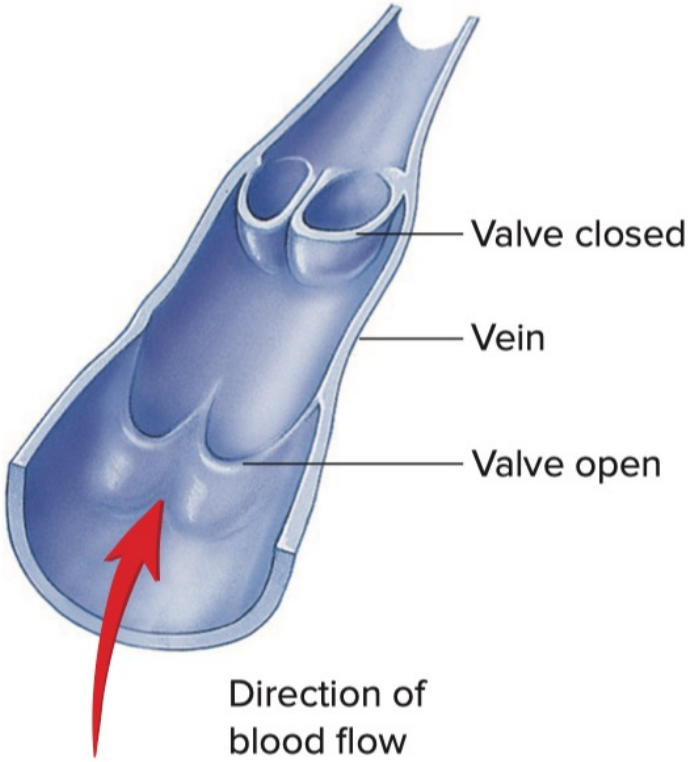


FIGURE 21.7 Valves
Folds in the tunica intima form the valves of veins, which allow blood to flow toward the heart but not in the opposite direction.

Blood Vessels

- Capillaries
- Smallest division of blood vessels
- However , largest total cross sectional area
- Contain approximately 5% of total blood volume
- Form extensive networks close to every cell in the body
- Function to deliver substances to cells and remove substances from cells
- Very slow blood flow (allows for full exchange of substances)
- Walls are one cell thick (optimizes diffusion)
- Walls are somewhat permeable
- Most have fenestrae – openings within endothelial cells
- Allows substances to more easily
- Diffuse and filter through

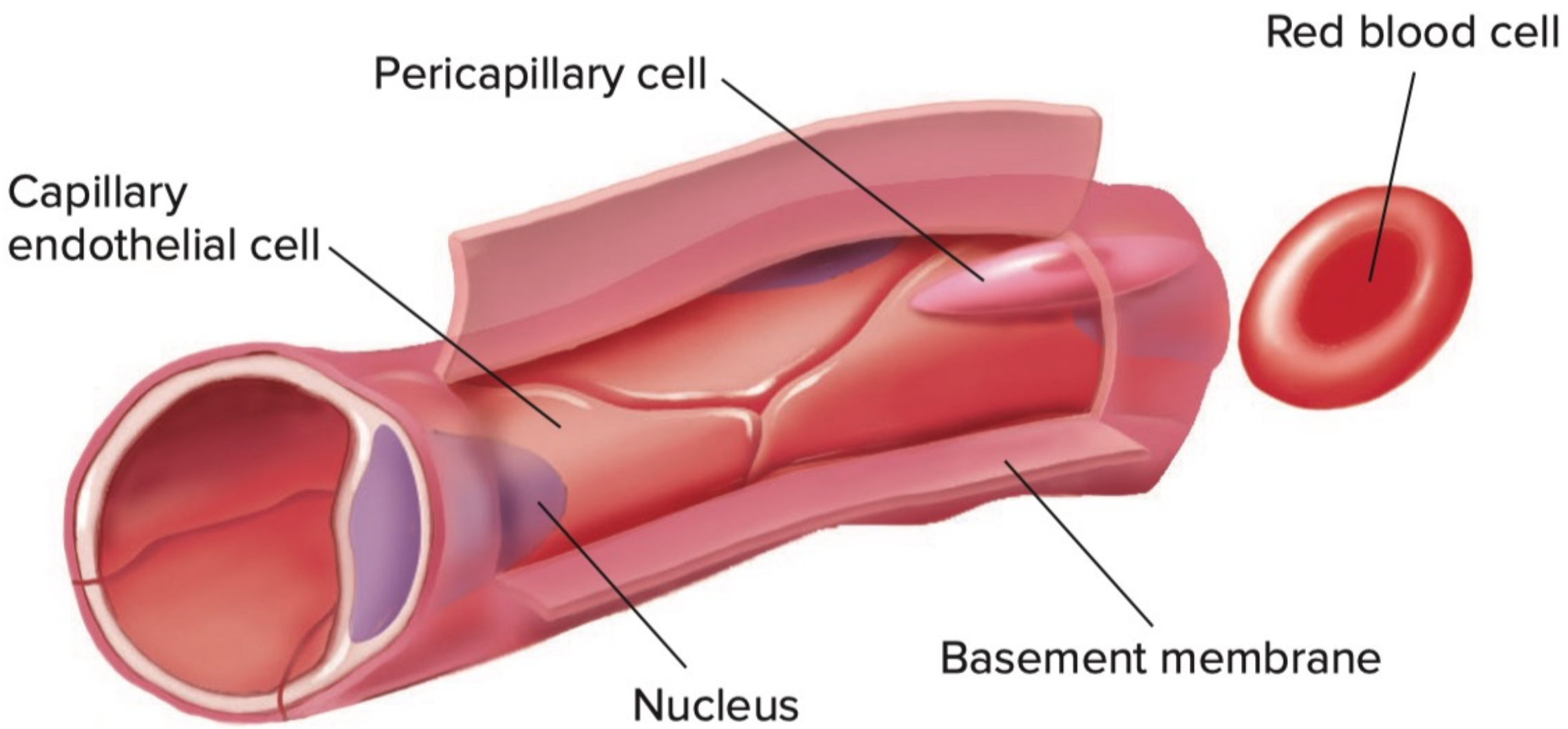


FIGURE 21.4 Capillary

Section of a capillary, showing that it is composed primarily of flattened endothelial cells.

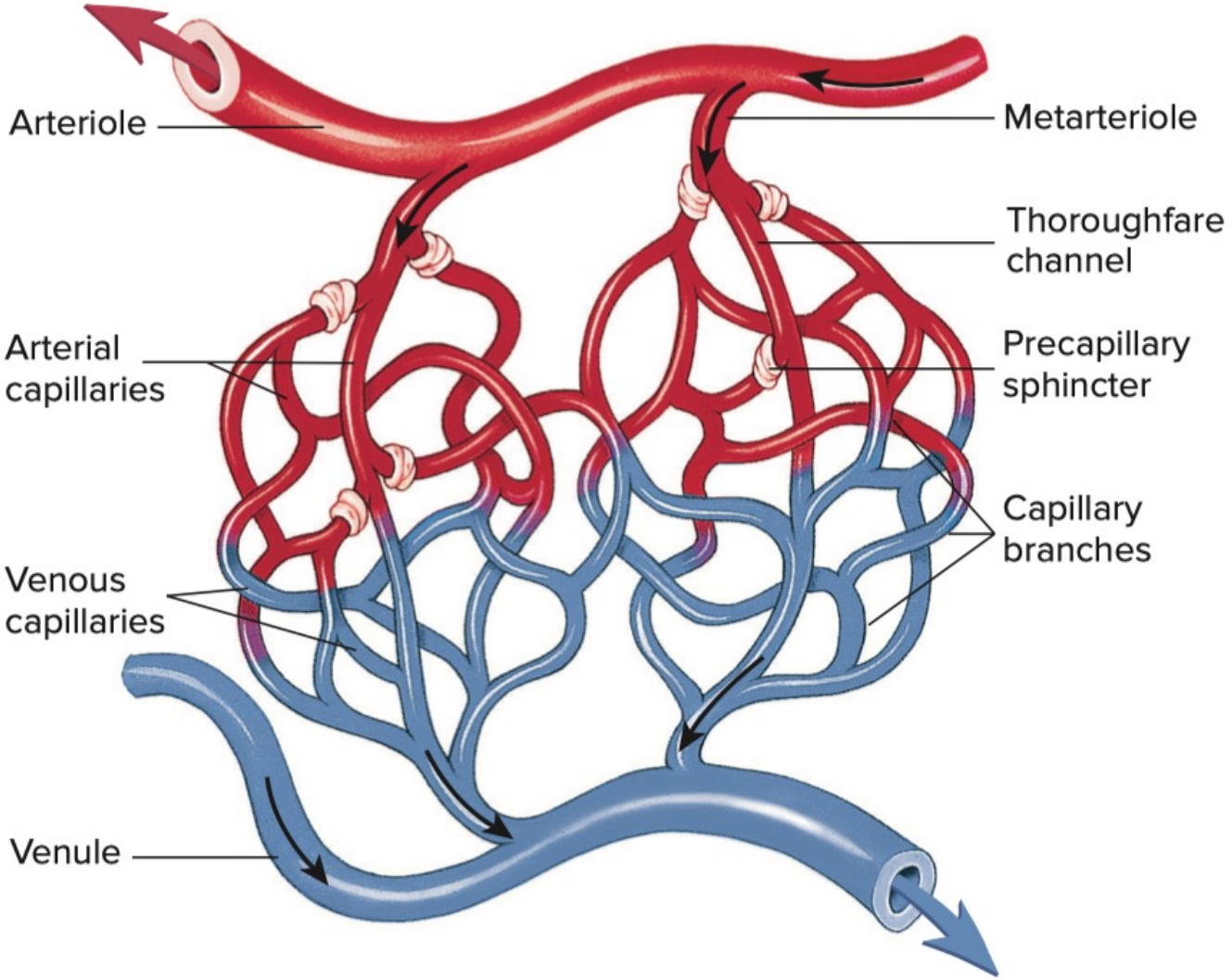


FIGURE 21.6 Capillary Network

A capillary network stems from an arteriole. Blood flows from the arteriole, through metarterioles, through the capillary network, to venules. Smooth muscle cells, called precapillary sphincters, regulate blood flow through the capillaries. Blood flow decreases when the precapillary sphincters constrict and increases when they dilate.

Permeability of Capillaries

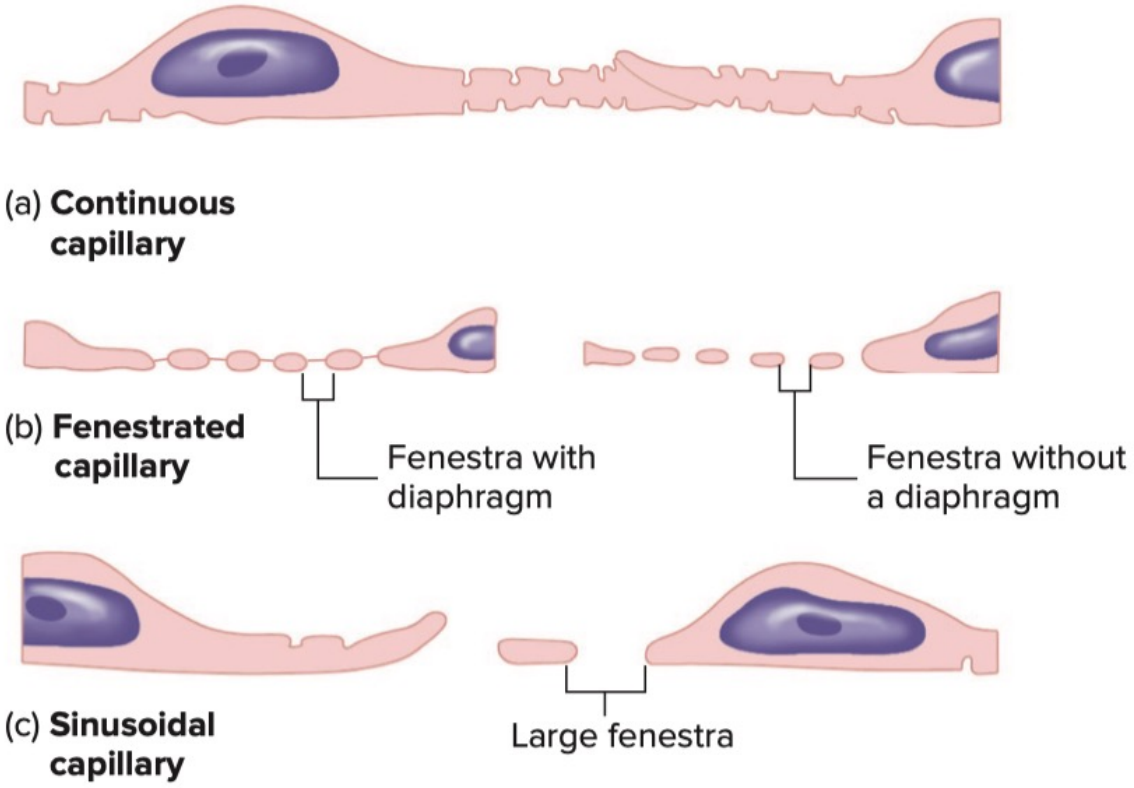
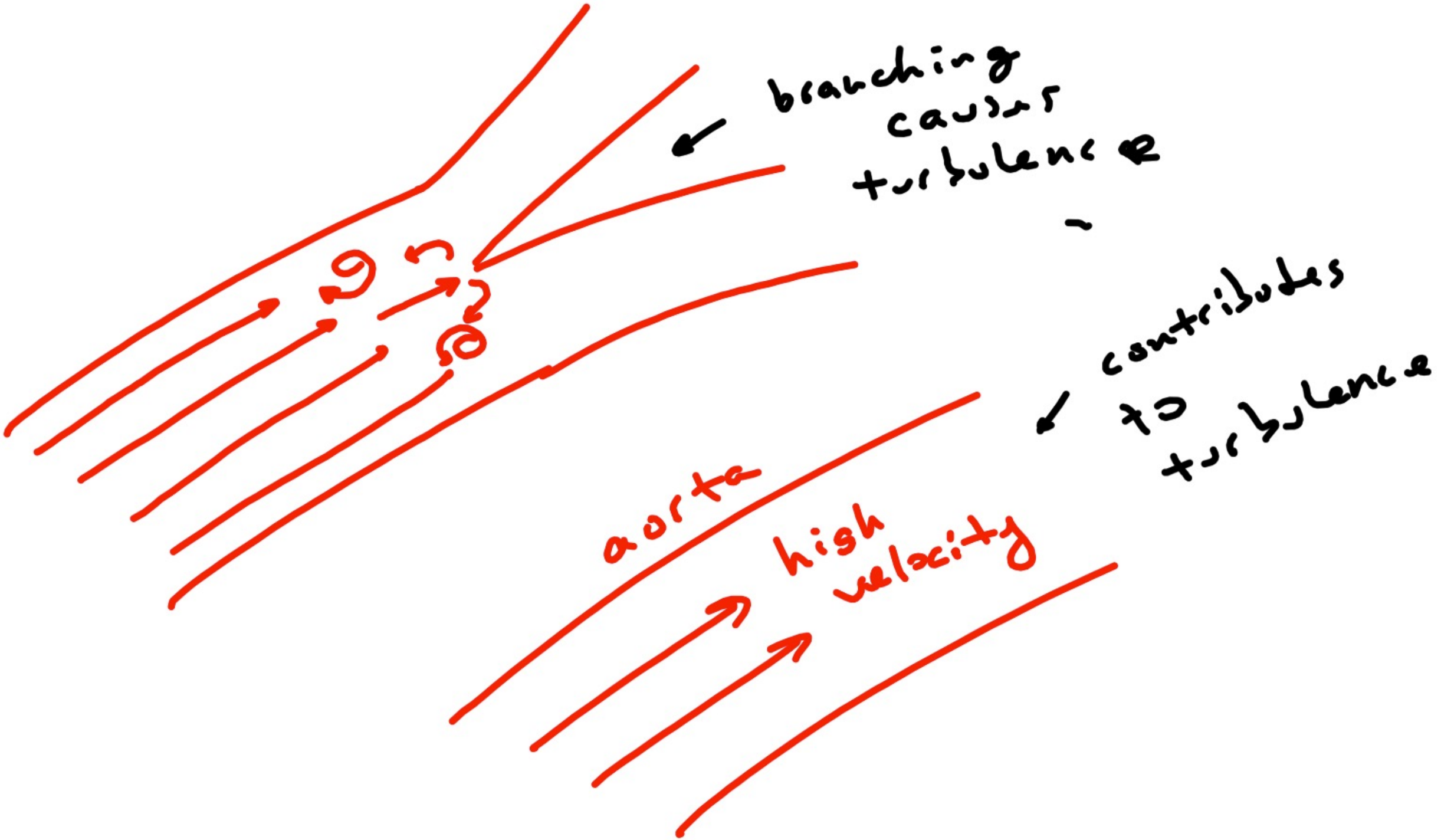


FIGURE 21.5 Structure of Capillary Walls

(*a*) Continuous capillaries have no gaps between endothelial cells and no fenestrae. They are common in muscle, nervous, and connective tissue. (*b*) Fenestrated capillaries have fenestrae 7–100 nm in diameter, covered by thin, porous diaphragms, which are not present in some capillaries. They are found in intestinal villi, ciliary processes of the eyes, choroid plexuses of the central nervous system, and glomeruli of the kidneys. (*c*) Sinusoidal capillaries have larger fenestrae without diaphragms and can have gaps between endothelial cells. They are found in endocrine glands, bone marrow, the liver, the spleen, and the lymphatic organs.

Blood Flow

- Two types of blood flow :
  - Laminar flow
    - Smooth blood flow occurring in parallel , concentric layers
    - Little resistance to blood flow
  - Turbulent flow
    - Rough , chaotic flow of blood
    - What can increase turbulence?
      - High velocity blood flow (biggest influence)
      - Thinner (lower viscosity) blood
      - Branching of arteries
  - Increases resistance to blood flow (harder for the heart to pump)
  - Can inflame and damage blood vessel walls
    - Can lead to thrombus formation and atherosclerosis
      - Atherosclerosis is hardening of arteries due to plaque formation


branching causes turbulence
contributes to turbulence
aorta
high velocity

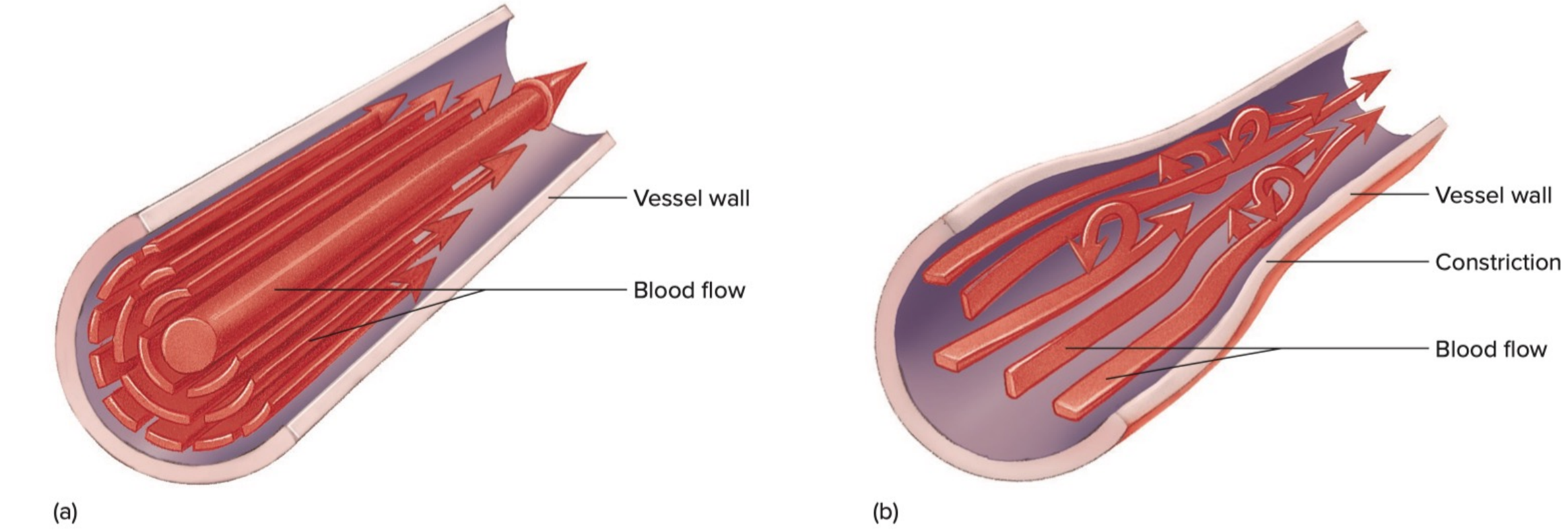


FIGURE 21.31 Laminar and Turbulent Flow

(*a*) In laminar flow, fluid flows in long, smooth-walled tubes as if it were composed of a large number of concentric layers. (*b*) Turbulent flow is caused by numerous small currents flowing crosswise or obliquely to the long axis of the vessel, resulting in flowing whorls and eddy currents.

Poiseuille's Law

Rate of blood flow in a blood vessel can be described by the following equation:

$$\text{Flow} = \frac{P_1 - P_2}{R}$$

P_1 = pressure at point one = upstream
P_2 = pressure at point two = downstream
R = resistance to flow

$$Resistance = \frac{8 * v * l}{\pi * r^4}$$

v = viscosity of a fluid (e.g. blood)
l = length of a tube (e.g. blood vessel)
r = radius of a tube (e.g. blood vessel)

$$Flow = \frac{(P_1 - P_2) * \pi * r^4}{8 * v * l}$$

Poiseuille's Law

Poiseuille's Law

- Blood pressure gradient
  - Blood pressure falls progressively as blood :
    - leaves the heart
      - flows through systemic and pulmonary circulations
        - then back to the heart
  - If blood pressure is too low , blood pressure gradient is too low
    - Blood flow to organs is impaired
      - Organ failure and death can occur

- Radius
  - Greatest effect on resistance to blood flow
  - Arterial radius affected by sympathetic tone and local factors
    - Vasoconstriction = decrease blood vessel radius
    - Vasodilation = increase blood vessel radius

Blood Pressure Gradient from Aorta to Right Atrium

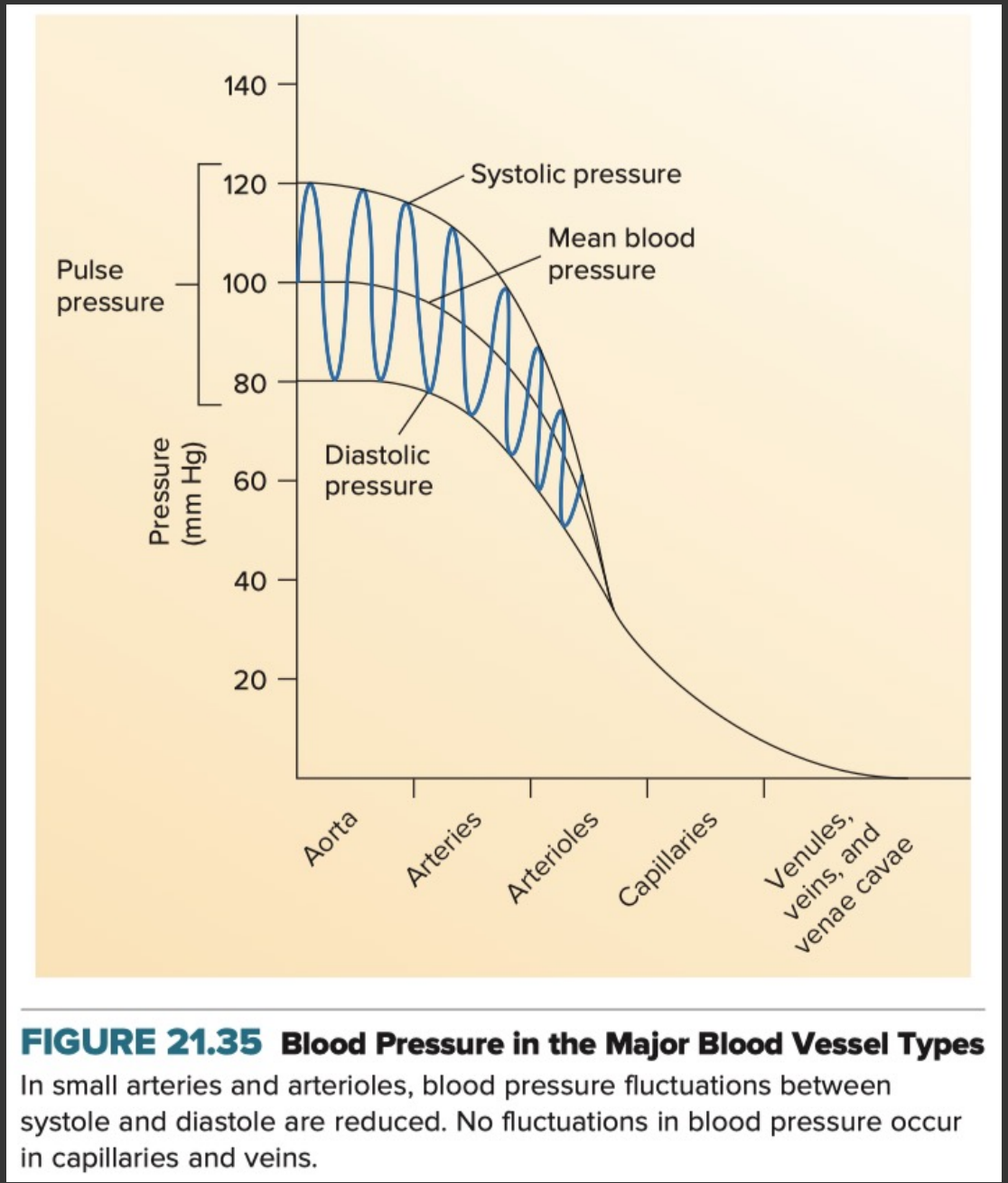


FIGURE 21.35 Blood Pressure in the Major Blood Vessel Types
In small arteries and arterioles, blood pressure fluctuations between systole and diastole are reduced. No fluctuations in blood pressure occur in capillaries and veins.

Effect of Radius on Blood Flow

decrease radius ➡ increase resistance

increase radius ➡ decrease resistance

decrease blood flow tremendously

increase blood flow tremendously

$$\text{Fluid Flow} = \frac{(P_1 - P_2)}{R}$$ ← pressure gradient

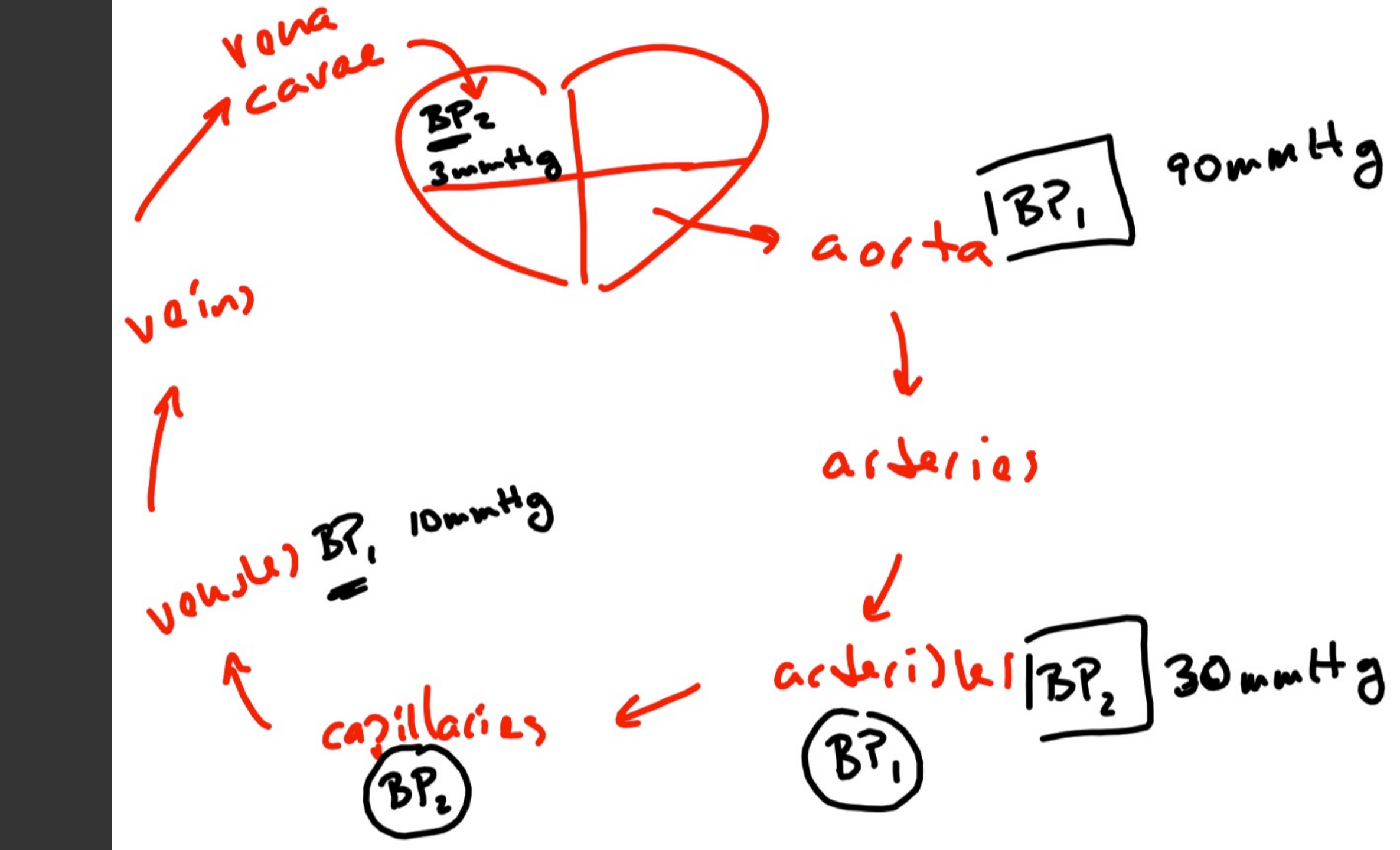
---

$$\text{Blood Flow} = \frac{(BP_1 - BP_2)}{R}$$

$$R \sim \frac{\nu}{r^4}$$

ν = viscosity of blood

r = radius of blood vessel


vena cavae
BP_2 = 3mmHg
aorta
BP_1
90mmHg
veins
arteries
venules BP_1 10mmHg
arterioles
BP_2
30mmHg
BP_1
capillaries
BP_2

↑ $(BP_1 - BP_2)$ → ↑ blood flow

↓ $(BP_1 - BP_2)$ → ↓ blood flow

If BP in arterial system is too low

↳ $(BP_1 - BP_2)$ is too small

↳ ↓↓ blood flow ← risk organ failure

↑ r → ↓ R → ↑ blood flow

↑ vasodilate

changes in radius have HUGE effects on blood flow

↓ vasoconstrict

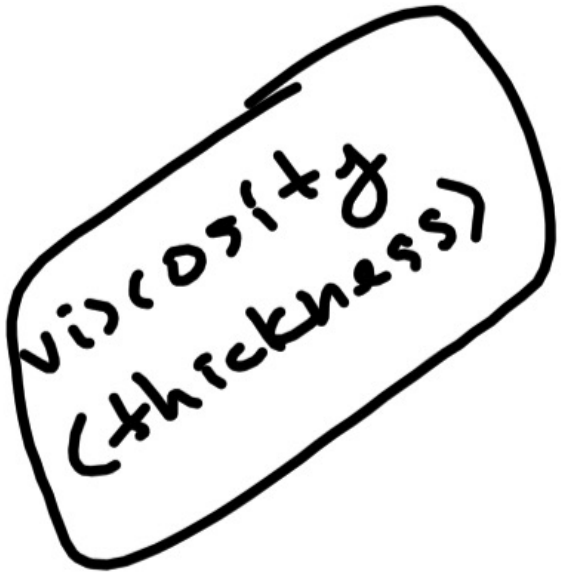
↓ r → ↑ R → ↓ blood flow

Poiseuille's Law

- Viscosity
  - Measure of the "thickness" of a fluid
  - Increase viscosity = increase resistance = decrease in blood flow
  - Factors that increase viscosity
    - Increase in hematocrit
      - Greatest influence on blood viscosity
    - Dehydration (concentrates substances in the blood)
    - High cholesterol and / or triglyceride levels
    - Stasis
      - Causes cell to cell and solute to cell adhesive interaction

↑ viscosity → ↑ R → ↓ blood flow

↓ viscosity → ↓ R → ↑ blood flow



* what affects blood viscosity the most?

↳ Hct

- ↑ Hct → ↑ viscosity
- ↓ Hct → ↓ viscosity

polycythemia

Control of Blood Flow

- Controlled via :
  - smooth muscle tone
- Vasomotor Centers
- Local Factors

Vasomotor Centers of the Brainstem

- Vasomotor centers in the brainstem
  - Output via the sympathetics (innervate vascular smooth muscle)
    - Stimulation of sympathetics
      - Vasoconstriction of skin and visceral blood vessels
        - Decreases blood flow to skin and viscera
      - Vasodilation of skeletal muscle blood vessels
        - Increases blood flow to skeletal muscle
    - Inhibition of sympathetics
      - Vasodilation of skin and visceral blood vessels
        - Increases blood flow to skin and viscera

Vasomotor Centers of the Brainstem

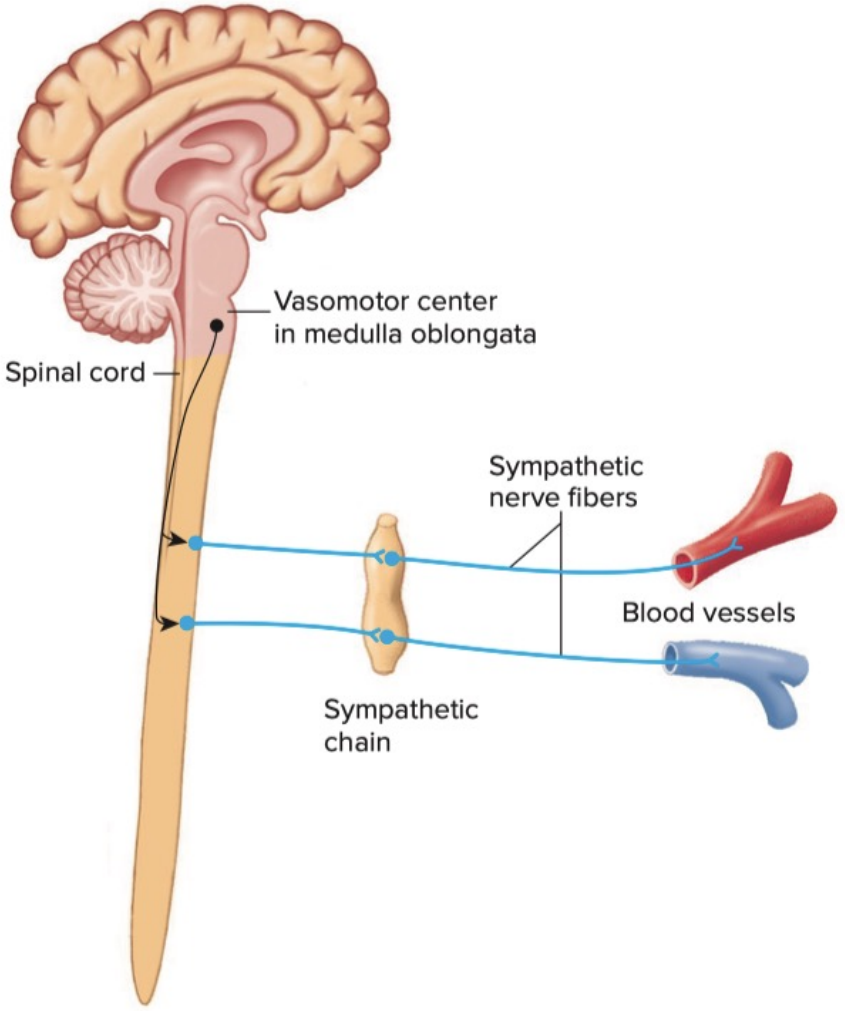
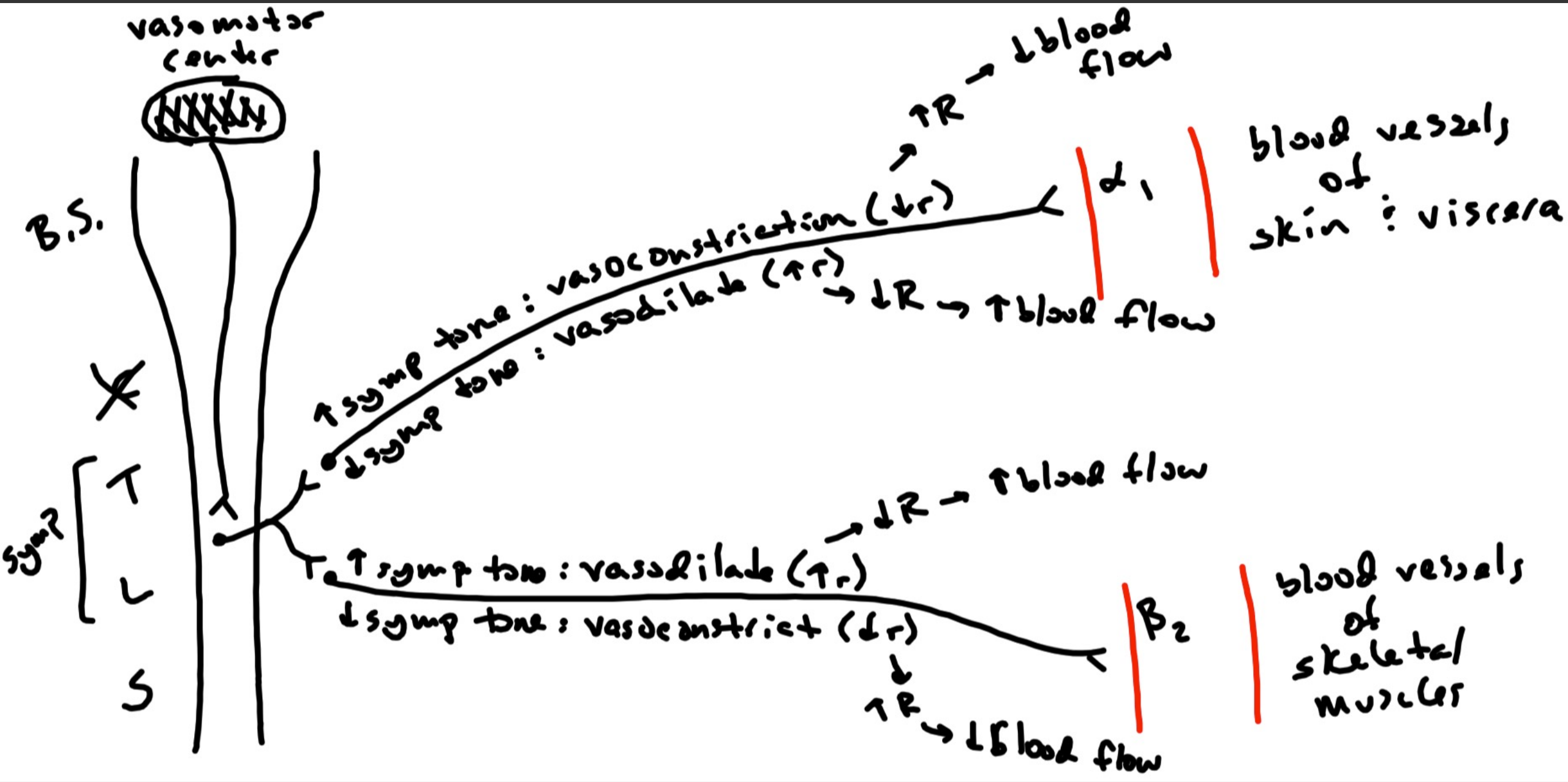
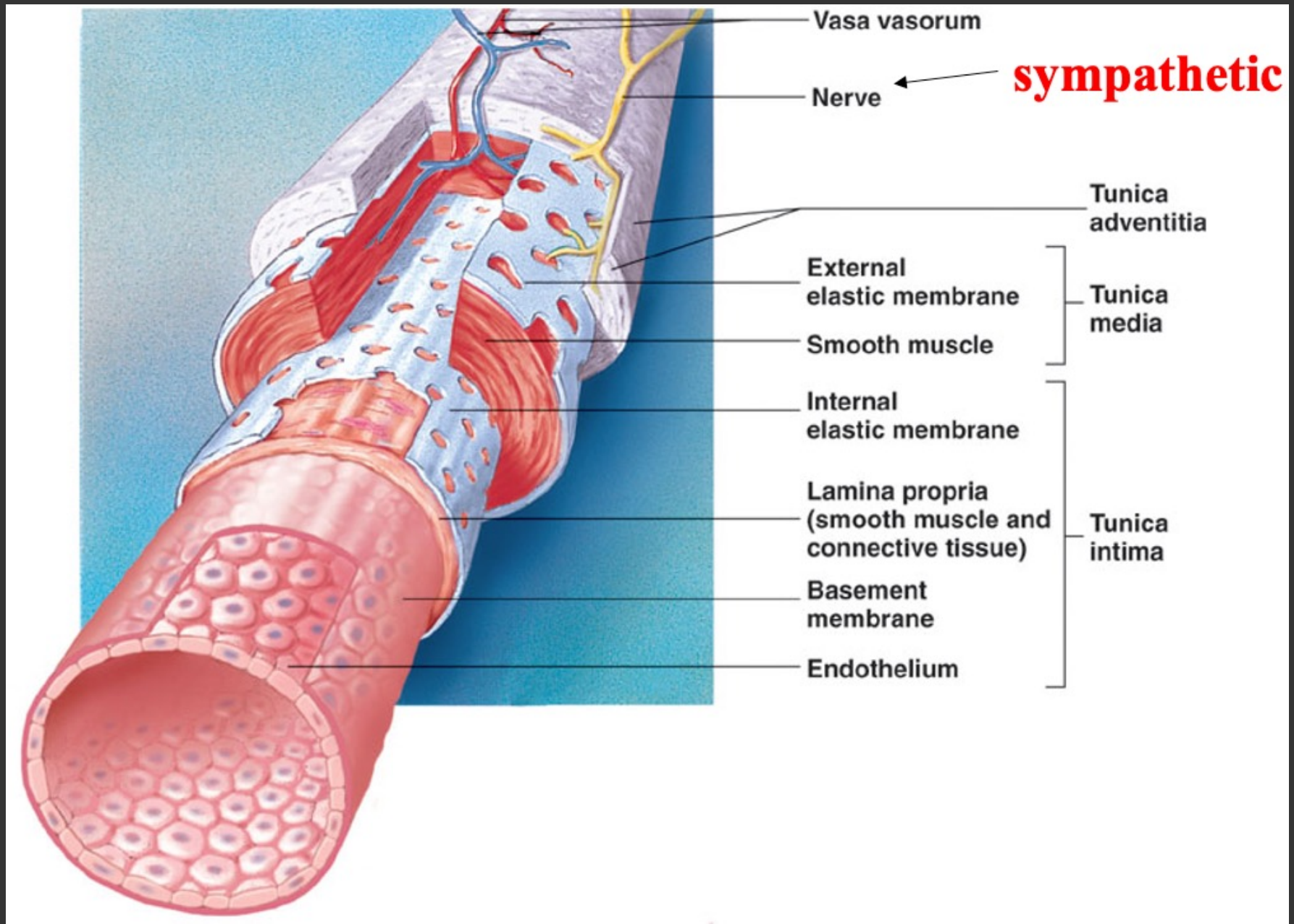


FIGURE 21.38 Nervous Regulation of Blood Vessels
Most blood vessels are innervated by sympathetic nerve fibers. The vasomotor center within the medulla oblongata plays a major role in regulating the frequency of action potentials in nerve fibers that innervate blood vessels.


vasomotor center
B.S.
sympT
sympL
S
↑symp tone : vasoconstriction (↓r)
↓symp tone : vasodilate (↑r)
↑R → ↓blood flow
↓R → ↑blood flow
α_1
blood vessels of skin & viscera
↑symp tone : vasodilate (↑r)
↓symp tone : vasoconstrict (↓r)
↓R → ↑blood flow
↑R → ↓blood flow
β_2
blood vessels of skeletal muscles

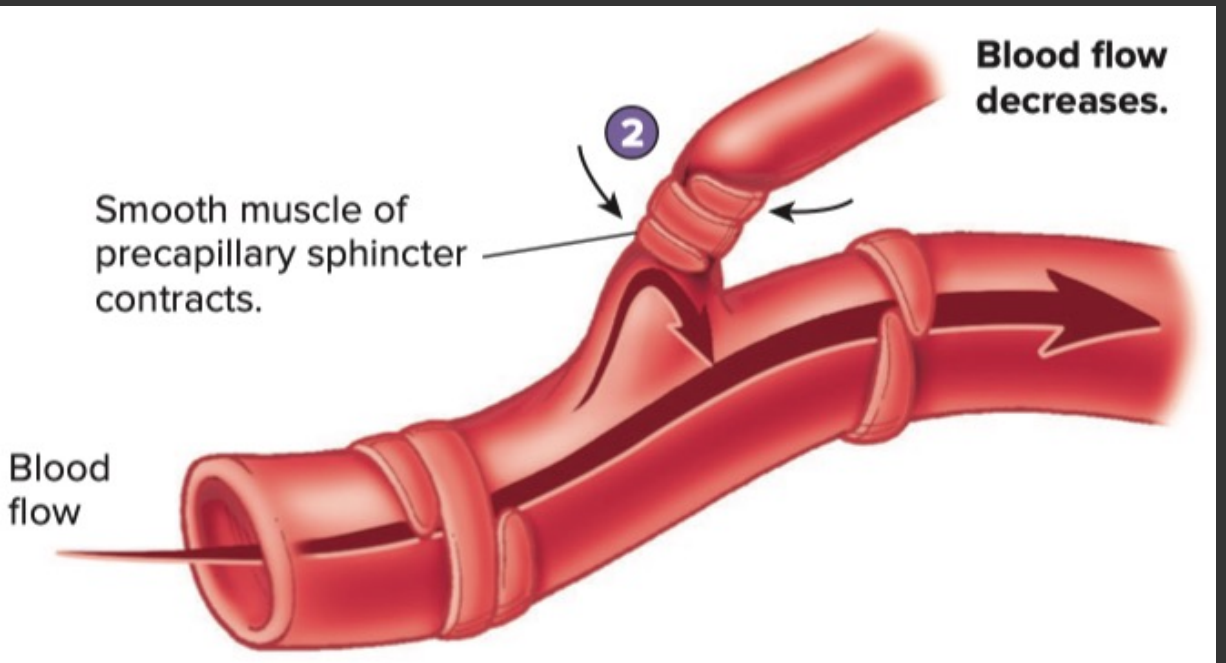
Sympathetic Innervation of Vascular Smooth Muscle

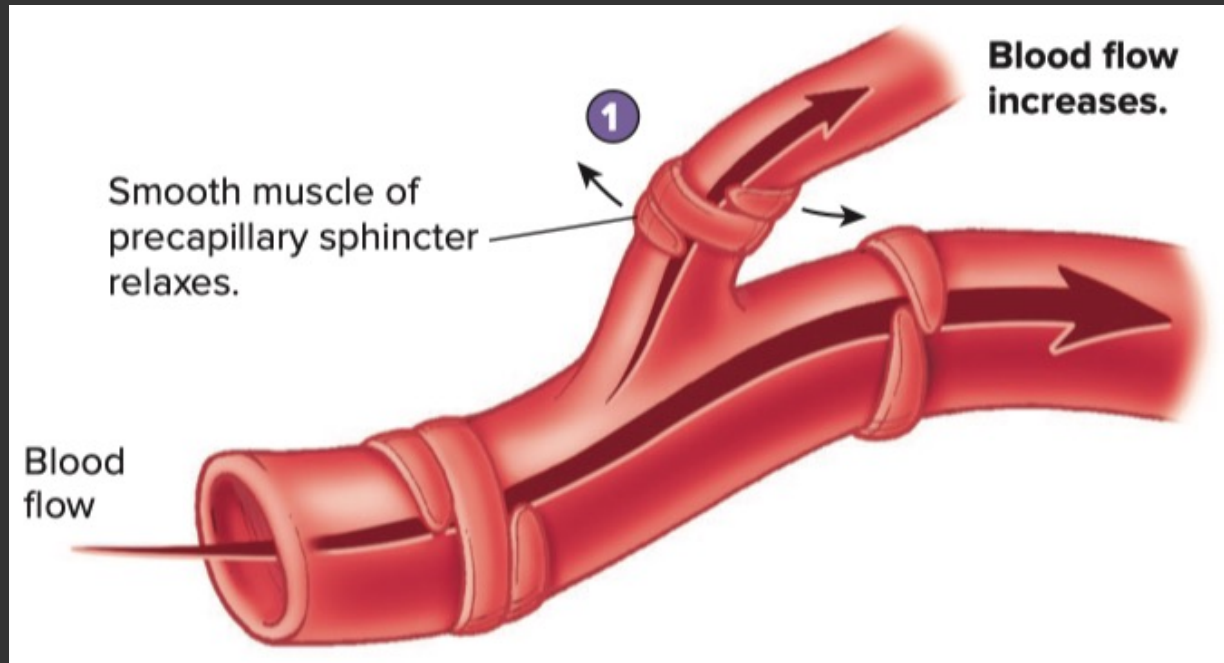


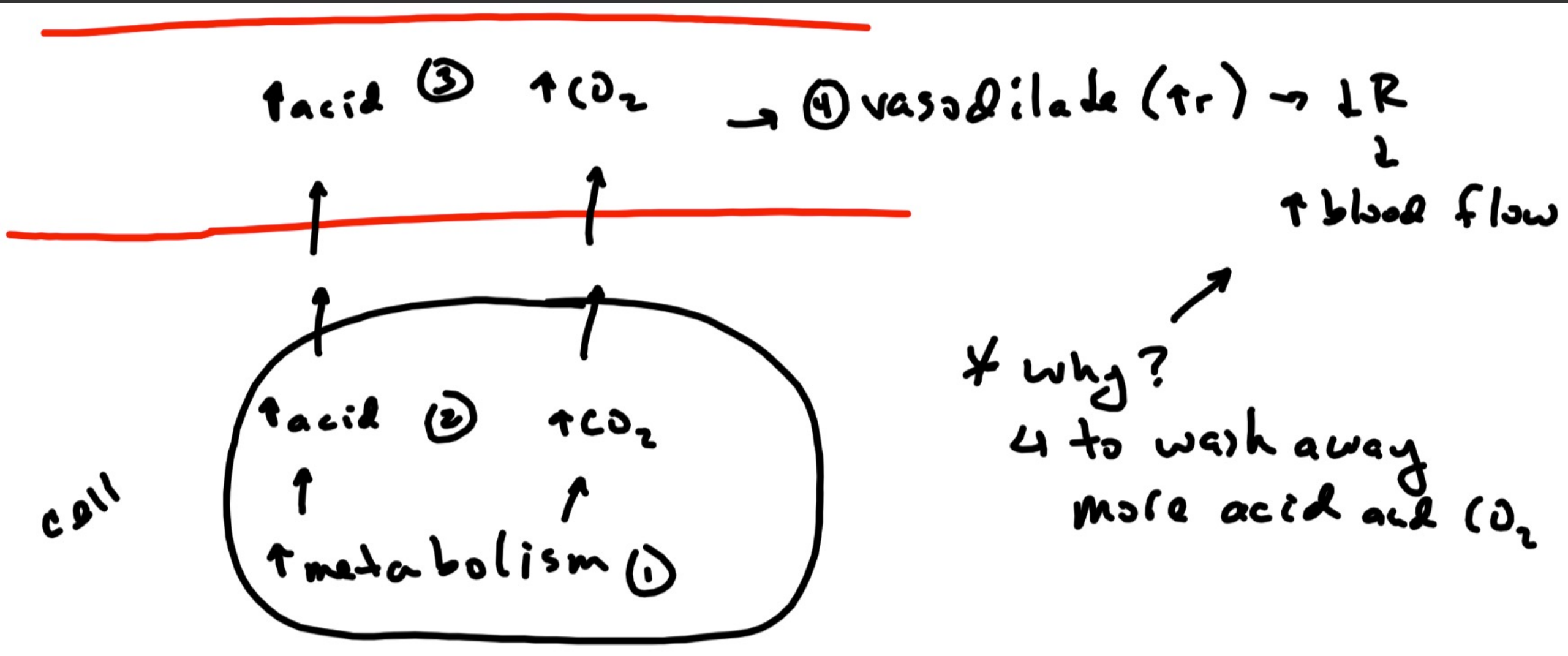
Effect of Radius on Blood Flow

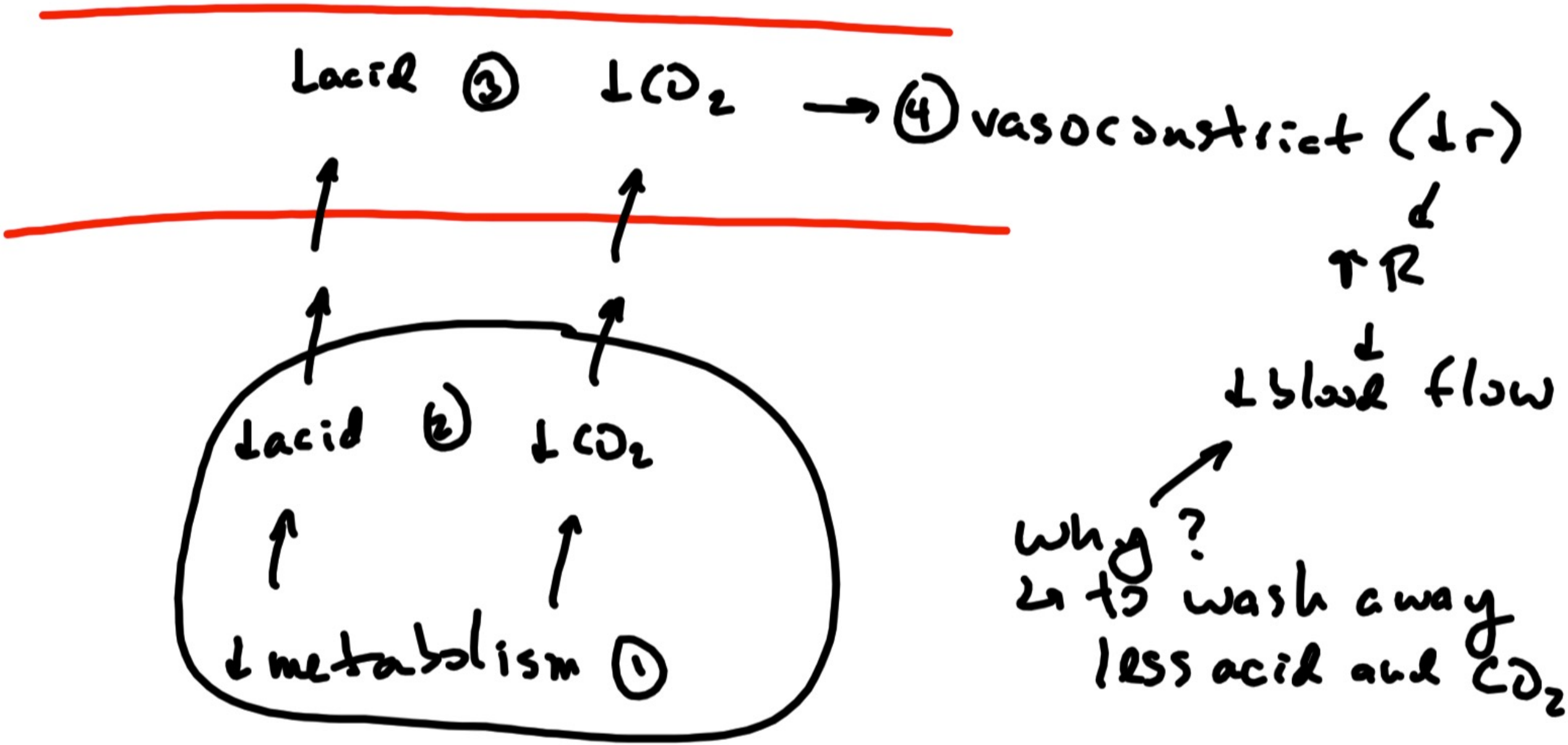
vasoconstriction

vasodilation





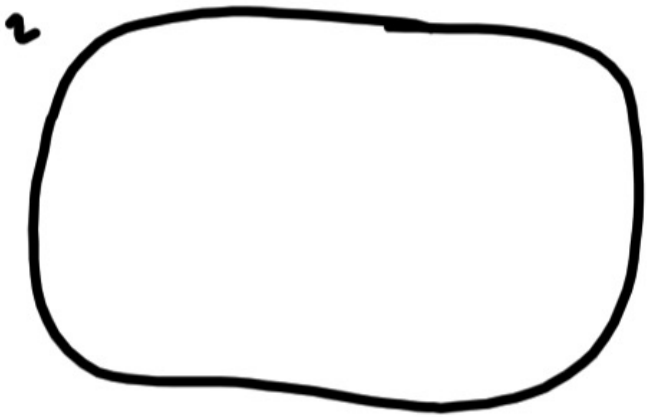

↑acid ③ ↑CO2 → ④ vasodilate (↑r) → ↓R
↓
↑blood flow
* why?
↳ to wash away
more acid and CO2
cell
↑acid ② ↑CO2
↑metabolism ①


Lacid ③ ↓CO2 → ④ vasoconstrict (↓r)
↓
↑R
↓
↓blood flow
↓acid ② ↓CO2
↓metabolism ①
why?
to wash away
less acid and CO2

Why?
↳ gets more O_2 to the cell
↓

② vasodilate (↑r) → ↓R_2
↑blood flow

① ↓O_2

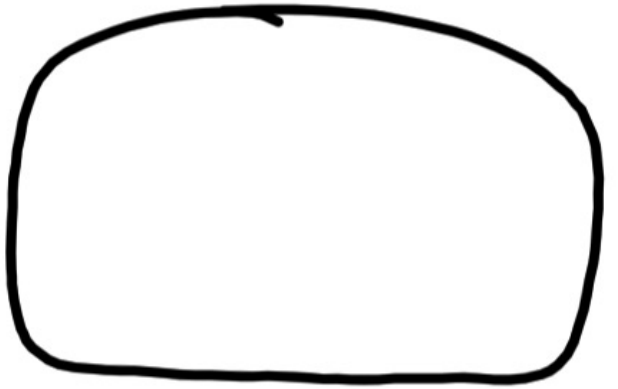


Why?
↳ blood (O_2) goes to other places that need it more →

↓blood flow
↑

② vasoconstrict (↓r) → ↑R

① ↑O_2



Local Factors

- Cause : smooth muscle relaxation (vasodilation) → increase blood flow
  - Nitric oxide
  - Histamine (part of inflammatory response and allergic reactions) (Basophil)
  - Decrease in pH
  - Increase in CO_2
  - Decrease in O_2

- Cause : smooth muscle contraction (vasoconstriction) → decrease blood flow
  - Thromboxane
  - Antidiuretic hormone / Vasopressin
  - Increase in pH
  - Decrease in CO_2
  - Increase in O_2

Blood Pressure

- Measure of the force that blood exerts against blood vessel walls
- Measure in millimeters of mercury (mm Hg)
- Systolic pressure: blood pressure during systole (i.e. the “top” number)
- Diastolic pressure: blood pressure during diastole (i.e. the “bottom” number)
- Pulse pressure: difference between systolic pressure and diastolic pressure

* systolic P : pressure during systole

* diastolic P : pressure during diastole

$$BP = \frac{110\ mmHg}{70\ mmHg}$$

* pulse pressure = (systolic P) - (diastolic P)

= 110mmHg - 70 mmHg

= 40 mmHg

Mean Arterial Pressure (MAP)

- Average blood pressure in the arterial circulation
- Normal range: 70 to 100 mmHg (although as low as 60 mmHg is ok)
- Weighted average: heart spends ⅓ of time in systole and ⅔ of time in diastole
- Calculation:

$$MAP = \tfrac{1}{3}\,(\,systolic\ pressure\,) \;+\; \tfrac{2}{3}\,(\,diastolic\ pressure\,)$$

OR

$$MAP = \frac{(\,systolic\ pressure\,) + 2\,(\,diastolic\ pressure\,)}{3}$$

Mean Arterial Pressure (MAP)

- Using a blood pressure of 110 mmHg / 70 mmHg as an example:

$$= \tfrac{1}{3} (110 \text{ mm Hg}) + \tfrac{2}{3} (70 \text{ mm Hg})$$

$$\approx 37 \text{ mm Hg} + 47 \text{ mm Hg}$$

$$\approx 84 \text{ mm Hg}$$

OR

$$= \frac{110 \text{ mm Hg} + 2 (70 \text{ mm Hg})}{3}$$

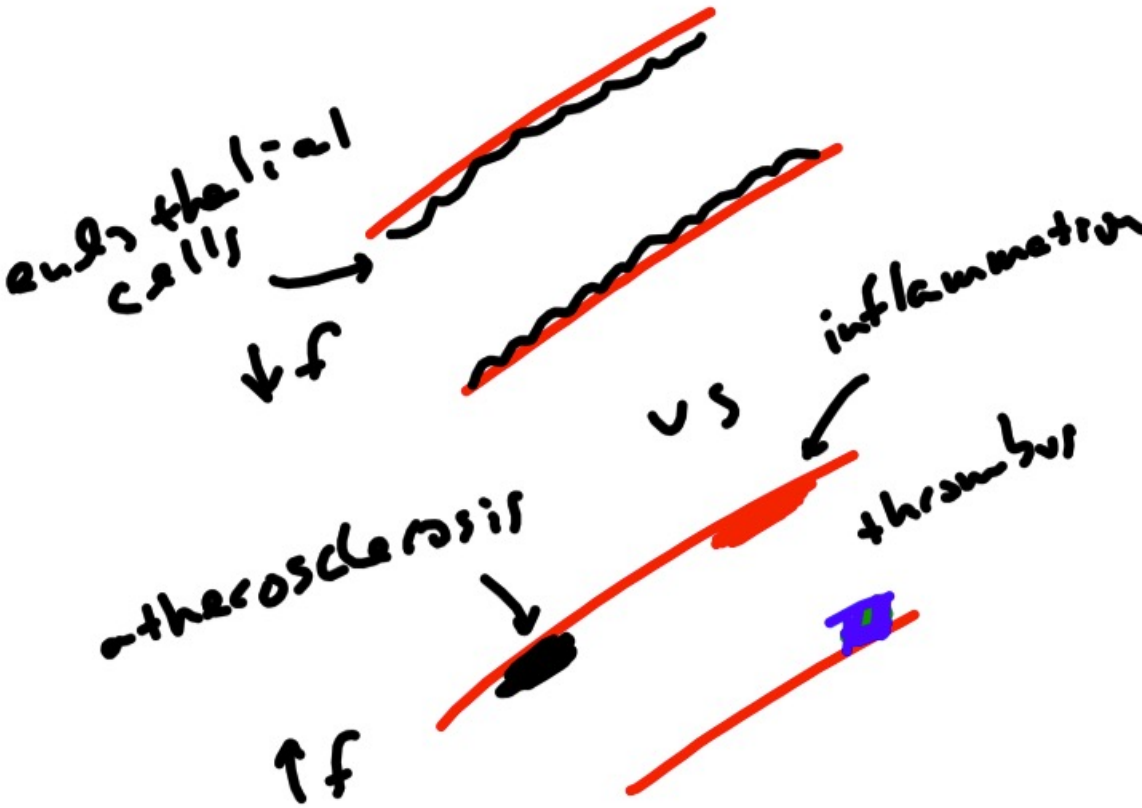
$$= \frac{250 \text{ mm Hg}}{3}$$

$$\approx 84 \text{ mm Hg}$$

(BP)

$$MAP = (CO) \times (TPR)$$

$$= (HR) \times (SV) \times (1/r) \times (v) \times (f)$$



Blood Pressure Gradient from Aorta to Right Atrium

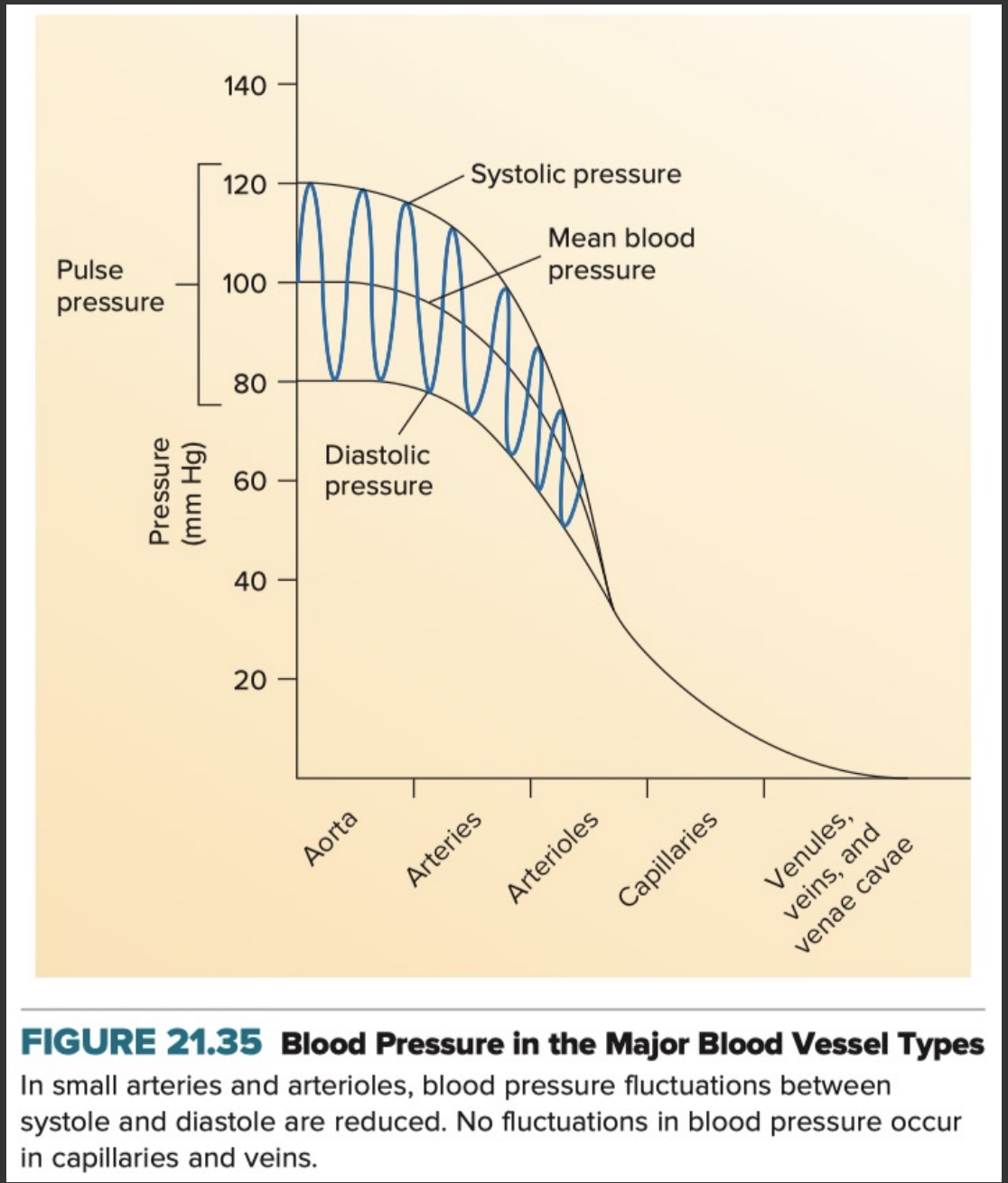


FIGURE 21.35 Blood Pressure in the Major Blood Vessel Types
In small arteries and arterioles, blood pressure fluctuations between systole and diastole are reduced. No fluctuations in blood pressure occur in capillaries and veins.

Mean Arterial Pressure (MAP)

- Also calculated using the following equation :
  - MAP = (Cardiac Output) x (Total Peripheral Resistance)
- Cardiac Output: product of stroke volume and heart rate
- Total Peripheral Resistance: opposing force to blood flow
- Factors affecting Total Peripheral Resistance (TPR) :
  - Arterial radius
    - Decrease radius = increase TPR
    - Increase radius = decrease TPR
  - Viscosity
    - Increase viscosity = increase TPR
    - Decrease viscosity = decrease TPR
  - Friction
    - Increase friction = increase TPR
    - Decrease friction = decrease TPR

Mean Arterial Pressure (MAP)

What increases BP	What decreases BP
Increase CO	Decrease CO
Increase SV	Decrease SV
Increase HR	Decrease HR
Increase TPR	Decrease TPR
Decrease radius	Increase radius
Increase viscosity	Decrease viscosity
Increase friction	Decrease friction

Regulation of Blood Pressure

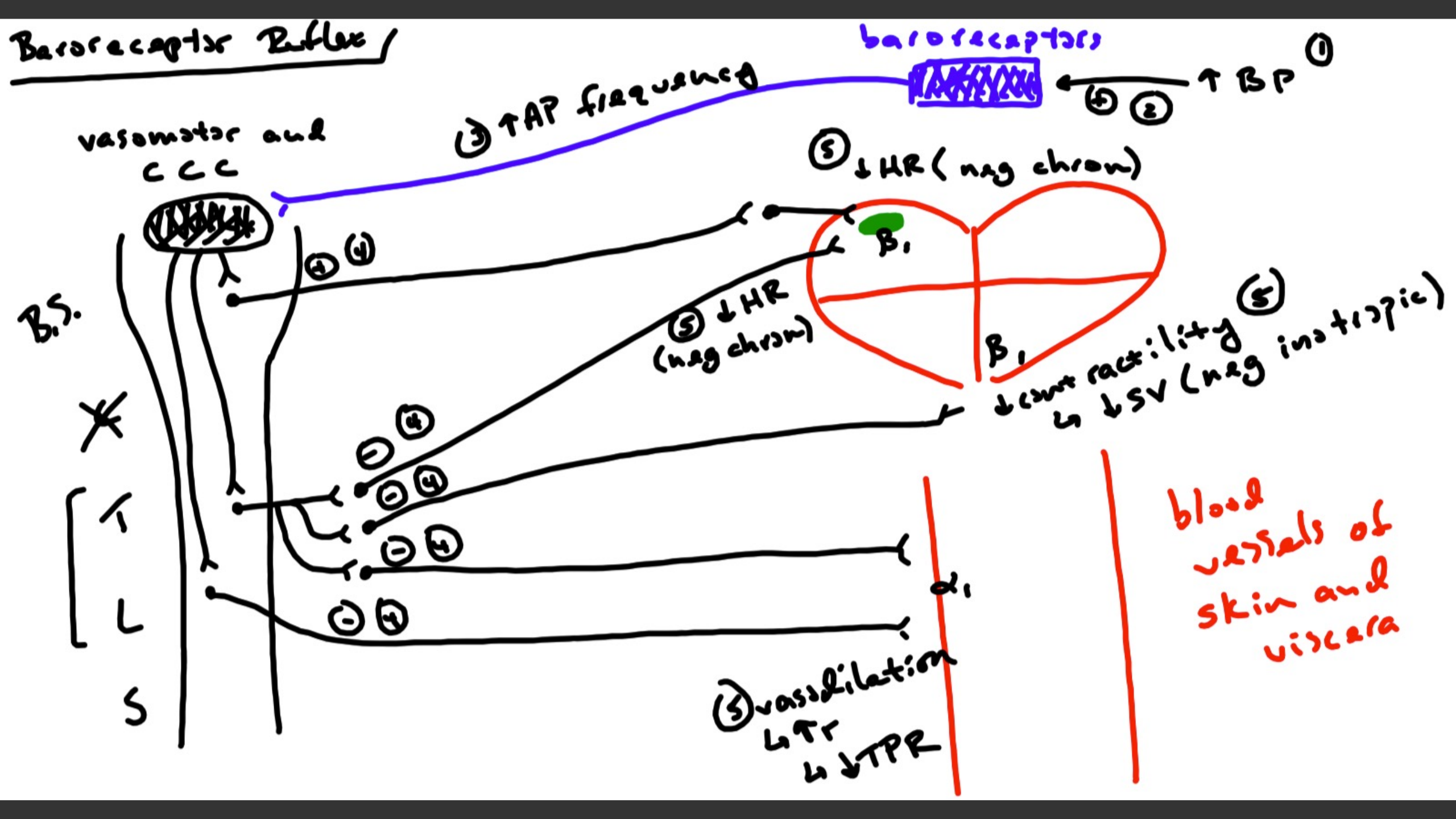
- Baroreceptor Reflex
- Renin-Angiotensin-Aldosterone System
- Atrial Natriuretic Hormone
- Antidiuretic Hormone (ADH) / vAsoPREssiN
- Epinephrine and Norepinephrine

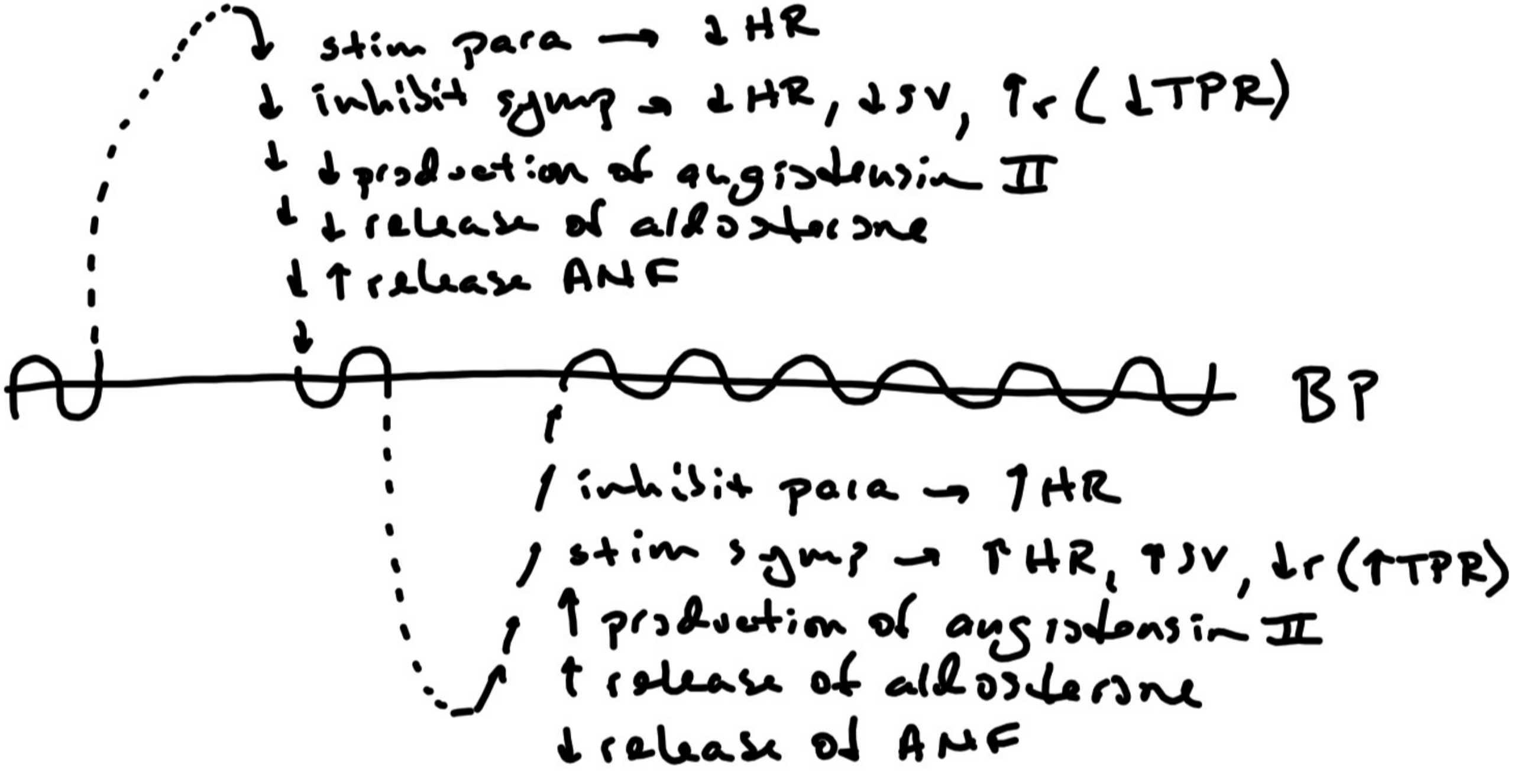
Regulation of Blood Pressure – Baroreceptor Reflex

- Most important short-term (second to second) regulatory mechanism
- Baroreceptors monitor blood pressure
  - Influence vasomotor center and cardiac control center

- With an increase in blood pressure :
  - Baroreceptors stimulated ⟵ proportional to the change in BP
    - Causes an increase in action potential frequency
      - Interpreted by vasomotor / cardiac control centers
        - Stimulates parasympathetics
          - Decreases Heart Rate
        - Inhibits sympathetics
          - Decreases heart rate
          - Decreases heart contractility
          - Vasodilation to skin and viscera
- With a decrease in blood pressure :
  - Baroreceptors inhibited ⟵ proportional to the change in BP
    - Causes a decrease in action potential frequency
      - Interpreted by vasomotor / cardiac control centers
        - Inhibits parasympathetics to increase BP
          - Increases heart rate
        - Stimulates sympathetics to increase BP
          - Increases heart rate
          - Increases heart contractility
          - Vasoconstriction to skin and viscera

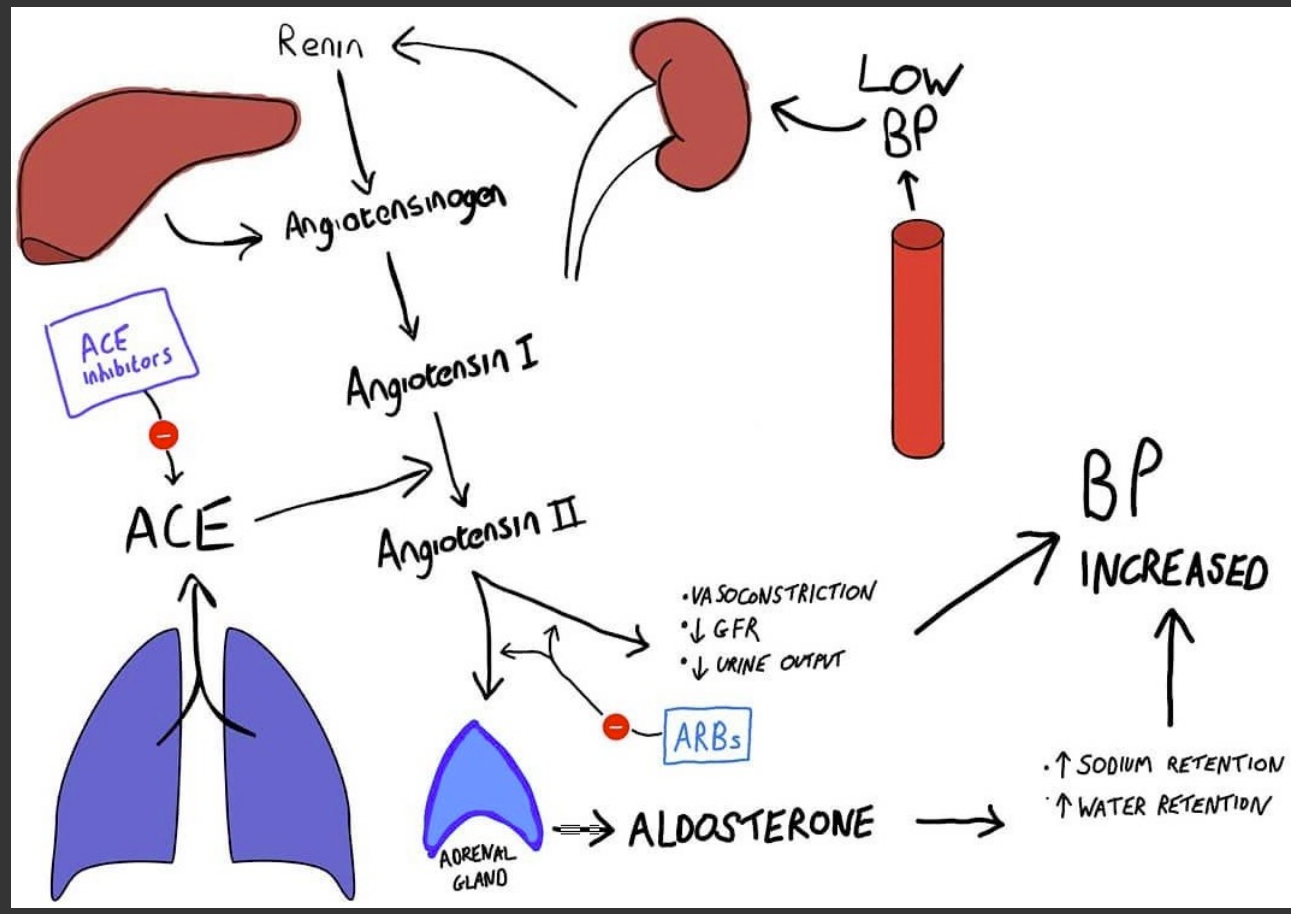
Baroreceptor Reflex

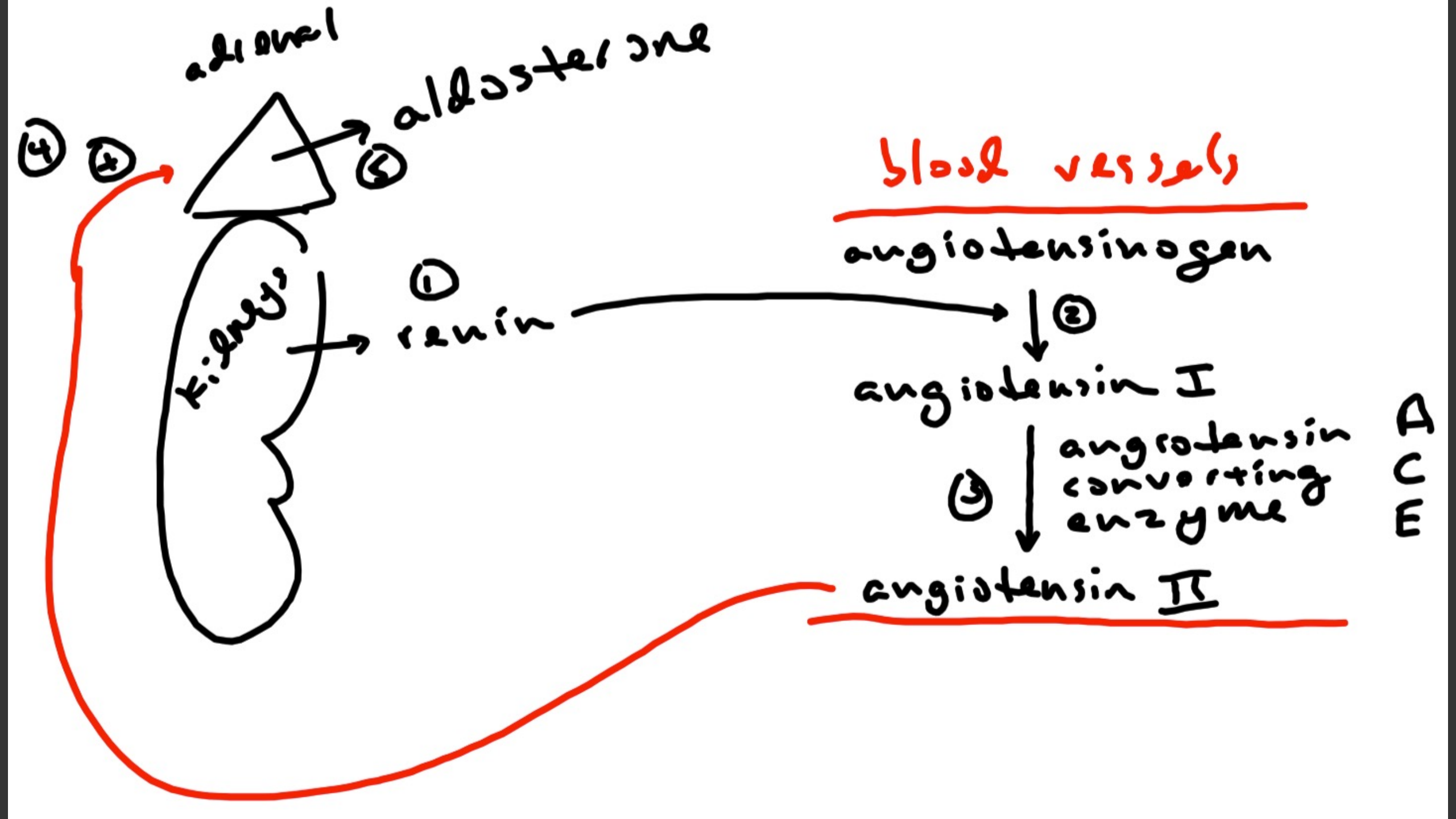



stim para → ↓HR
↓ inhibit symp → ↓HR, ↓SV, ↑r (↓TPR)
↓ ↓production of angiotensin II
↓ ↓release of aldosterone
↓ ↑release ANF
BP
↑ inhibit para → ↑HR
↑ stim symp → ↑HR, ↑SV, ↓r (↑TPR)
↑ ↑production of angiotensin II
↑ ↑release of aldosterone
↑ release of ANF

Regulation of Blood Pressure – Renin-Angiotensin-Aldosterone System

- Most important long-term regulatory mechanism
- Angiotensinogen converted to angiotensin I via renin
  - Angiotensin I converted to angiotensin II
    - Conversion via angiotensin converting enzyme (ACE)
- Effects of angiotensin II
  - Vasoconstriction to skin and viscera
    - Increases blood pressure
  - Increases aldosterone release from the adrenal gland
    - Increases blood volume and thus blood pressure
  - Increases antiduretic hormone secretion by the pituitary gland
- With an increase in blood pressure
  - Decrease angiotensin II production / decrease aldosterone release
    - Decreases blood pressure towards normal
- With a decrease in blood pressure
  - Increase angiotensin II production / increase aldosterone release
    - Increases blood pressure towards normal




adrenal
aldosterone
blood vessels
kidneys
renin
angiotensinogen
angiotensin I
angiotensin converting enzyme
ACE
angiotensin II
1
2
3
4
5

* Angiotensin II
  - causes release aldosterone → ↑blood volume, vasoconstricts
  - causes vasoconstriction
  - causes release of ADH → ↑blood volume, vasoconstriction

---

* ↑ production of angiotensin II and ↑ release of aldosterone
  - ↑BP
* ↓ production of angiotensin II and ↓ release of aldosterone
  - ↓BP

Regulation of Blood Pressure – Natriuretic Factor / Peptide / Hormone

- Released by atria of the heart in response to high blood pressure
- Stimulates the kidneys to transport sodium out of the blood
  - Water follows sodium osmotically
    - Decreases blood volume ➡️ blood pressure towards normal

Emergency Situations – When Blood Pressure is Too Low (ie , Hypotension)

- Antidiuretic hormone (ADH) / Vasopressin
  - Increases blood pressure via:
    - Increased blood volume
    - Vasoconstriction

- Epinephrine and norepinephrine
  - Increases blood pressure via:
    - Increased heart rate
    - Increased heart contractility and thus increased stroke volume
    - Vasoconstriction

Factors That Affect Blood Pressure- Compliance

- Measurement of the stretchiness/stiffness of a structure
- Arterial wall distends (stretches) during systole
  - Dissipates the increased pressure during systole
- Arterial wall recoils (“snaps back”) during diastole
    - Increases pressure during diastole to help maintain blood flow
- Decrease in arterial compliance indicates the arteries are more stiff
  - Causes an increase in systolic blood pressure
  - Causes a decrease in diastolic blood pressure
  - Seen with arteriosclerosis: “hardening” of the arteries
- Stretch of arterial wall during systole allows for measurement of a pulse
  - Measured at surface arteries (e.g. radial and carotid)
  - Measurement of a pulse gives the heart rate

* Compliance of blood vessels
  - how "stretchy" ... how stiff a blood vessel is

* with normal compliance:
  - during systole:
    - arteries stretch
      - benefit:
        - decreases force against walls of blood vessel (LBP a little)
  - during diastole:
    - arteries recoil ("snap back") (TBP a little)
      - benefit
        - increase force against walls of blood vessels

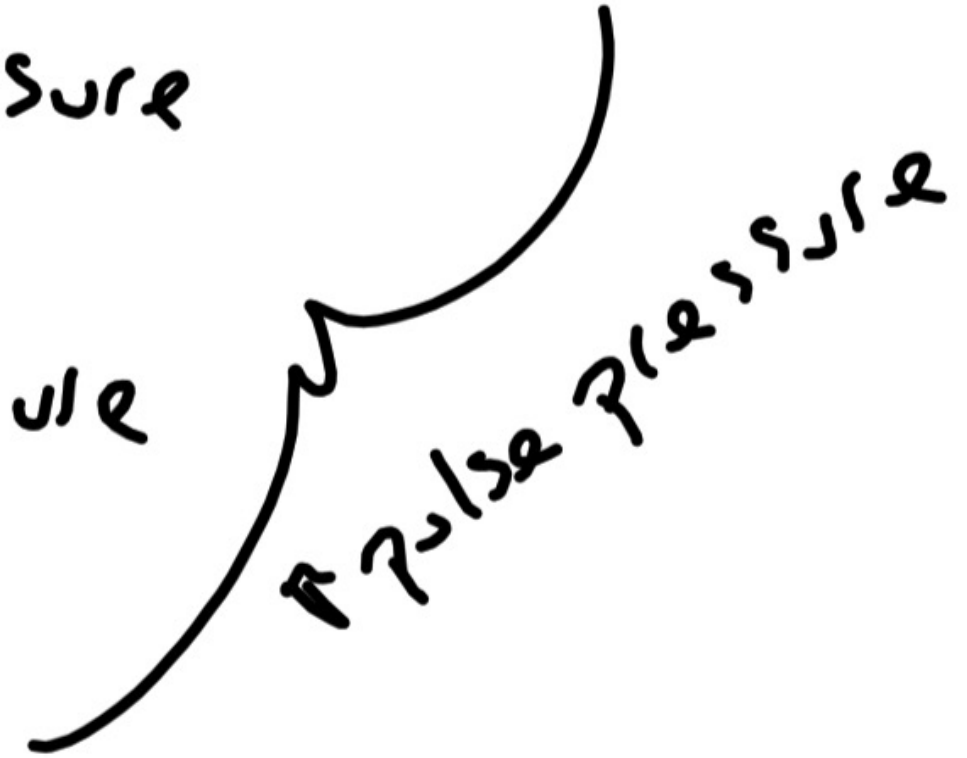
* ↓ compliance → too stiff

↳ during systole:

↳ ↑ systolic pressure

↳ during diastole:

↳ ↓ diastolic pressure



cause:

↳ arteriosclerosis ← hardening of arteries

Arterial Compliance and Pulse

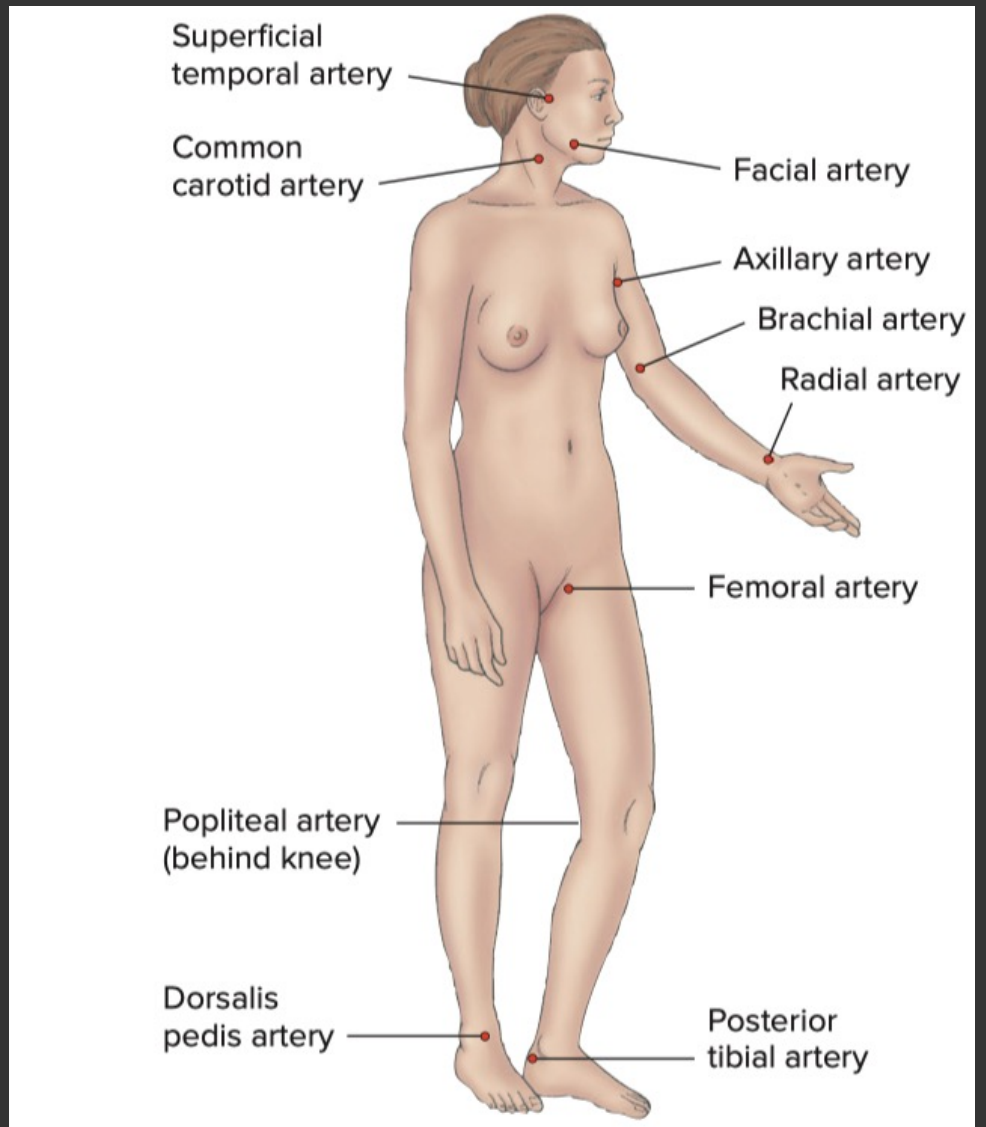


FIGURE 21.34 Major Points at Which the Pulse Can Be Monitored

Each pulse point is named after the artery on which it occurs.

* Weak pulse:

- ↓compliance (↑↑ stiff) ← arteriosclerosis
- ↓↓BP

Hypotension

- Abnormally low blood pressure
- Dangerous (and life threatening) for two main reasons :
  1) Blood pressure gradient is decreased
  2) Critical closing pressure is reached

Hypotension

1) Blood pressure gradient for blood flow is decreased :
   - Blood flow to tissues is decreased
     - Tissues can die with decreased blood flow
       - Organ failure and death is possible
2) Critical closing pressure is reached :
   - Blood pressure at which blood vessels collapse
     - Causes ischemia: no blood flow to tissues
       - Tissue dies without blood flow
         - Organ failure and death is possible
   - Laplace's Law
     - Force that prevents blood vessel collapse
     - Is equal to the product of blood pressure and diameter of the blood vessel
     - Force = P x D
       - P: blood pressure
       - D: diameter of blood vessel
   - In other words: blood pressure must be high enough to prevent the collapse of blood vessels

Circulatory Shock

- Severe decrease in blood pressure , which leads to inadequate blood flow
  - Large decrease in blood pressure gradient
  - Critical closing pressure reached

- Types of circulatory shock :
  - Hypovolemic shock – loss of blood volume decreases blood pressure
  - Vasodilatory shock – vasodilation decreases blood pressure
  - Cardiogenic shock – inadequate cardiac output decreases blood pressure

Reasons for Circulatory Shock

- Hemorrhagic shock (hypovolemic)
  - Bleeding that causes loss of blood volume
- Anaphylactic shock (vasodilatory)
  - Severe allergic response that causes massive release of inflammatory chemicals
    - Cause severe vasodilation
- Septic shock (hypovolemic , vasodilatory , and cardiogenic)
  - Develops from sepsis (inflammatory response to infection)
    - Bleeding caused by leaky capillaries
    - Vasodilation
    - Decreases heart rate and contractility
- Emotional shock (vasodilatory and cardiogenic)
  - Sudden emotional trauma
    - Causes strong parasympathetic stimulation
      - Decreases heart rate
    - Causes strong sympathetic inhibition
      - Vasodilation
      - Decreases heart rate and contractility
- Neurogenic shock (vasodilatory and cardiogenic)
  - Causes strong inhibition of the sympathetics
    - Vasodilation
    - Decreases heart rate and contractility

Response of the Body to Hypotension

- Increase heart rate and contractility (if the heart is not the cause)
  - Attempts to increase cardiac output
    - Increases blood pressure
- Vasoconstriction (if the blood vessels are not the cause)
  - Attempts to increase total peripheral resistance
    - Increases blood pressure

Treatments for Hypotension

- IV fluids and/or blood transfusion to increase blood volume
- Administration of pressors to increase blood pressure
  - Epinephrine (most powerful)
    - β-agonist
      - Increases contractility (i.e. SV) and heart rate
    - α-agonist
      - Vasoconstriction
  - Dopamine
    - β-agonist (at medium doses)
      - Increases contractility (i.e. SV) and heart rate
    - β-agonist and α-agonist (at high doses)
      - Increases contractility (i.e. SV) and heart rate
        - Vasoconstriction
  - Phenylephrine (neo synephrine)
    - α-agonist
      - Vasoconstriction
  - Vasopressin
    - Increases blood volume
    - α-agonist
      - Vasoconstriction

Hypertension

- Elevated (borderline / pre-hypertension no longer exists as categories)
  - Systolic pressure from 120 to 129 mmHg
  - Diastolic pressure not taken into account
- Stage 1 hypertension :
  - Systolic pressure from 130 to 139 mmHg

    and / or
  - Diastolic pressure from 80 to 89 mmHg
- Stage 2 hypertension :
  - Systolic pressure of 140 mmHg or above

    and / or
  - Diastolic pressure of 90 mmHg or above
- Hypertensive crisis :
  - Systolic pressure above 180 mmHg

    and / or
  - Diastolic pressure above 120 mmHg
- Essential / Primary hypertension :
  - Cause is unknown (majority of cases)
- Secondary hypertension :
  - Cause is known (e.g. renal disease)

Complications Due to Hypertension

- Heart failure
  - Heart must work harder to pump blood against a higher afterload
- Inflammation of blood vessels
  - Can lead to thrombi
  - Can lead to atherosclerosis
- Aneurysm
  - Blood vessels weaken and bulge from higher pressures
  - Rupture of aneurysm is life-threatening
- Ruptured blood vessels from higher pressures
  - Can lead to organ dysfunction
  - Can lead to blindness

* stage I hypertension

132*/72

126/86*

*134/*82

* stage II hypertension

148*/82

130/92*

*152/*96

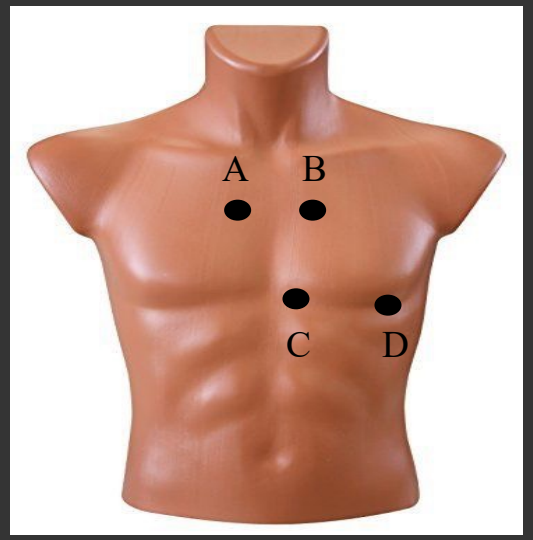
* Symptoms of hypertension:

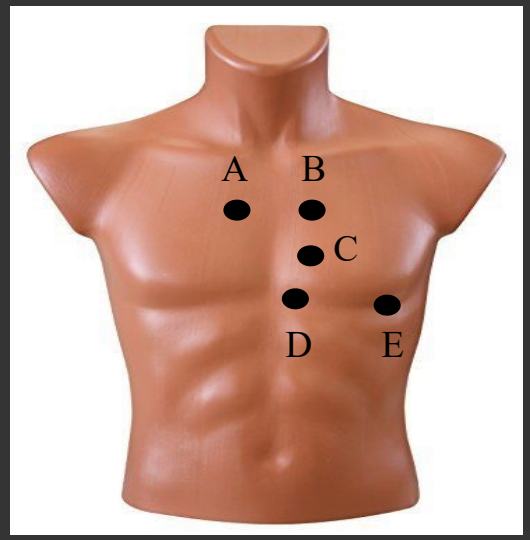
- There are none

"The Silent Killer"

Treatments for Hypertension

- **Change of lifestyle to get into better shape and lose weight**
  - e.g. 75% of overweight people that lose weight are cured
- **β-blockers**
  - Decrease contractility (i.e. SV) and heart rate
- **α-blockers**
  - Decrease peripheral resistance
    - Vasodilation
- **Calcium channel blockers**
  - Inhibit calcium channels of conduction system and cardiac muscle
    - Decrease contractility (i.e. SV) and heart rate
  - Inhibit calcium channels of vascular smooth muscle
    - Decrease peripheral resistance
      - Vasodilation
- **Diuretics**
  - Cause the excretion of fluids from the body
    - Decrease blood volume
- **ACE inhibitors**
  - Inhibit production of angiotensin II
  - Decrease peripheral resistance
    - Vasodilation
  - Decrease blood volume (via inhibition of aldosterone)
- **Angiotensin II receptor blockers**
  - Decrease peripheral resistance
    - Vasodilation
  - Decrease blood volume (via inhibition of aldosterone)
- **Renin inhibitors**
  - Decrease peripheral resistance
    - Vasodilation
  - Decrease blood volume (via inhibition of aldosterone)


A
B
C
D


A
B
C
D
E

Sinoatrial Node- Action Potential- Pacemaker

1.) Pacemaker Potential

.) HCN **Sodium** Leak Channels are Open

.) **Sodium** Enters ∴ more positive , less negative

.) Voltage-Gated **Potassium** Channels Close

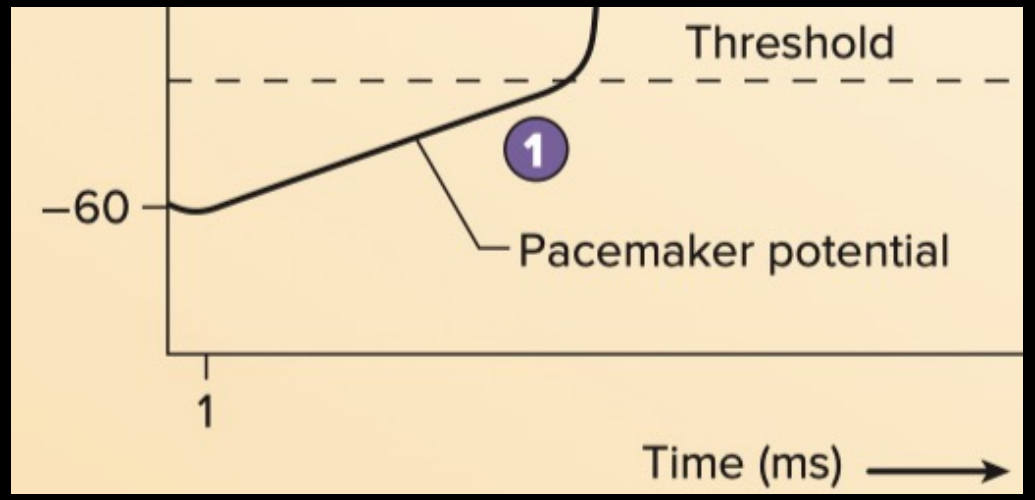
.) Less **Potassium** ∴ more negative , less positive

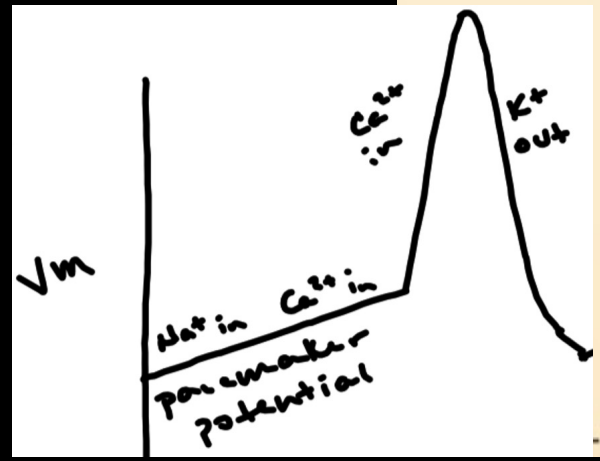
.) Voltage-Gated **Calcium** Channels Open

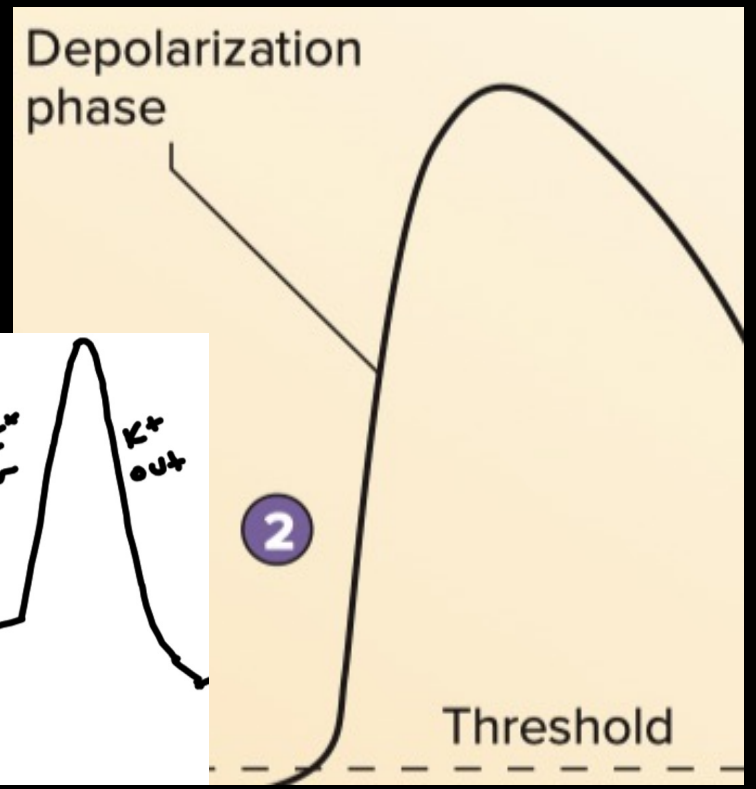
2.) Depolarization Phase

.) **Calcium** Enters

.) Voltage-Gated **Potassium** Channels Close







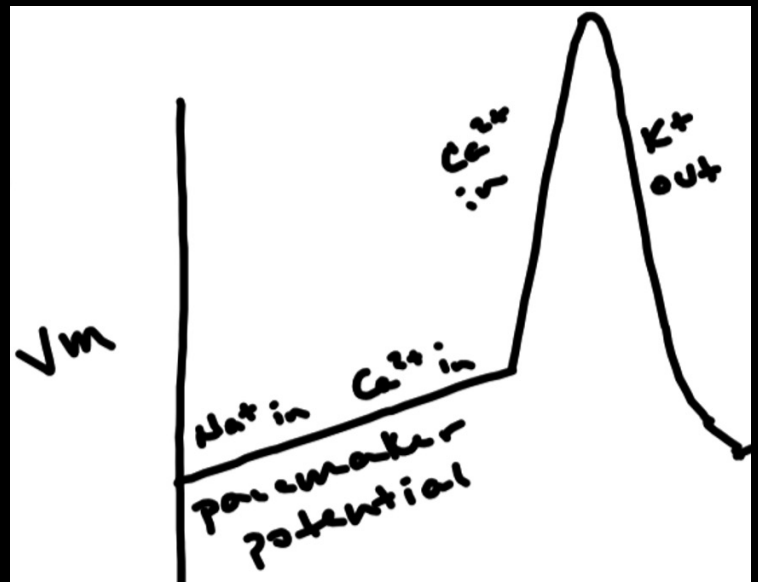
Sinoatrial Node- Action Potential- Pacemaker

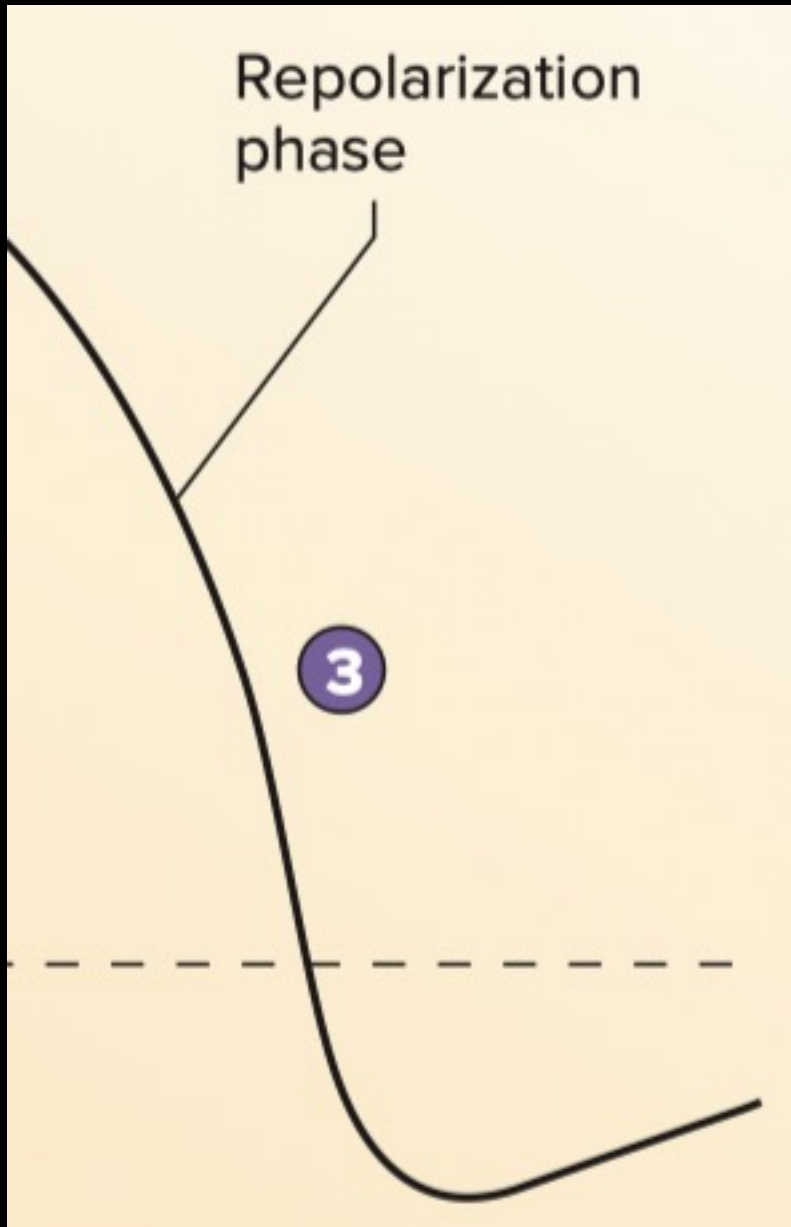
3.) Repolarization Phase

.) Voltage-Gated **Calcium** Channels Close

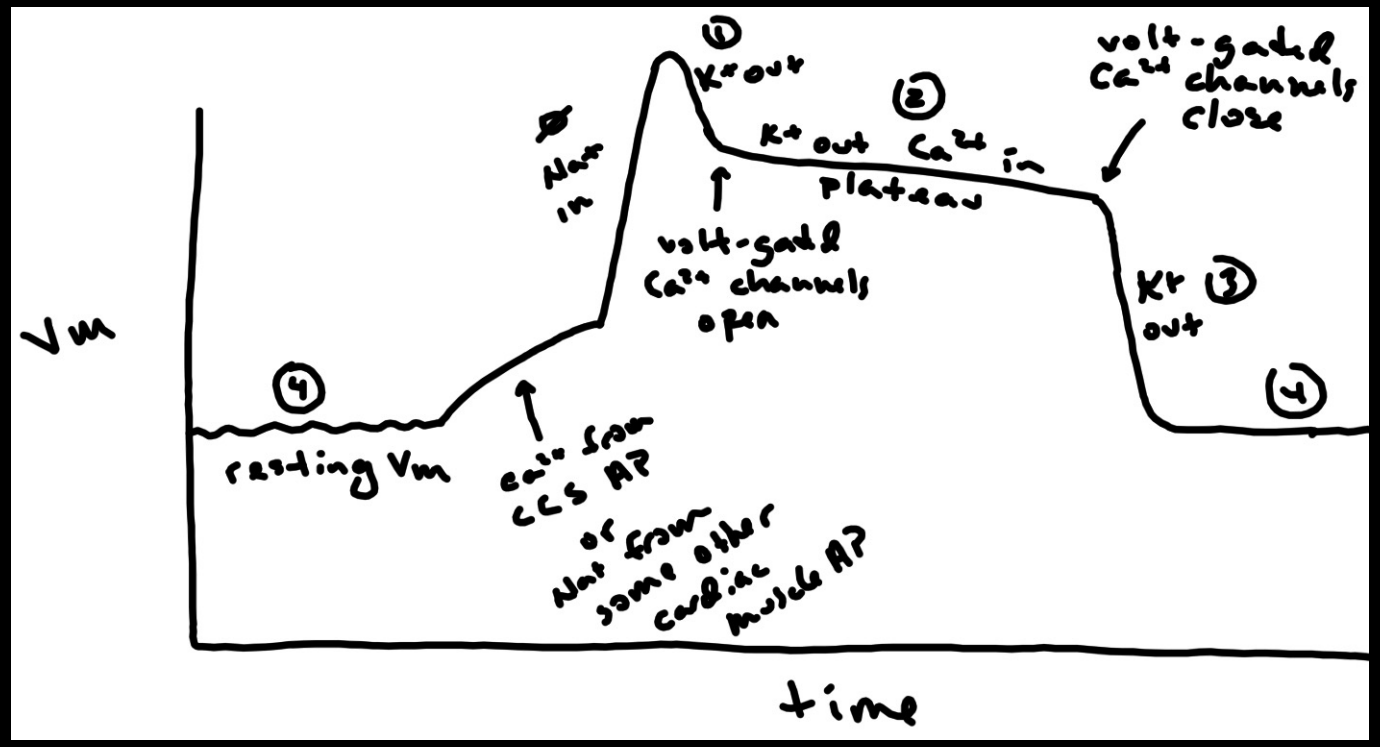
.) Voltage-Gated **Potassium** Channels Open

.) Less **Potassium** ∴ more negative , less positive





Cardiac Muscle - Action Potential



Threshold reached via:

Action potential from cardiac conduction system

Action potential from adjacent cardiac muscle cell

1.) Voltage-Gated **Sodium** Channels Open

2.) **Sodium** Enters

3.) Voltage-Gated **Potassium** Channels Open

4.) **Potassium** Leaves

5.) Voltage-Gated **Calcium** Channels Open

6.) **Calcium** Enters

7.) Voltage-Gated **Calcium** Channels Close

8.) **Potassium** Leaves

Cardiac Muscle- Action Potential

Threshold reached via:

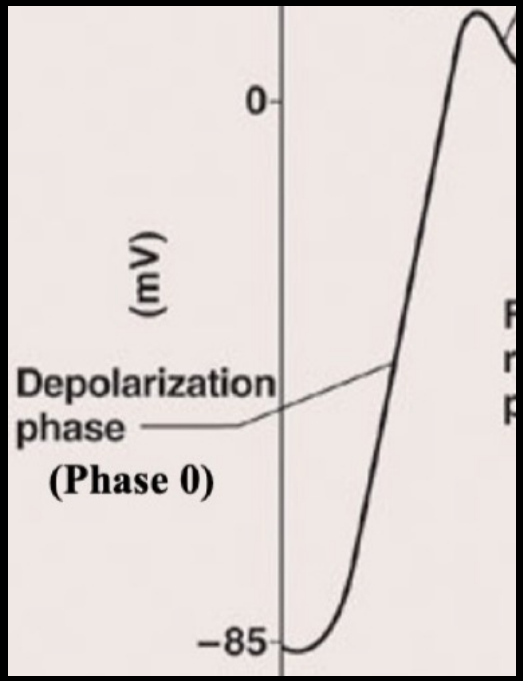
Action potential from cardiac conduction system

Action potential from adjacent cardiac muscle cell

Phase 0 - fast depolarization phase

1.) Voltage-Gated Sodium Channels Open

2.) Sodium Enters

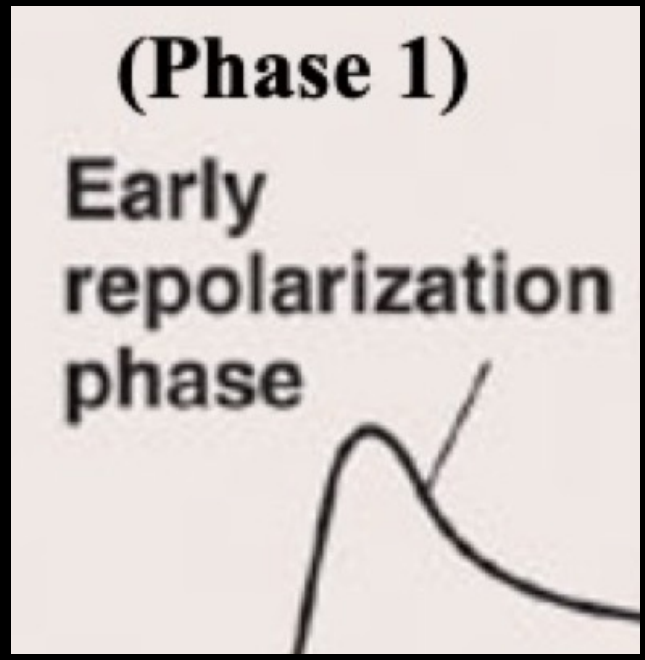


Cardiac Muscle- Action Potential

Phase 1 - early fast repolarization

3.) Voltage-Gated Potassium Channels Open

4.) Potassium Leaves



Cardiac Muscle - Action Potential

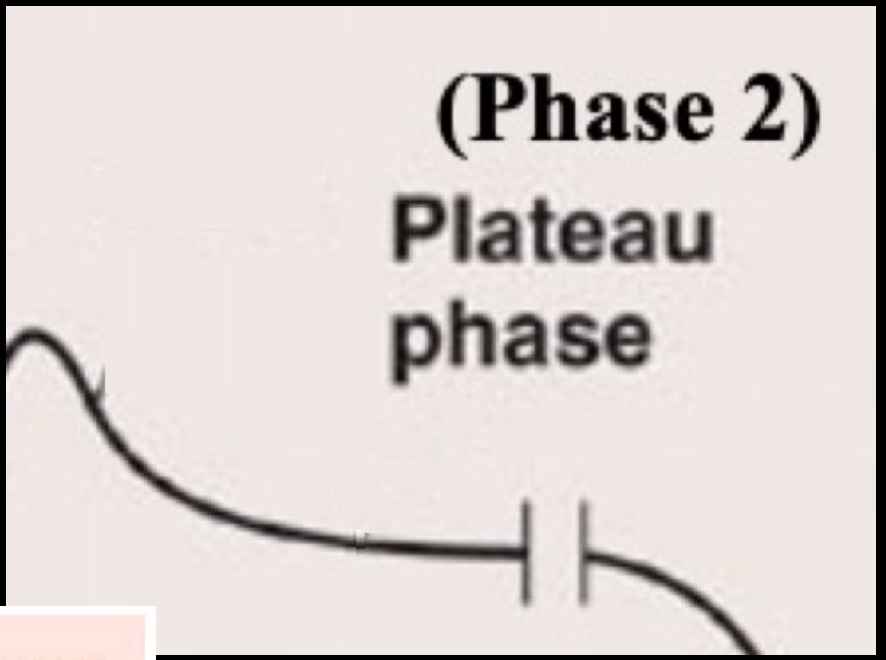
Phase 2 - Plateau Phase

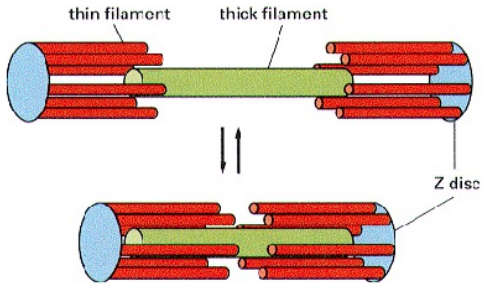
5.) Voltage-Gated **Calcium** Channels Open

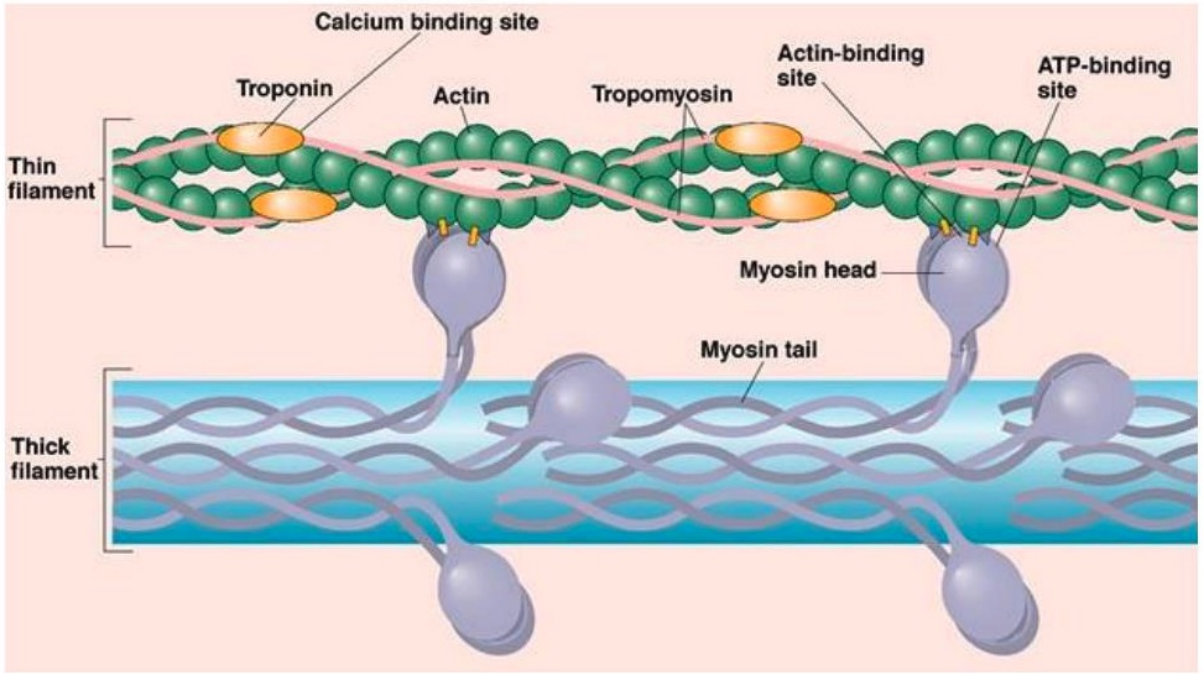
Potassium is still leaving

6.) **Calcium** Enters

Called **Trigger Calcium**





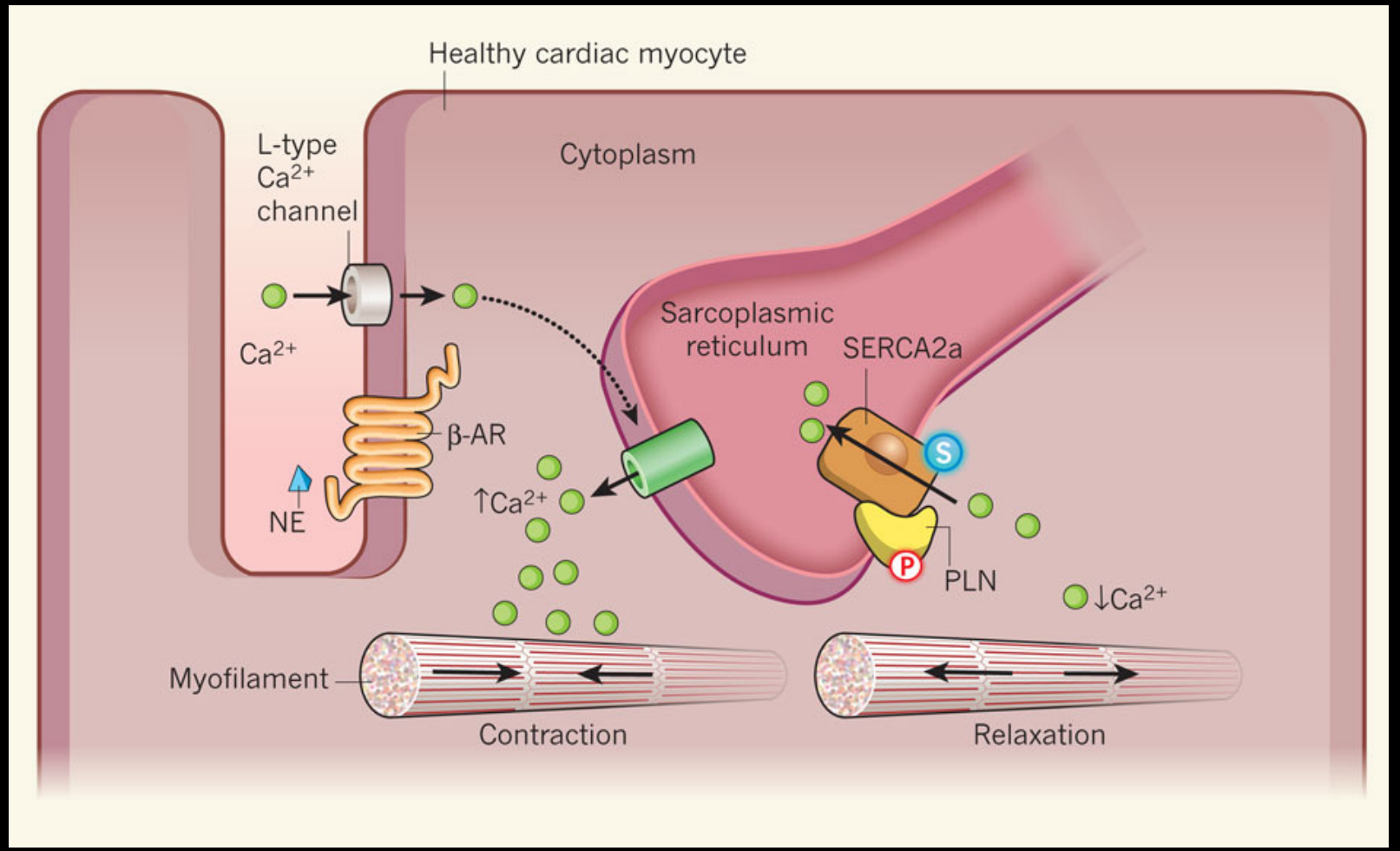


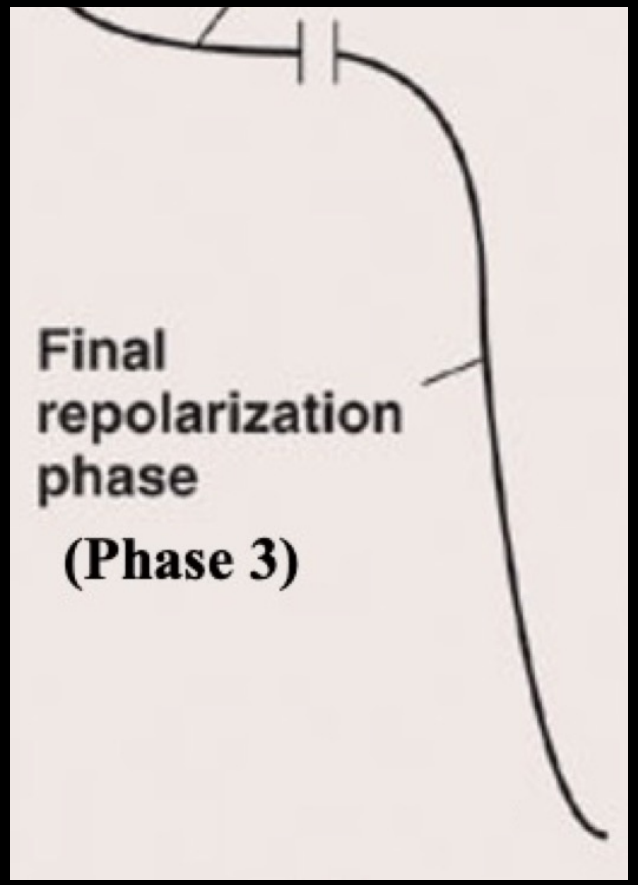
Cardiac Muscle - Action Potential

Phase 3 - Final Fast Repolarization Phase

7.) Potassium Leaves

8.) Voltage-Gated Calcium Channels Close





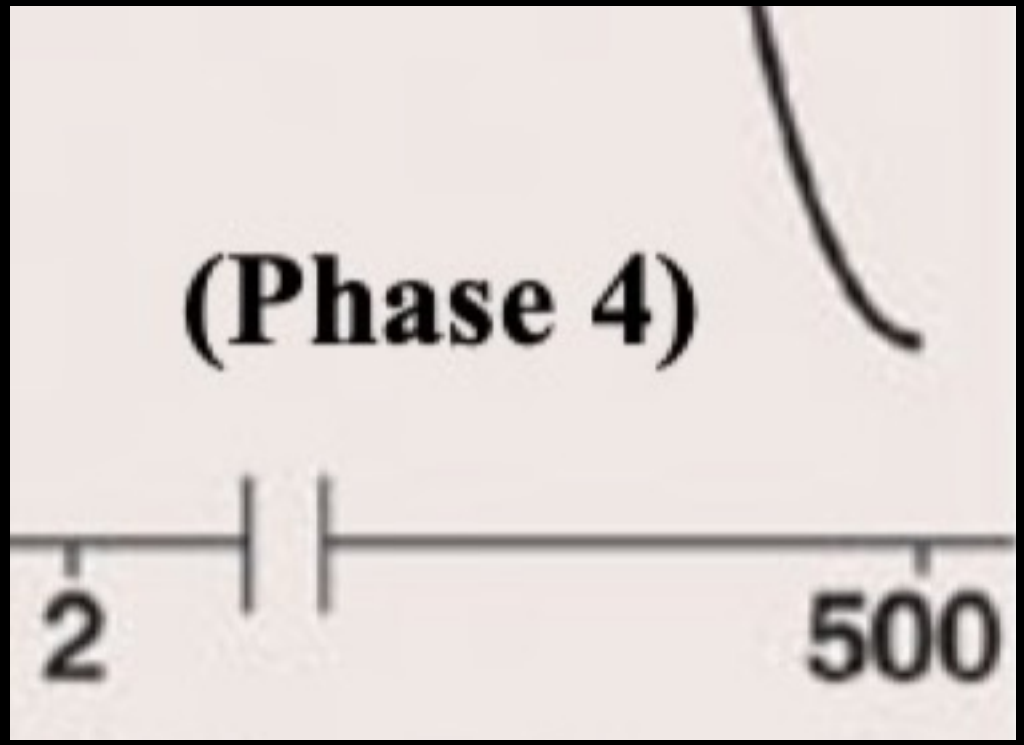
Contraction stops when calcium is pumped back into the sarcoplasmic reticulum

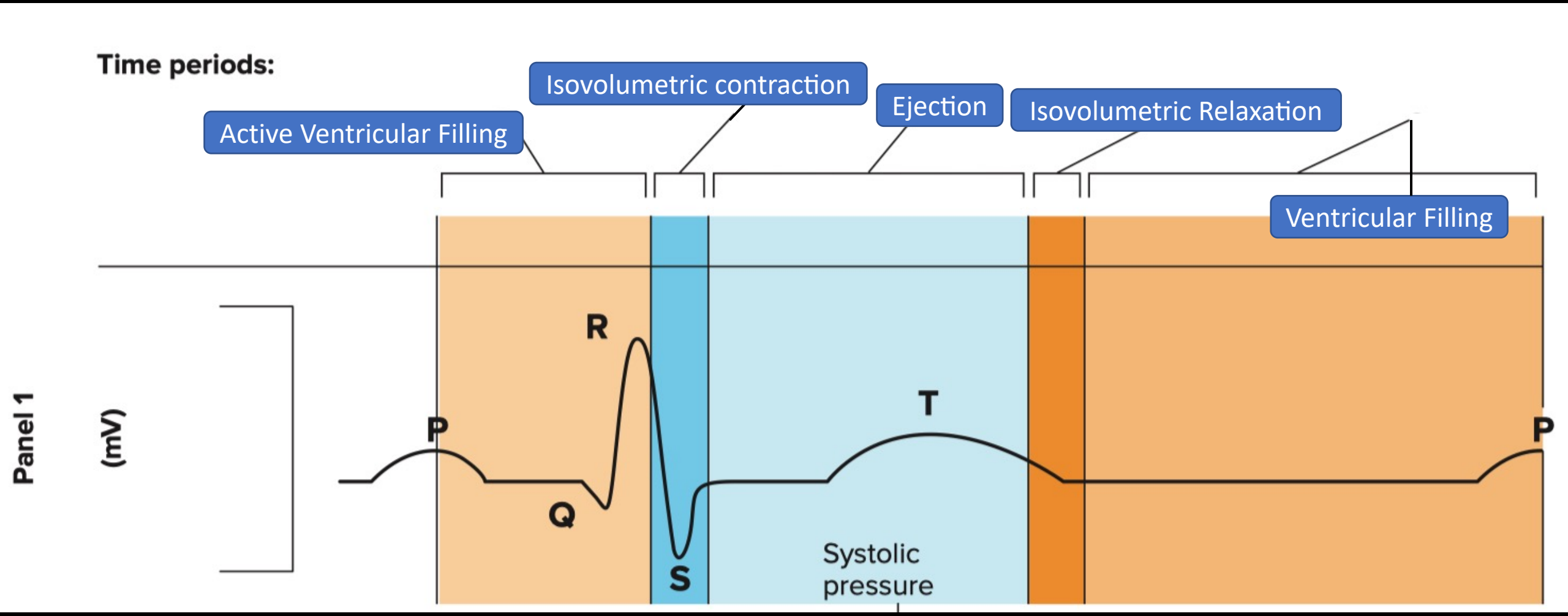
Cardiac Muscle - Action Potential

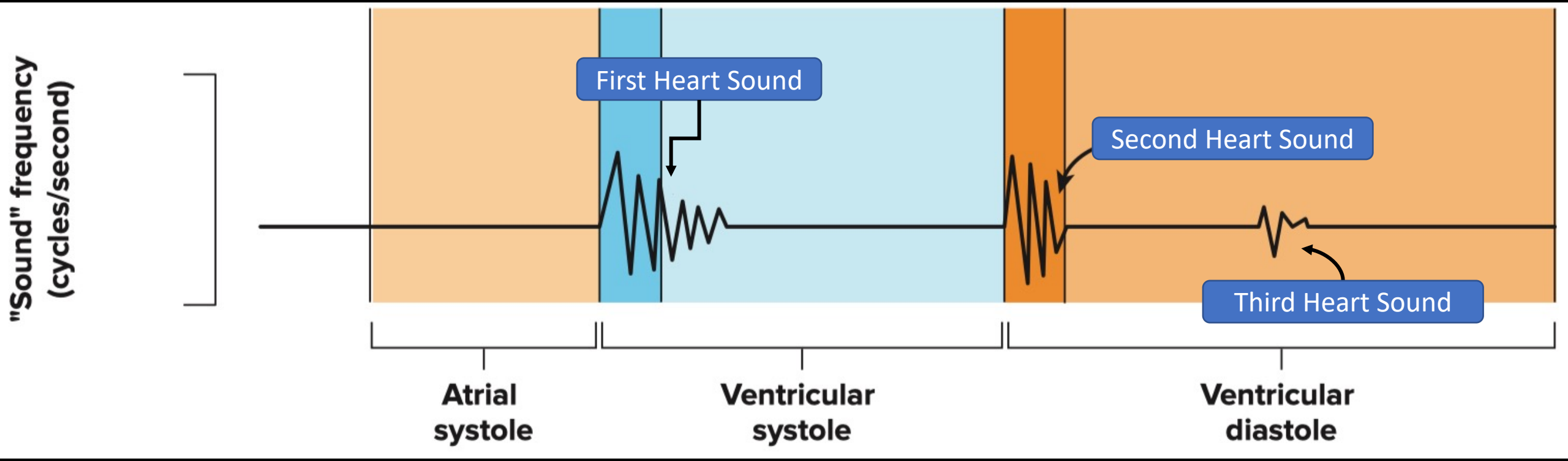
Phase 4 - resting V_m

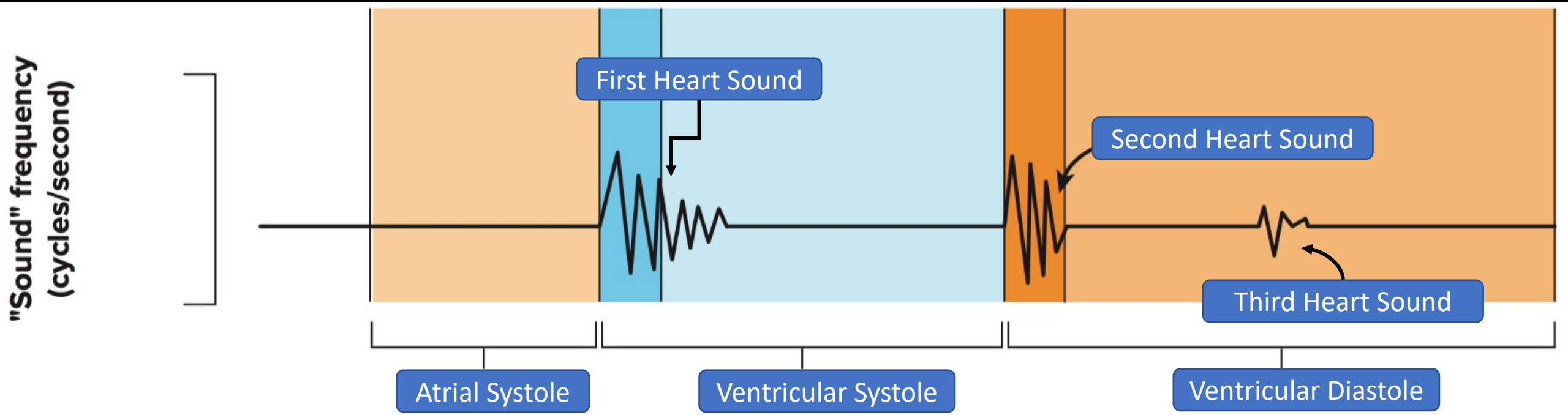
9.) Potassium Channels Eventually Close

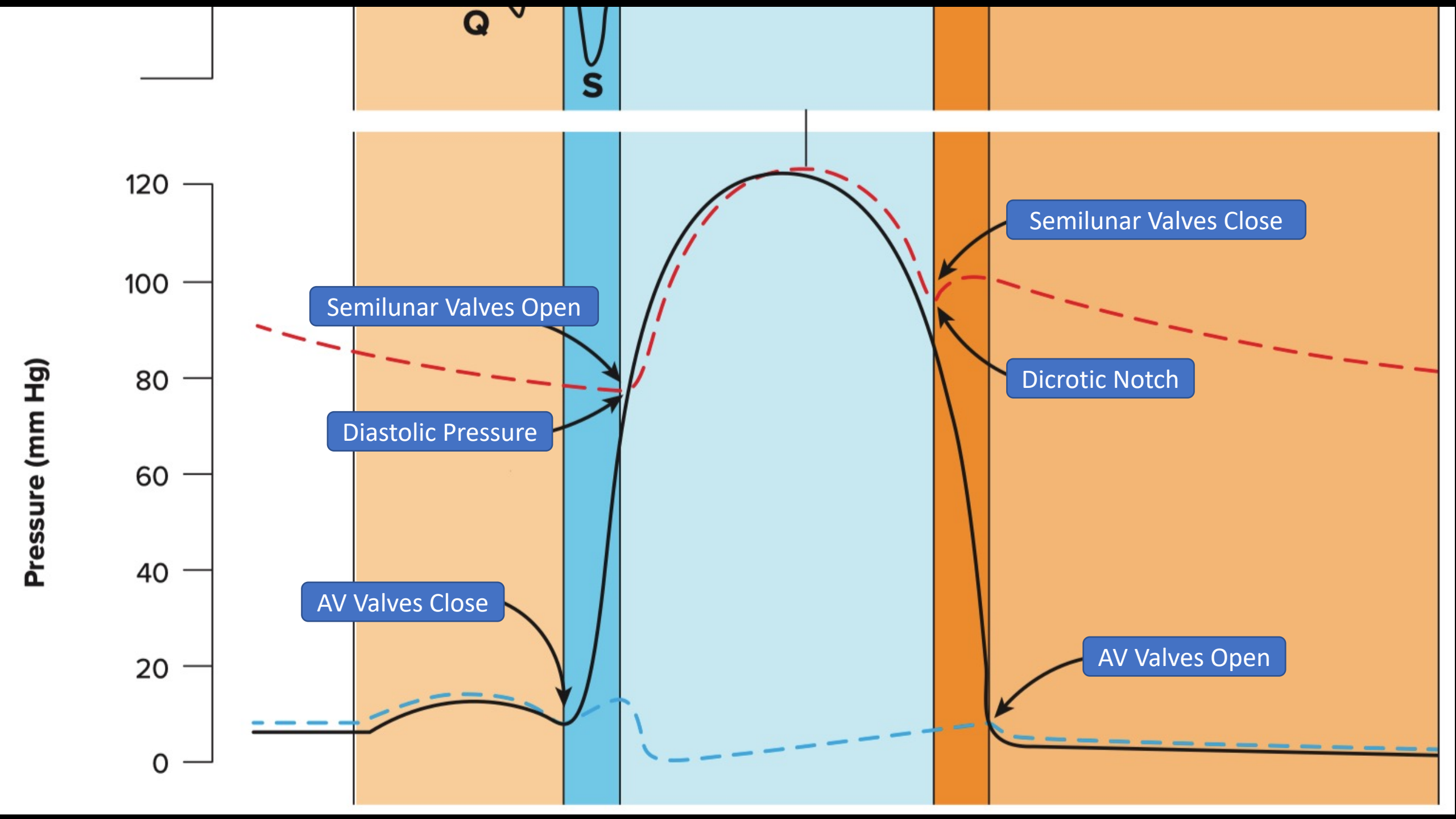
10.) Resting Vm is established

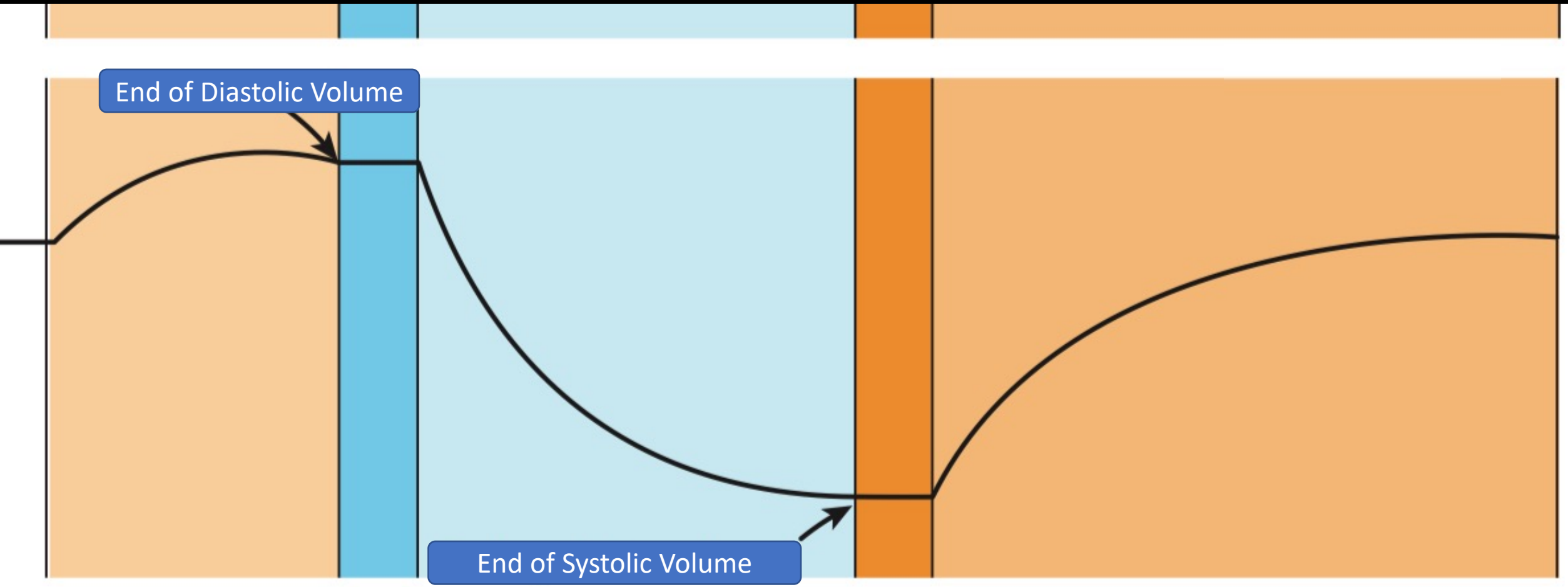



Time periods:
Active Ventricular Filling
Isovolumetric contraction
Ejection
Isovolumetric Relaxation
Ventricular Filling
Panel 1
(mV)
R
P
T
P
Q
S
Systolic
pressure

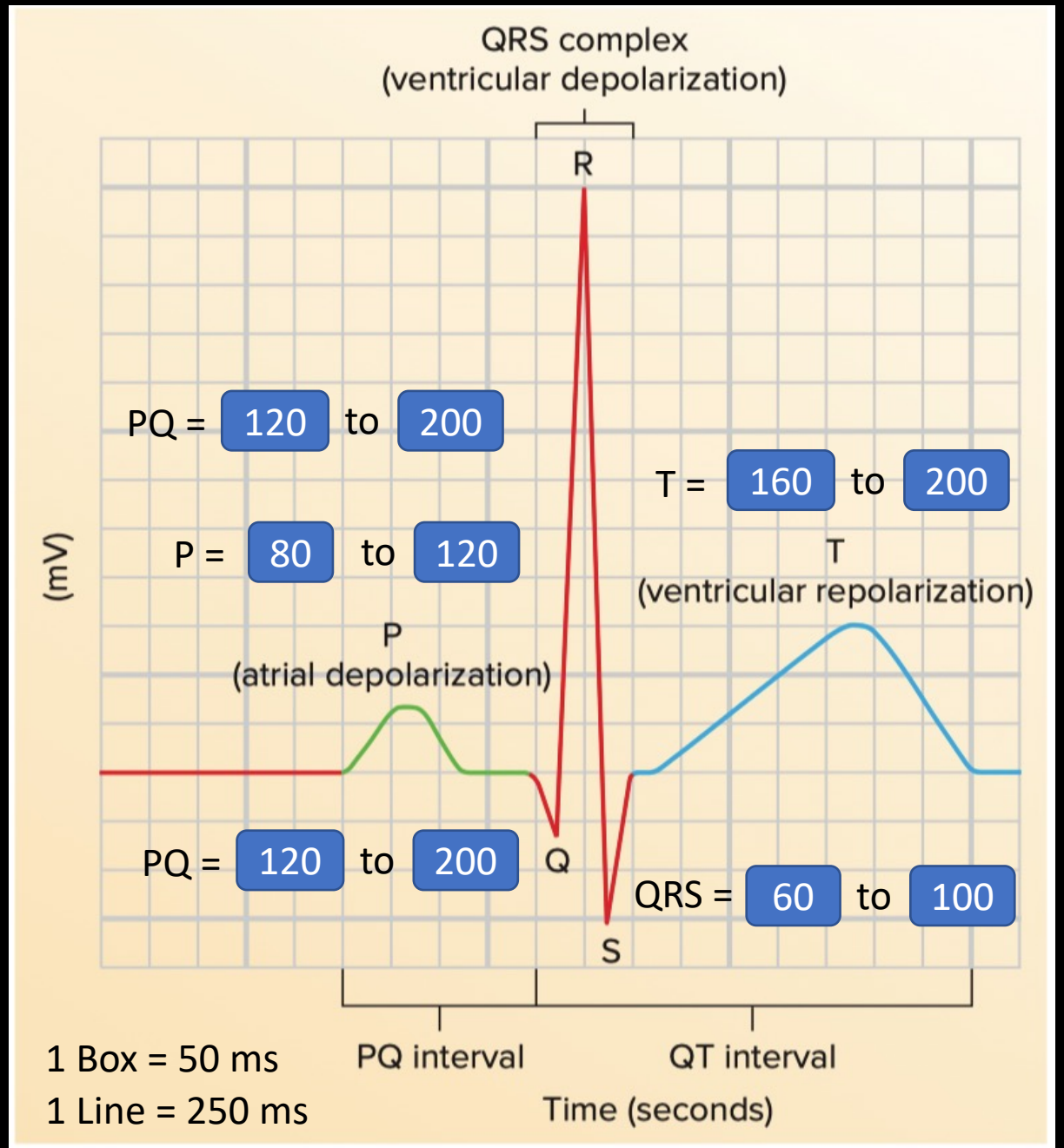

"Sound" frequency (cycles/second)
First Heart Sound
Second Heart Sound
Third Heart Sound
Atrial systole
Ventricular systole
Ventricular diastole


"Sound" frequency (cycles/second)
First Heart Sound
Second Heart Sound
Third Heart Sound
Atrial Systole
Ventricular Systole
Ventricular Diastole


Q
S
120
100
80
60
40
20
0
Pressure (mm Hg)
Semilunar Valves Open
Diastolic Pressure
AV Valves Close
Semilunar Valves Close
Dicrotic Notch
AV Valves Open


End of Diastolic Volume
End of Systolic Volume

Trace


QRS complex
(ventricular depolarization)
R
PQ = 120 to 200
T = 160 to 200
P = 80 to 120
T
(ventricular repolarization)
(mV)
P
(atrial depolarization)
PQ = 120 to 200
Q
QRS = 60 to 100
S
1 Box = 50 ms
1 Line = 250 ms
PQ interval
QT interval
Time (seconds)

Sinoatrial node (SA node)

"Pacemaker" of the heart

Spontaneously generates action potentials at a rate of about to 70 to 80 per minute

Ectopic Pacemaker

AV Node would slow heart rate to approximately 40 to 60 beats / min

Purkinje Fibers would slow heart rate to approximately 25 to 45 beats / min

Limb Leads – Standard Bipolar

- **Standard bipolar limb leads**
  - Measure the electrical activity of the heart in a frontal plane
  - Lead I: Right Arm (–) to Left Arm left arm (+)
    - Measures electrical activity across the heart at a 0° angle
  - Lead II: Right Arm (–) to Left Leg (+)
    - Measures electrical activity across the heart at a +60° angle
  - Lead III: Left Arm (–) to Left Leg (+)
    - Measures electrical activity across the heart at a +120° angle

- **Augmented unipolar limb leads**
  - Measure the electrical activity of the heart in a frontal plane
  - aVR: Right Arm (+) to central terminal ground lead (joining of left arm and left leg)
    - Measures electrical activity across the heart at a –150° angle
  - aVL: Left Arm (+) to central terminal ground lead (joining of left leg and right arm)
    - Measures electrical activity across the heart at a –30° angle
  - aVF: Left Leg (+) to central terminal ground lead (joining of right arm and left arm)
    - Measures electrical activity across the heart at a +90° angle

- **Chest leads / Precordial leads**
  - Measure the electrical activity of the heart in a transverse plane
  - V1 , V2 , V3 , V4 , V5 , V6 leads arranged across the chest

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  - Measure the electrical activity of the heart in a frontal plane
  - Lead 1 : right arm (–) to left arm (+)
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    - Measures electrical activity across the heart at a +60° angle
  - Lead 3 : left arm (–) to left leg (+)
    - Measures electrical activity across the heart at a +120° angle

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    - Measures electrical activity across the heart at a –30° angle
  - aVF left leg (+) to central terminal ground lead (joining of right arm and left arm)
    - Measures electrical activity across the heart at a +9° angle

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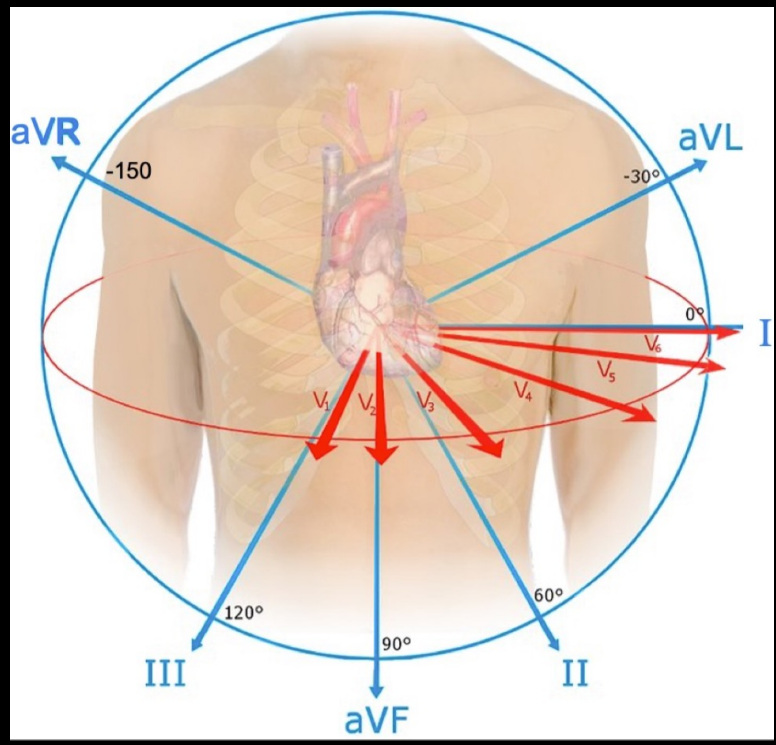
  - Measure the electrical activity of the heart in a transverse plane
  - V1 , V2 , V3 , V4 , V5 , V6 leads arranged across the chest

Limb Leads – Standard Bipolar



- **Standard bipolar limb leads**
  - Measure the electrical activity of the heart in a Frontal Plane
  - Lead 1 : Right Arm (–) to Left Arm (+)
    - Measures electrical activity across the heart at a 0 ° angle
  - Lead 2 : Right Arm (–) to Left Leg (+)
    - Measures electrical activity across the heart at a +60 ° angle
  - Lead 3 : Left Arm (–) to Left Leg (+)
    - Measures electrical activity across the heart at a +120 ° angle

- **Augmented unipolar limb leads**
  - Measure the electrical activity of the heart in a frontal Frontal Plane plane
  - aVR : Right Arm (+) to central terminal ground lead (joining of left arm and left leg)
    - Measures electrical activity across the heart at a -150 ° angle
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    - Measures electrical activity across the heart at a -30 ° angle
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    - Measures electrical activity across the heart at a +90 ° angle

- **Chest leads / Precordial leads**
  - Measure the electrical activity of the heart in a Transverse plane
  - V1 , V2 , V3 , V4 , V5 , V6 leads arranged across the chest

Hypertension

- Elevated (borderline / pre-hypertension no longer exists as categories)
  - Systolic pressure from 120 to 129 mmHg
  - Diastolic pressure not taken into account
- Stage 1 hypertension :
  - Systolic pressure from 130 to 139 mmHg

    and / or
  - Diastolic pressure from 80 to 89 mmHg
- Stage 2 hypertension :
  - Systolic pressure from 140 mmHg or above
  - Diastolic pressure of 90 mmHg or above
- Hypertensive crisis :
  - Systolic pressure above 140 mmHg

    and / or
  - Diastolic pressure above 120 mmHg
- Essential / Primary hypertension :
  - Cause is unknown (majority of cases)
- Secondary hypertension :
  - Cause is known (e.g. renal disease)