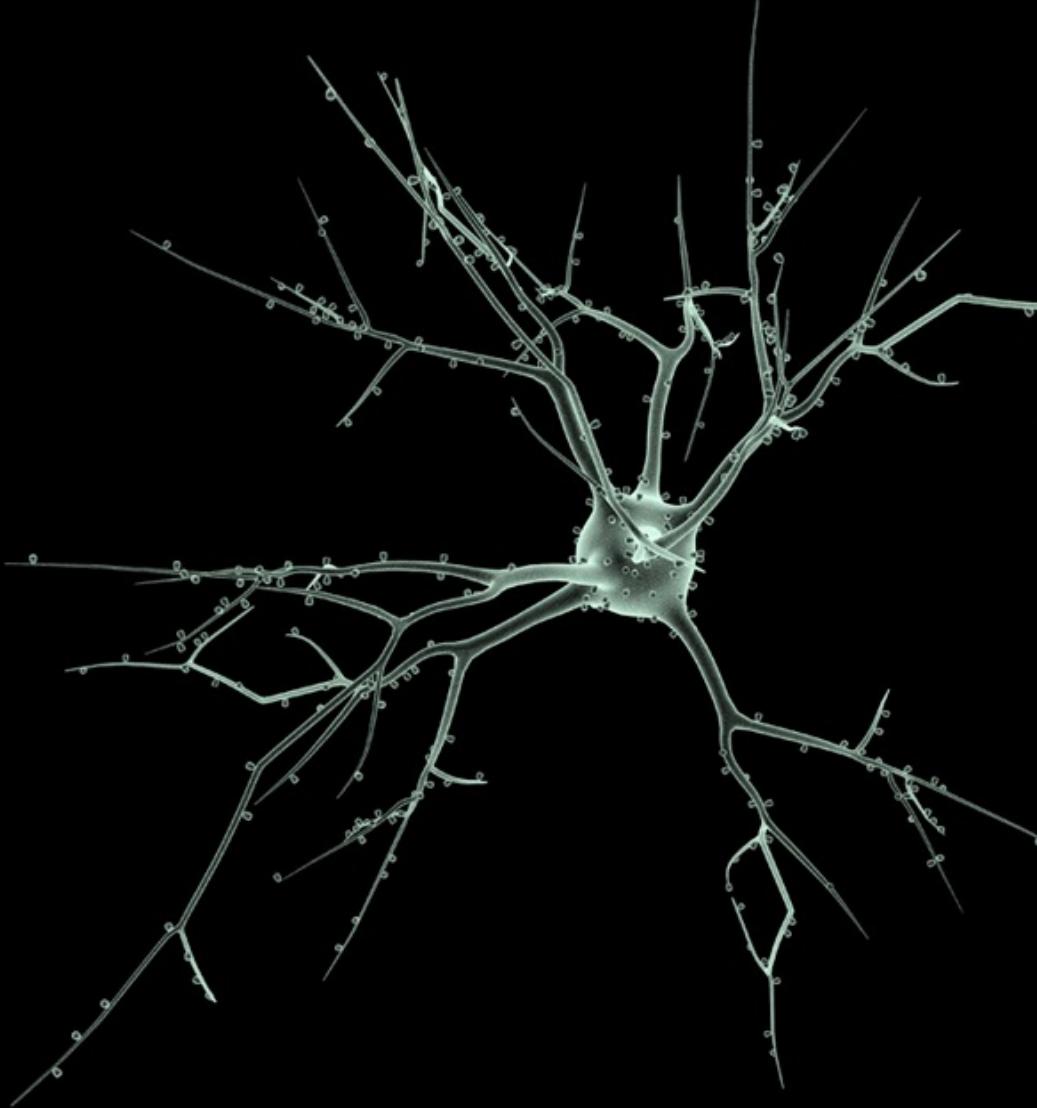


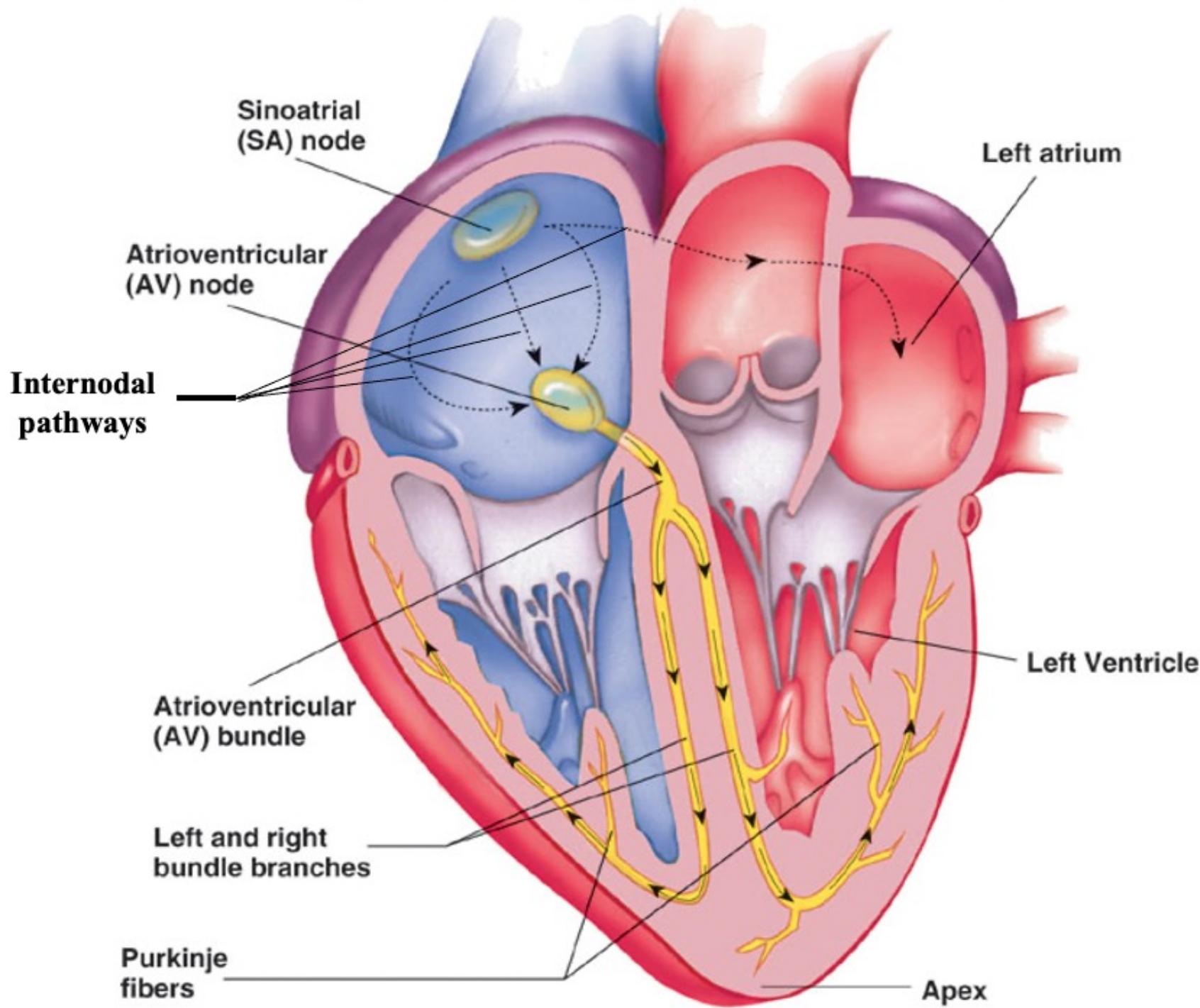
# ANT 3120

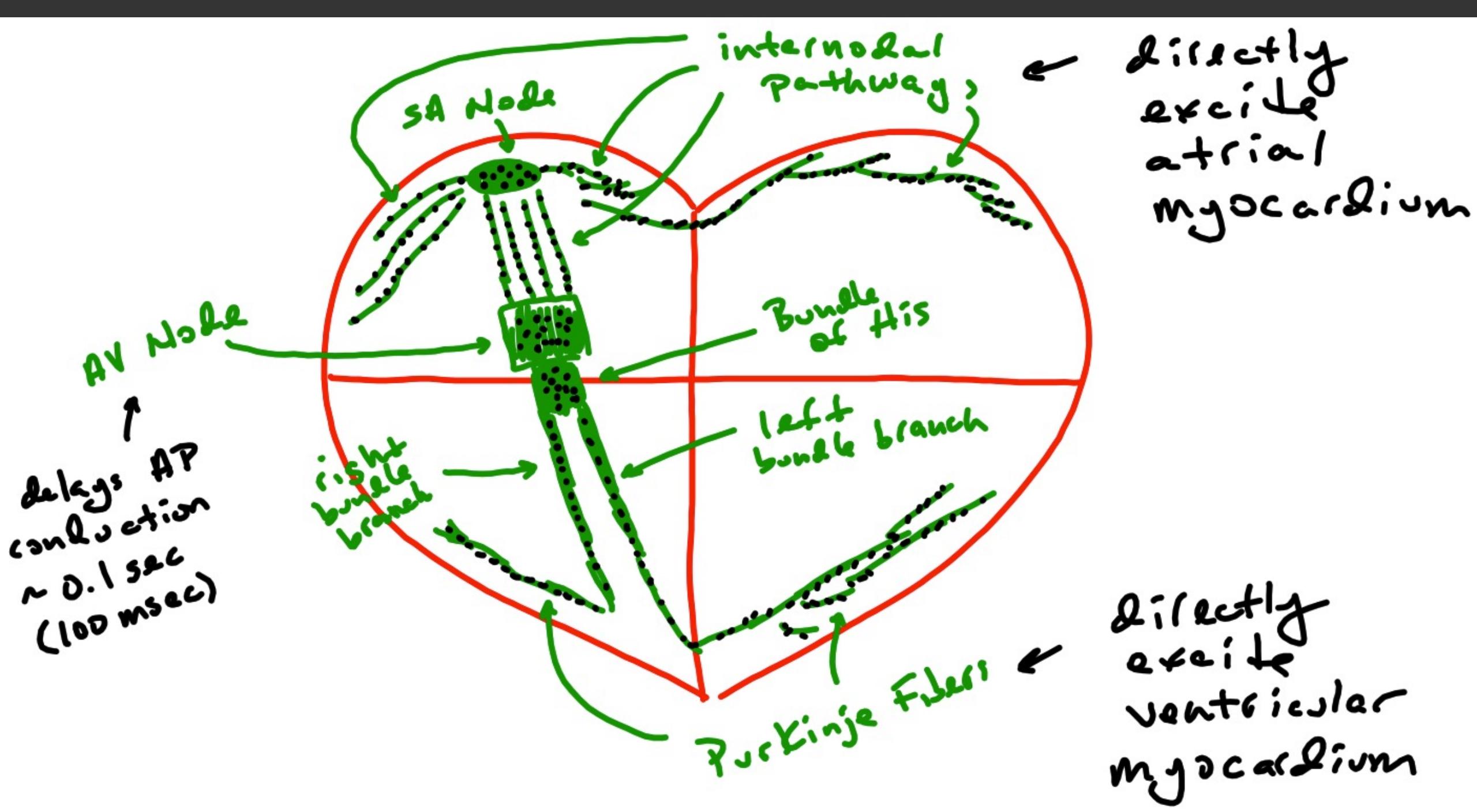
## Physiology – Exam 3



# 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

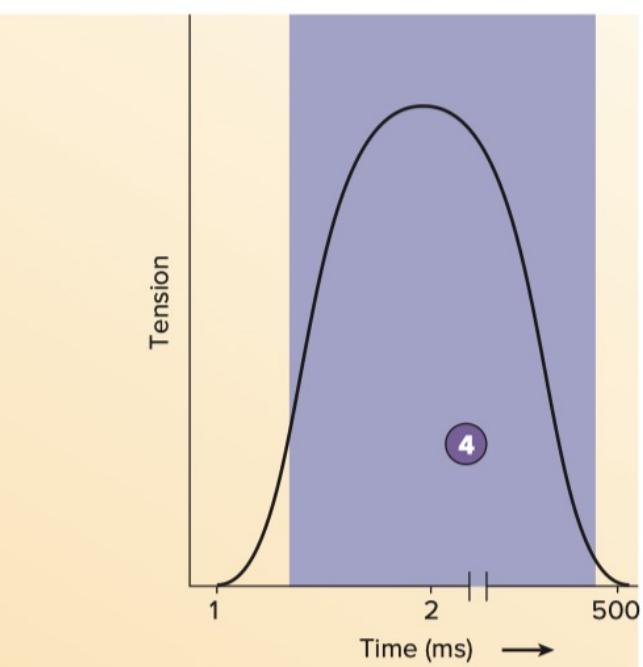
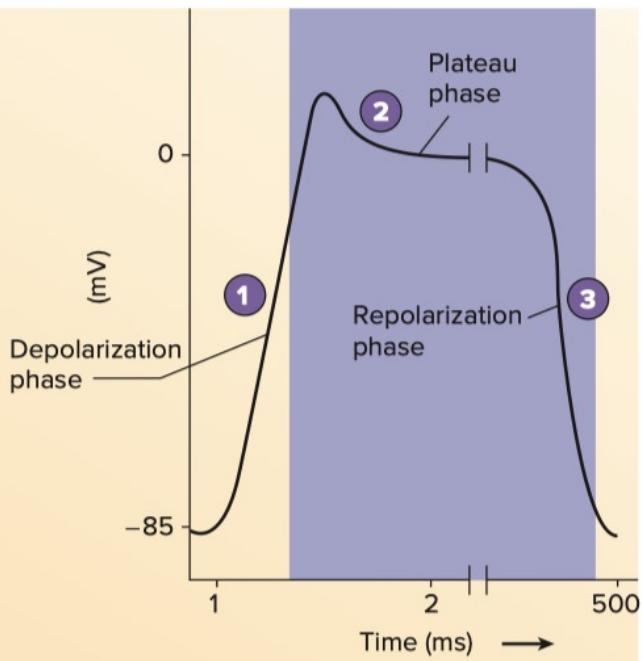




# Cardiac Conduction System – Action Potential

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

### Cardiac Muscle



#### 1 Depolarization phase

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

#### 2 Early repolarization and plateau phases

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

#### 3 Final repolarization phase

- Voltage-gated  $\text{Ca}^{2+}$  channels close.
- Many voltage-gated  $\text{K}^+$  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

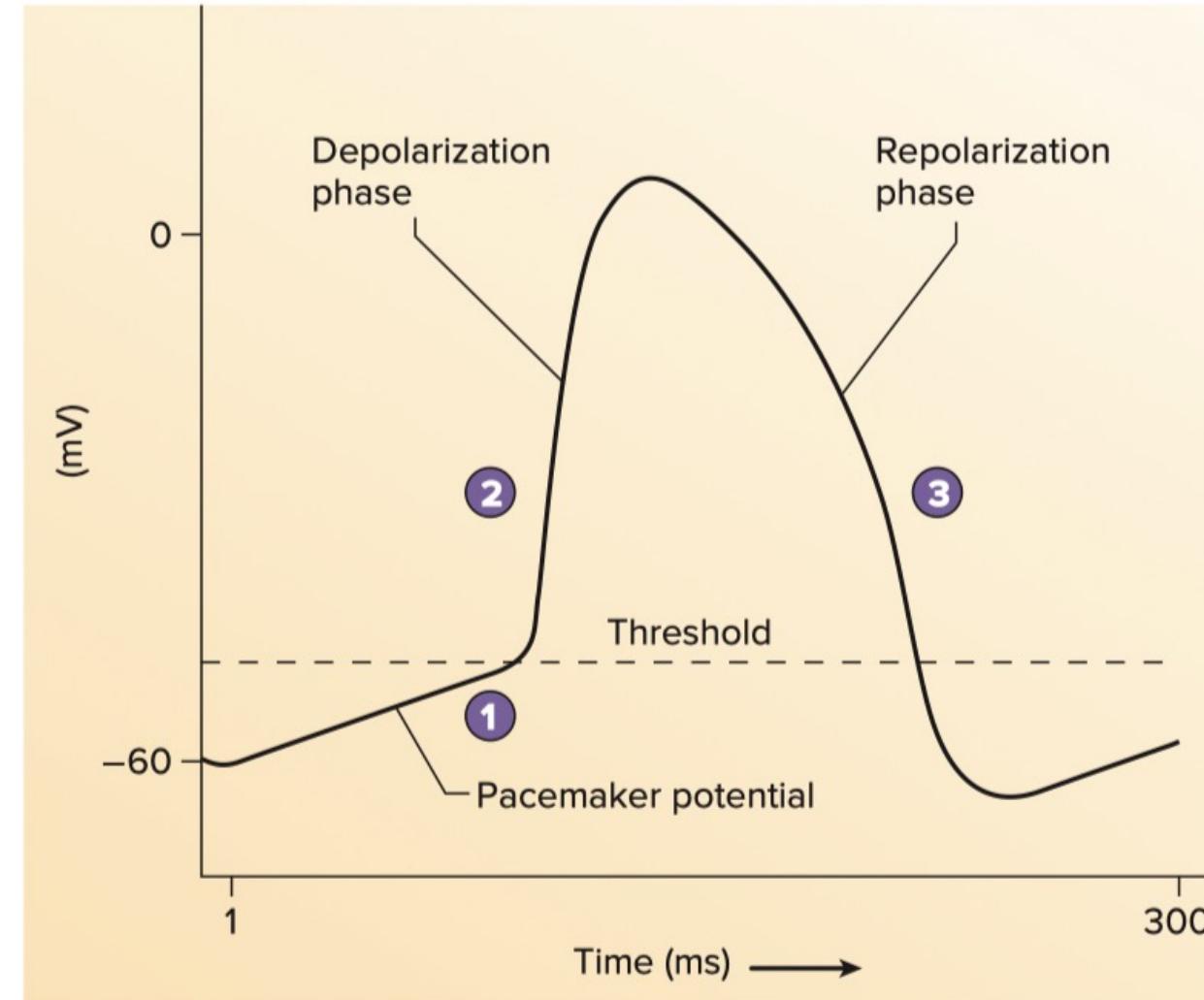
- A small number of  $\text{Na}^+$  channels are open.
- Voltage-gated  $\text{K}^+$  channels that opened in the repolarization phase of the previous action potential are closing.
- Voltage-gated  $\text{Ca}^{2+}$  channels begin to open.

### 2 Depolarization phase

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

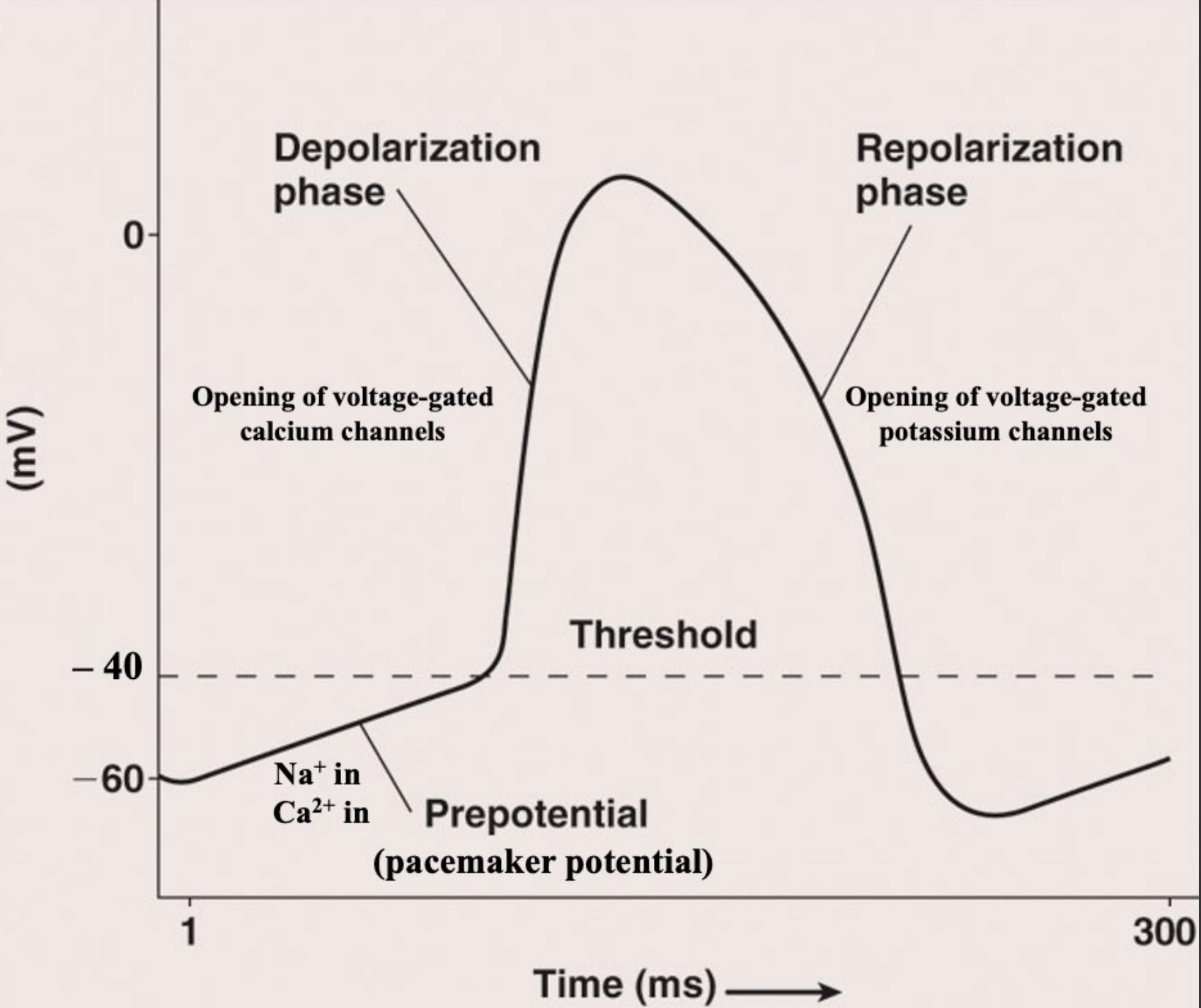
### 3 Repolarization phase

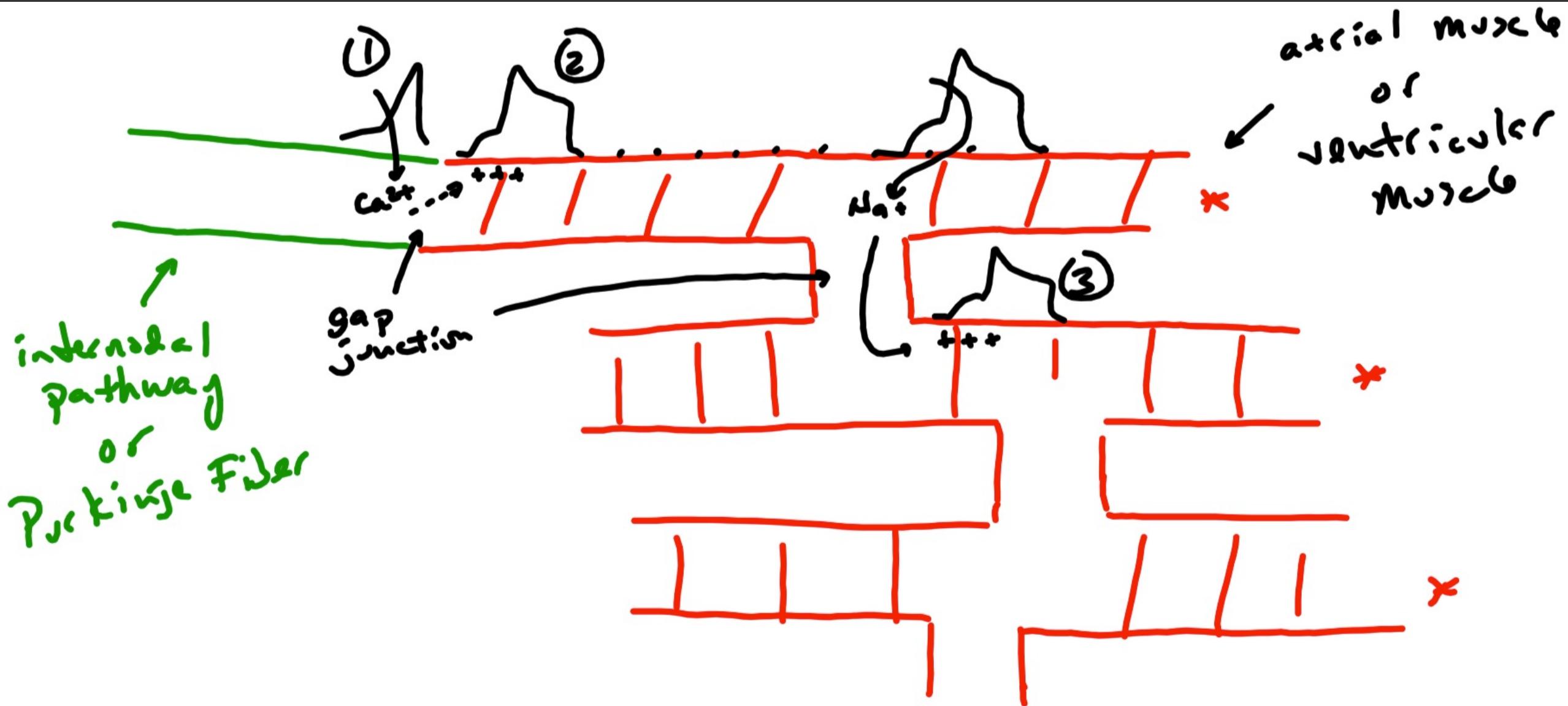
- Voltage-gated  $\text{Ca}^{2+}$  channels close.
- Voltage-gated  $\text{K}^+$  channels open.

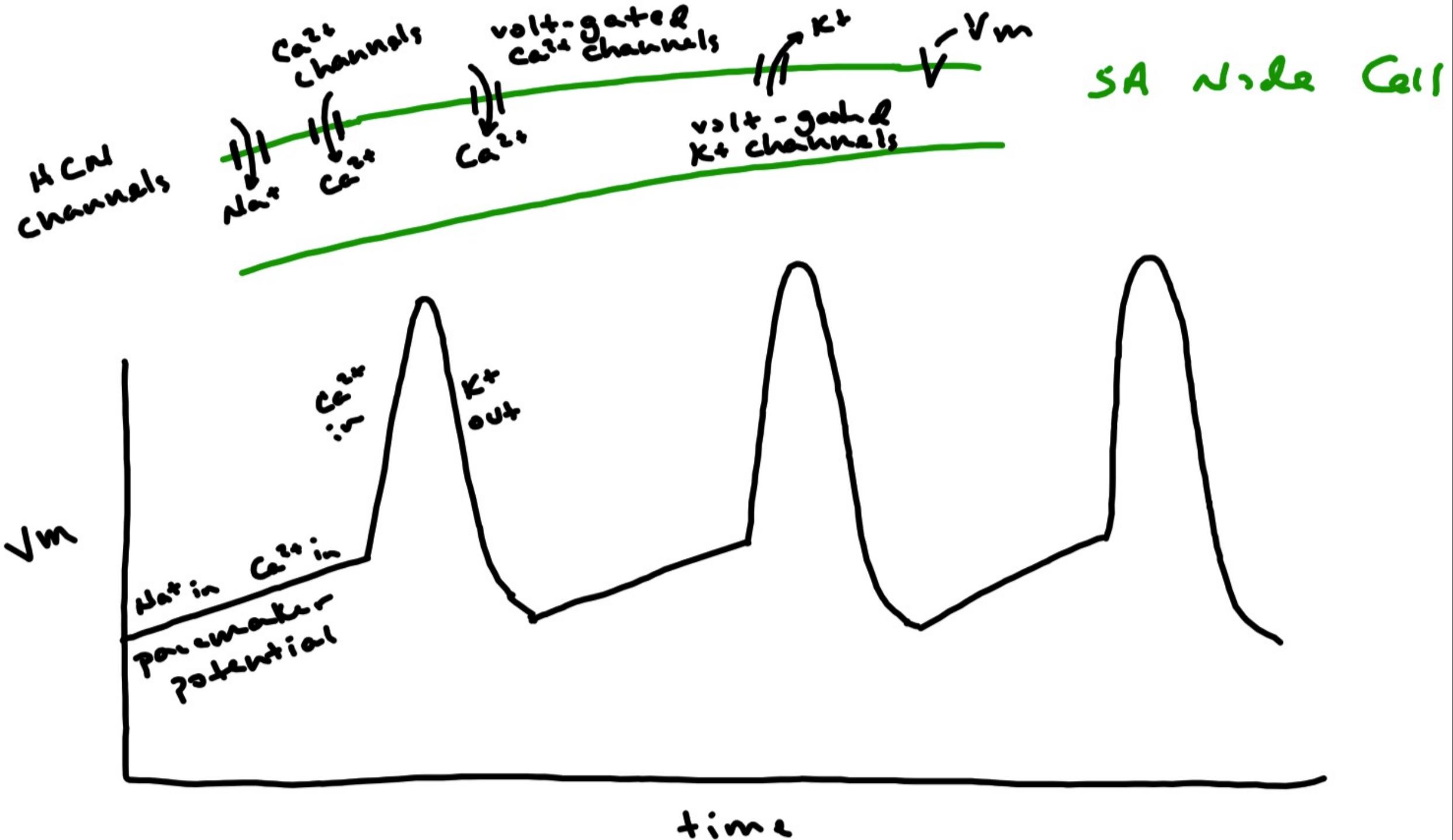


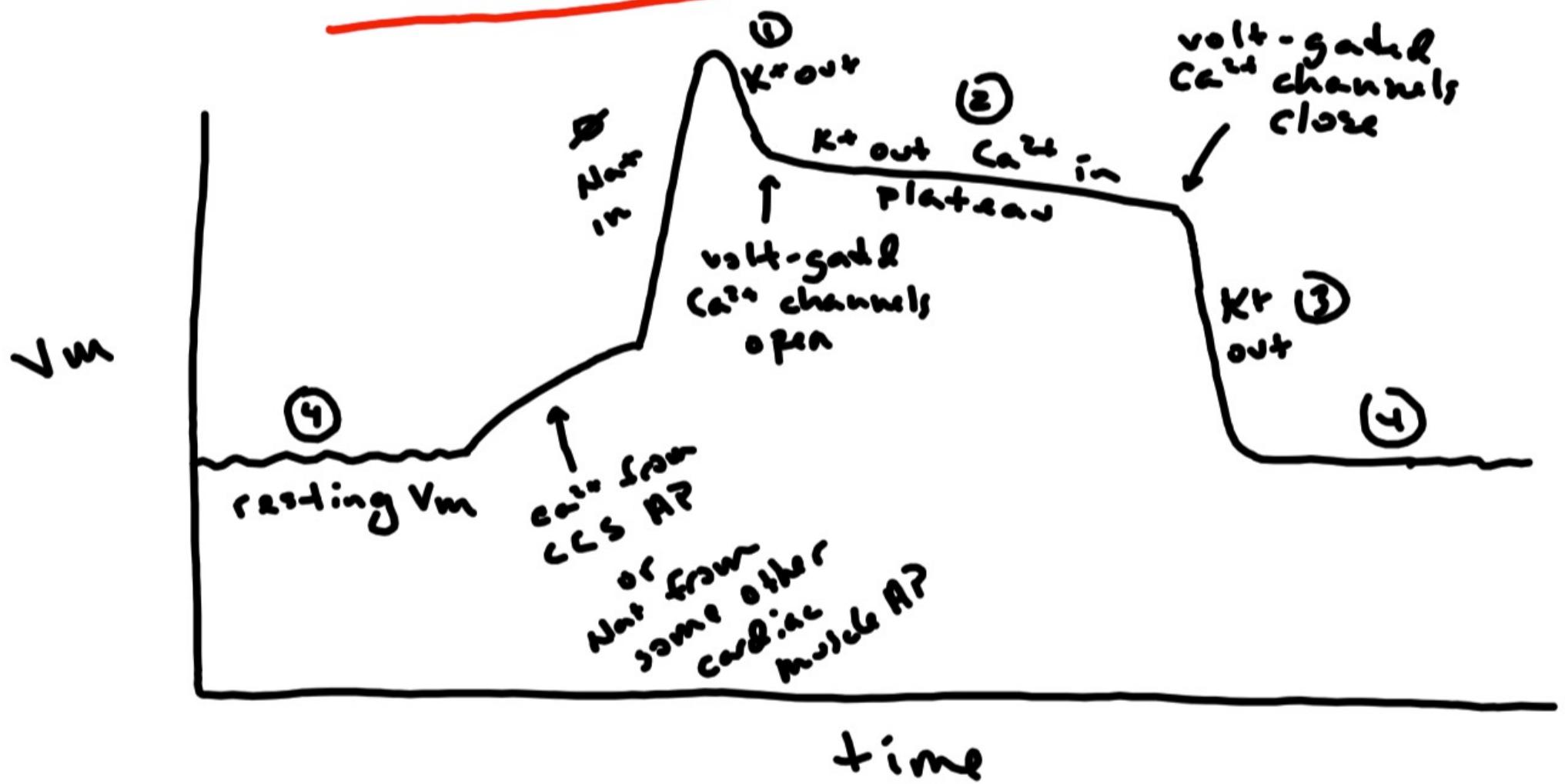
## PROCESS FIGURE 20.15 Pacemaker Potential

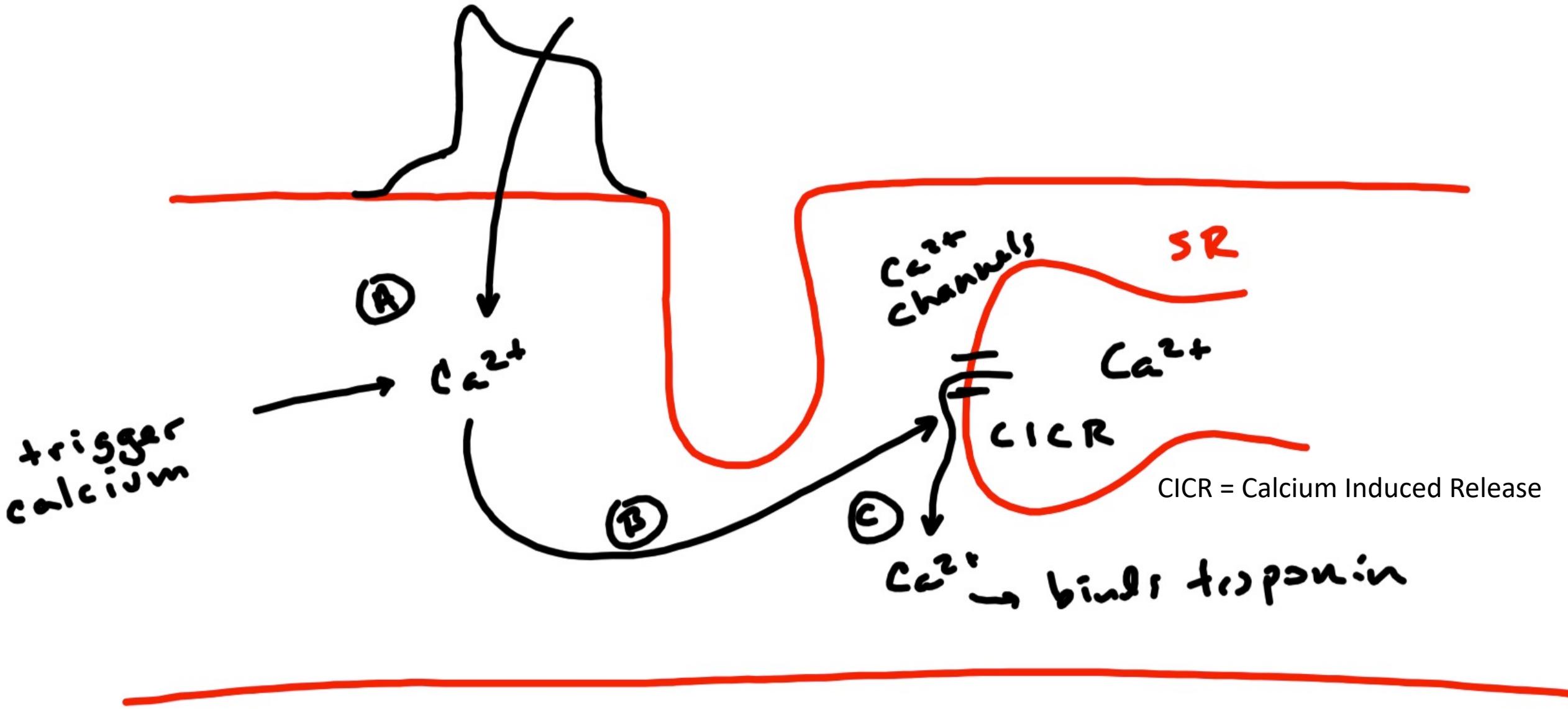
The production of action potentials by the pacemaker cells of the SA node is responsible for the autorhythmicity of the heart.







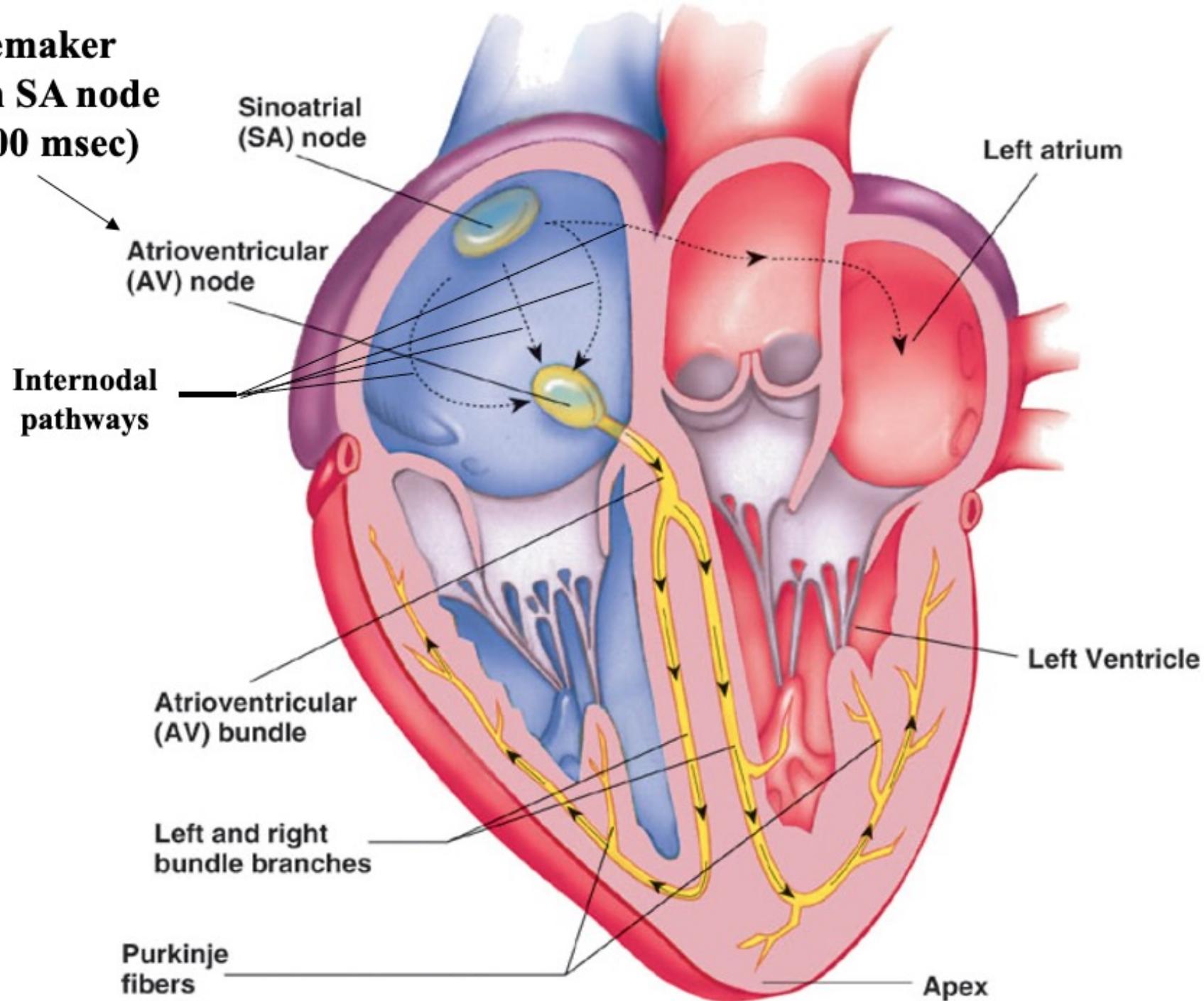




# Cardiac Conduction System

- **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)**

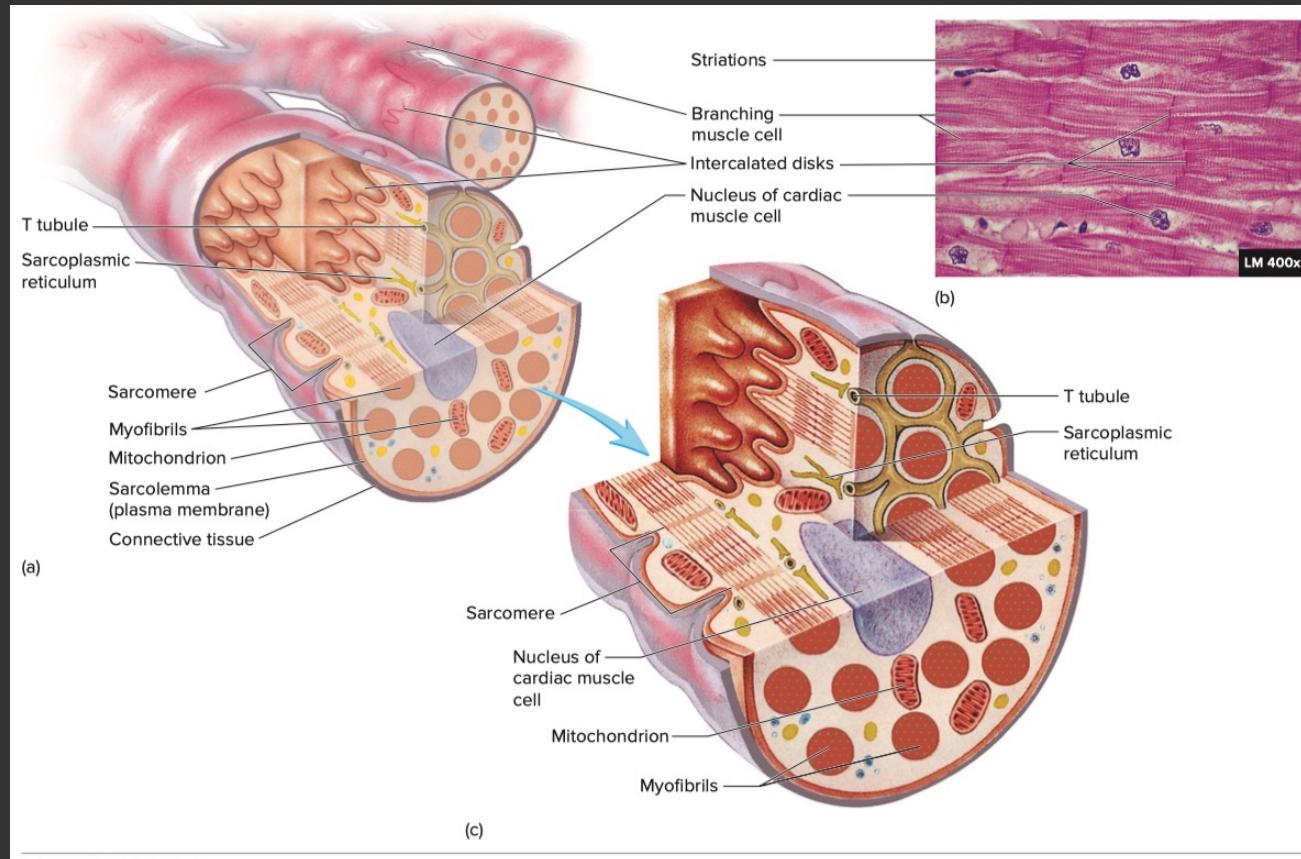


# Ectopic Pacemaker

- 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

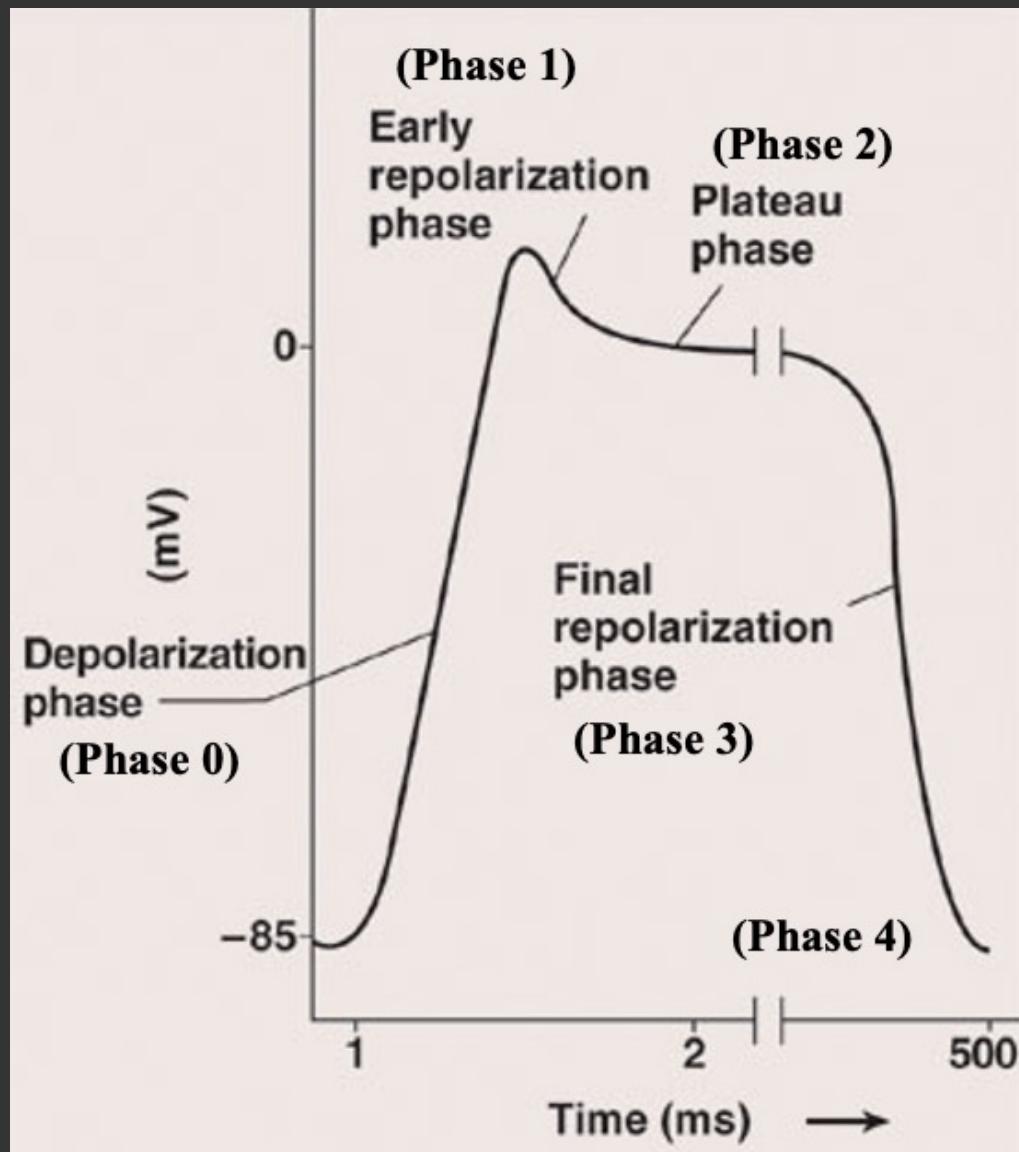


**FIGURE 20.12 Histology of the Heart**

(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 )

- Recording of the electrical activity ( i.e. action potentials ) of cardiac muscle
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      - 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**

# 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

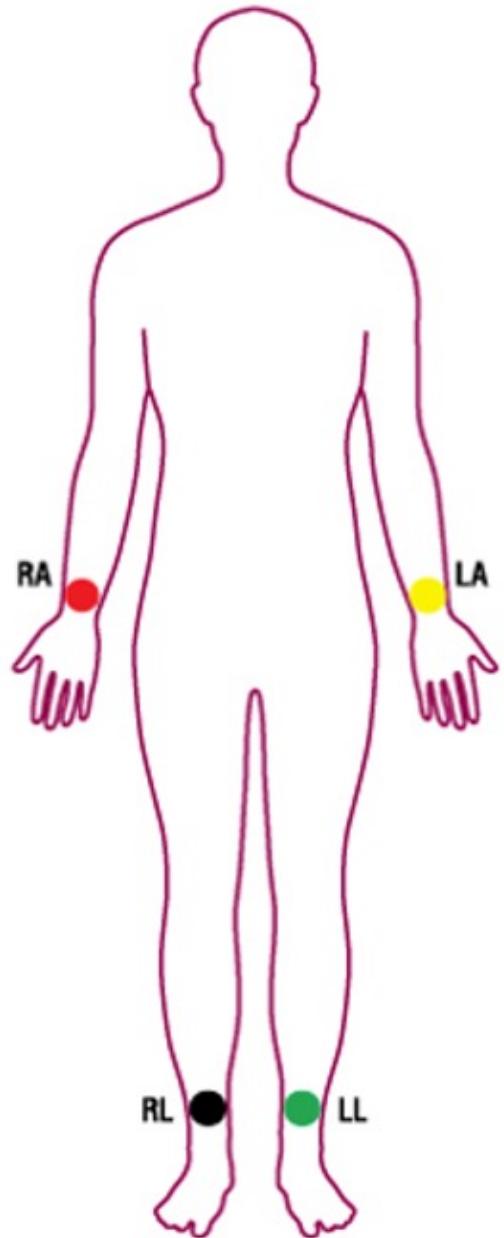
## ➤ Augmented unipolar limb leads

- 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

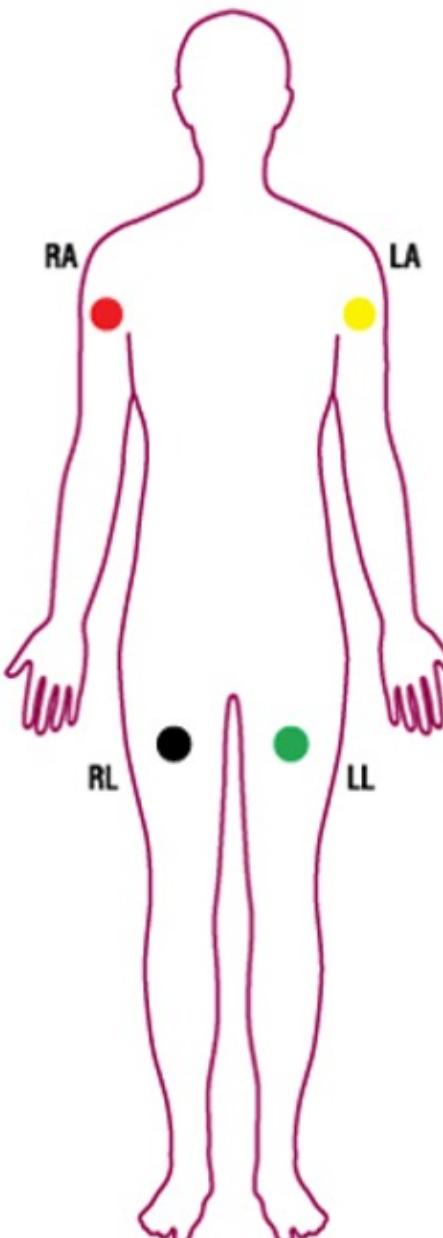
## ➤ Chest leads / Precordial leads

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

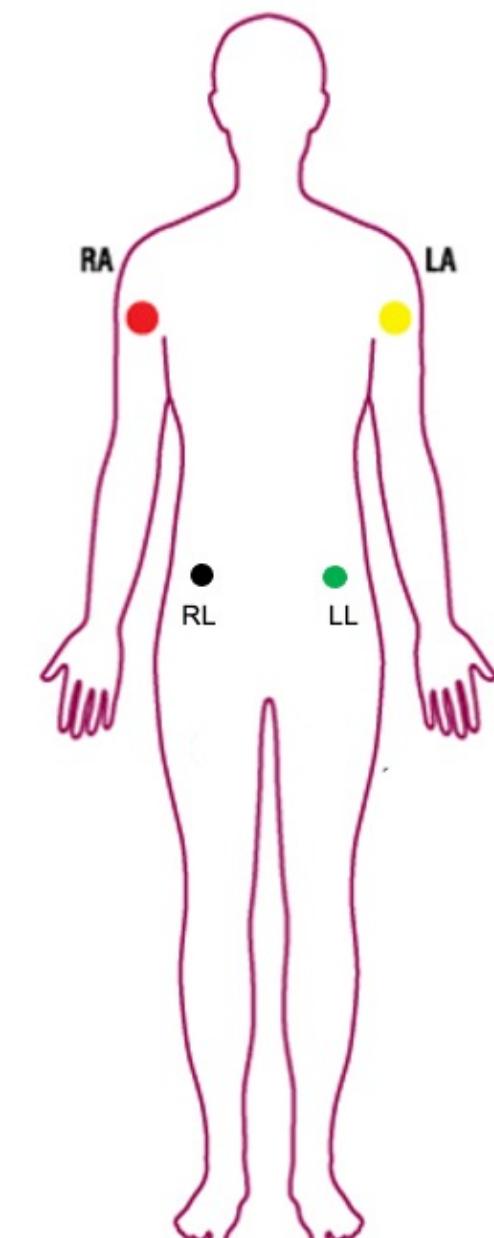
# ECG – Limb Lead Electrode Placement



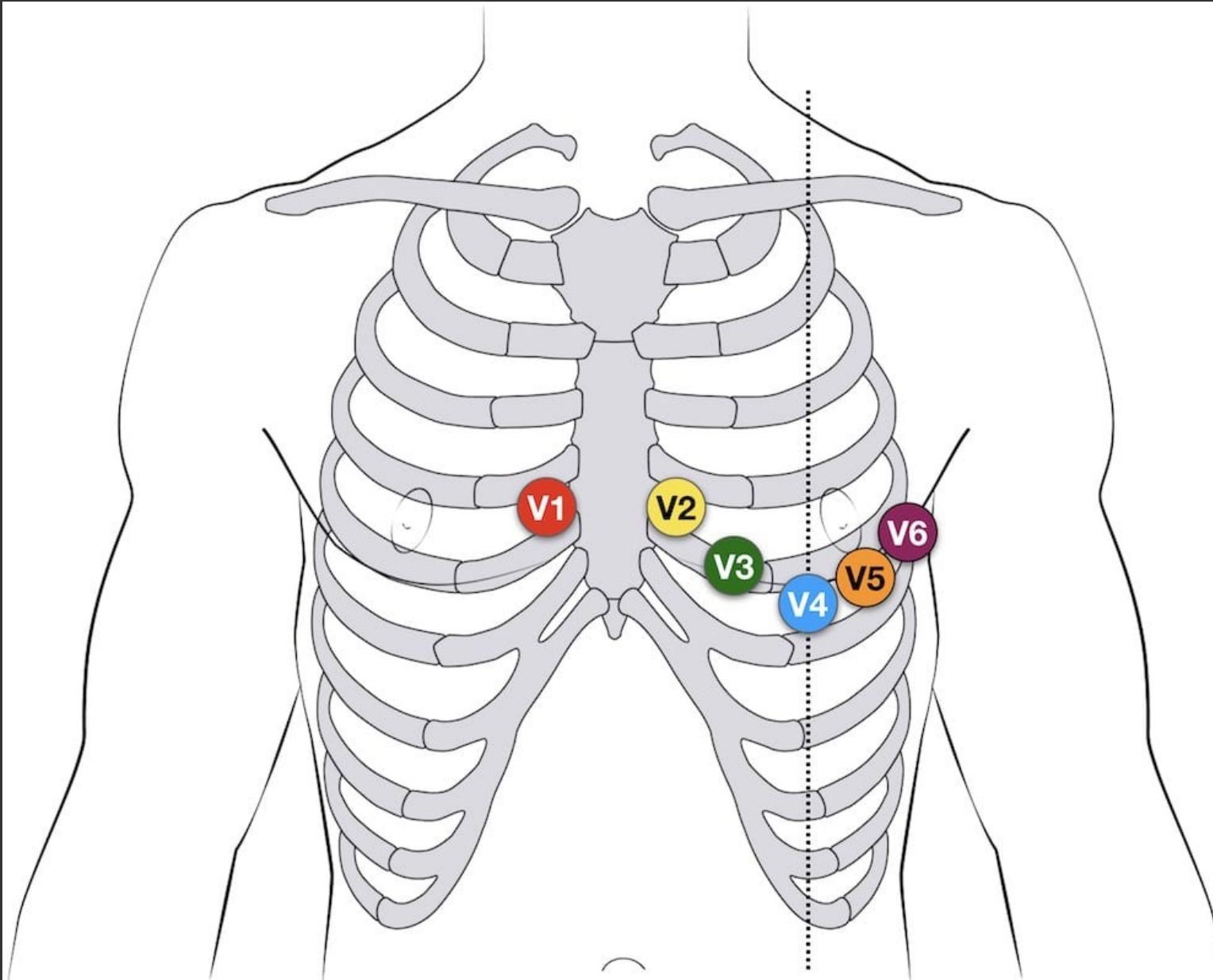
OR

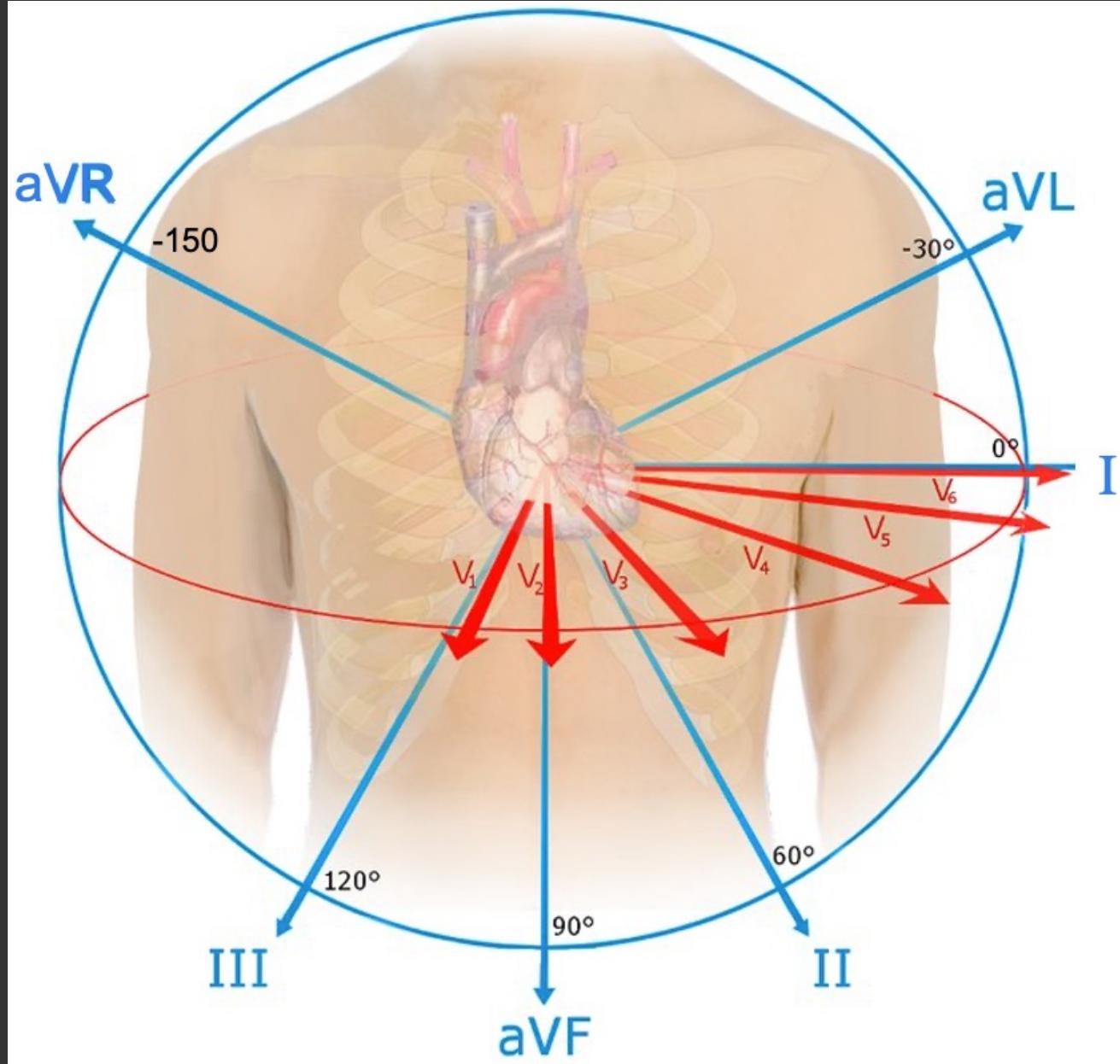


OR

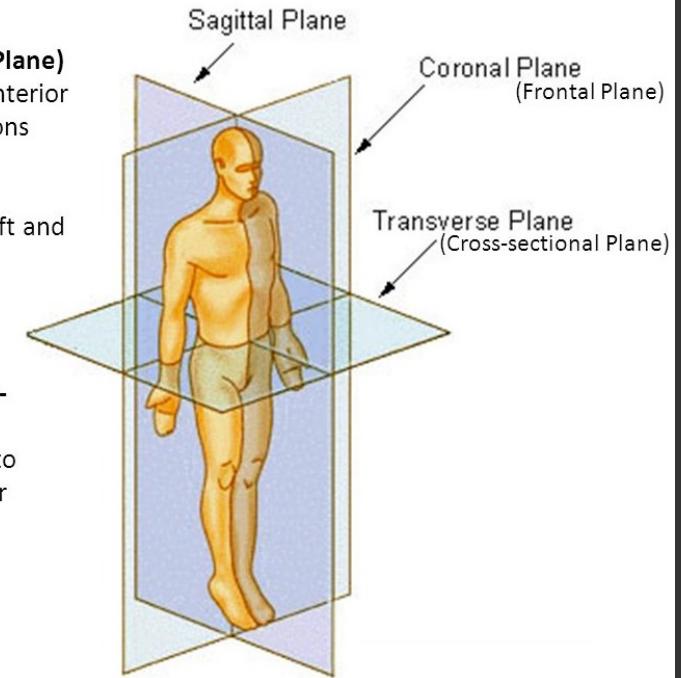


# ECG – Chest / Precordial Lead Electrode Placement



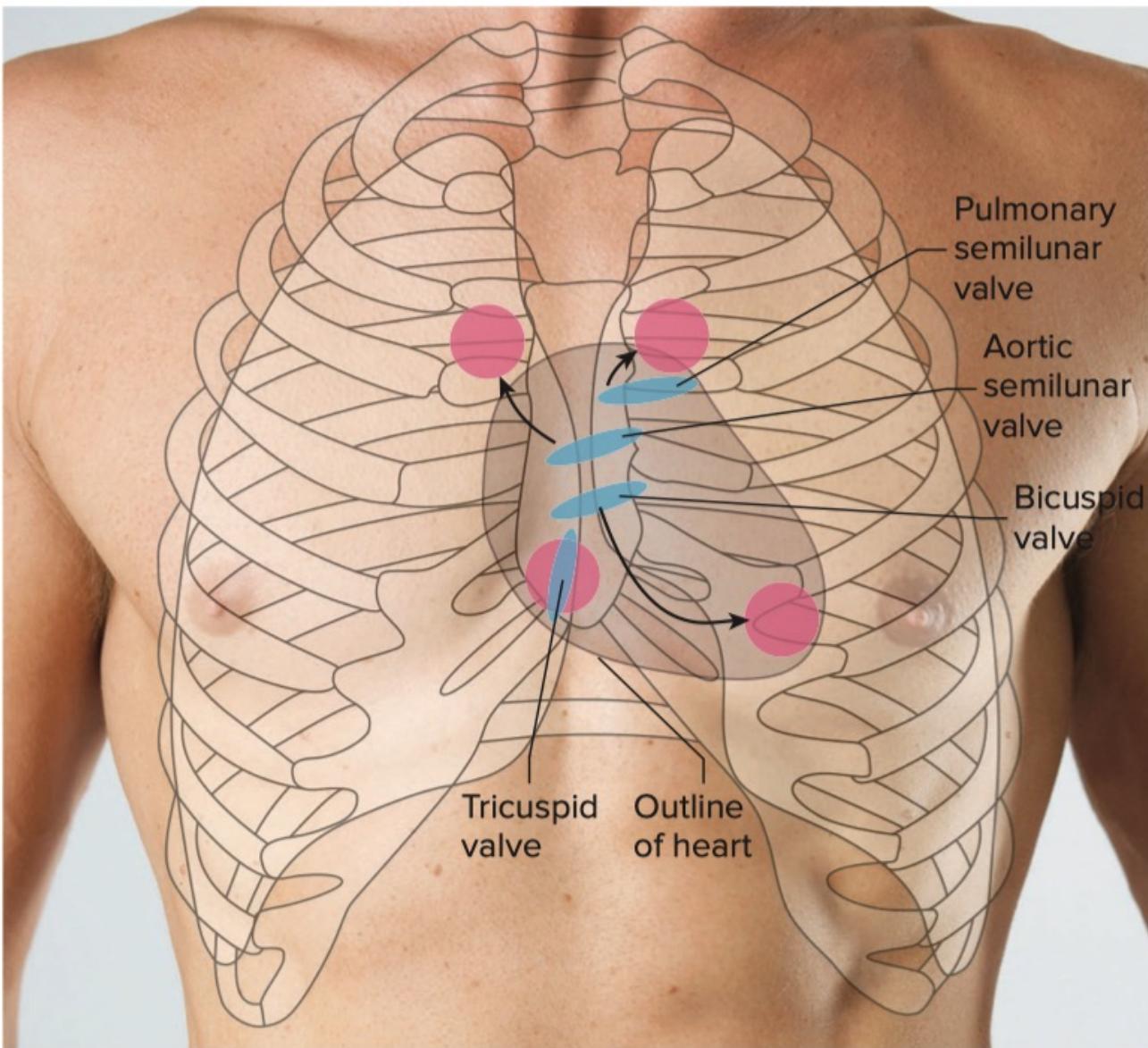


- **Coronal Plane (Frontal Plane)**
  - Divides body into anterior and posterior portions
  
- **Sagittal Plane**
  - Divides body into left and right portions
  - Midsagittal Plane
  - Parasagittal Plane
  
- **Transverse Plane (Cross-sectional Plane)**
  - Divides the body into superior and inferior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



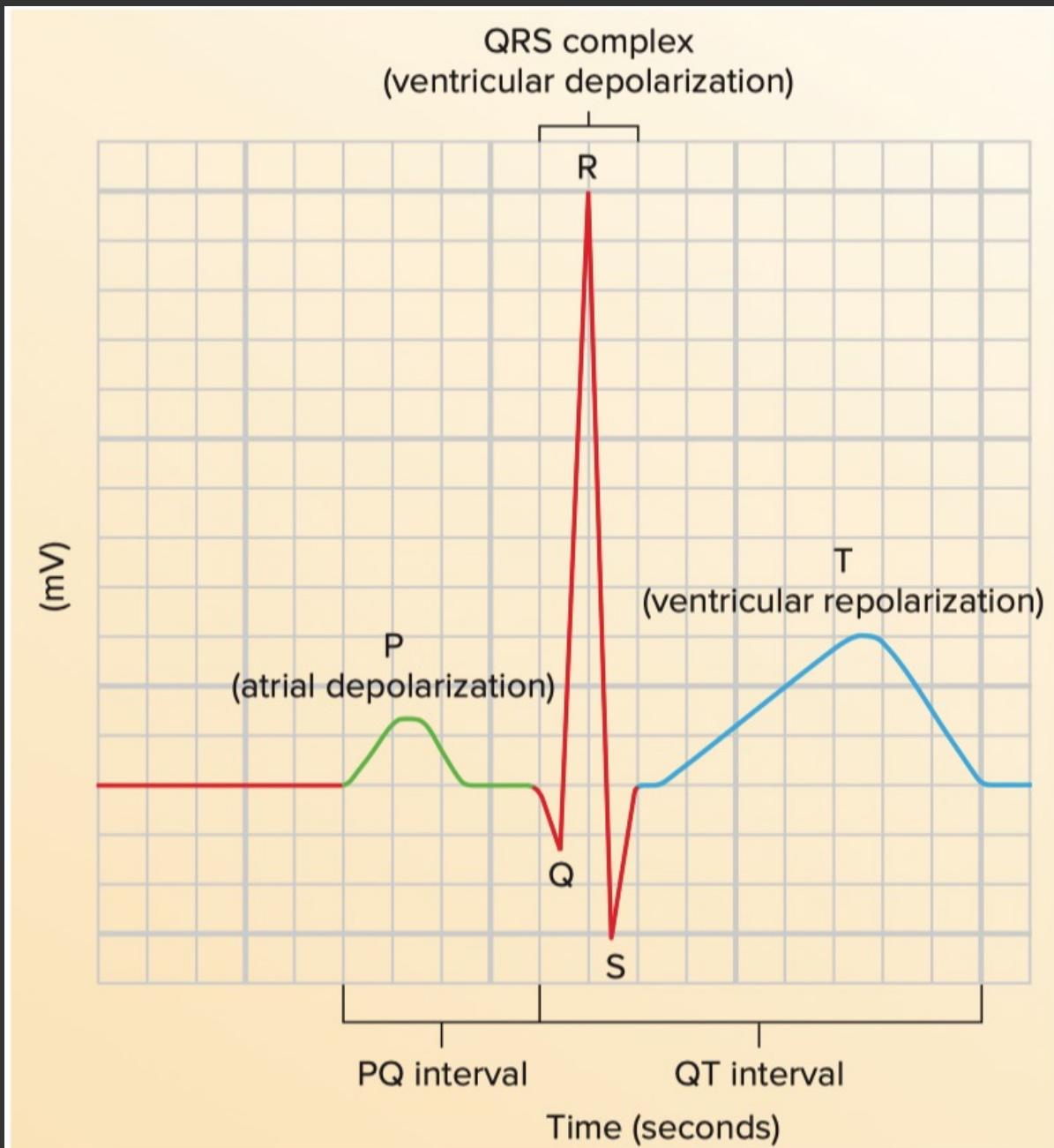
**FIGURE 20.20 Location of the Heart Valves in the Thorax**

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*.

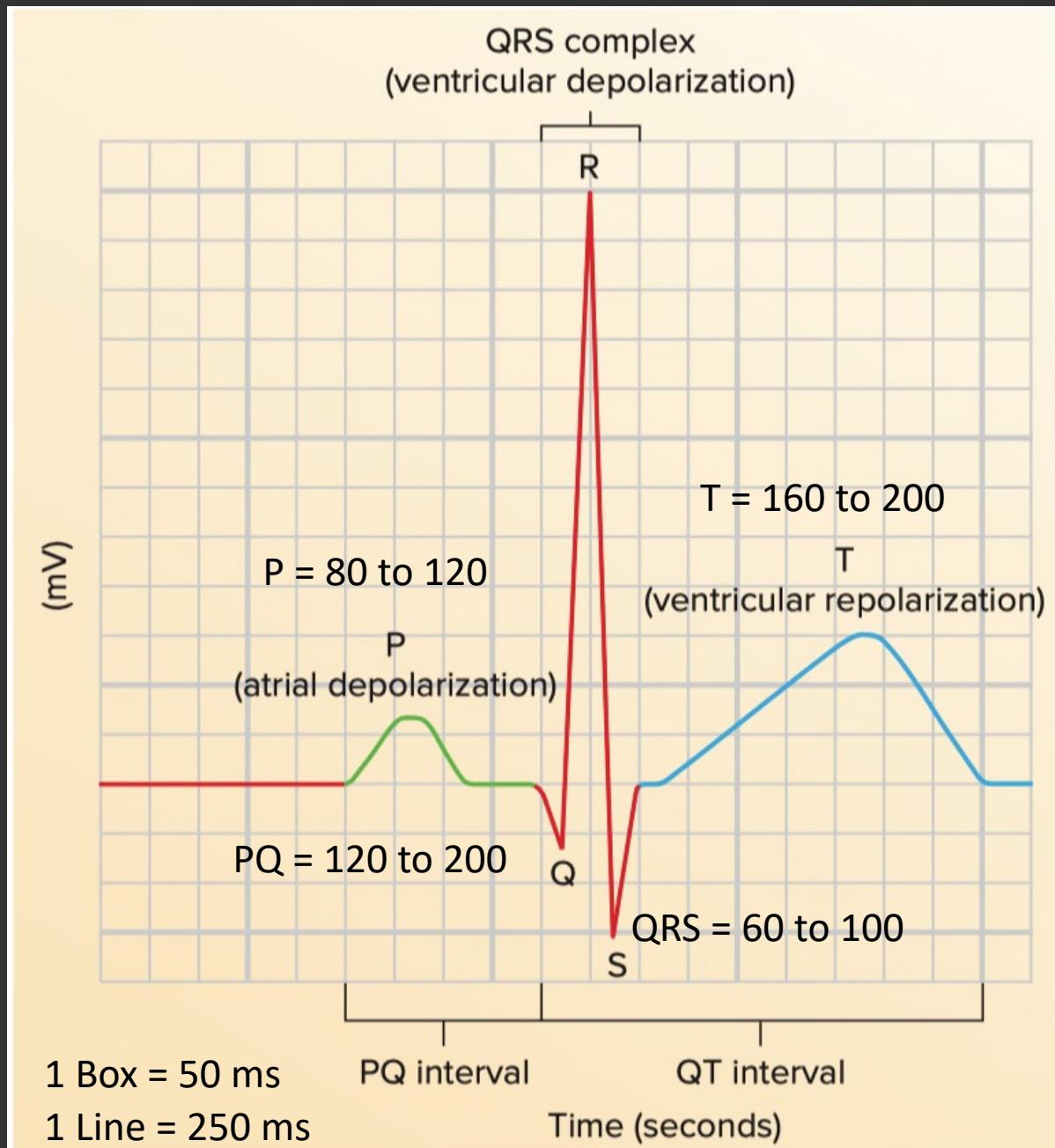
# ECG – Trace

- 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

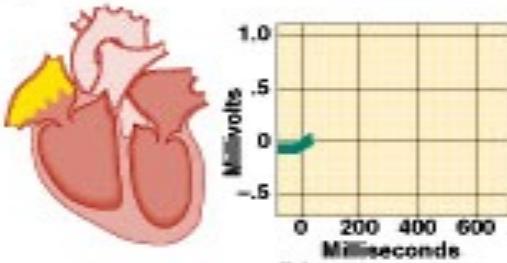


# ECG – Trace

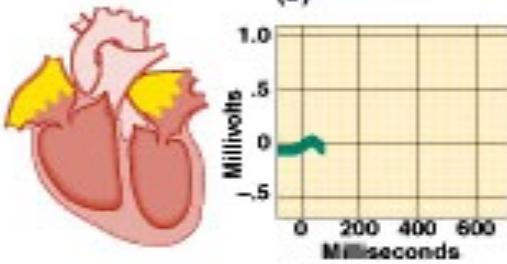




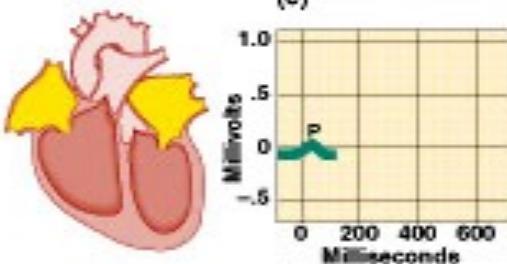
(a)



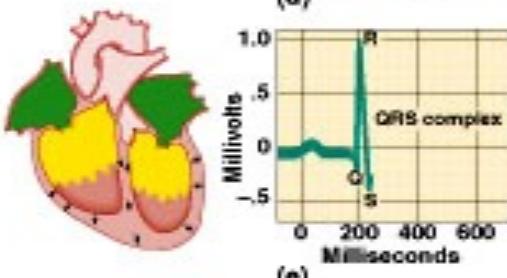
(b)



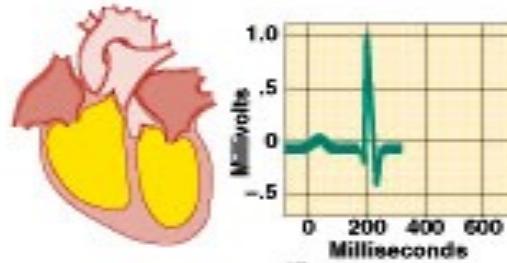
(c)



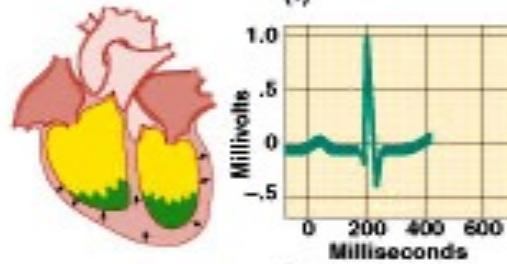
(d)



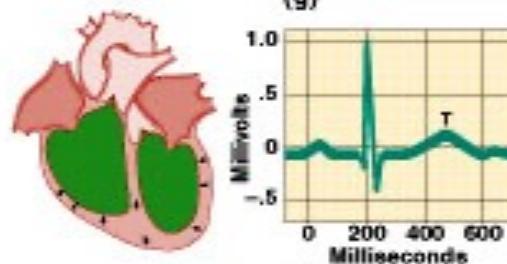
(e)



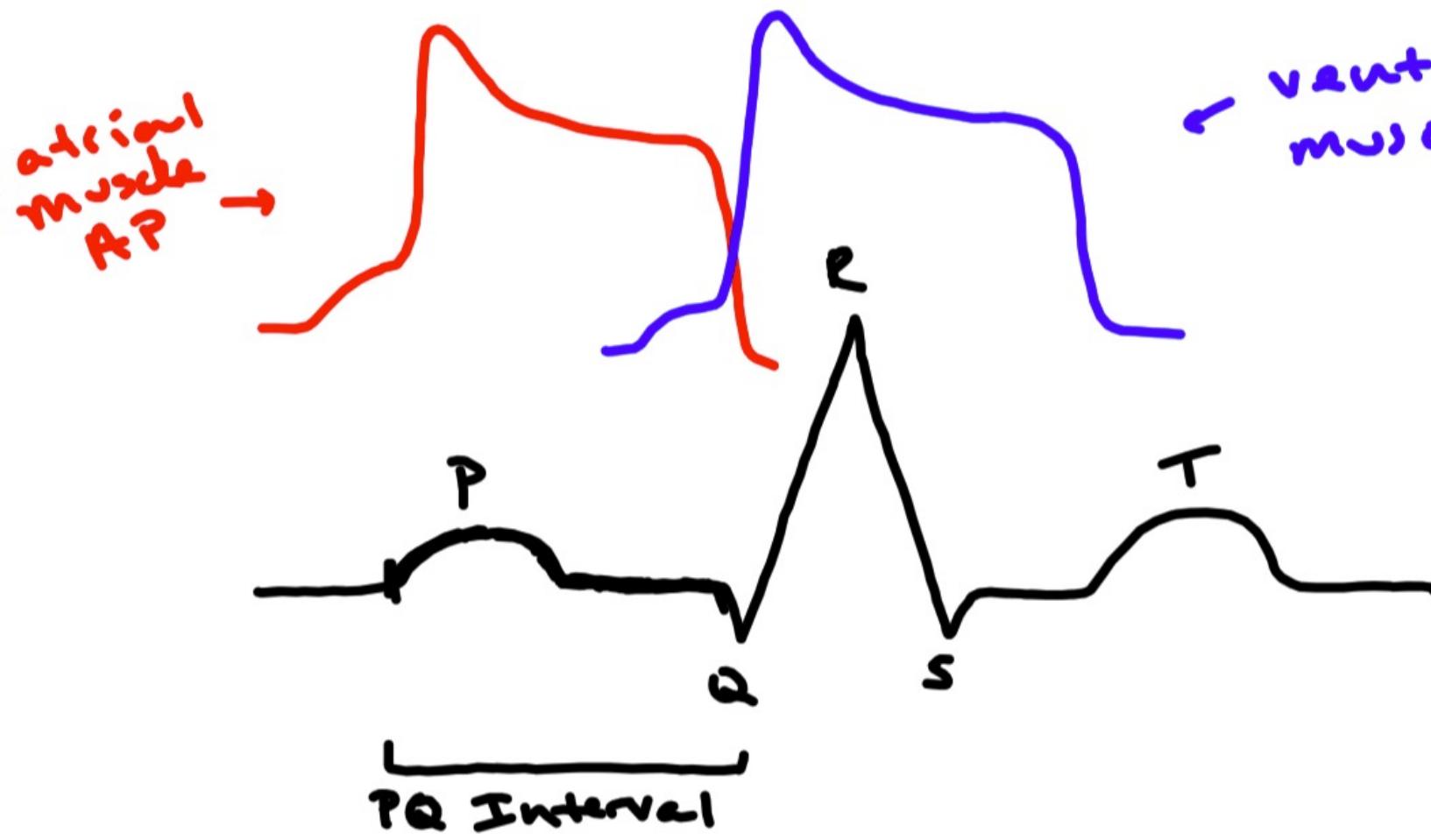
(f)



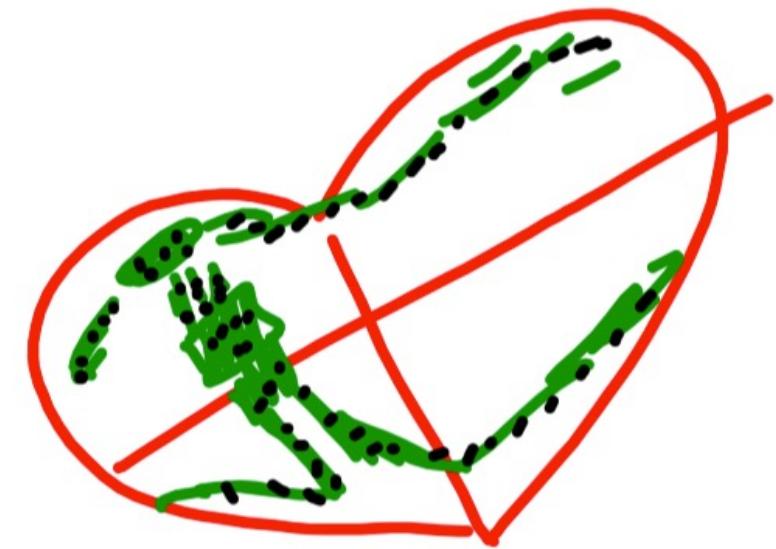
(g)



(h)



ventricular  
muscle AP



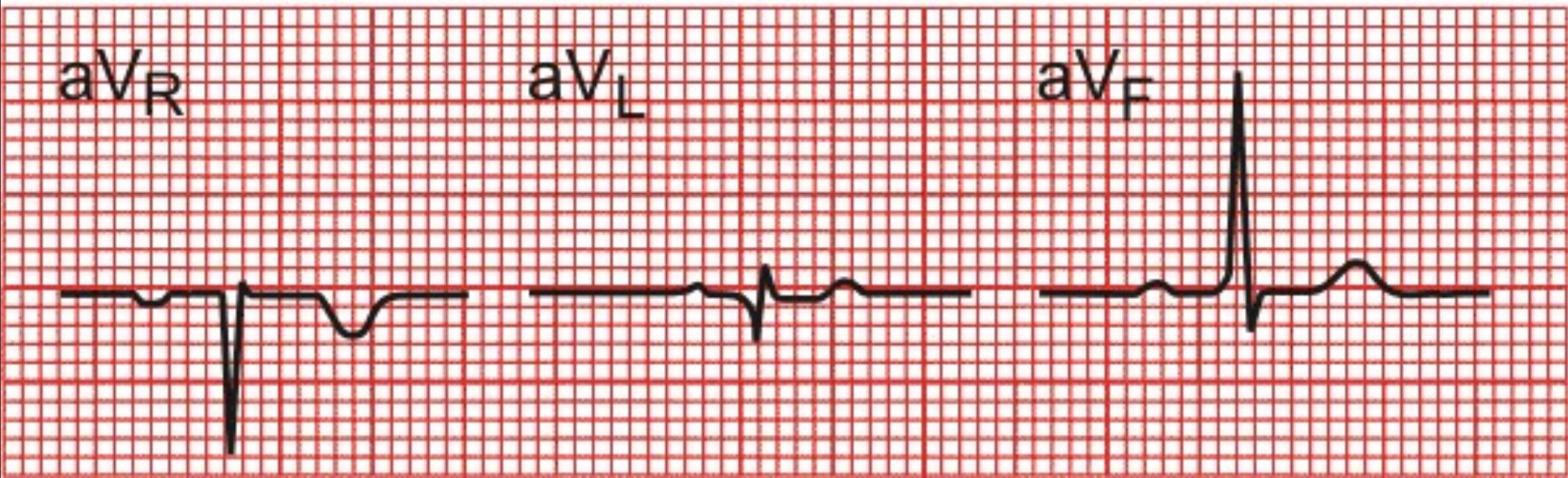
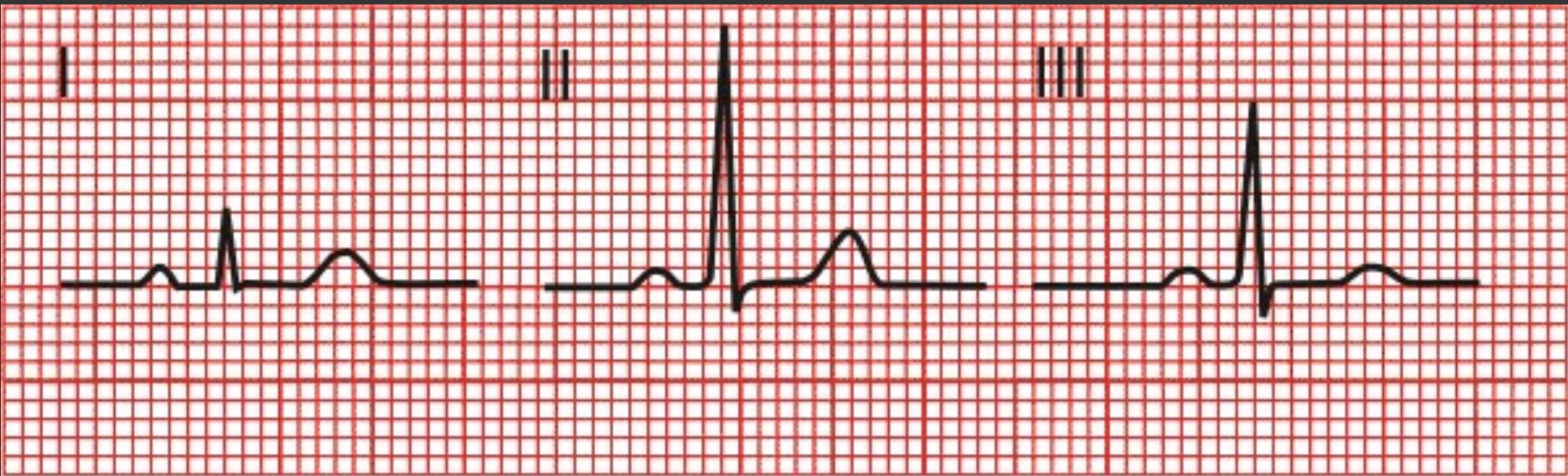
P wave: dep of atria

QRS : dep of ventricles;

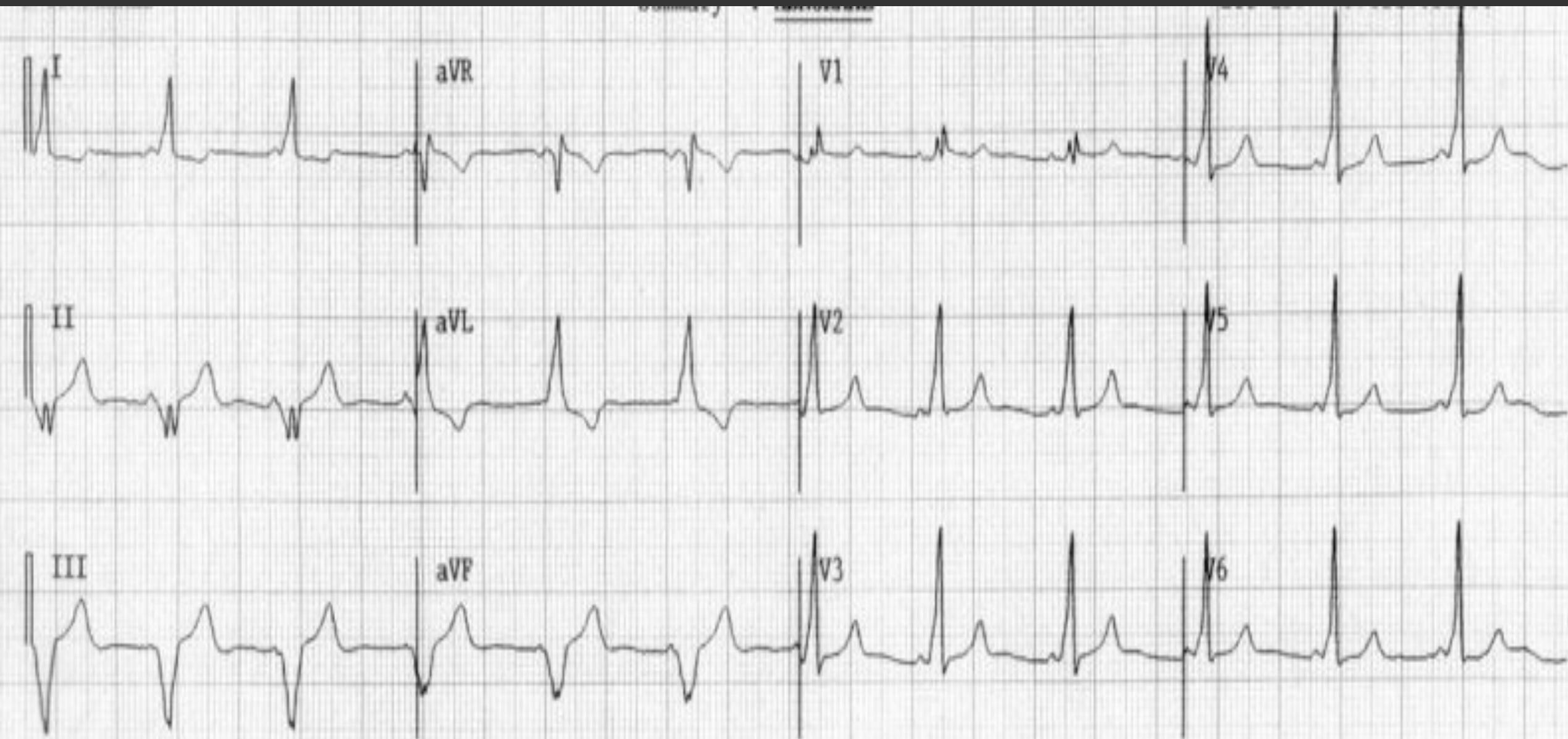
T wave : rep of ventricles

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

# ECG – Trace of Limb Leads

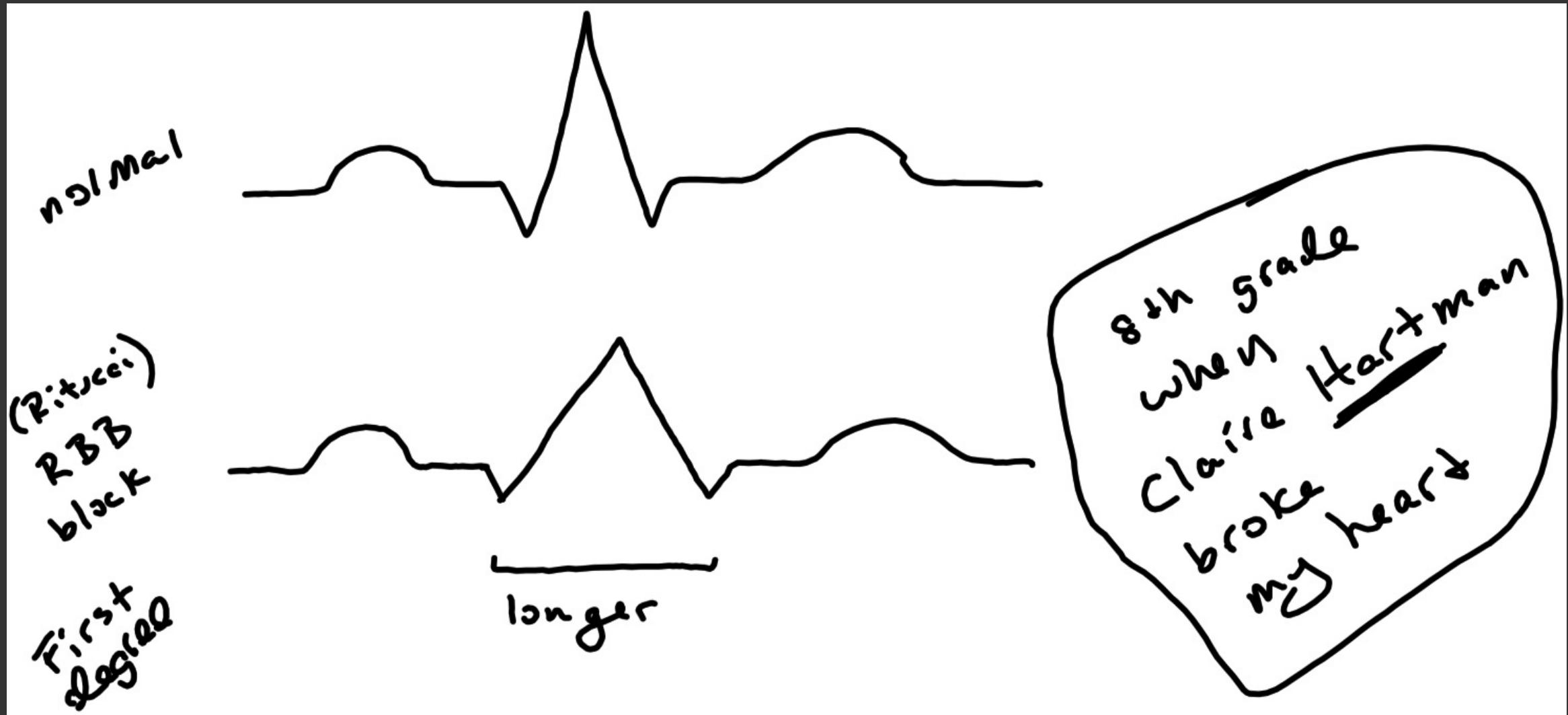


# ECG – 12 Lead Trace



# Arrhythmia / Dysrhythmia

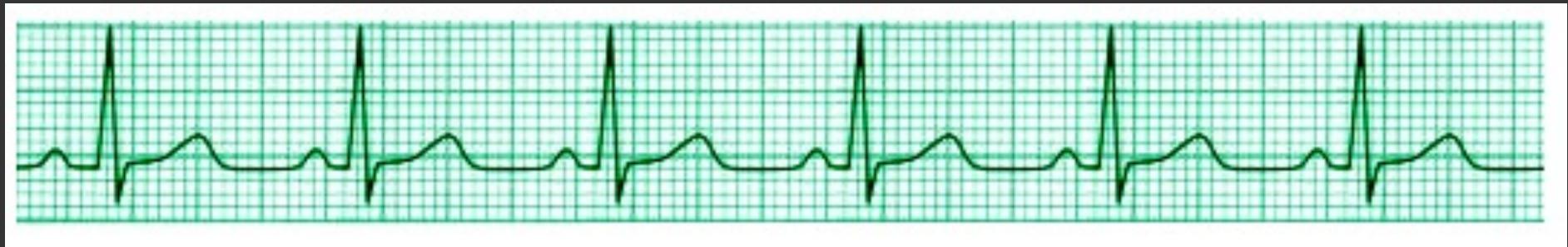
- Condition where the electrical activity is irregular



# Tachycardia

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

normal

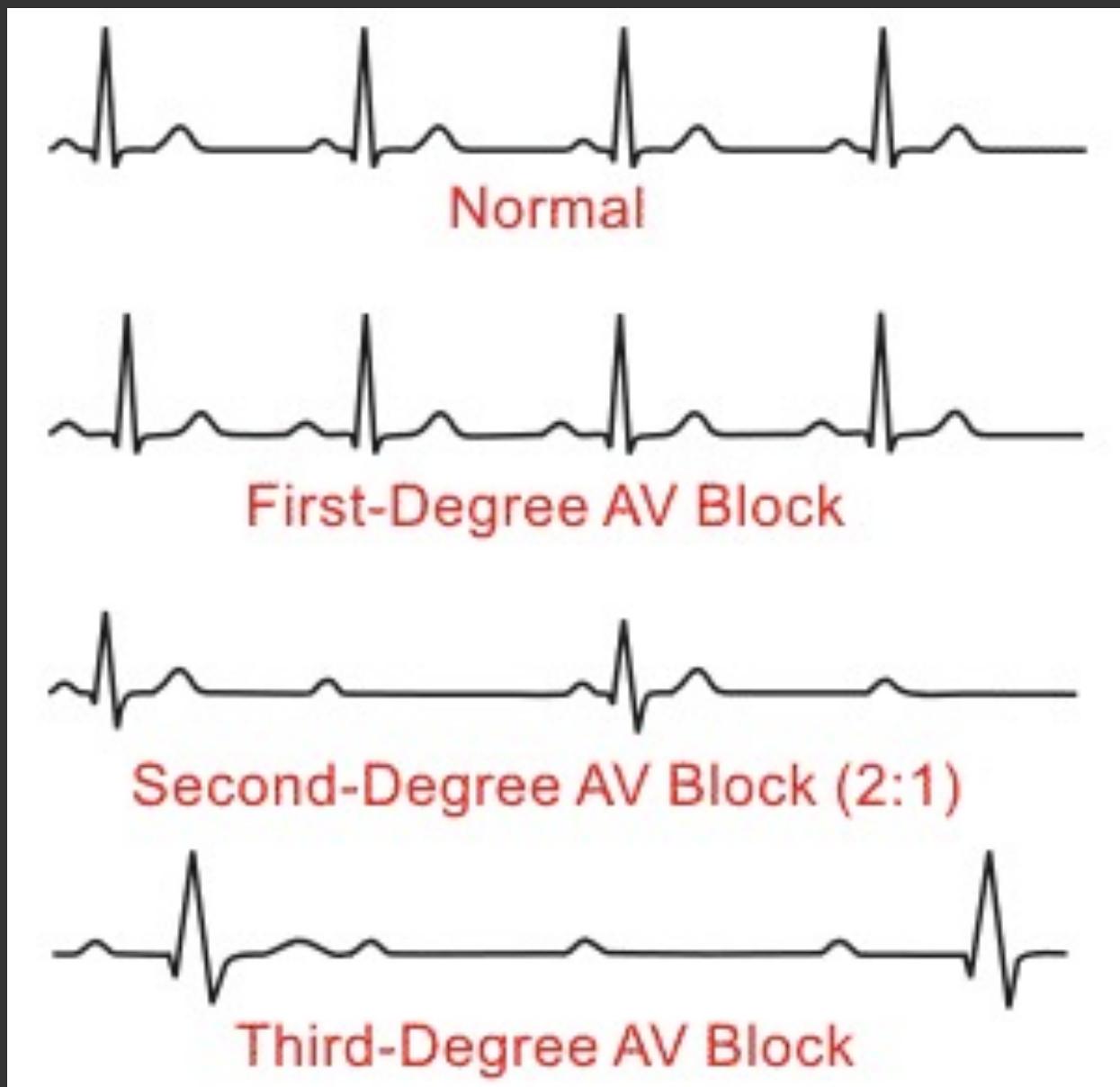


tachycardia



# 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 deg<sup>1/2</sup>  
Type I



2nd deg<sup>1/2</sup>  
Type II



probably going to need an artificial pacemaker

3rd deg<sup>1/2</sup>



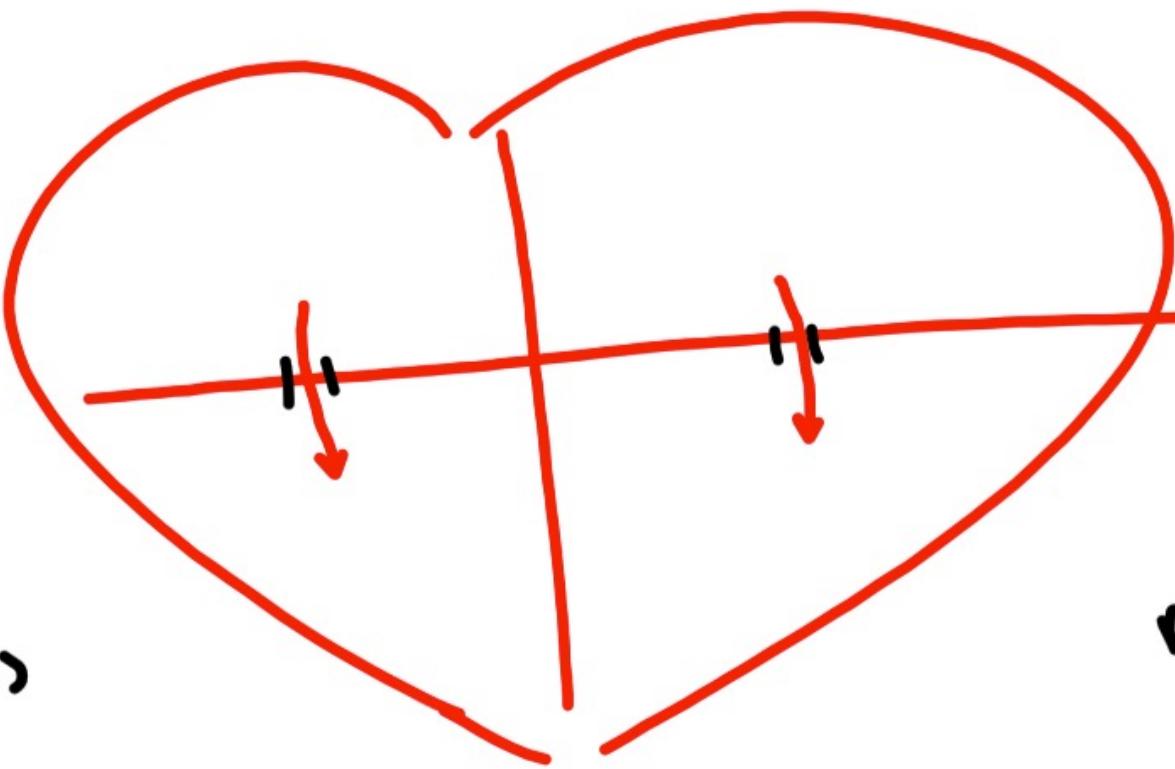
will require a pacemaker

# 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 Fib



X can lead  
to blood clots  
(in atria)

excess blood  
sits in atria  
(stasis)

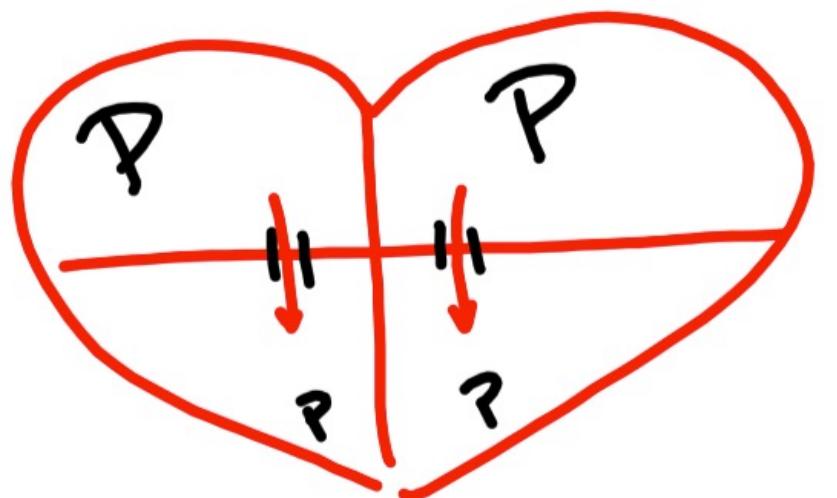
\* might not  
present  
with symptoms  
↳ Why?

most blood in atria  
delivering from  
no ventricles who  
atria contracting

# Opening and Closing of Heart Valves

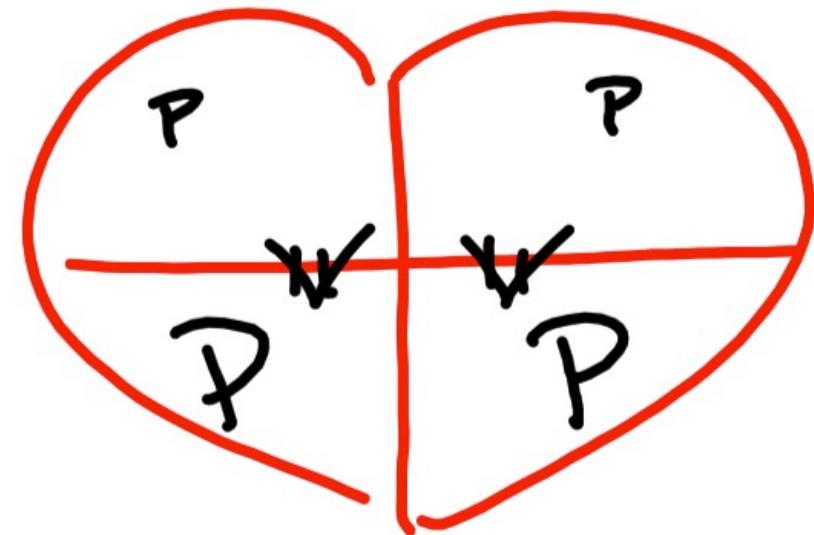
- *Valves function to allow the flow of blood through the heart in only one direction*
  - *Blood from atria to ventricles*
  - *Blood from ventricles to great arteries*
- *Opening and closing of valves is passive*
  - *Dictated by pressure changes in the chambers of the heart and great arteries*
  - *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

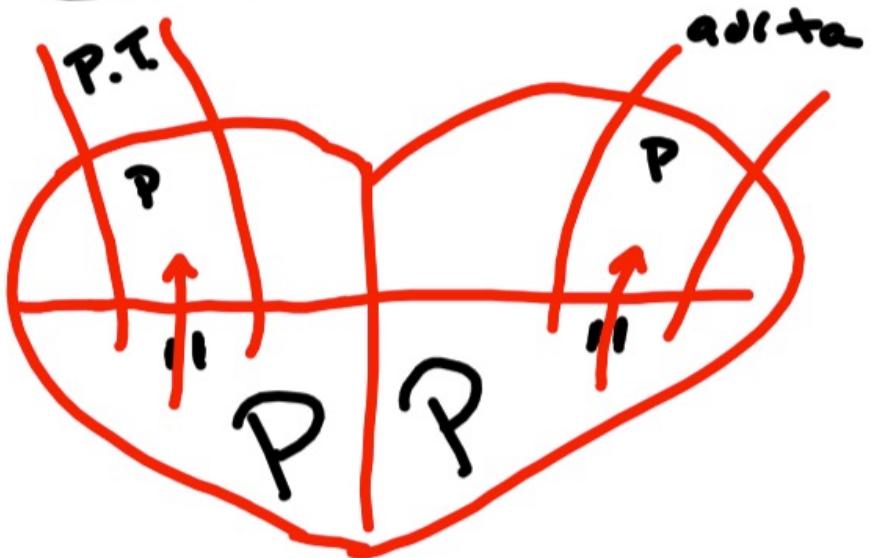
## Closing of AV valves



vent P > atria P

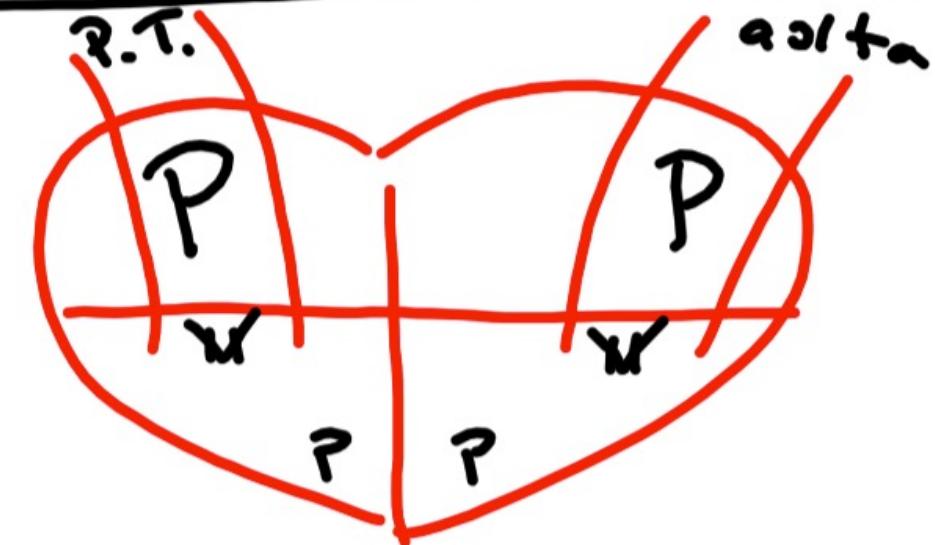
first heart sound ( $S_1$ )

## Opening of Semilunar Valves



vent P > Great Artery P

## Closing of Semilunar Valves



Great P > vent P  
Artery      |  
second heart sound ( $S_2$ )

Normal

stenosis (narrowed)



closed



opened



closed



or



heart works harder

Normal

Ins / Reg (<sup>imp's per</sup>  
closing)

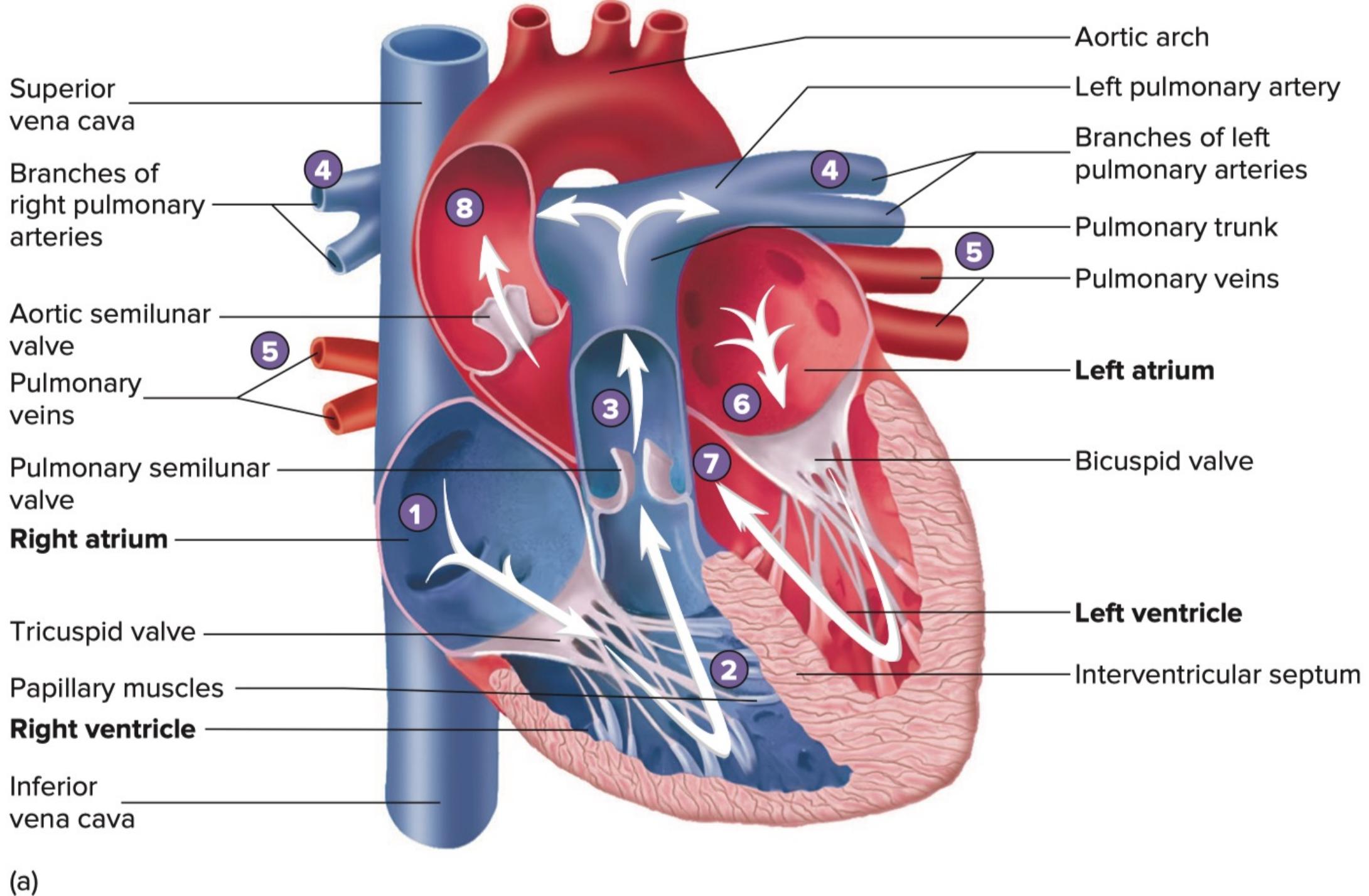
opened

closed

opened

closed

heart works harder



# 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

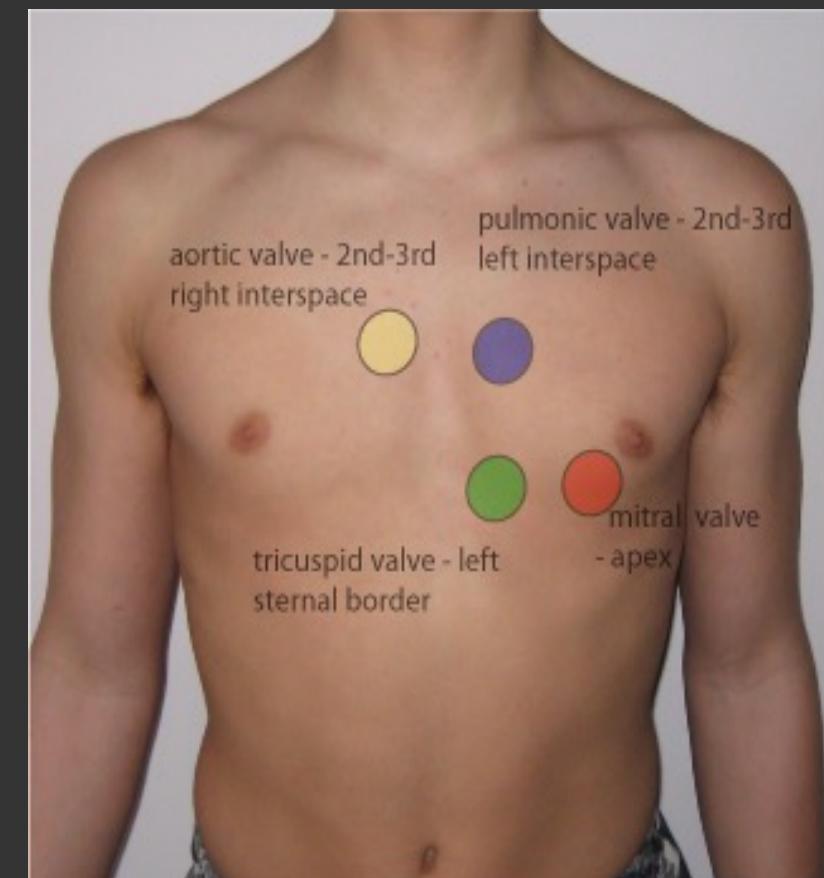
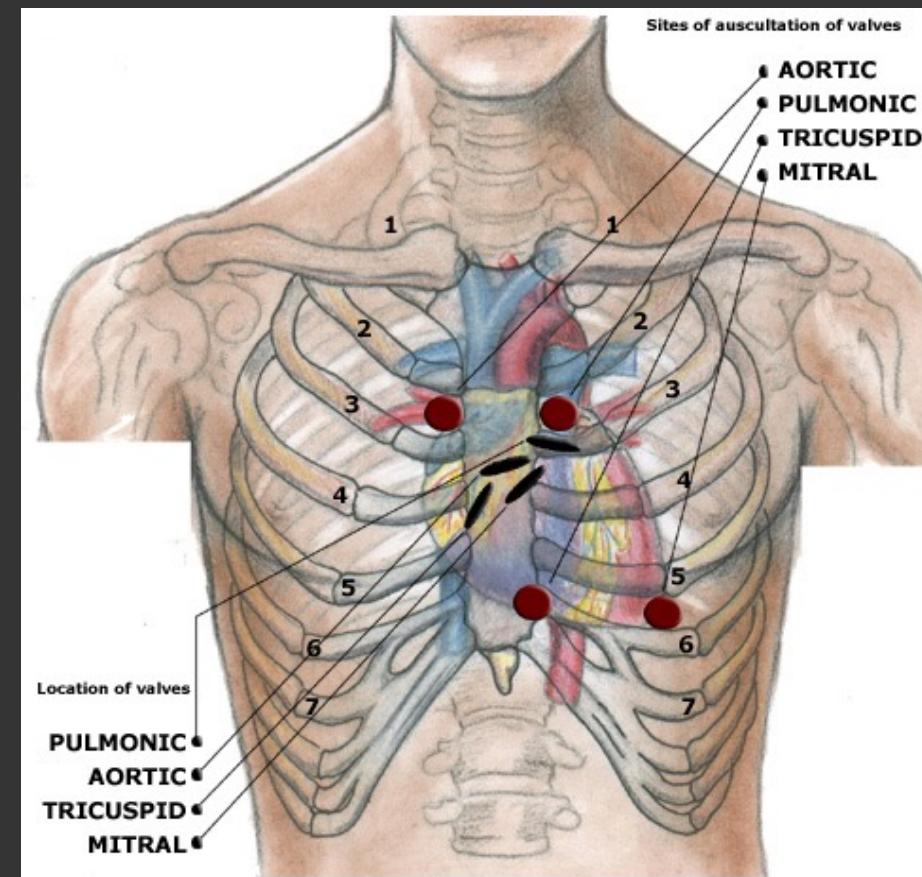
# Auscultations of the Heart

## ➤ 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  $\frac{1}{3}$  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  $\frac{2}{3}$  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

Isovolumic Relaxation

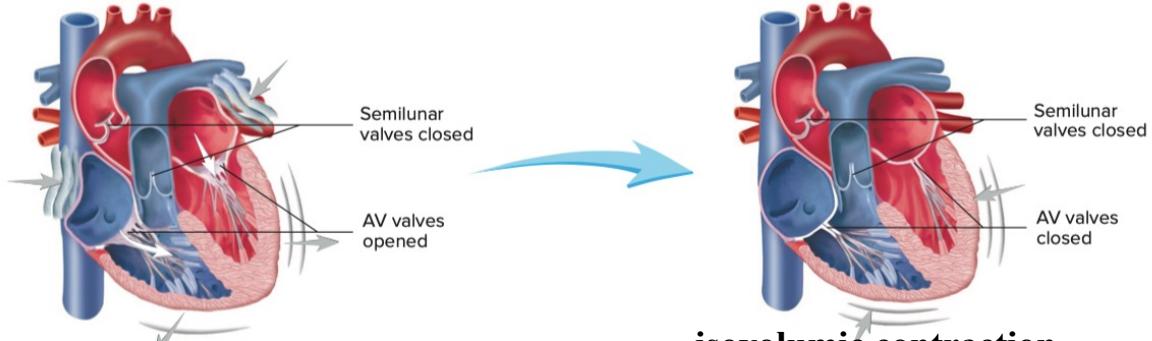
Passive Ventricular Filling

Active Ventricular Filling

} systolic

} diastolic

# Systole

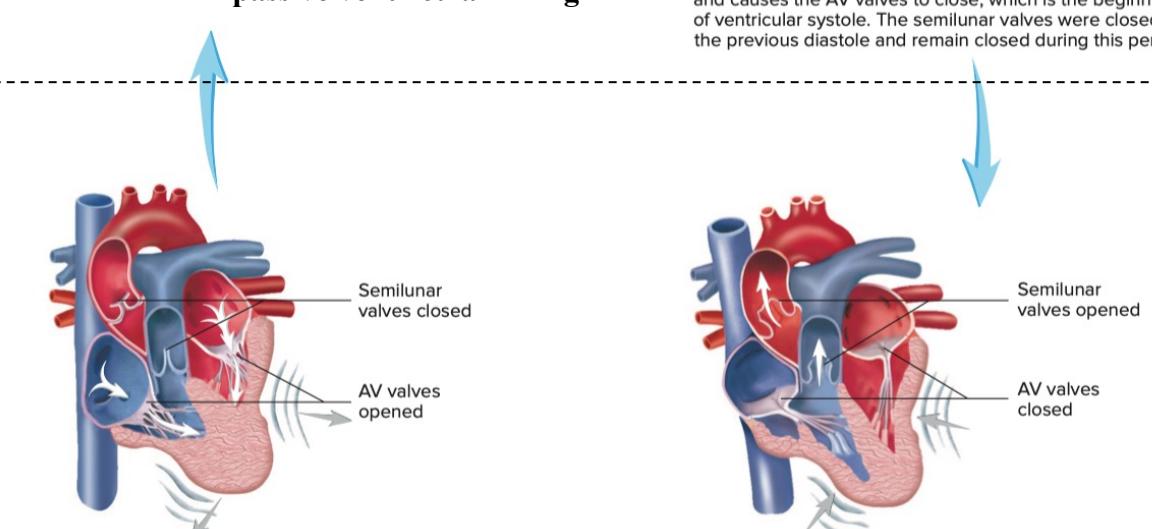


- ① Atrial systole: active ventricular filling. The atria contract, increasing atrial pressure and completing ventricular filling while the ventricles are relaxed.

## passive ventricular filling

- ② Ventricular systole: period of isovolumetric contraction. The atria are relaxed, and blood flows into them from the veins. Ventricular contraction causes ventricular pressure to increase and causes the AV valves to close, which is the beginning of ventricular systole. The semilunar valves were closed in the previous diastole and remain closed during this period.

# Diastole

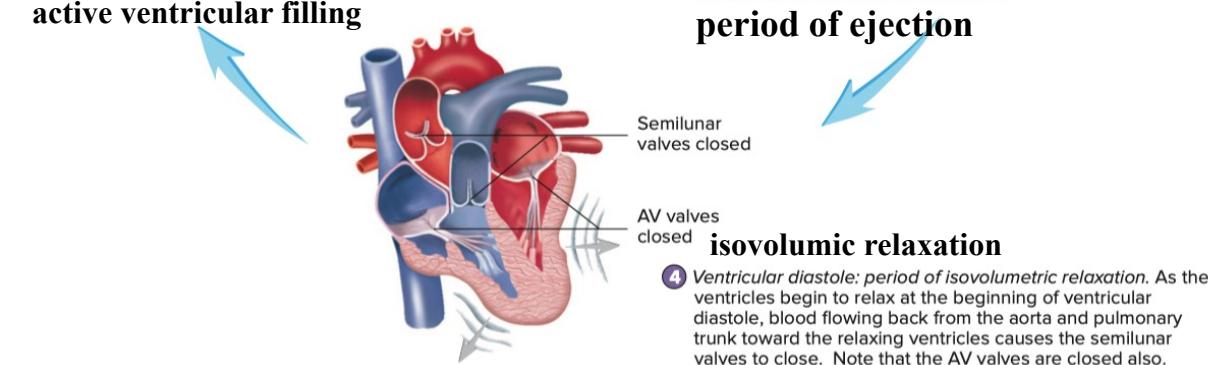


- ⑤ Ventricular diastole: passive ventricular filling. As ventricular relaxation continues, the AV valves open, and blood flows from the atria into the relaxing ventricles, accounting for most of the ventricular filling.

## active ventricular filling

- ③ Ventricular systole: period of ejection. Continued ventricular contraction causes a greater increase in ventricular pressure, which pushes blood out of the ventricles, causing the semilunar valves to open.

## period of ejection



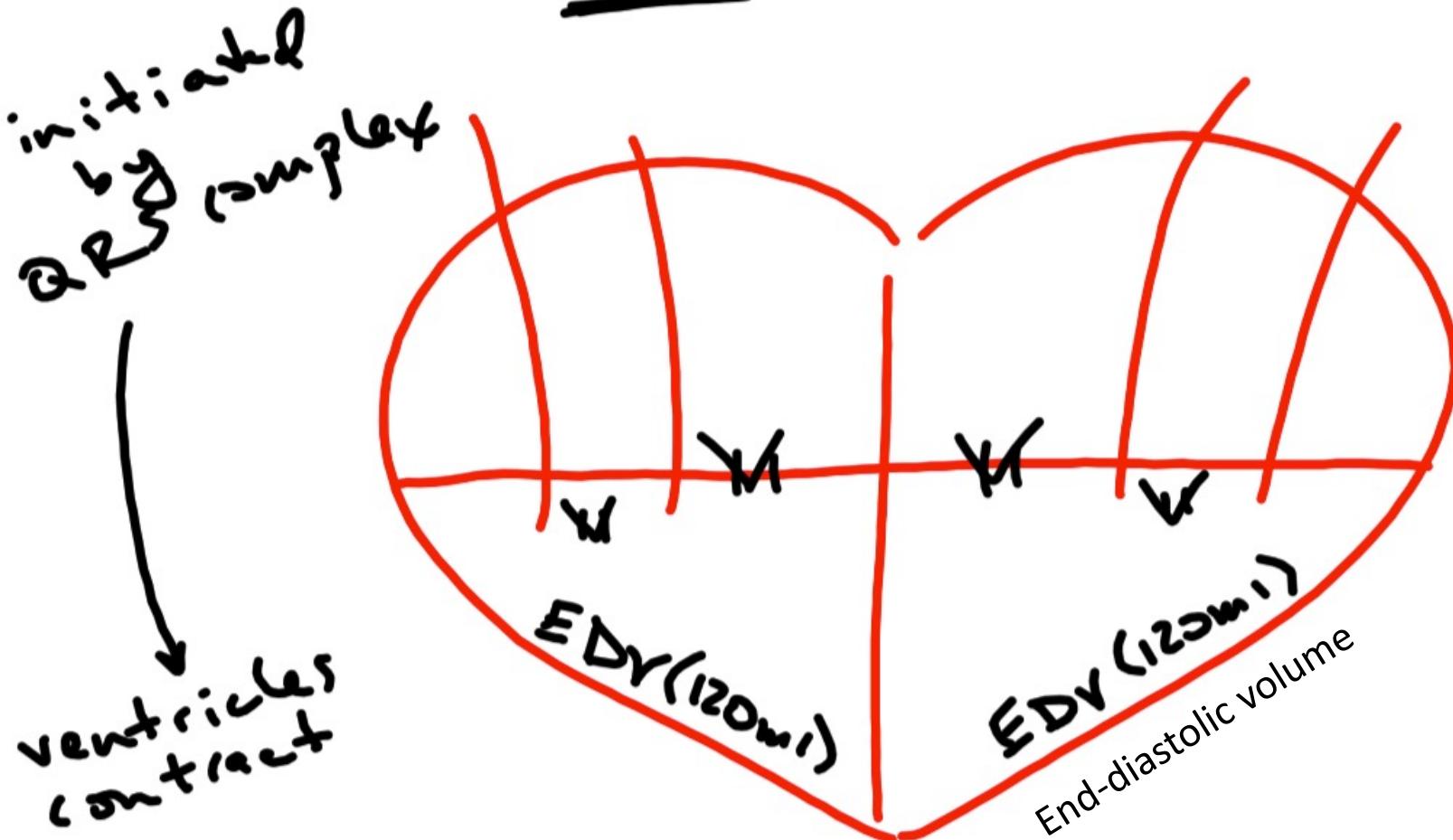
- ④ Ventricular diastole: period of isovolumetric relaxation. As the ventricles begin to relax at the beginning of ventricular diastole, blood flowing back from the aorta and pulmonary trunk toward the relaxing ventricles causes the semilunar valves to close. Note that the AV valves are closed also.

## isovolumic relaxation

# 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



$T_{vent P} \rightarrow vent P > atrial P \rightarrow AV valves close (S.)$

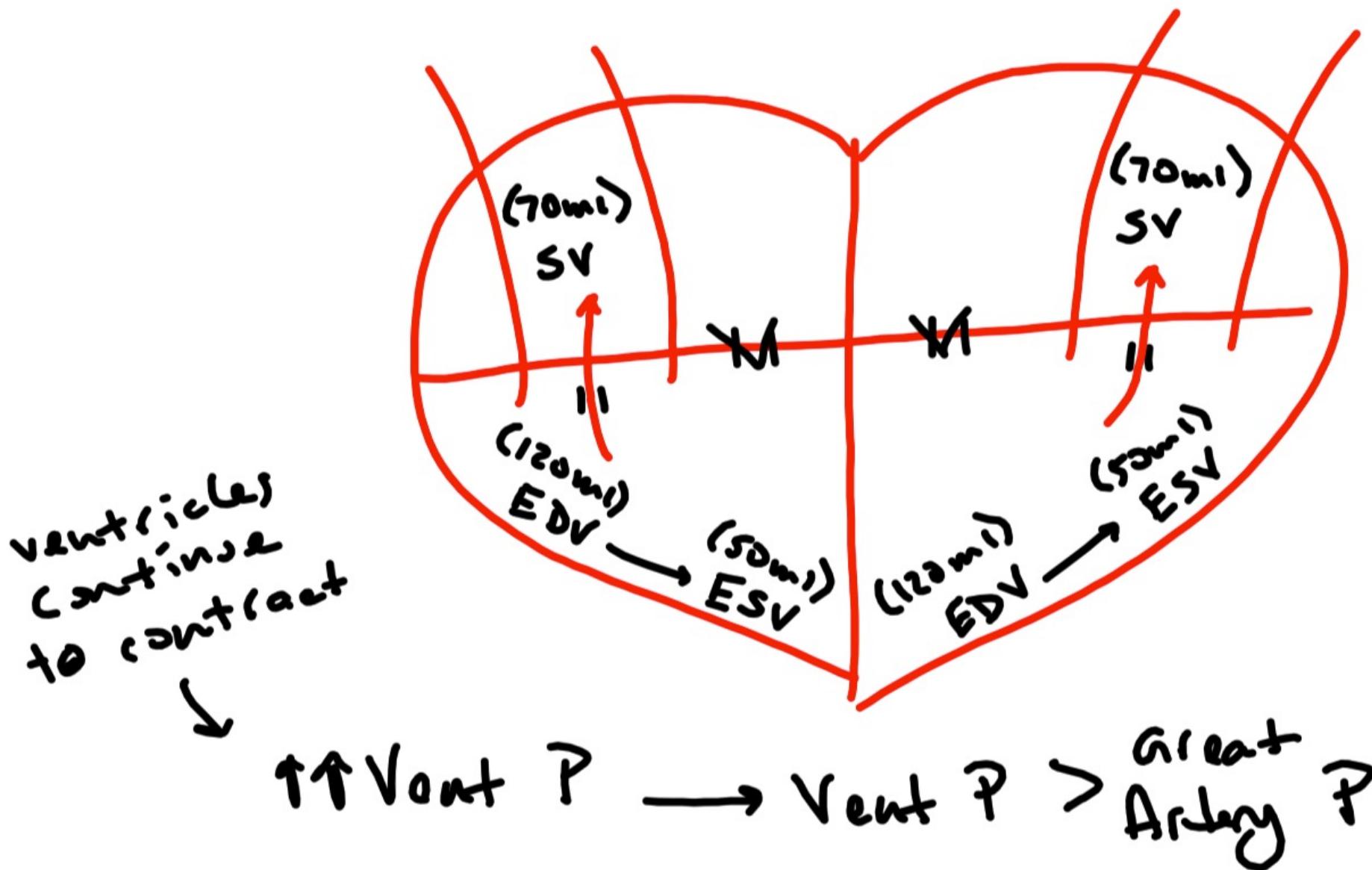
mitral & 2 reg  
an AV ins reg  
could be heal &

semilunar  
valves would  
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 )
  - $EF = ( SV ) / ( EDV ) \times 100$
- 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



maximum QRS  
to aortal  
semilunar  
sinusos;

$\uparrow\uparrow V_{out} P$  → Vent P > <sup>great</sup> Artery P → open semilunar valves

# Cardiac Cycle – Diastole

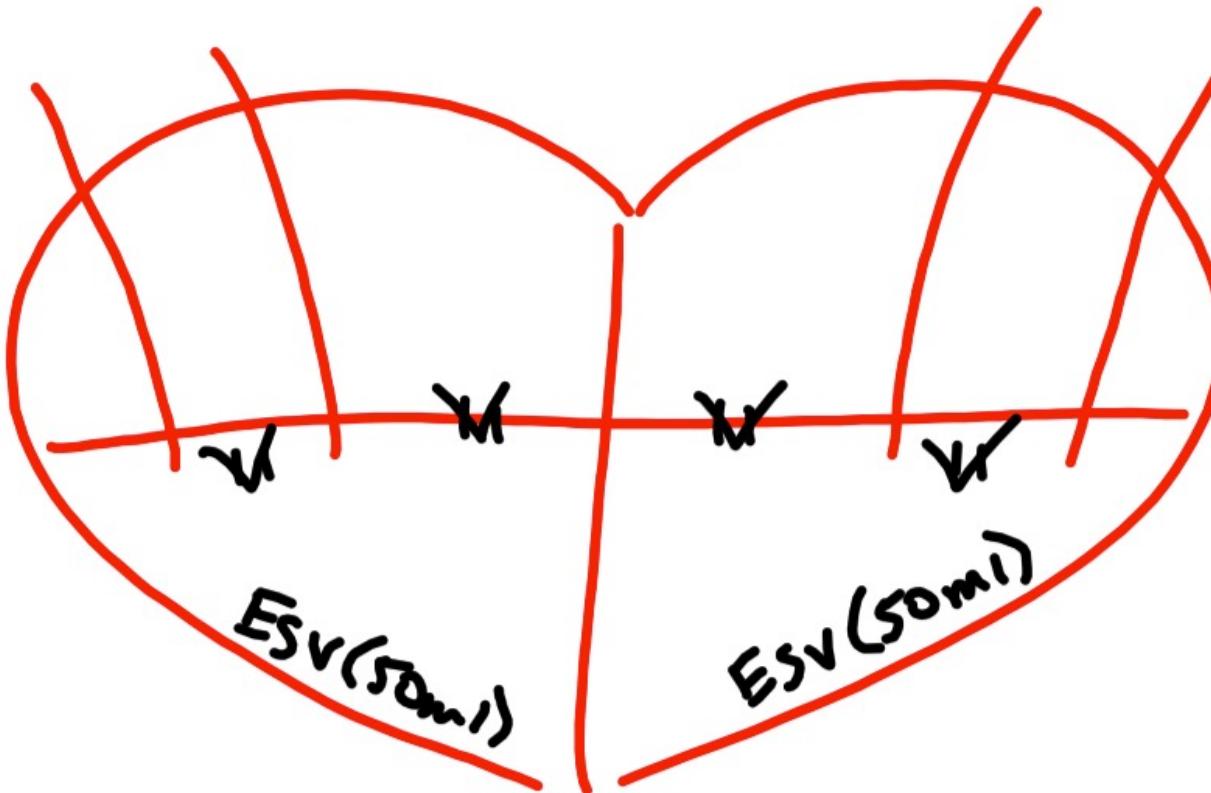
- Isovolumic relaxation ( part of diastole )
  - Relaxation of ventricles
    - Ventricular pressure decreases below pressure of great arteries
      - Semilunar valves snap shut ( all heart valves are now closed )
        - Ventricular blood volume does not change
        - Second heart sound ( S2 )
        - 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  $\leftarrow$  Diastole

target artery of SV  
because of SV

ventricles are relaxed  
↓ ventricular blood volume

$\downarrow V_{\text{vent}} P \rightarrow$  Great Artery  $P > \text{vent}?$



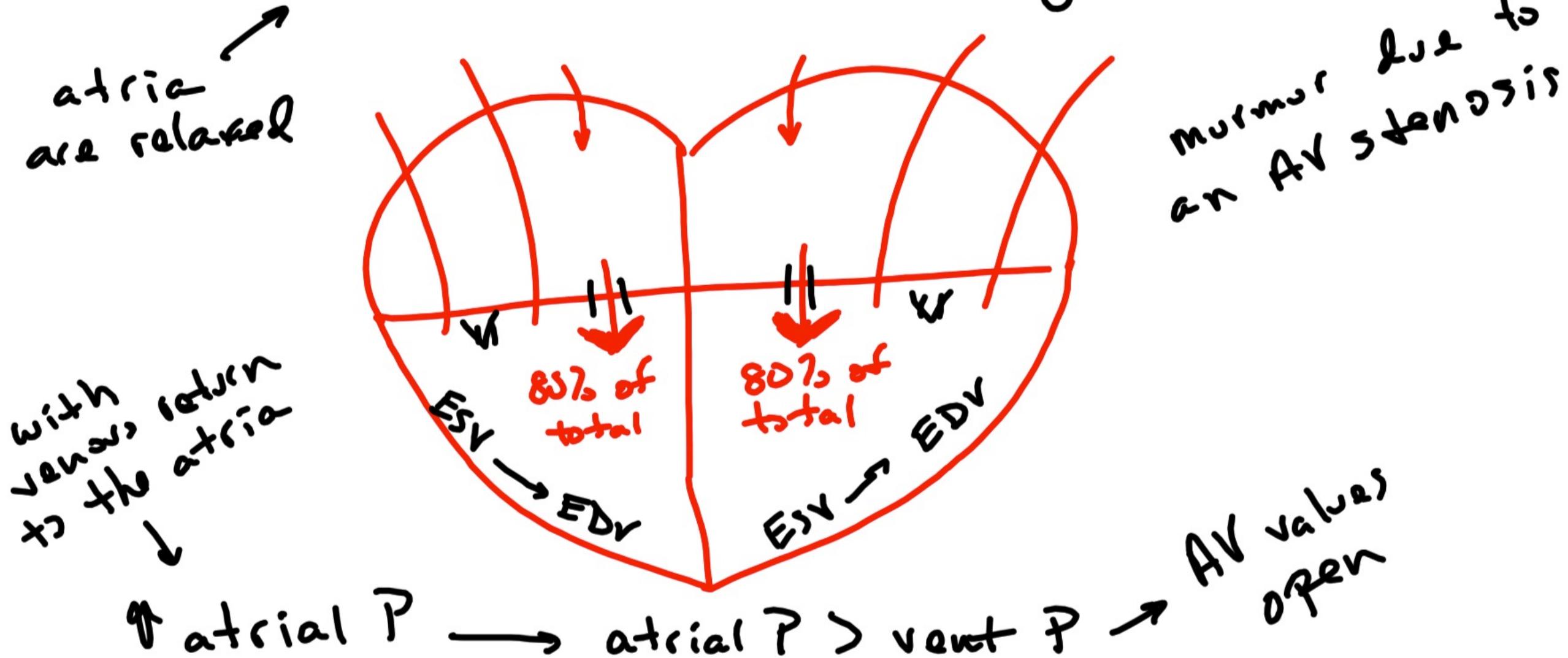
murmur due to a semilunar valve

(S2) close semilunar values

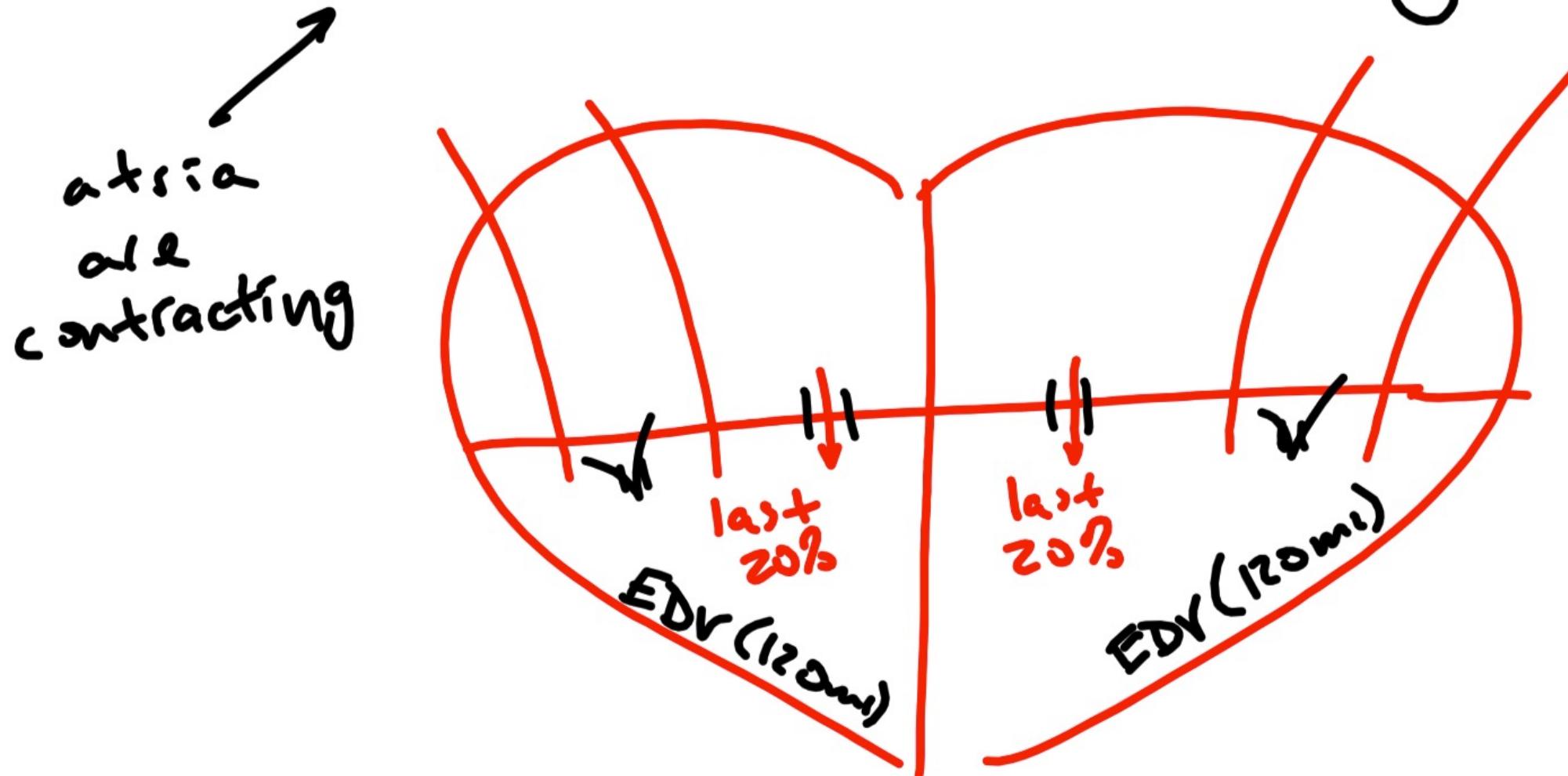
AV valves already closed

all valves closed

# Passive Ventricular Filling ← diastole



# Active Ventricular Filling ← Diastole



# Cardiac Output ( CO )

- Volume of blood pumped by either the left or right ventricle per minute
- $CO = ( SV ) \times ( HR )$
- $= ( 70 \text{ ml blood / heart beat} ) \times ( 70 \text{ heart beats / minute} )$
- $= 4,900 \text{ ml blood / minute or } 4.9 \text{ liters blood / minute}$
- 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

$$ESV = EDV - SV$$

$$SV = EDV - ESV$$

$$EDV = ESV + SV$$

---

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

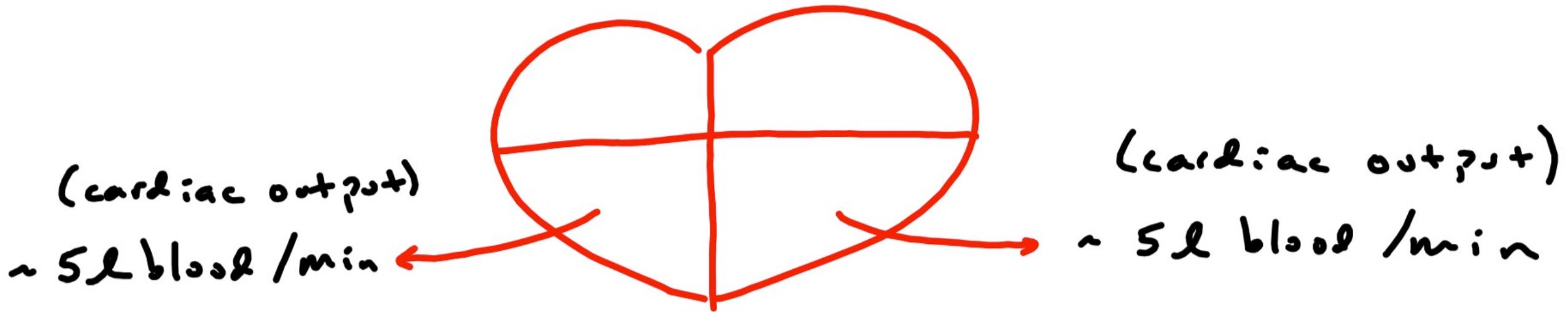
an E.F. greater than  
50% is normal

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

\* unhealthy heart (i.e. heart failure)  
↳ weak heart muscle

$$E.F. = \frac{35\text{ml}}{120\text{ml}} \cdot 100$$

$$= 25\%$$



$$CO = HR \times SV$$

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

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

\* Efficient Heart:

↓ HR      ↗ TSV



$$\text{CO} = \frac{\cancel{50 \text{ beats}}}{\text{min}} \times \frac{\cancel{100 \text{ ml blood}}}{\cancel{\text{beat}}} \\ = 5 \text{l/min}$$

# 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

<b>Preload</b>	<b>Stroke Volume</b>	<b>Afterload</b>	<b>Stroke Volume</b>
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

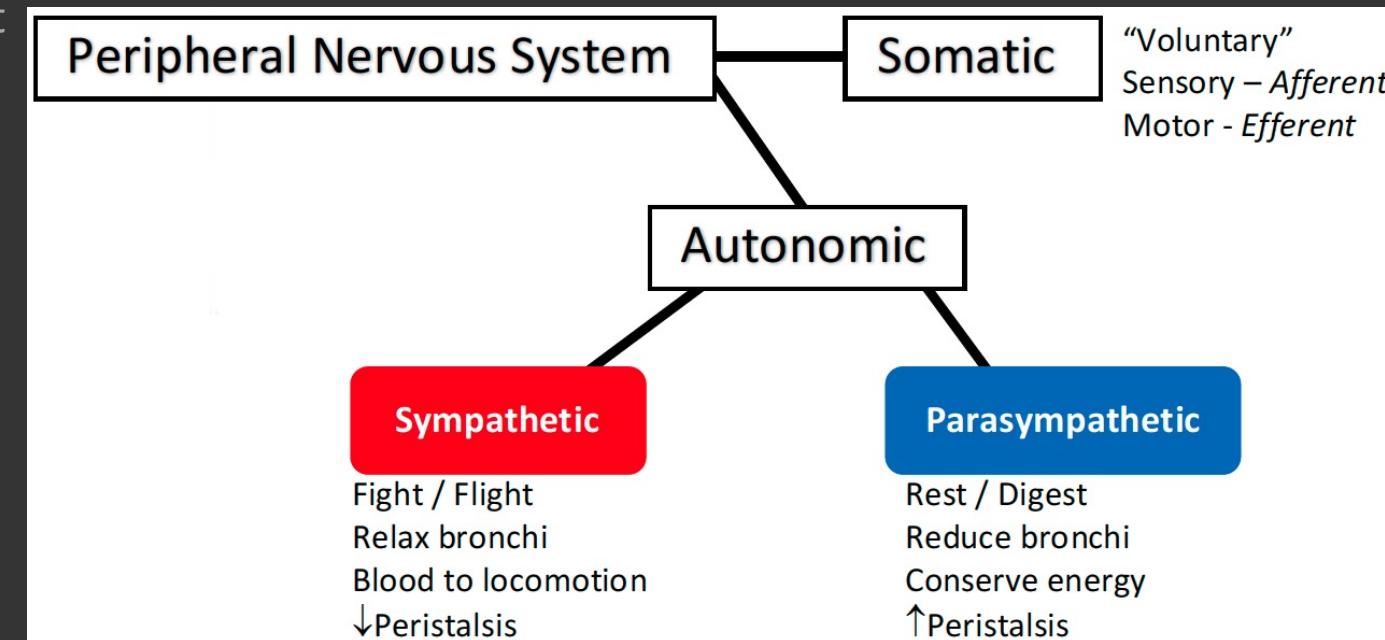
- Primary controller of resting heart rate

- Innervates SA node , AV node , atrial myocardium

- Has an inhibitory influence on the heart

- Decreases heart rate

- Negative chronotropic effect

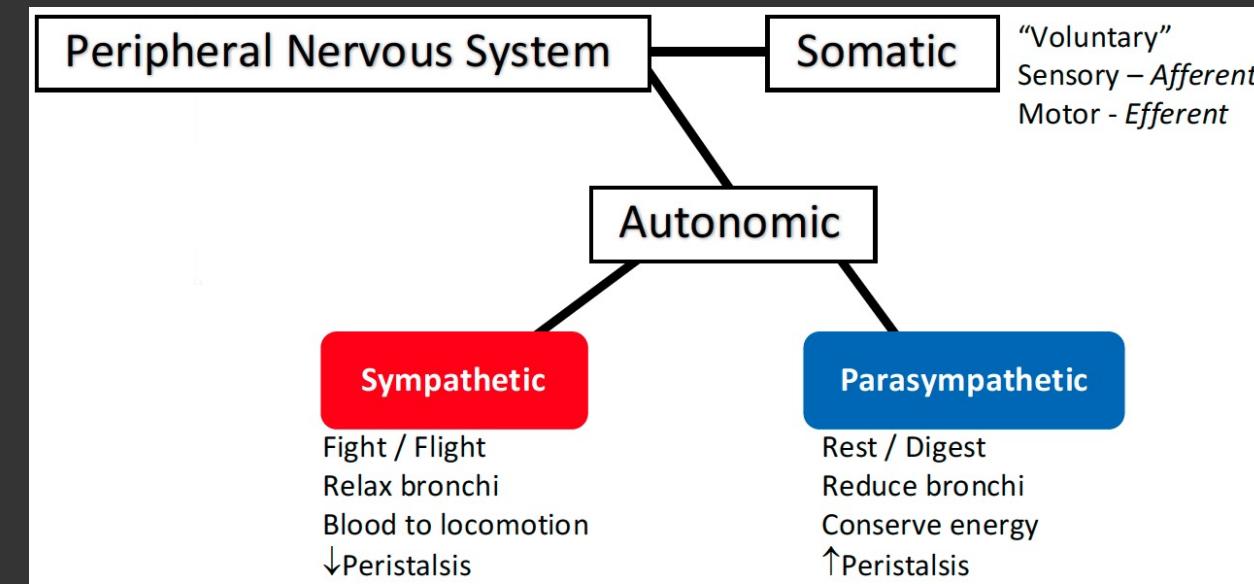


# Extrinsic Factors – Cardiac Control Center

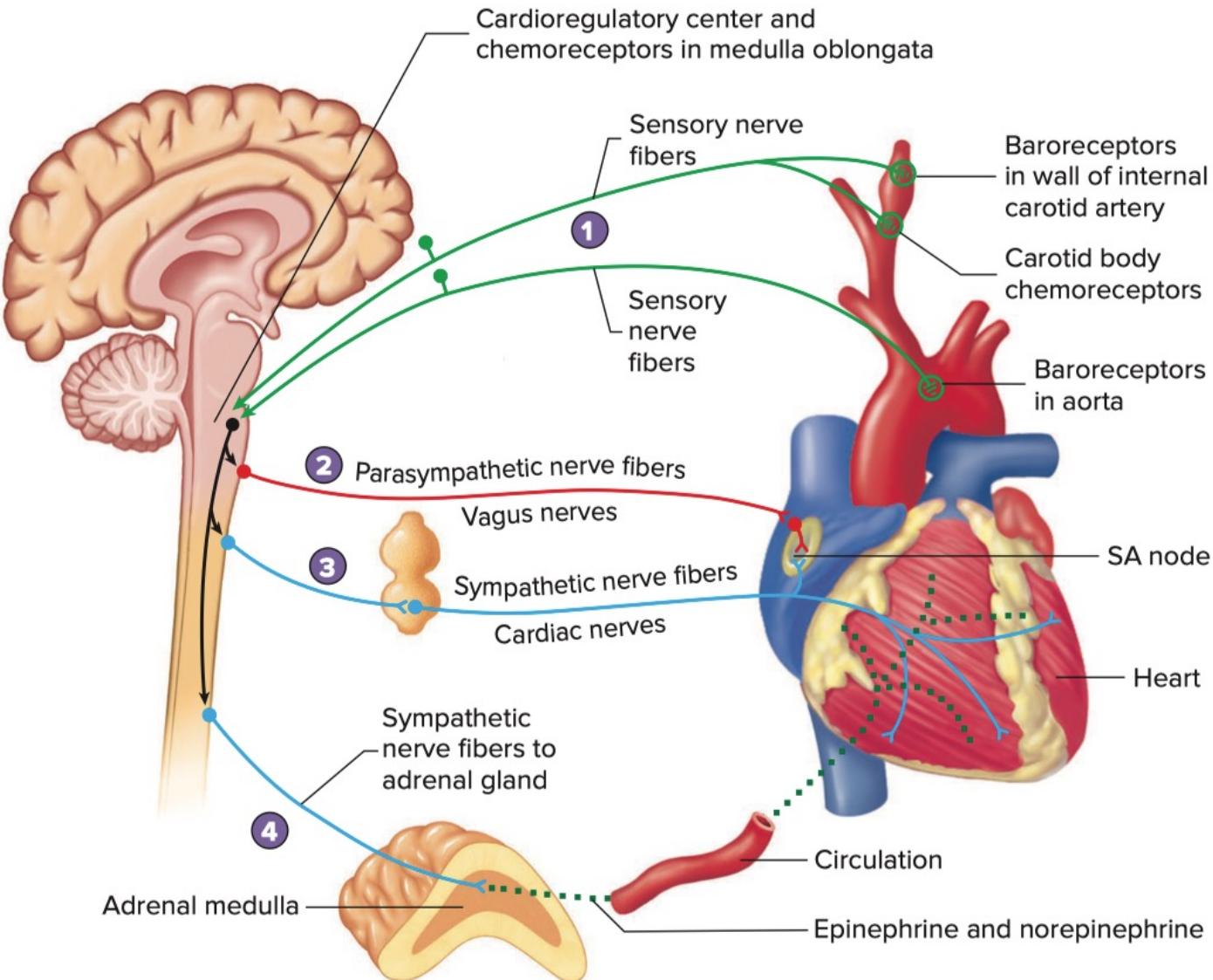
➤ Control via :

➤ 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 heart contractility
    - Positive ionotropic effect



- 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.
- 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.
- 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.
- 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<sub>2</sub>, and blood CO<sub>2</sub> 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).

- \* Positive inotrope is something that causes an increase in heart contractility ( $\uparrow SV$ )
- \* Negative inotrope is something that causes a decrease in heart contractility ( $\downarrow SV$ )
- \* 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

↑  
weak

↳ ↓ SV

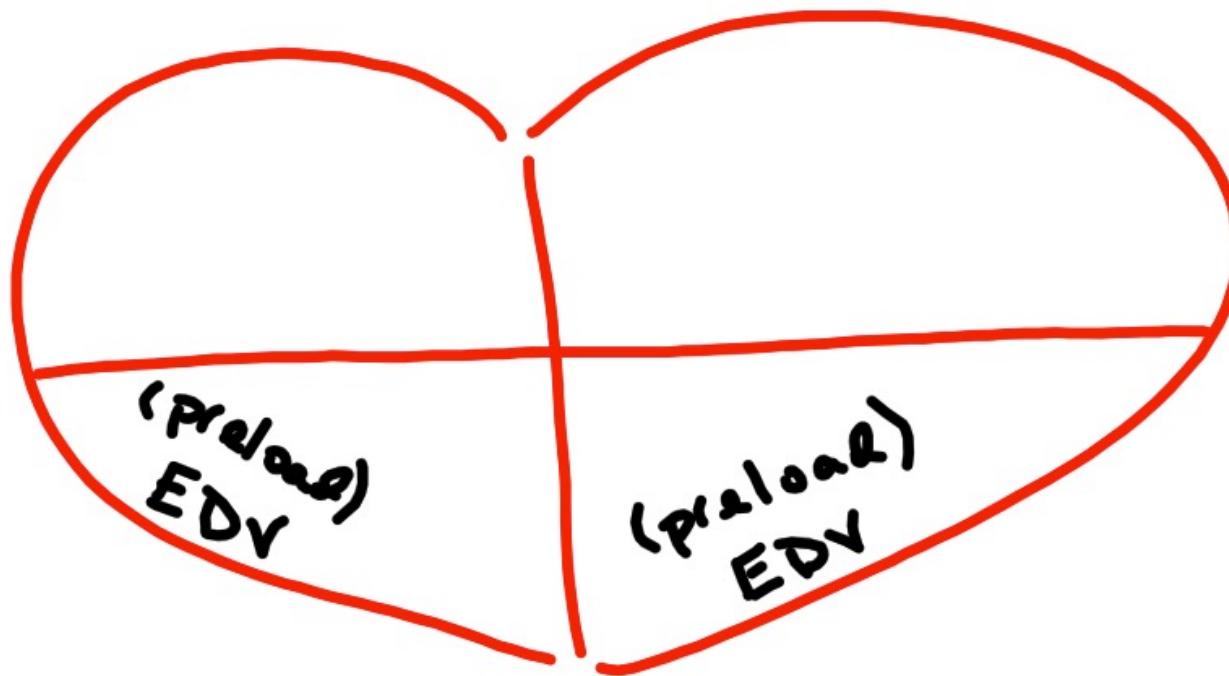
heart muscle

↑  
body responds by: ↑ HR



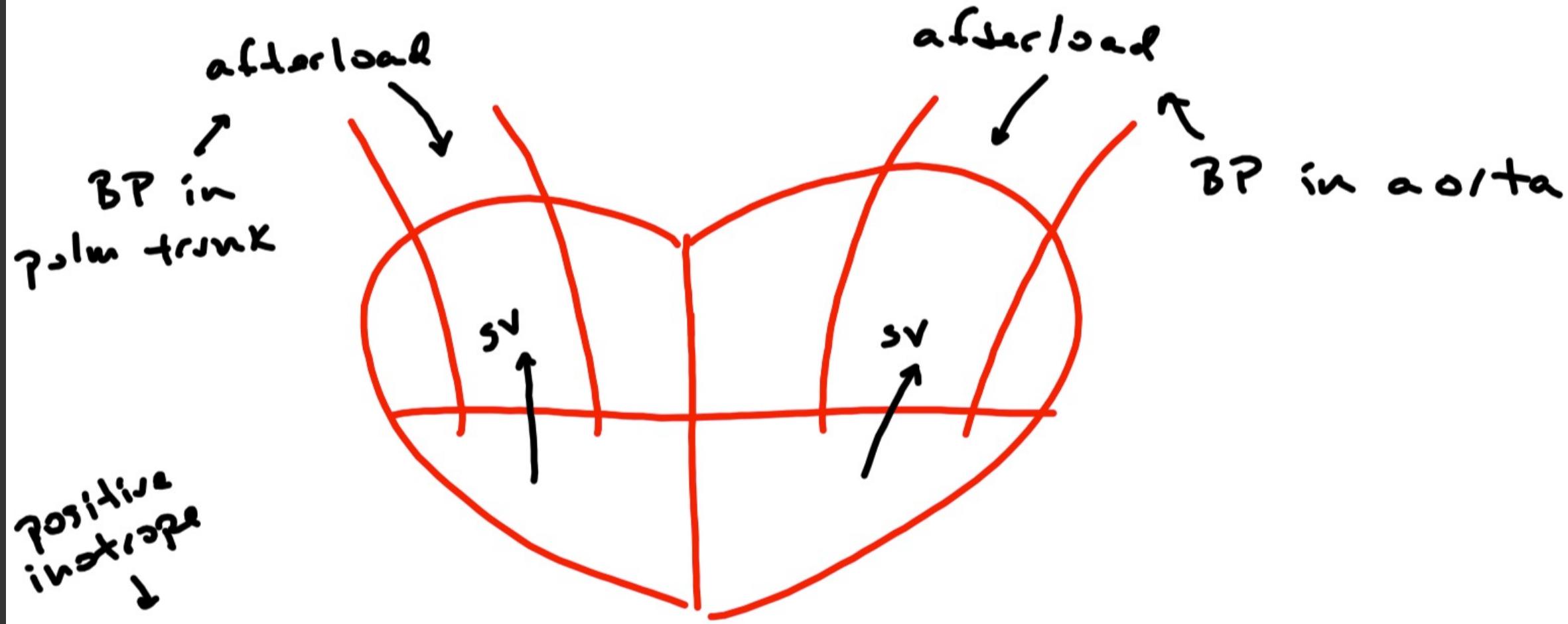
inefficient + hard

positive  
inotrope  
↓



↑ preload → ↑ contractility → ↑ SV

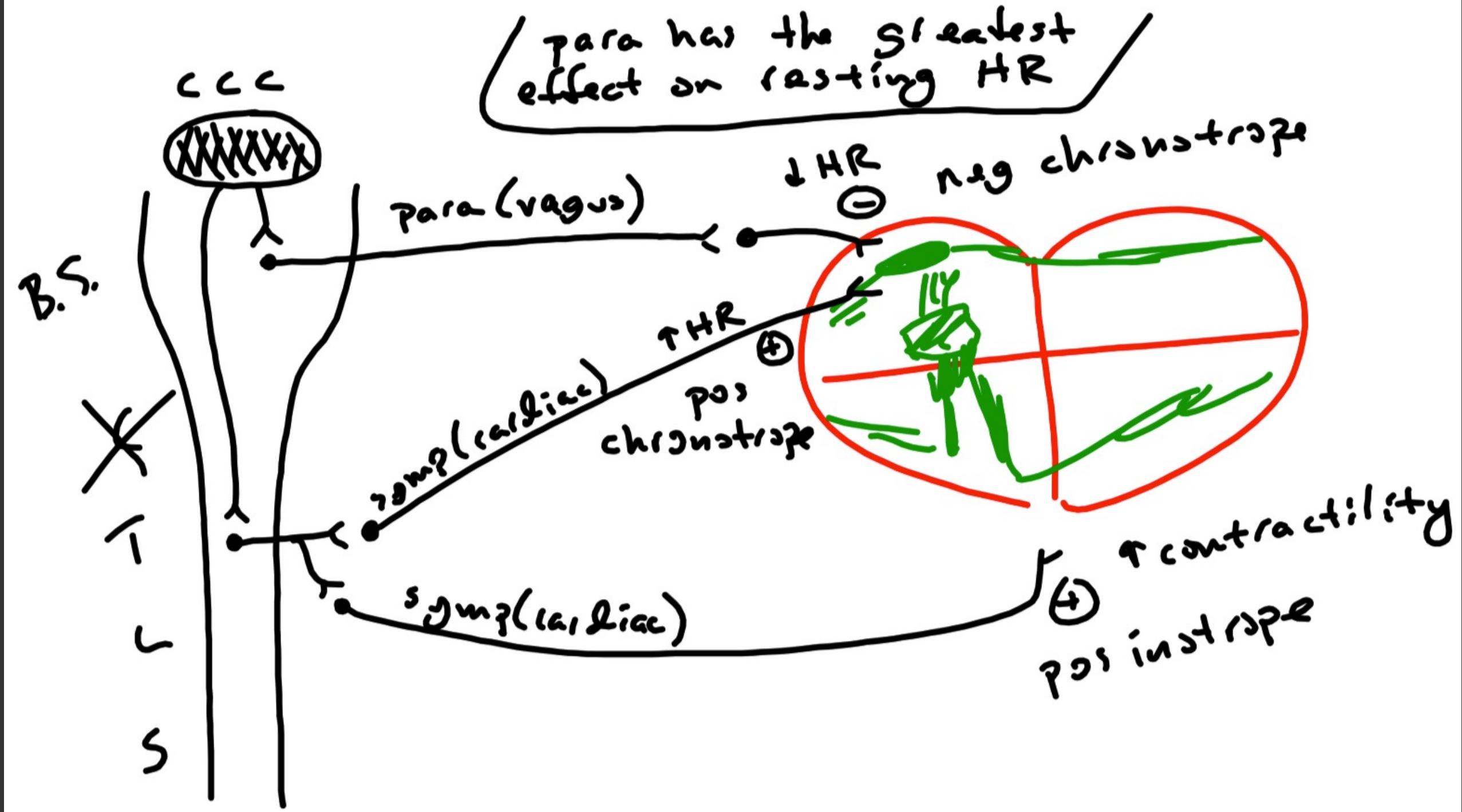
↑ afterload → ↓ contractility → ↓ SV  
negative inotrope



↑ afterload  $\rightarrow$  ↑ heart contractility

↓ afterload  $\rightarrow$  ↓ heart contractility

↑ negative inotrope

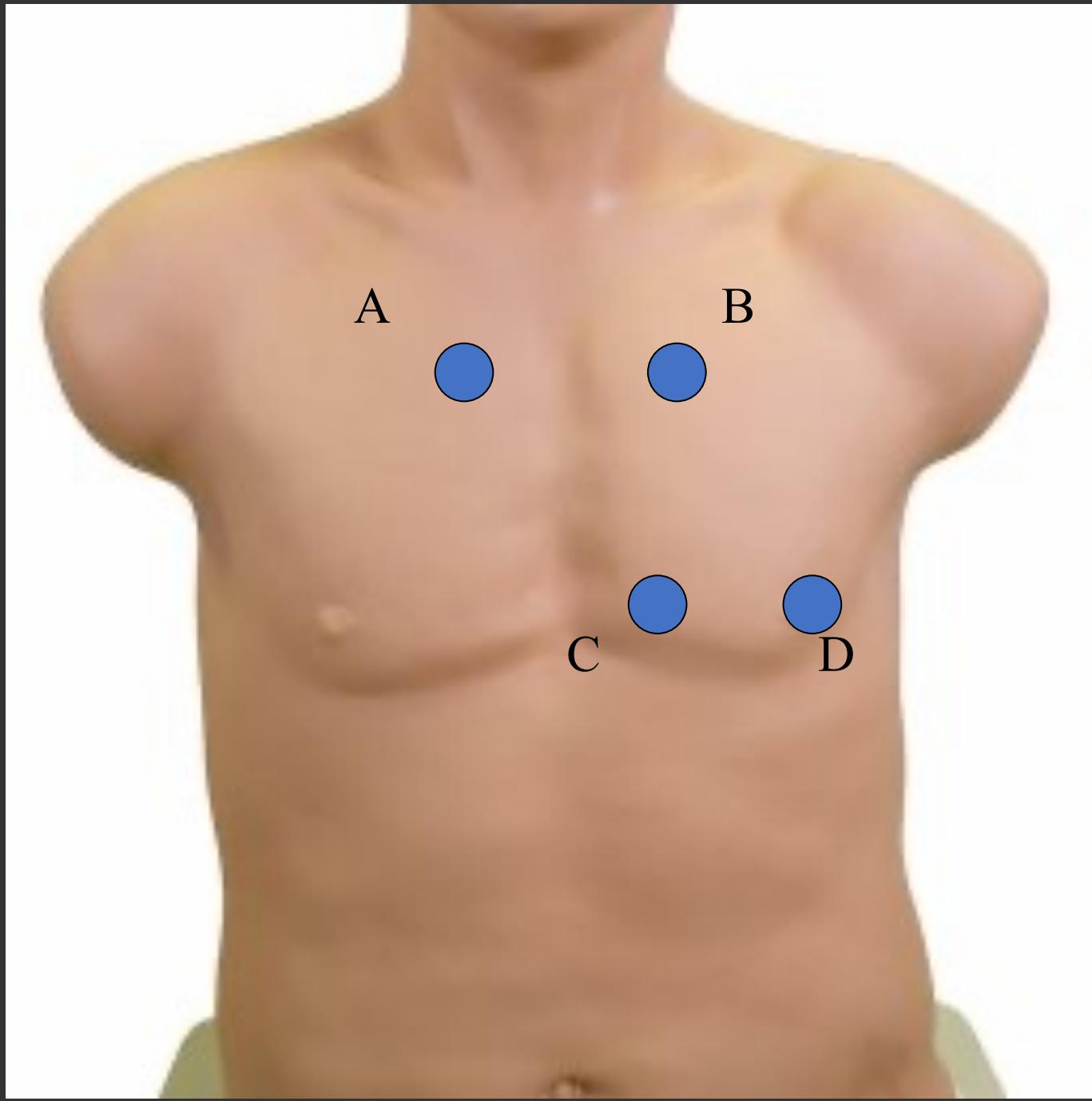


# Extrinsic Factors – Hormones

- 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

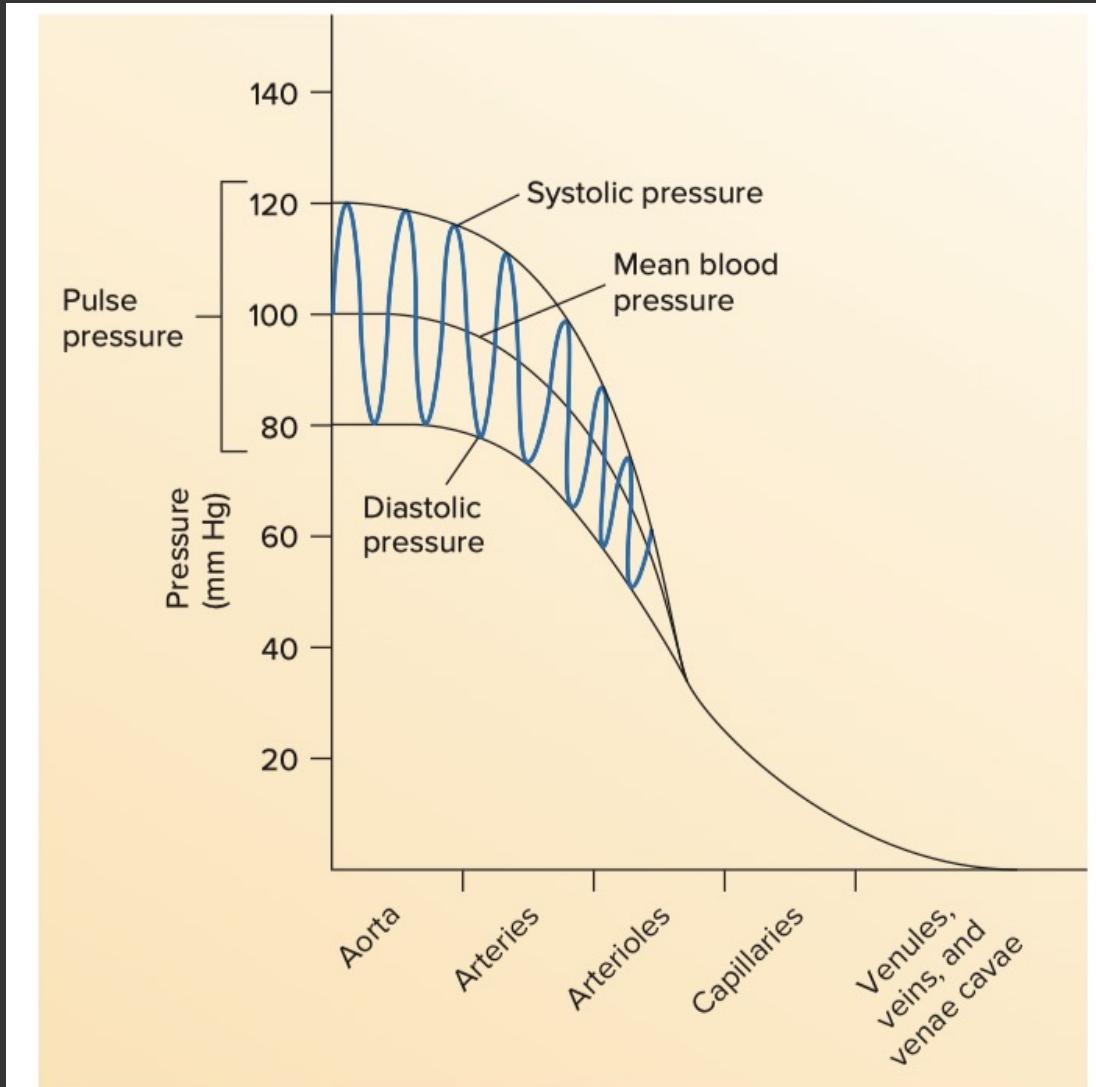
- A change of 1°C changes heart rate approximately 10 beats per minute
  - Affects action potential generation and conduction
- Increase temperature
  - Increase heart rate
    - Positive chronotropic effect
  - Decrease temperature
    - Decrease heart rate
      - Negative chronotropic effect



# 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

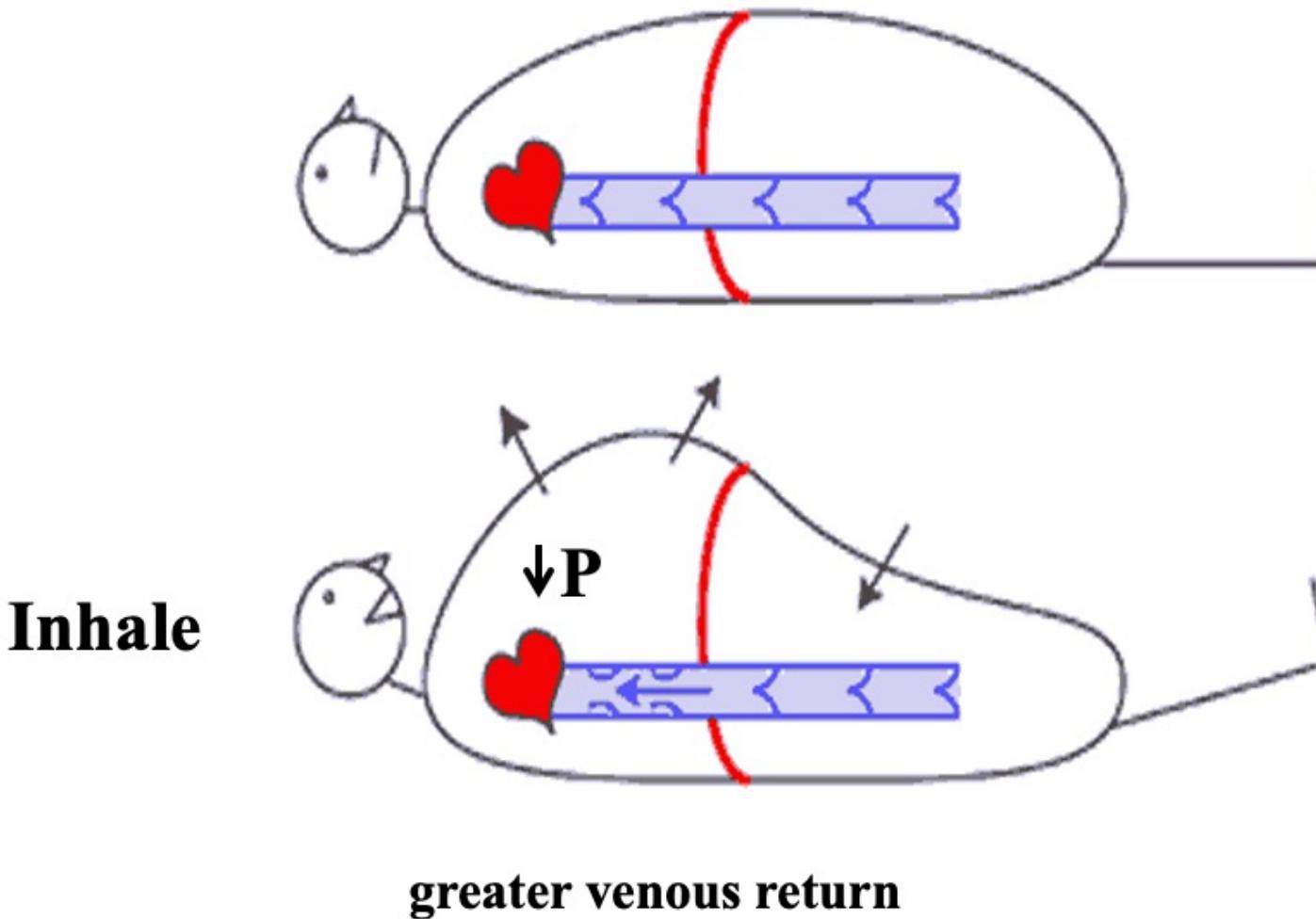


**FIGURE 21.35** Blood Pressure in the Major Blood Vessel Types

In small arteries and arterioles, blood pressure fluctuations between systole and diastole are reduced. No fluctuations in blood pressure occur in capillaries and veins.

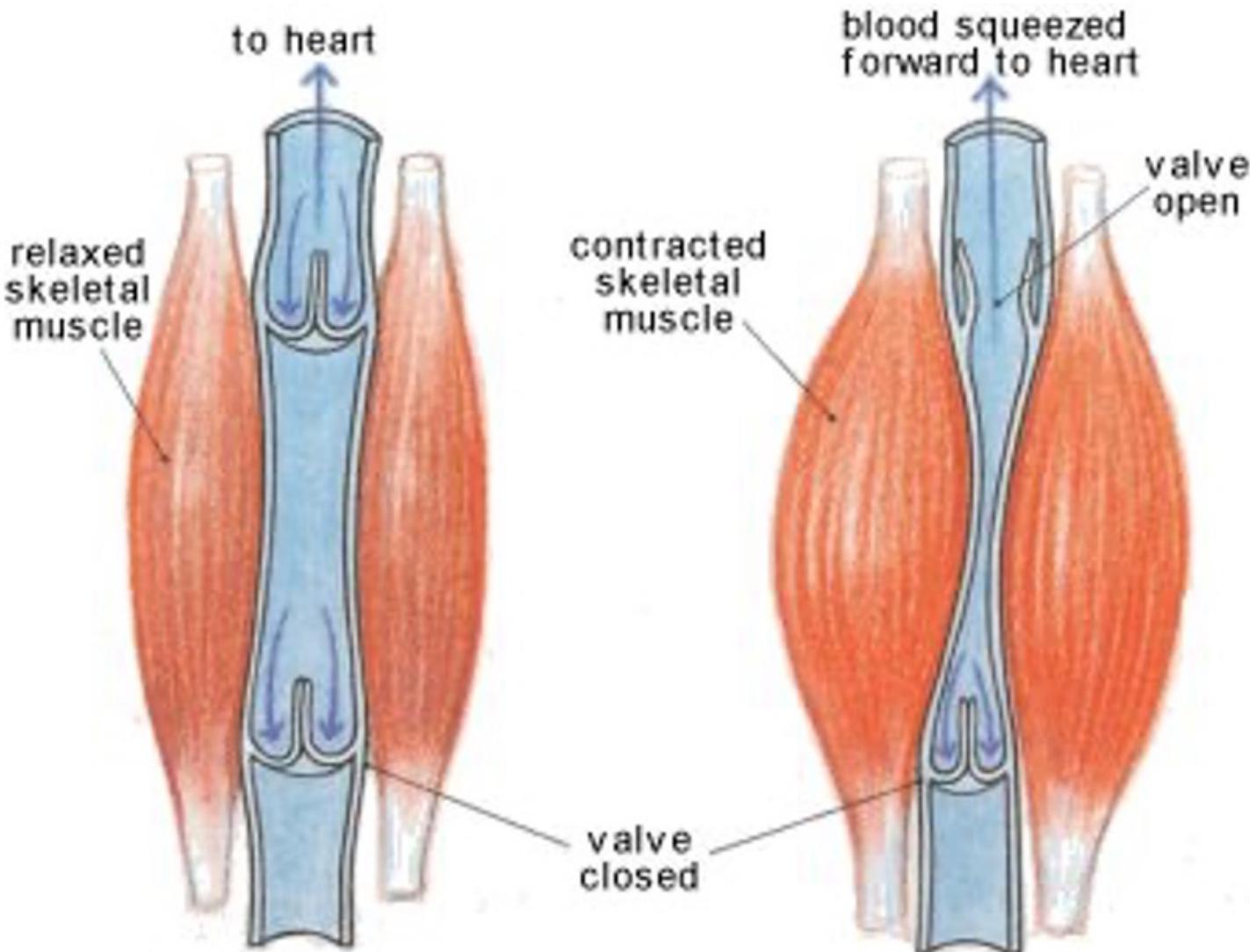
# Factors Contributing to Venous Return

## Respiratory Pump

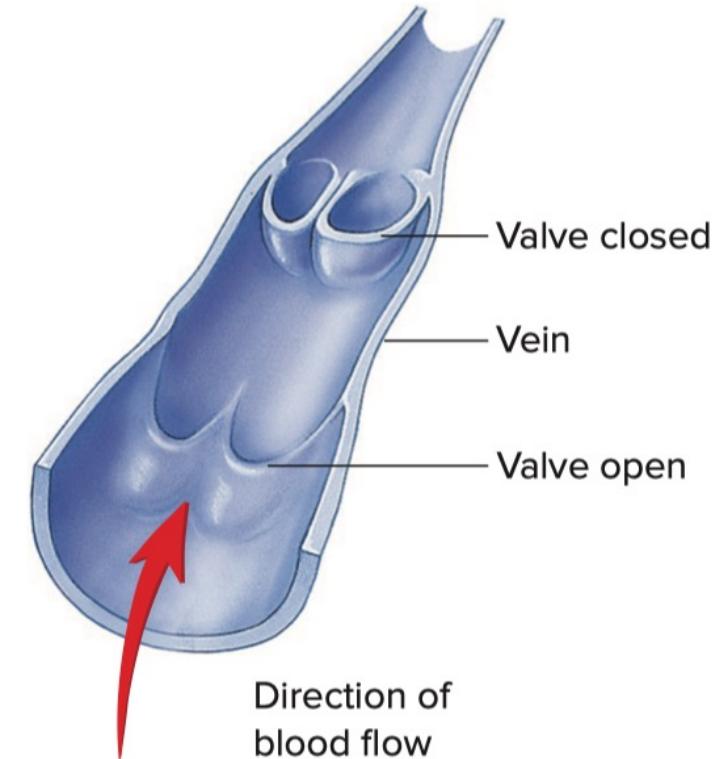
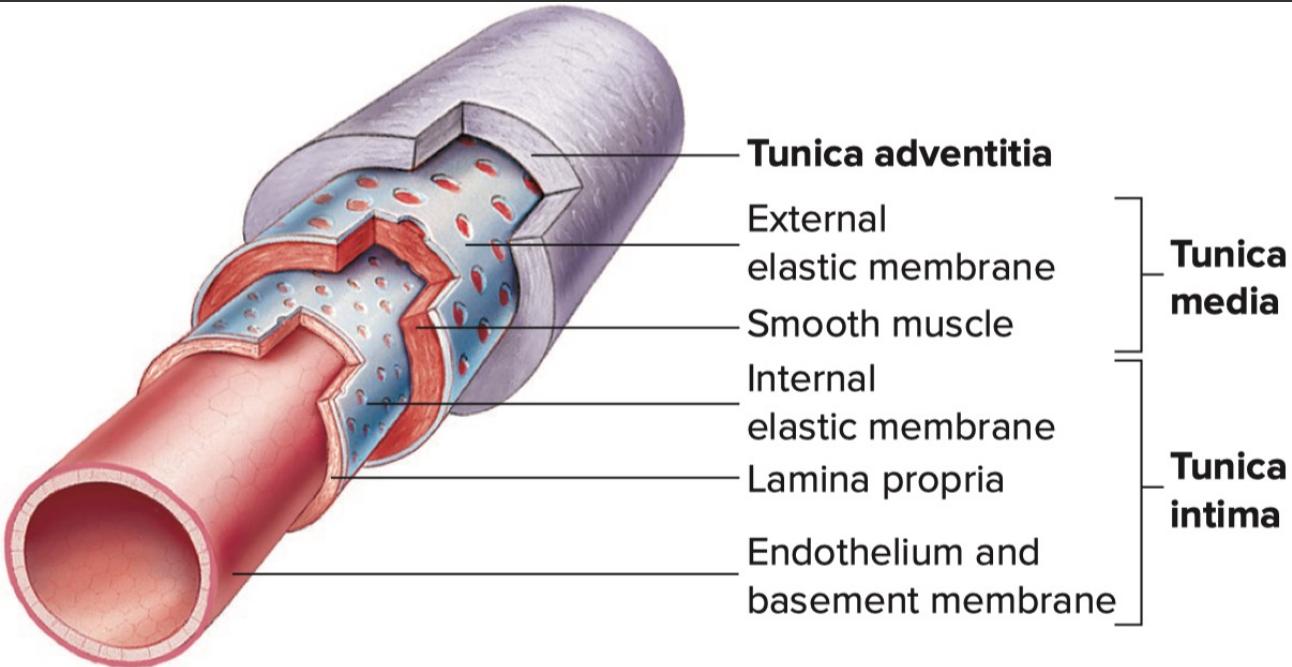


# Factors Contributing to Venous Return

## Muscle Contraction



# Veins and Venules

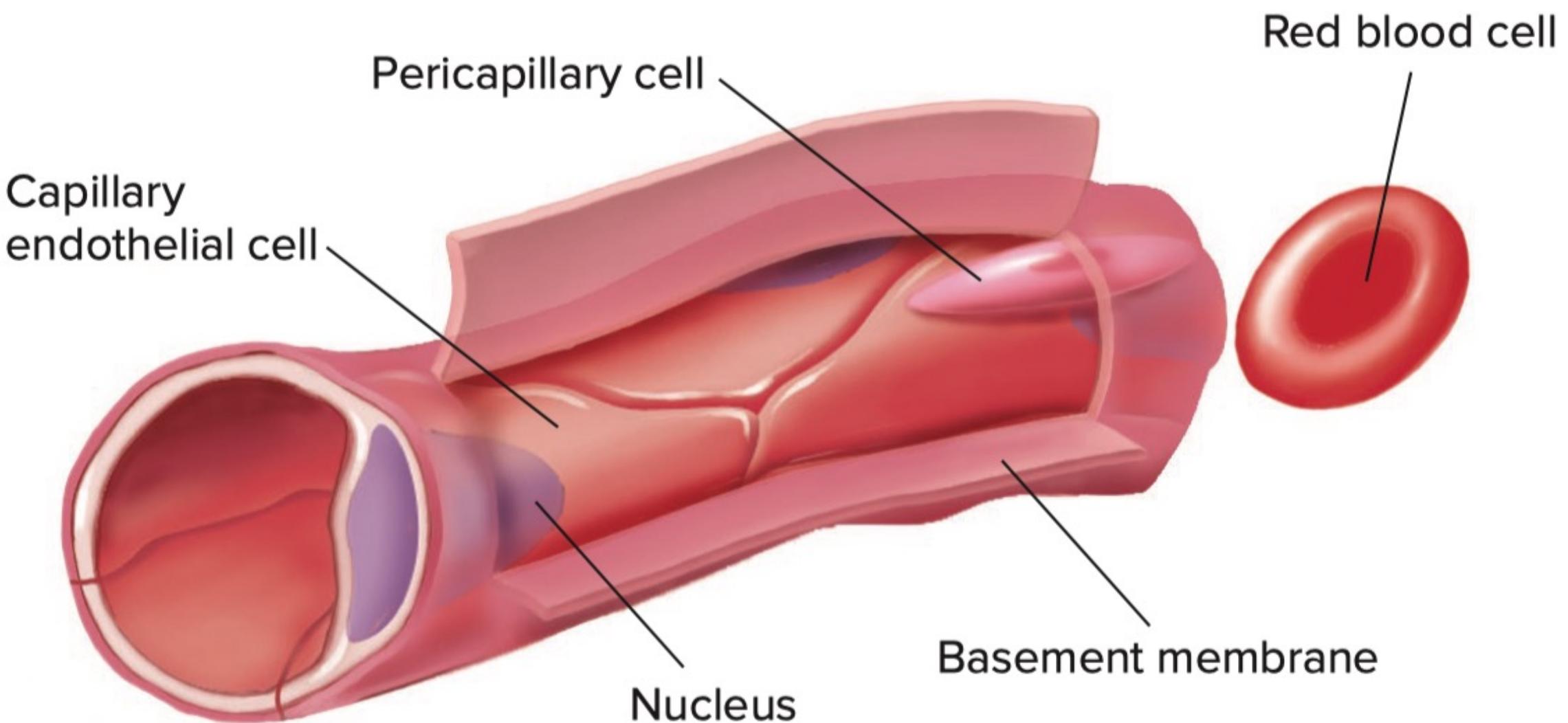


**FIGURE 21.7 Valves**

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

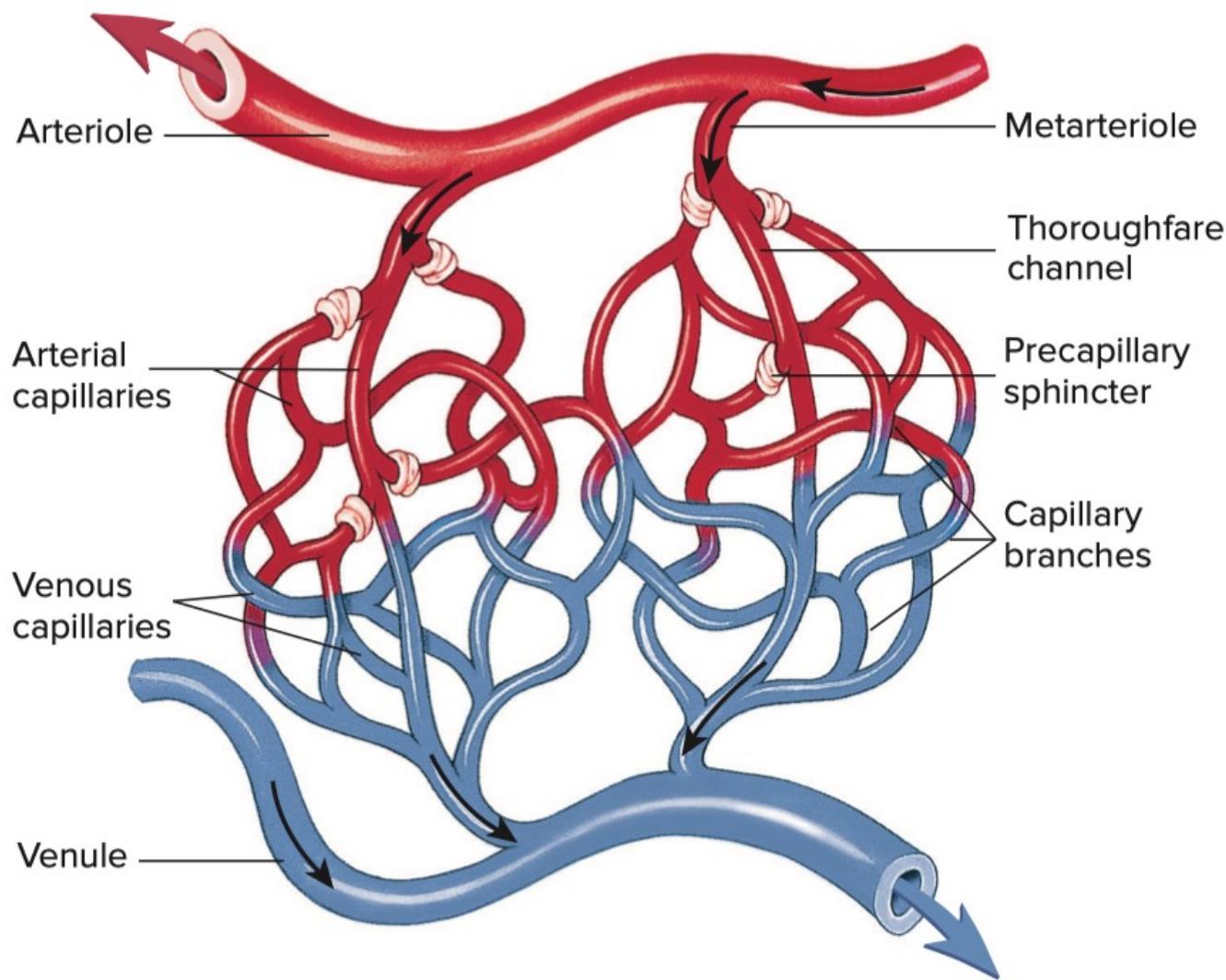
# Blood Vessels

- 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 move easily
- Diffuse and filter through



## FIGURE 21.4 Capillary

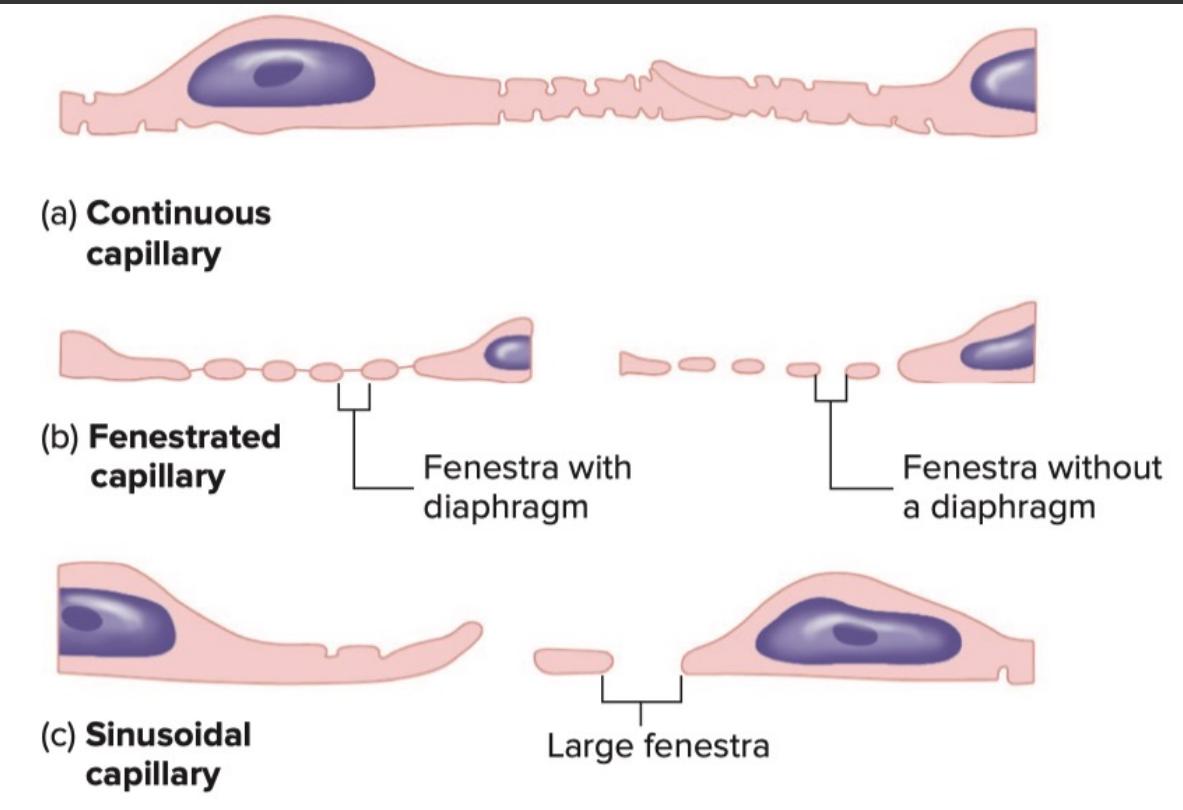
Section of a capillary, showing that it is composed primarily of flattened endothelial cells.



## 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

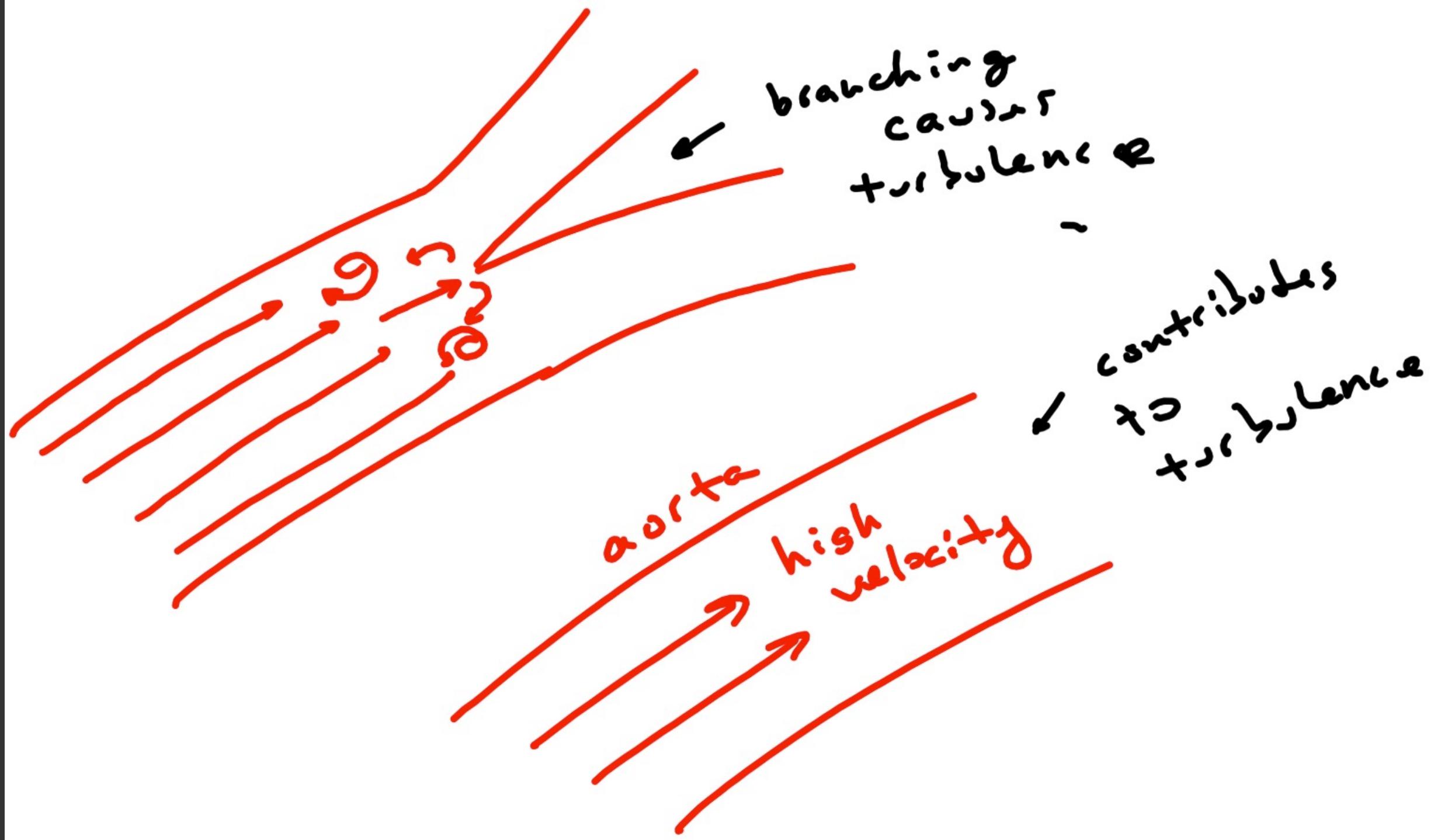


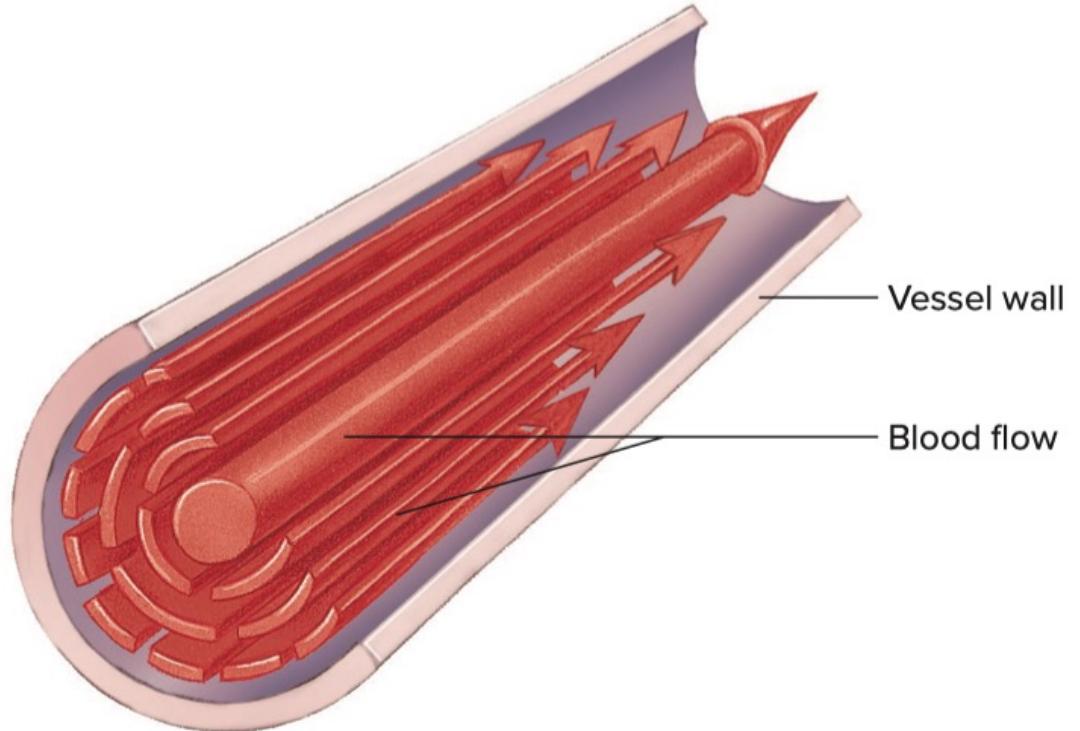
**FIGURE 21.5 Structure of Capillary Walls**

- (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.

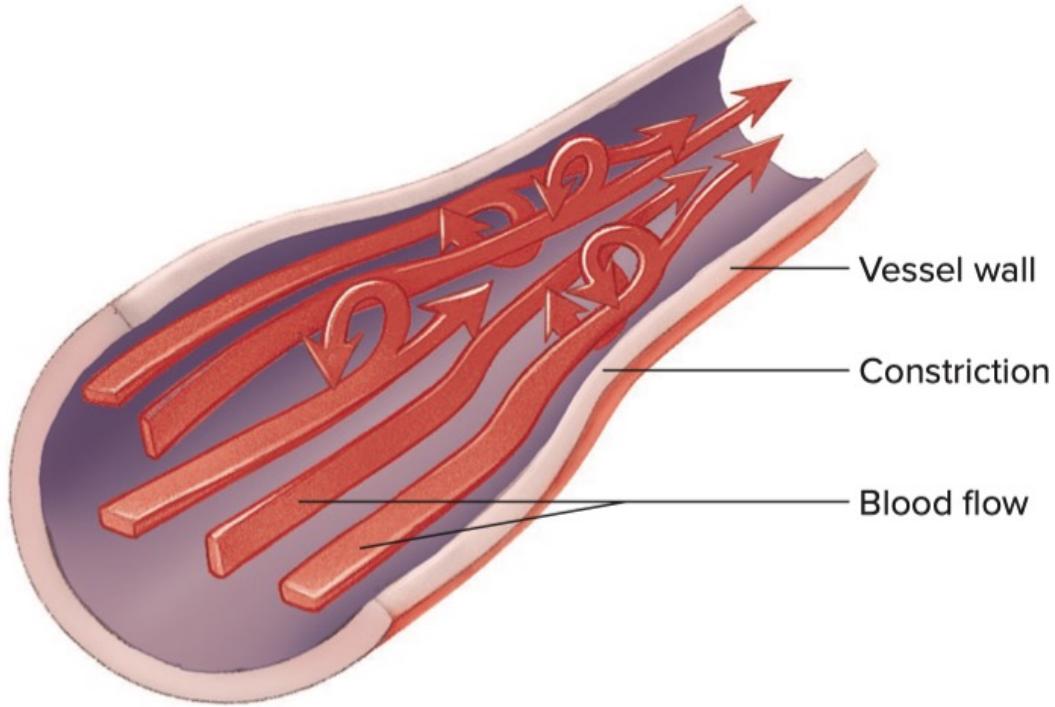
# Blood Flow

- 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





(a)



(b)

### 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

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

$\nu$  = 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 )

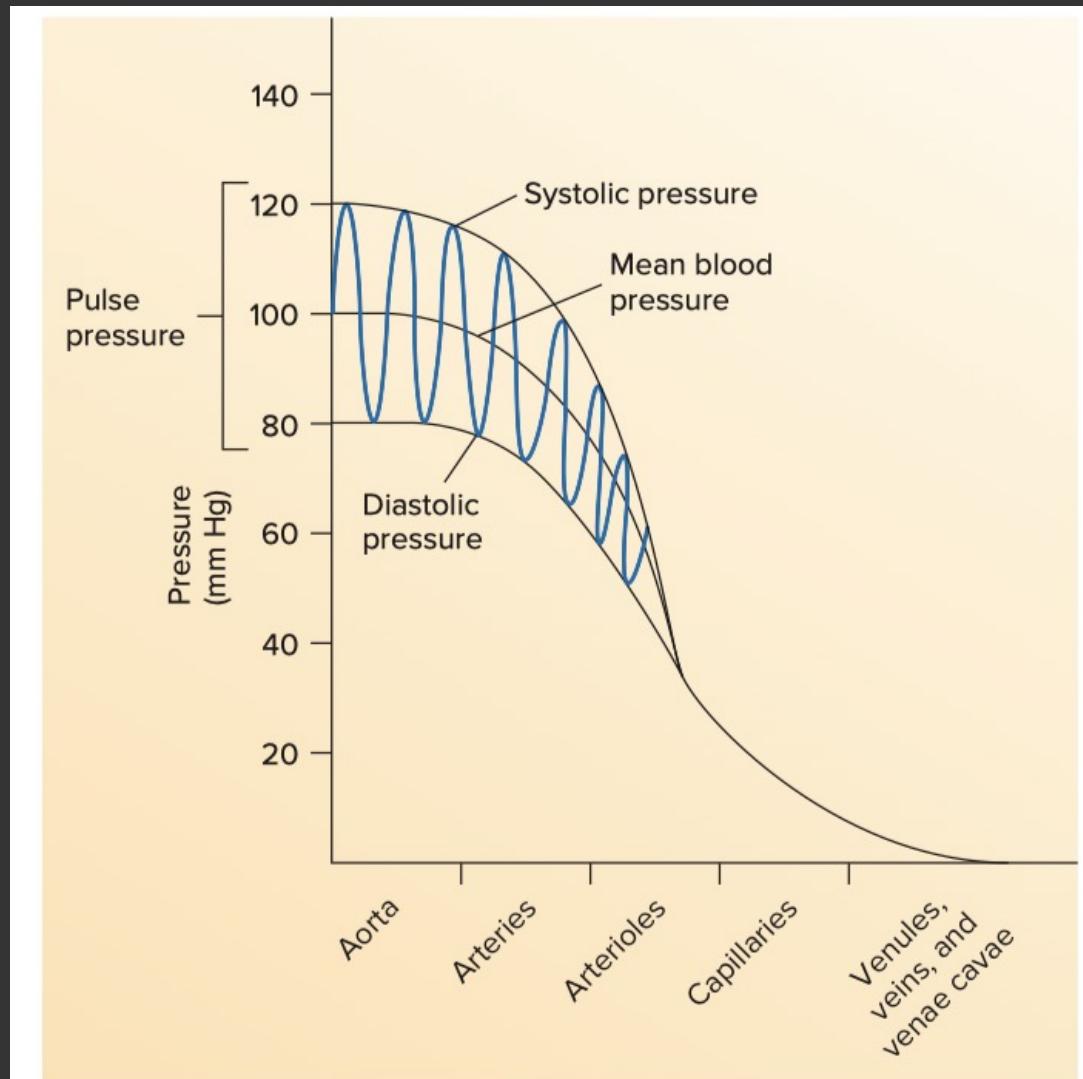
$$\text{Flow} = \frac{(P_1 - P_2) * \pi * r^4}{8 * \nu * l}$$

*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.

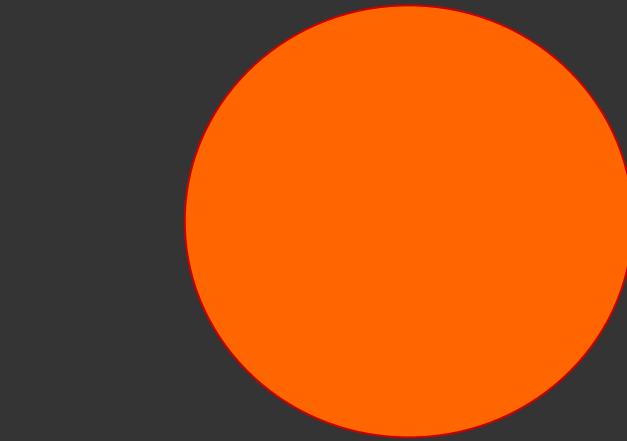
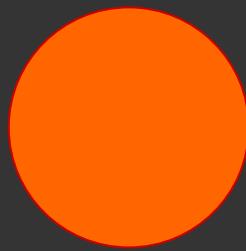
# Effect of Radius on Blood Flow



decrease radius ➔ increase resistance



decrease blood flow tremendously



increase radius ➔ decrease resistance



increase blood flow tremendously

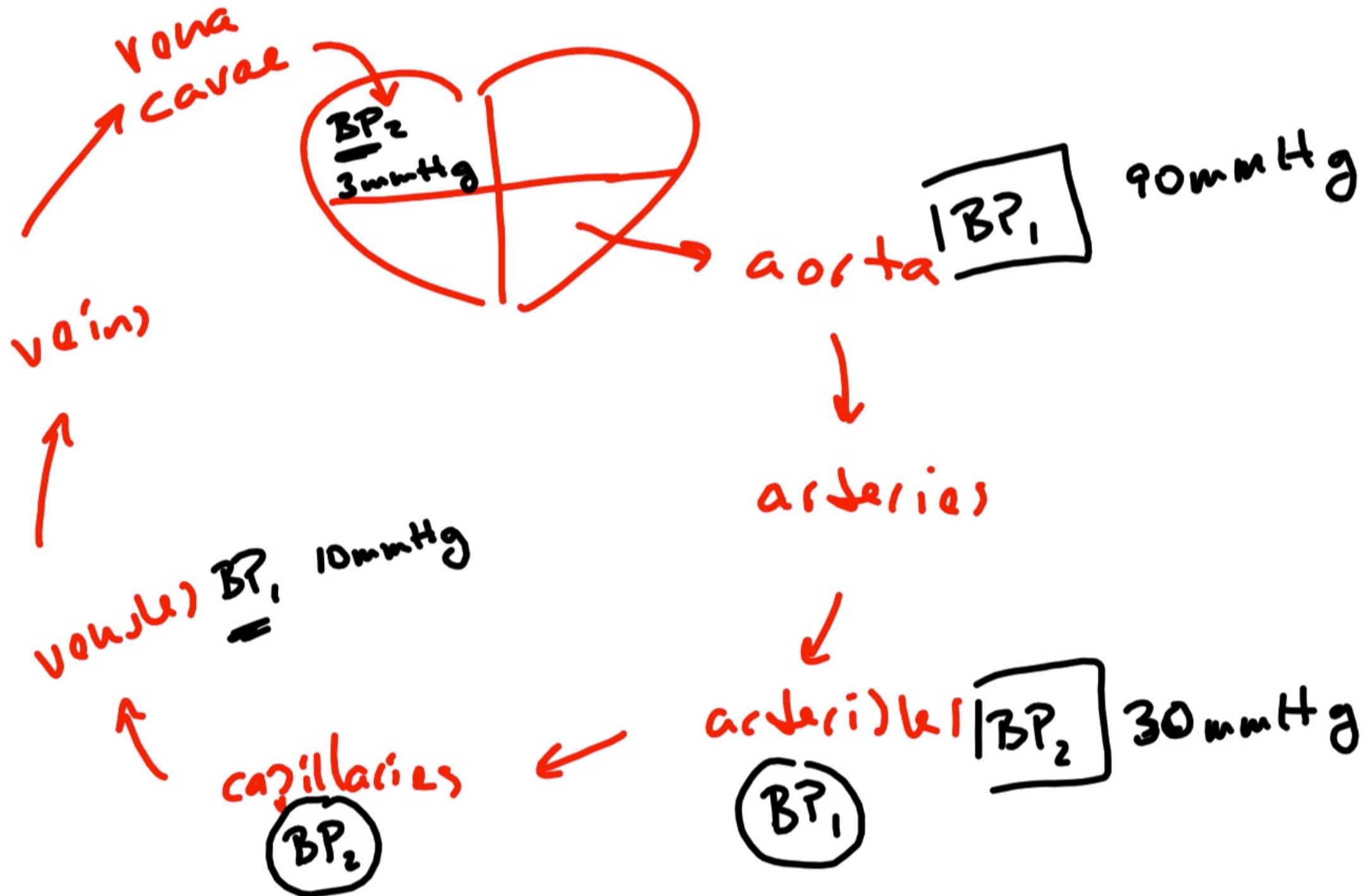
$$\text{Fluid Flow} = \frac{(P_1 - P_2)}{R}$$

← pressure gradient

---

$$\text{Blood Flow} = \frac{(BP_1 - BP_2)}{R}$$
$$R \sim \frac{\eta}{r^4}$$

$\eta$  = viscosity of blood  
 $r$  = radius of blood vessel



$\uparrow (BP_1 - BP_2) \rightarrow \uparrow$  blood flow

$\downarrow (BP_1 - BP_2) \rightarrow \downarrow$  blood flow

If BP in arterial system is too low

$\hookrightarrow (BP_1 - BP_2)$  is too small

$\hookrightarrow \downarrow$  blood flow  $\leftarrow$  risk organ failure

$\uparrow r \rightarrow \downarrow R \rightarrow \uparrow \text{blood flow}$

$\uparrow$  vaso dilate

$\downarrow$  vaso constrict

changes in radius  
have ~~HUGE~~  
effects on  
blood flow

$\downarrow r \rightarrow \uparrow R \rightarrow \downarrow \text{blood flow}$

# 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

$\uparrow$  viscosity  $\rightarrow$   $\uparrow R \rightarrow \downarrow$  blood flow

$\downarrow$  viscosity  $\rightarrow$   $\downarrow R \rightarrow \uparrow$  blood flow

\* What affects blood viscosity the most?

↳ Hct

-  $\uparrow$  Hct  $\rightarrow$   $\uparrow$  viscosity

polycythemia

-  $\downarrow$  Hct  $\rightarrow$   $\downarrow$  viscosity



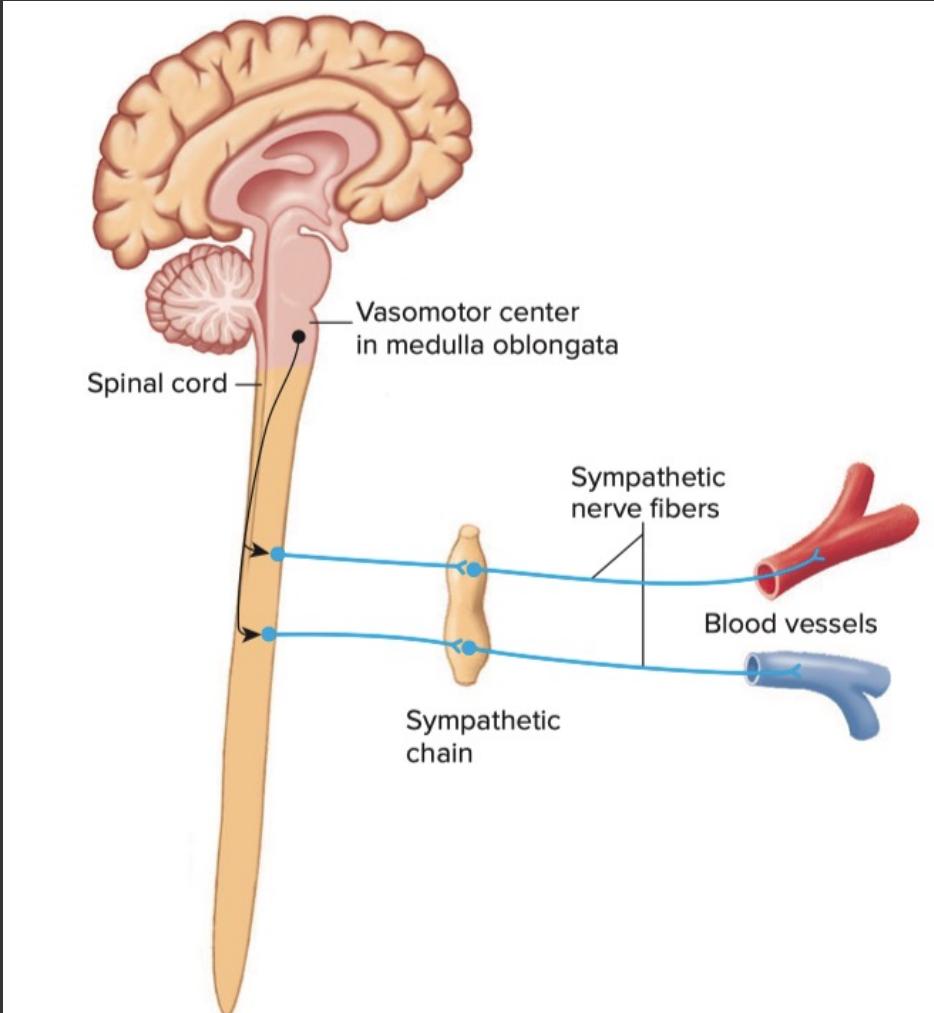
# Control of Blood Flow

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

# Vasomotor Centers of the Brainstem

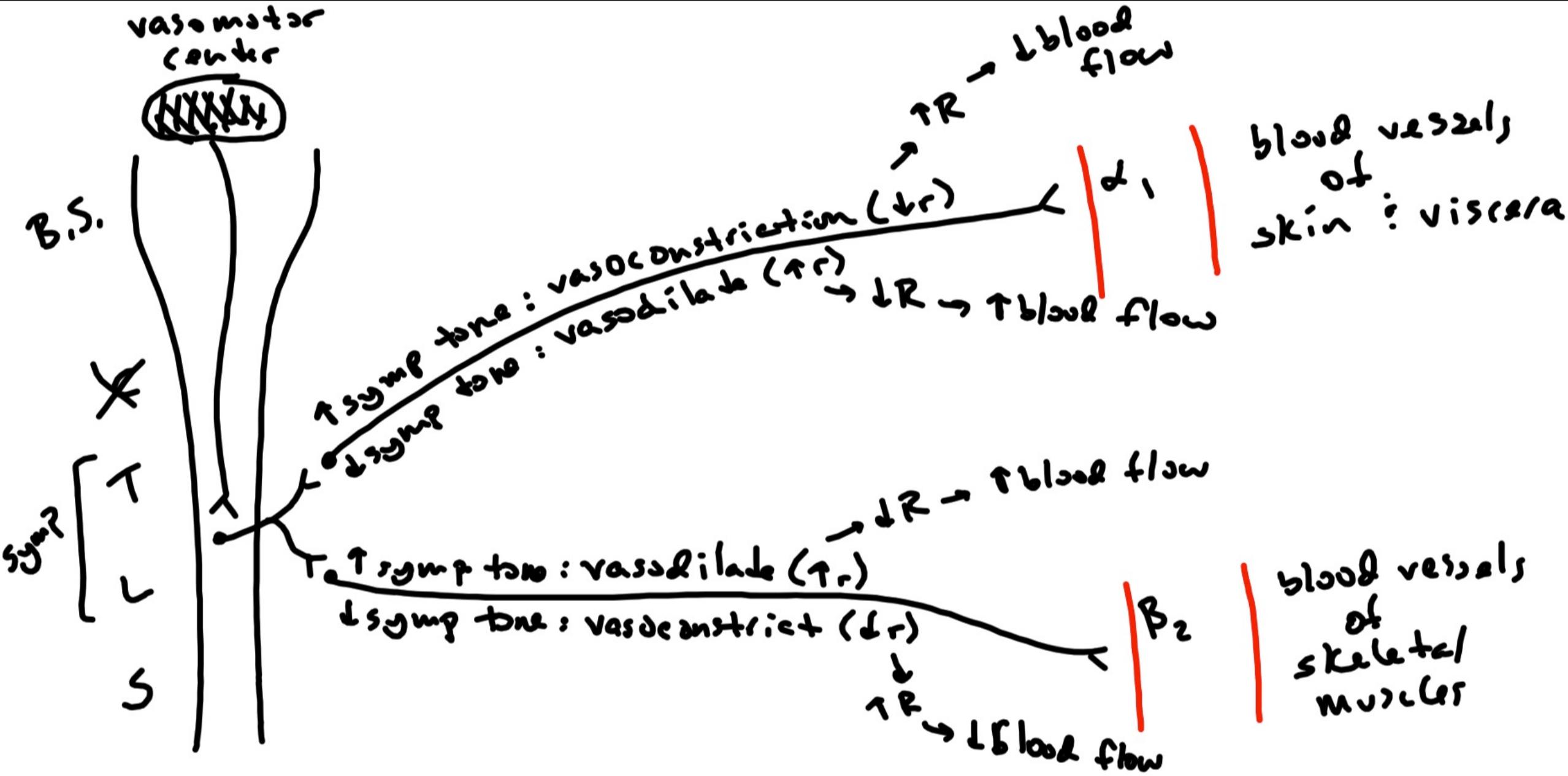
- Vasomotor centers in the brainstem
- Output via the sympathetics ( innervate vascular smooth muscle )
  - Stimulation of sympathetics
    - Vasoconstriction of skin and visceral blood vessels
      - Decreases blood flow to skin and viscera
    - Vasodilation of skeletal muscle blood vessels
     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

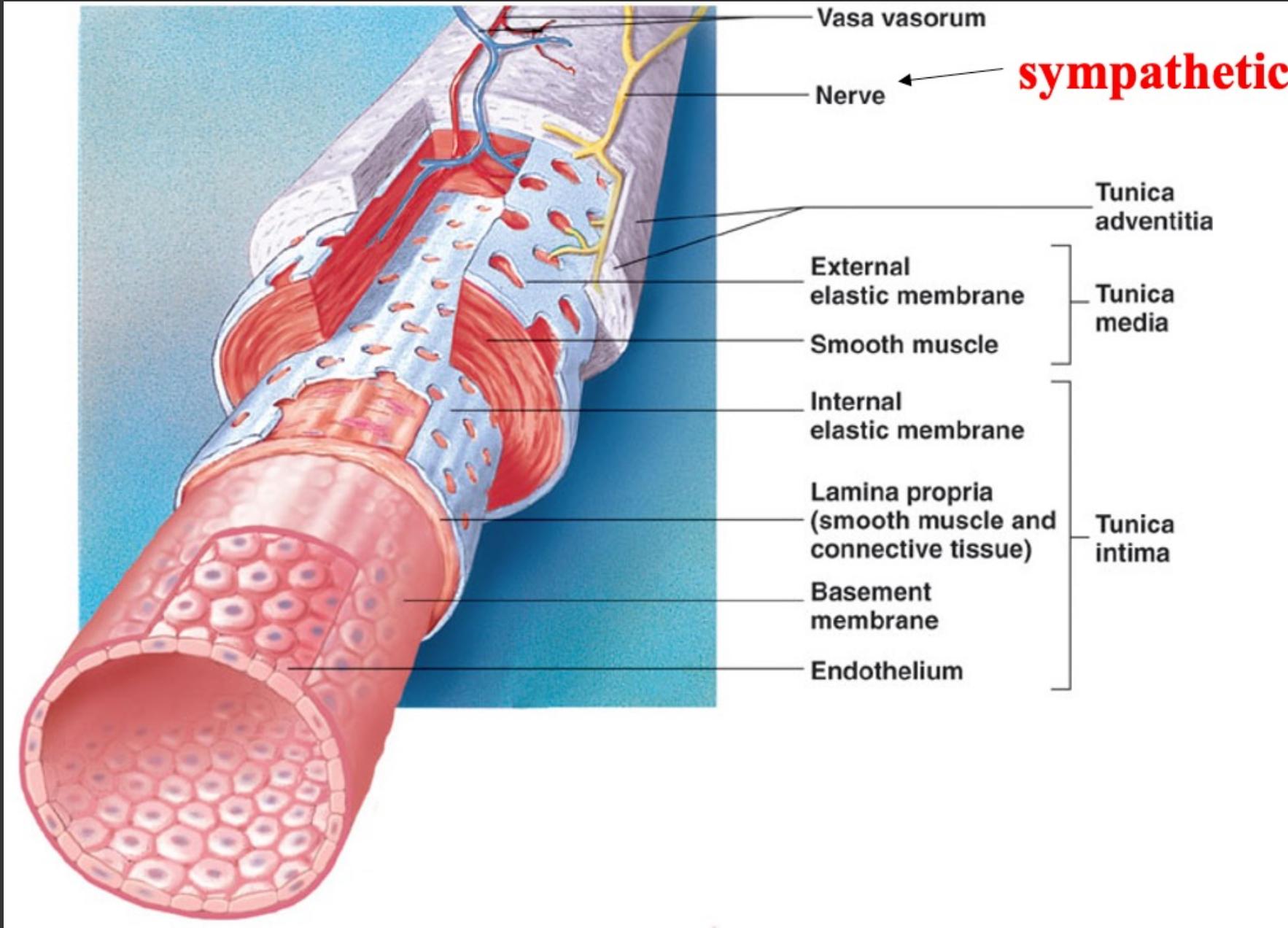


**FIGURE 21.38 Nervous Regulation of Blood Vessels**

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.



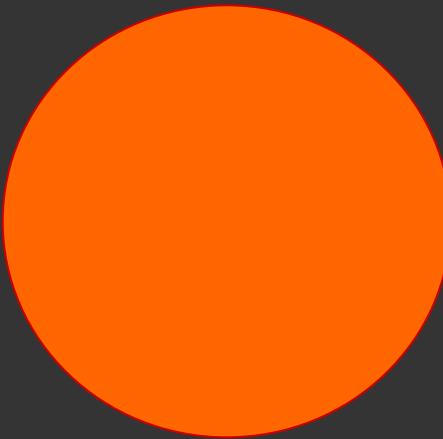
# Sympathetic Innervation of Vascular Smooth Muscle



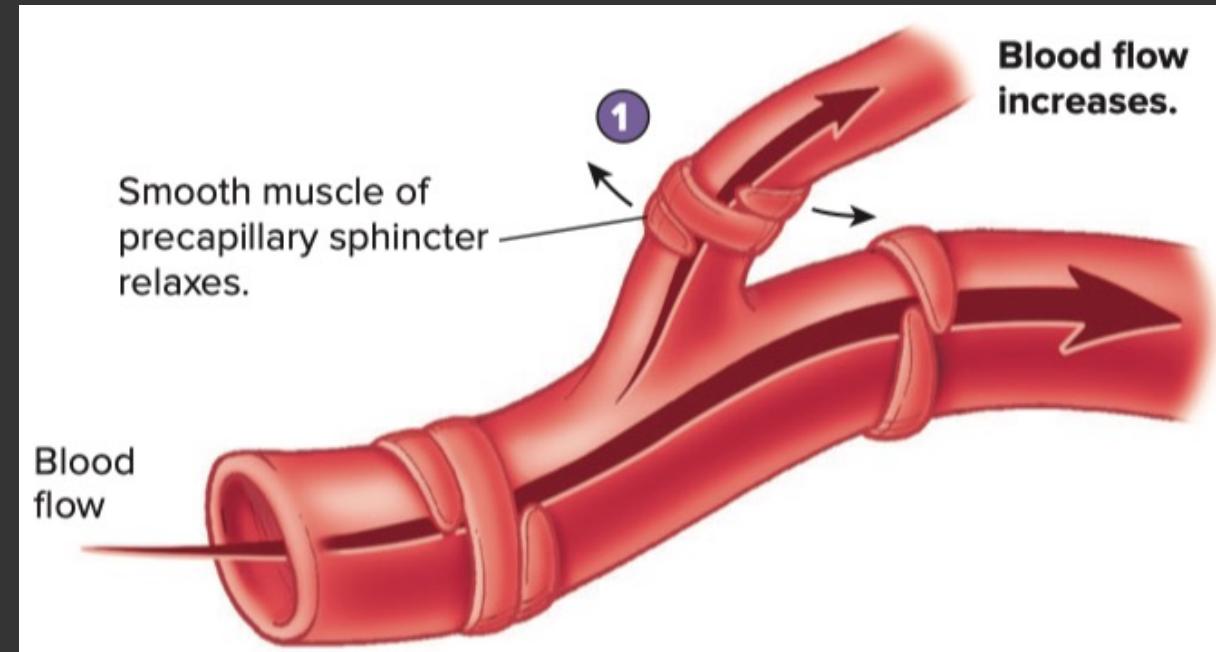
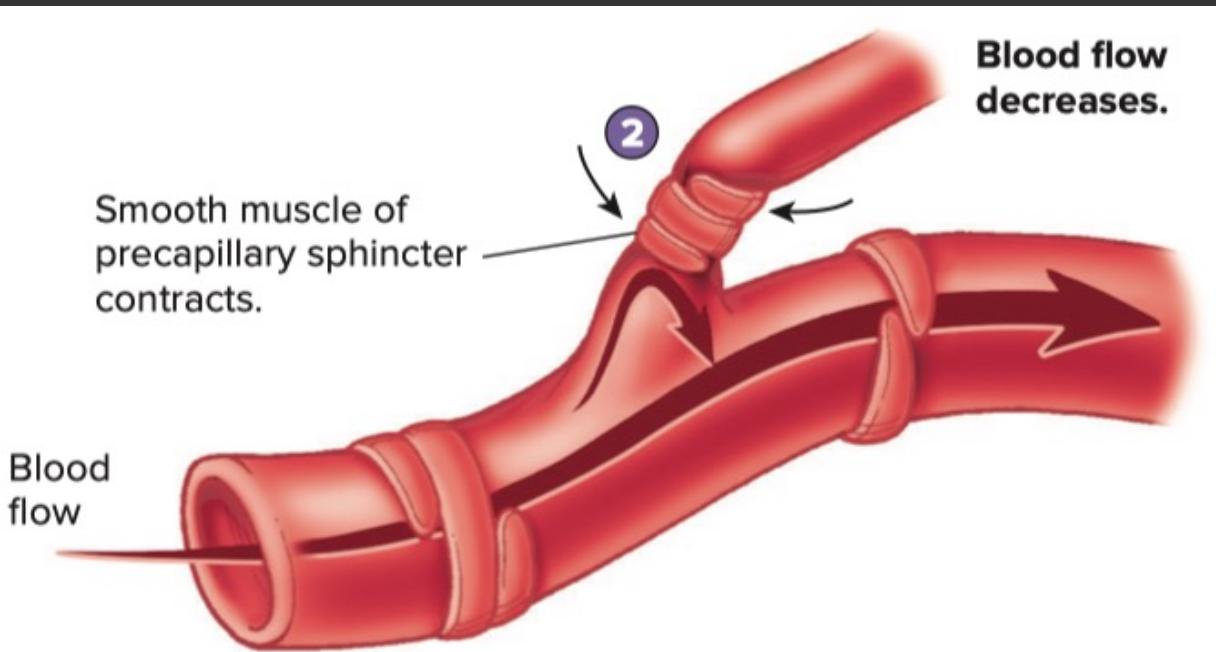
# Effect of Radius on Blood Flow



vasoconstriction

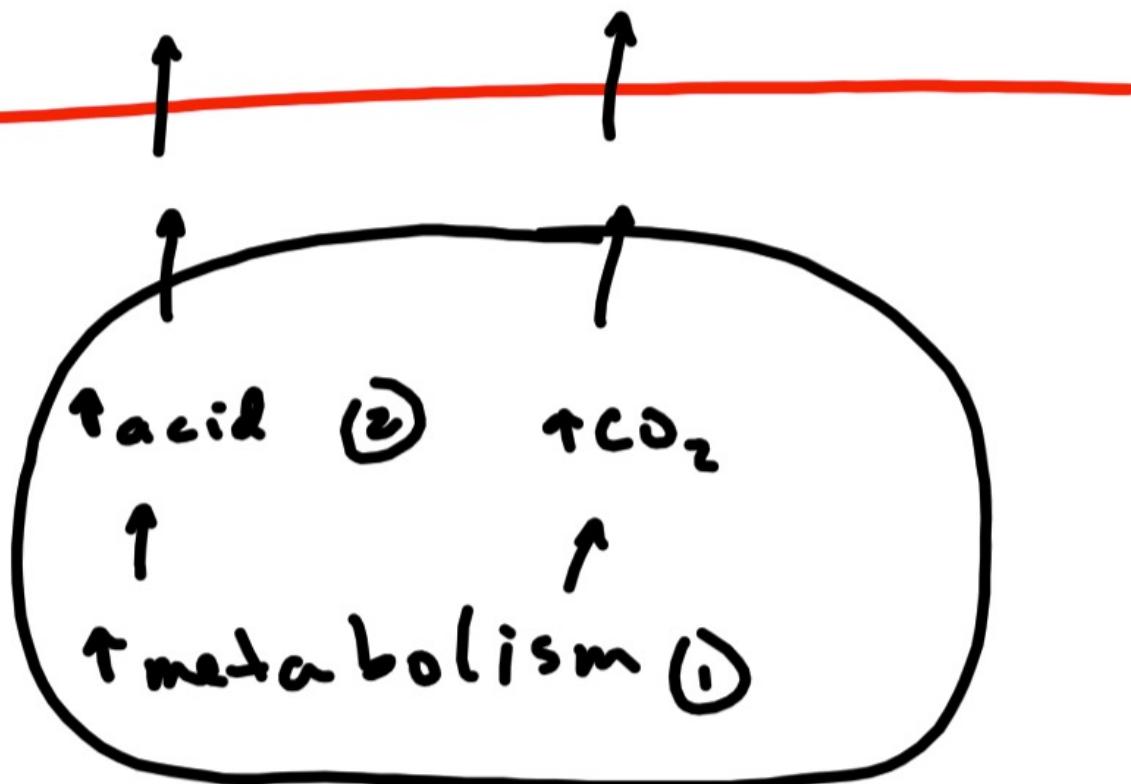


vasodilation



$\text{↑ acid } \textcircled{3}$     $\text{↑ CO}_2$     $\rightarrow \textcircled{4} \text{ vasodilate (tr)}$   $\rightarrow \text{LR}$

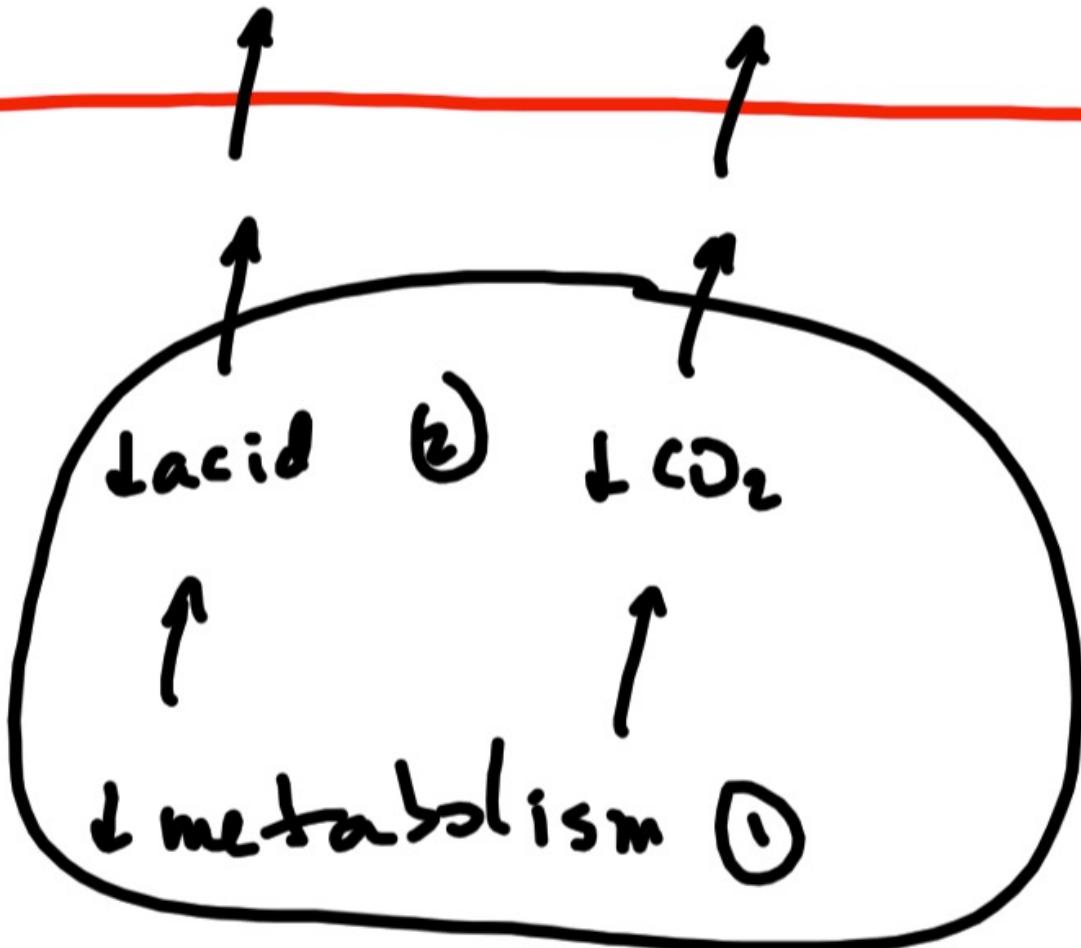
$\downarrow$   
 $\text{↑ blood flow}$



\* why?  
↳ to wash away  
more acid and CO<sub>2</sub>

cell

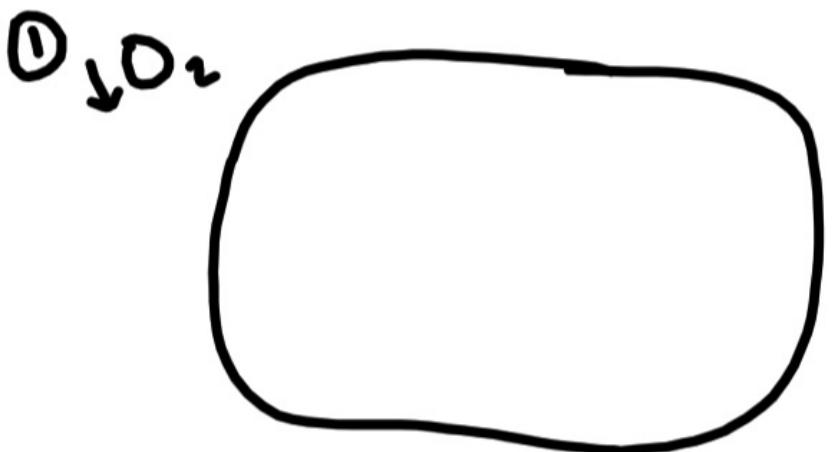
Lacid ③  $\downarrow$   $\text{CO}_2$   $\rightarrow$  ④ vasoconstrict (dr)



$\uparrow$  TR  
 $\downarrow$  blood flow  
why?  
↳ to wash away  
less acid and  $\text{CO}_2$

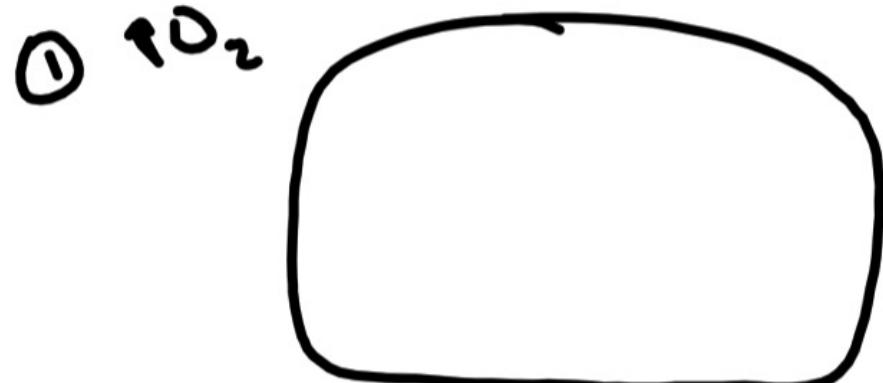
Why?  
It gets more O<sub>2</sub> to the cell  
↓

② vasoconstrict ( $\uparrow r$ )  $\rightarrow \downarrow R$   
 $\uparrow$  blood flow



Why?  
It's blood (O<sub>2</sub>) goes to other places that need it more ↓ blood flow

② vasoconstrict ( $\downarrow r$ )  $\rightarrow \uparrow R$



# 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<sub>2</sub>
  - Decrease in O<sub>2</sub>
- Cause : smooth muscle contraction ( vasoconstriction ) → decrease blood flow
  - Thromboxane
  - Antidiuretic hormone / Vasopressin
  - Increase in pH
  - Decrease in CO<sub>2</sub>
  - Increase in O<sub>2</sub>

# 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

$$BP = \frac{110 \text{ mm Hg}}{70 \text{ mm Hg}}$$

$$\text{* pulse pressure} = (\text{systolic P}) - (\text{diastolic P})$$

$$\begin{aligned}&= 110 \text{ mm Hg} - 70 \text{ mm Hg} \\&= 40 \text{ mm Hg}\end{aligned}$$

# 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  $\frac{1}{3}$  of time in systole and  $\frac{2}{3}$  of time in diastole
- Calculation:

$$MAP = \frac{1}{3} (\text{systolic pressure}) + \frac{2}{3} (\text{diastolic pressure})$$

OR

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

# Mean Arterial Pressure ( MAP )

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

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

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

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

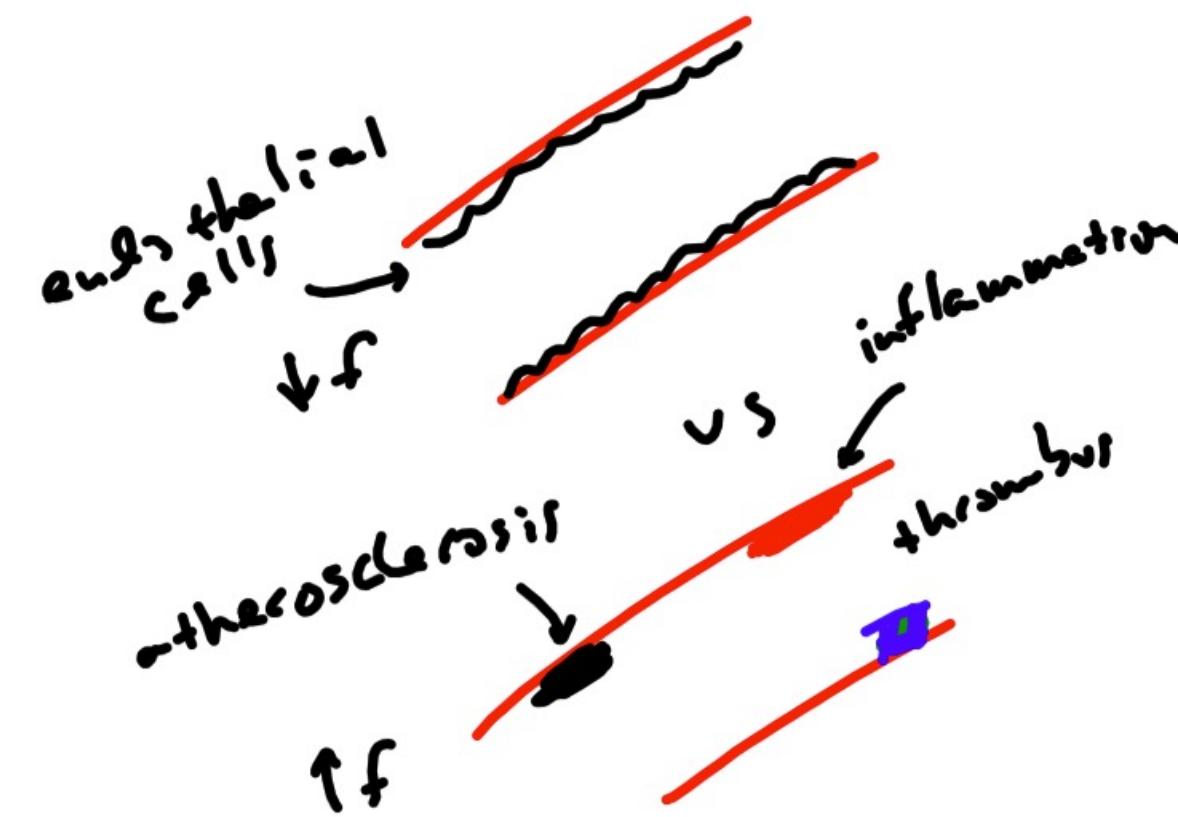
OR

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

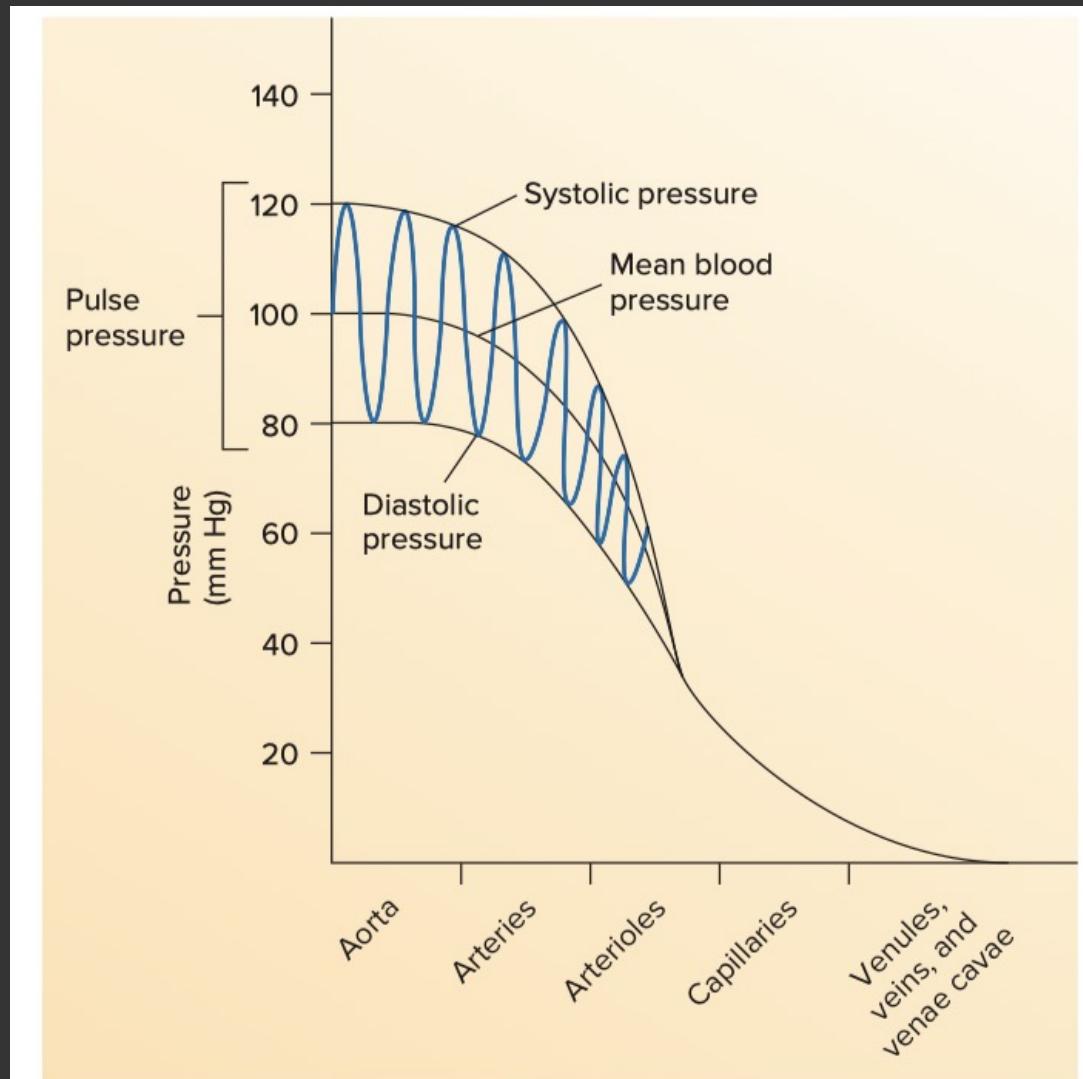
$$= \frac{250 \text{ mm Hg}}{3}$$

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

$$\begin{aligned} & (BP) \\ MAP &= (CO) \times (TPR) \\ &= (HR) \times (SV) \times (\%_r) \times (r) \times (f) \end{aligned}$$



# 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 :
  - $\text{MAP} = (\text{Cardiac Output}) \times (\text{Total Peripheral Resistance})$
- 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

Increase CO

Increase SV

Increase HR

Increase TPR

Decrease radius

Increase viscosity

Increase friction

## What decreases BP

Decrease CO

Decrease SV

Decrease HR

Decrease TPR

Increase radius

Decrease viscosity

Decrease friction

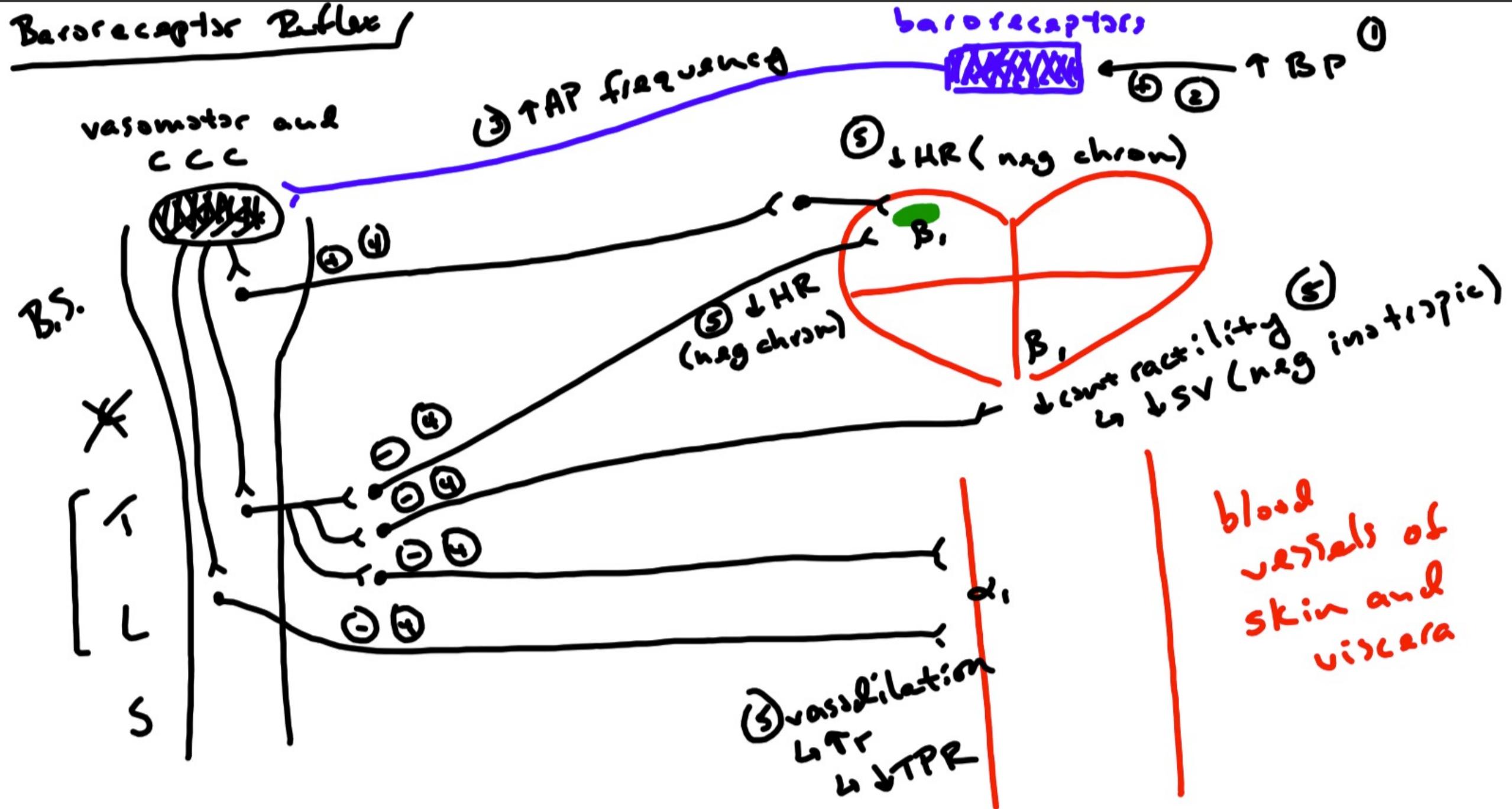
# Regulation of Blood Pressure

- 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



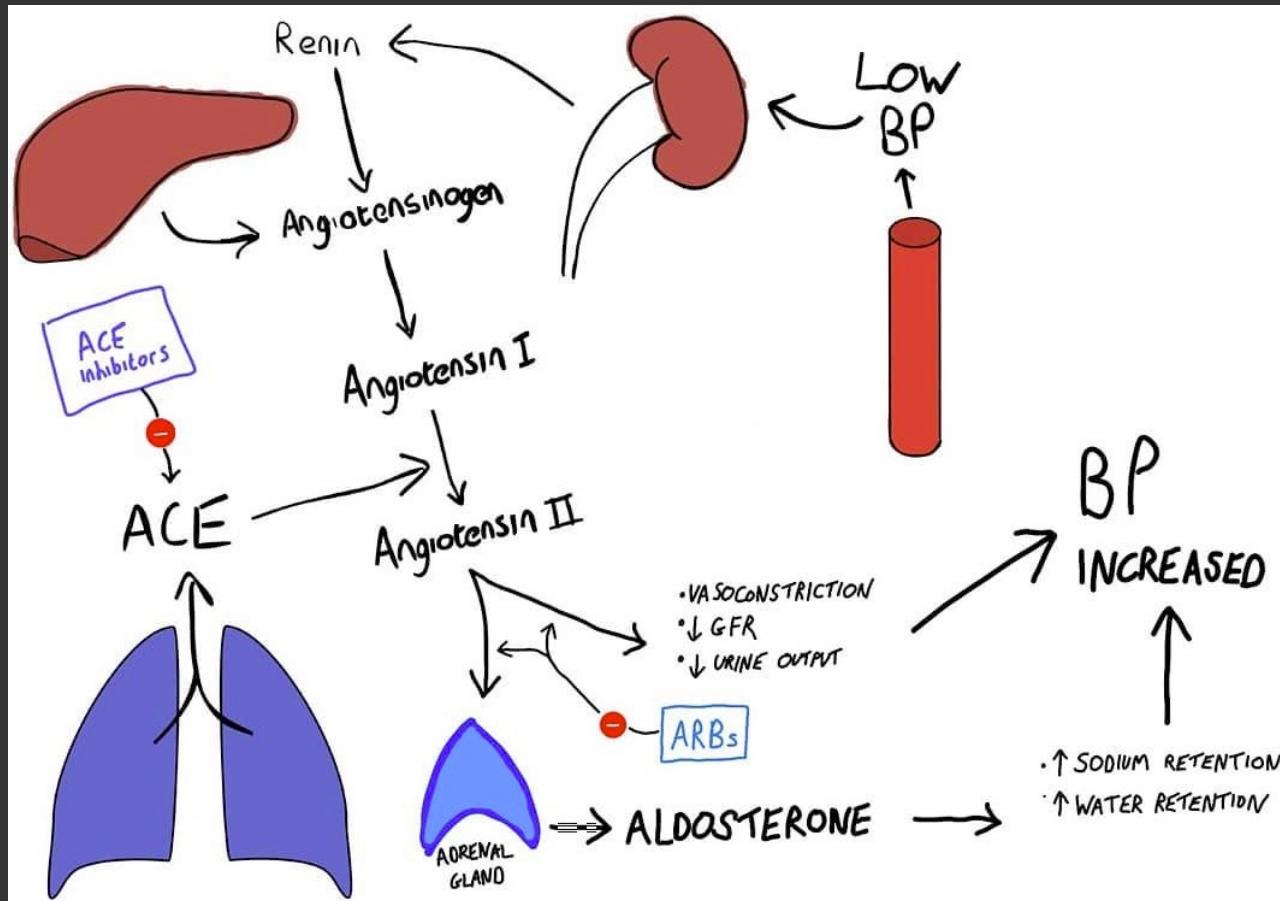
- } stim para  $\rightarrow$  ↓ HR  
↓ inhibit sympathetic  $\rightarrow$  ↓ HR, ↓ SV, ↑ r (↓ TPR)  
↓ ↓ production of angiotensin II  
↓ ↓ release of aldosterone  
↓ ↑ release ANF

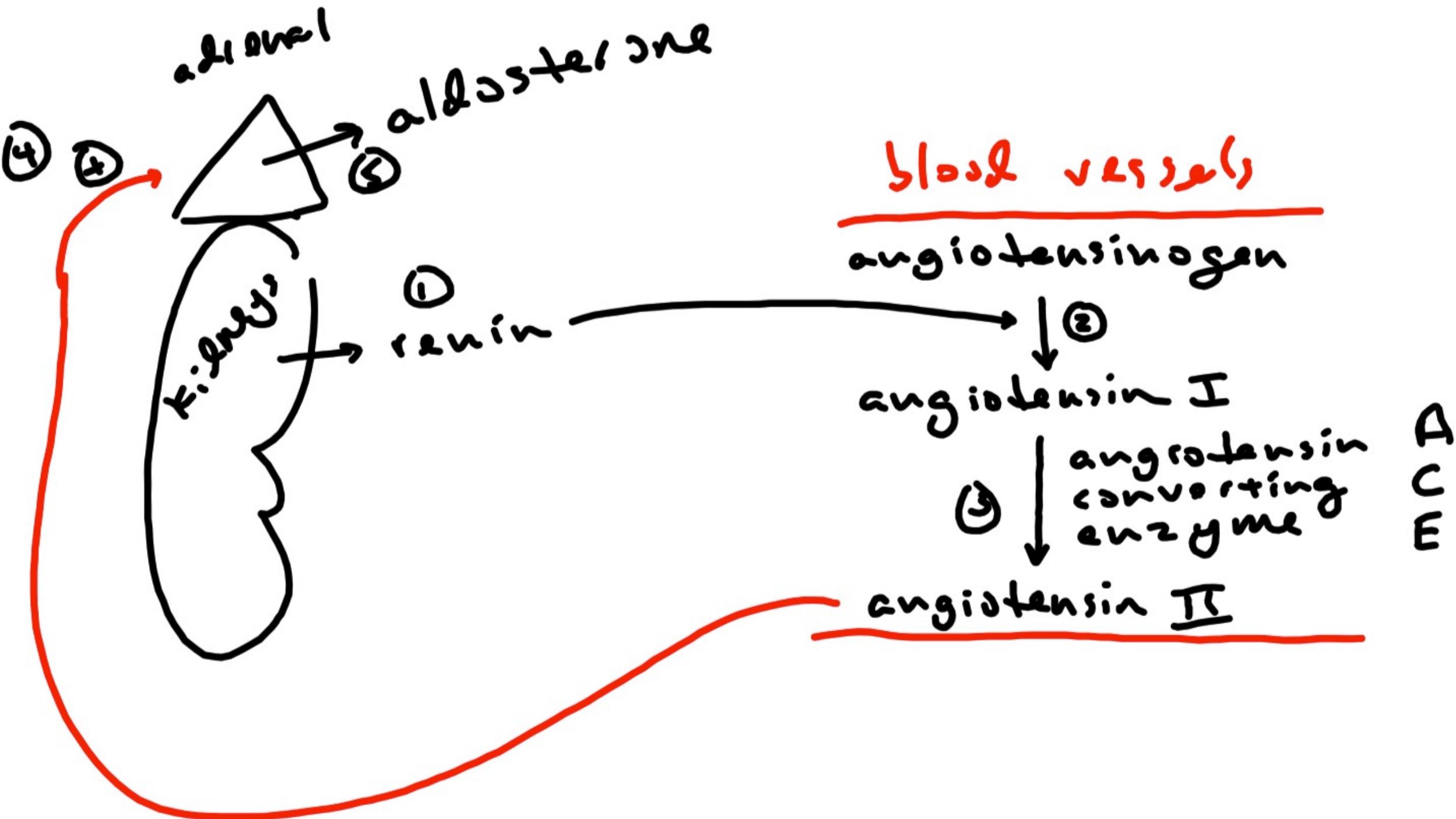


- ↓ inhibit para  $\rightarrow$  ↑ HR  
↓ stim sympathetic  $\rightarrow$  ↑ HR, ↑ SV, ↓ r (↑ TPR)  
↑ production of angiotensin II  
↑ release of aldosterone  
↓ release of ANF

# 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 antidiuretic 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





## \* Angiotensin II

↑ blood volume

↳ causes release aldosterone  $\rightarrow$  vasoconstricts

↳ causes vasoconstriction

↳ causes release of ADH  $\rightarrow$  ↑ blood volume  
 $\rightarrow$  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")

↳ benefit:

↳ increase force against walls of blood vessels

(TBP a little)



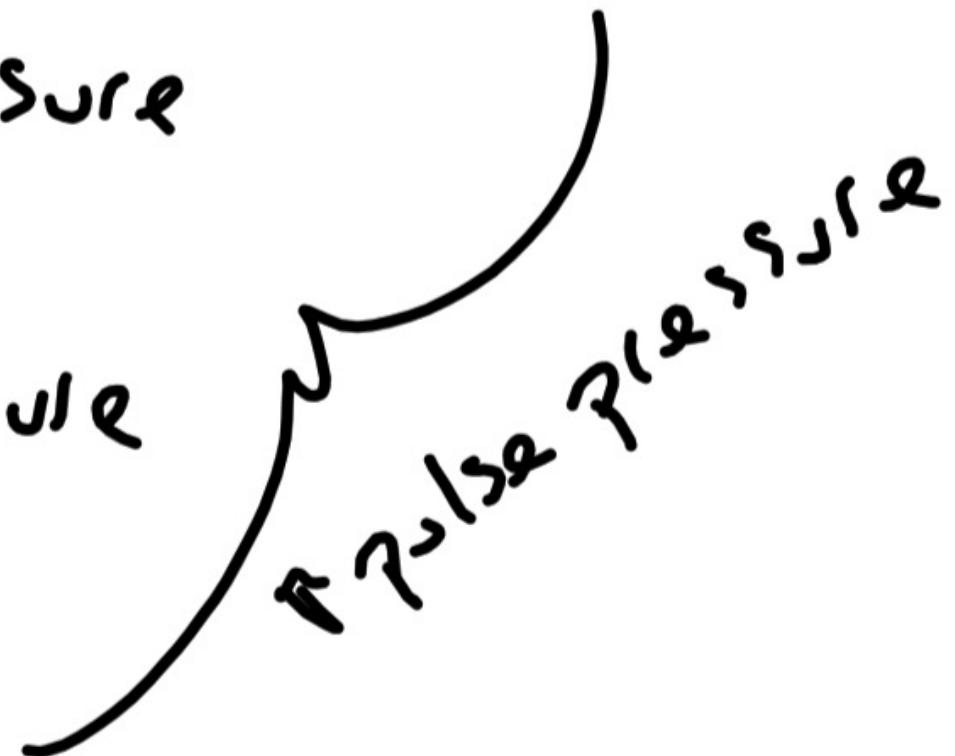
\* ↓ compliance → too stiff

↳ During systole:

↳ ↑ systolic pressure

↳ During Diastole:

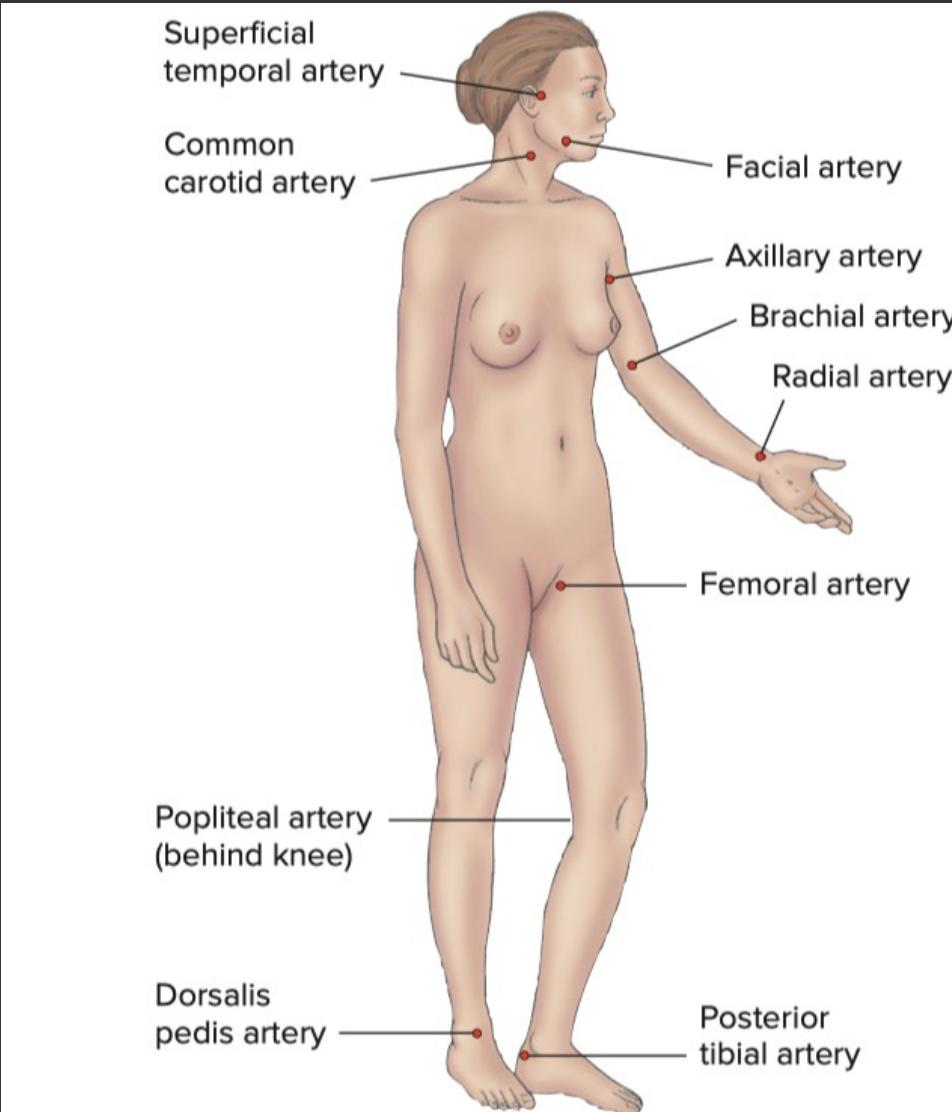
↳ ↓ diastolic pressure



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 (→ too 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
  - $\text{Force} = P \times D$ 
    - 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 )
    - $\beta$ -agonist
      - Increases contractility ( i.e. SV ) and heart rate
    - $\alpha$ -agonist
      - Vasoconstriction
  - Dopamine
    - $\beta$ -agonist ( at medium doses )
      - Increases contractility ( i.e. SV ) and heart rate
    - $\beta$ -agonist and  $\alpha$ -agonist ( at high doses )
      - Increases contractility ( i.e. SV ) and heart rate
        - Vasoconstriction
  - Phenylephrine ( neo synephrine )
    - $\alpha$ -agonist
      - Vasoconstriction
  - Vasopressin
    - Increases blood volume
    - $\alpha$ -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 :

In 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

## ➤ $\beta$ -blockers

- Decrease contractility ( i.e. SV ) and heart rate

## ➤ $\alpha$ -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

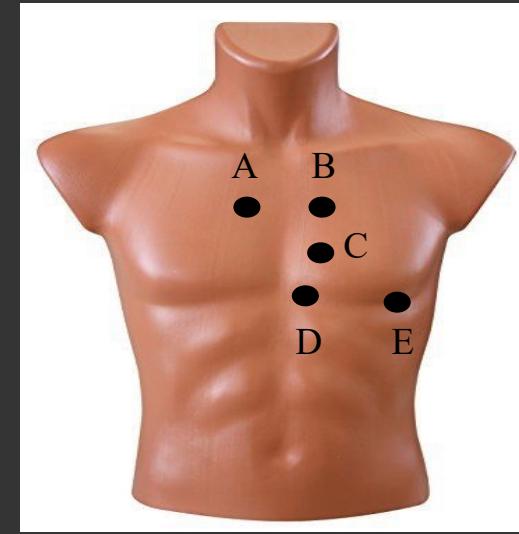
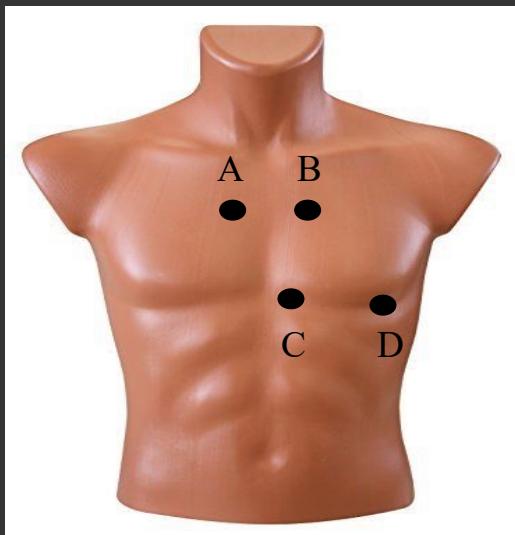
- Inhibit production of angiotensin II
- Decrease peripheral resistance
  - Vasodilation
- Decrease blood volume ( via inhibition of aldosterone )

## ➤ Angiotensin II receptor blockers

- Decrease peripheral resistance
  - Vasodilation
- Decrease blood volume ( via inhibition of aldosterone )

## ➤ Renin inhibitors

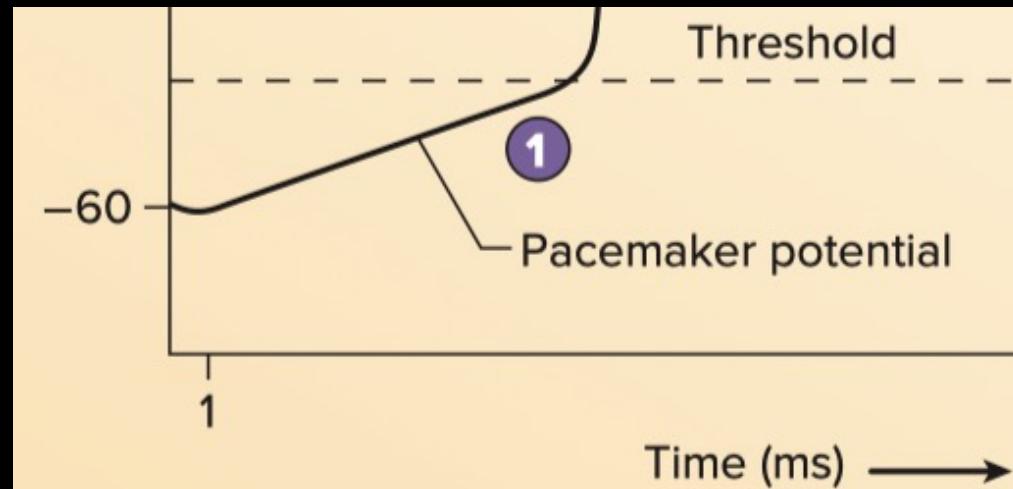
- Decrease peripheral resistance
  - Vasodilation
- Decrease blood volume ( via inhibition of aldosterone )



# Sinoatrial Node- Action Potential- Pacemaker

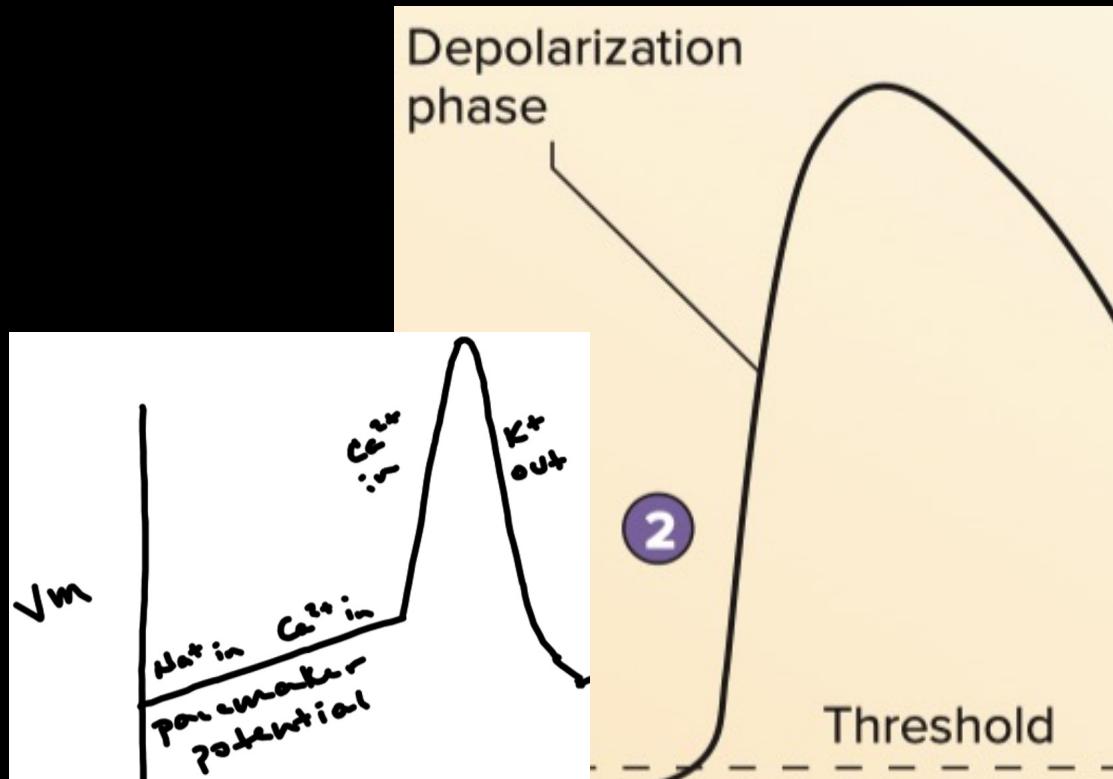
## 1.) Pacemaker Potential

- .) 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

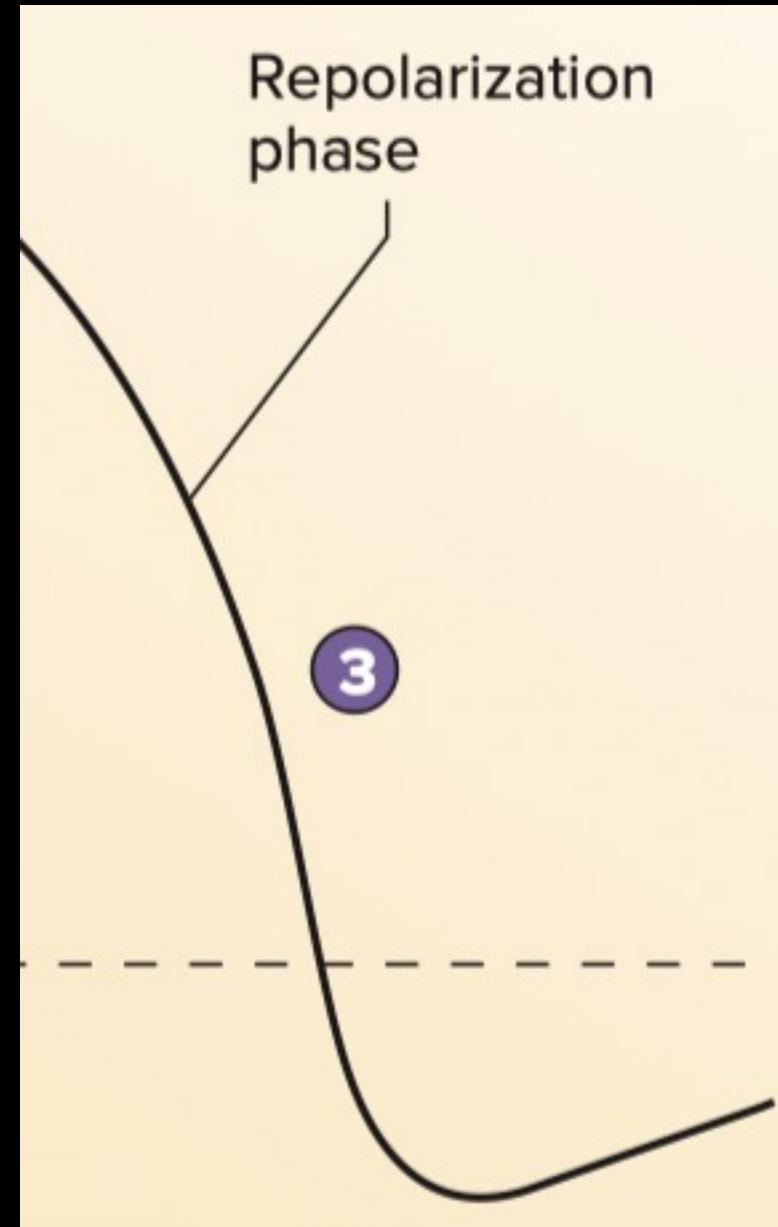
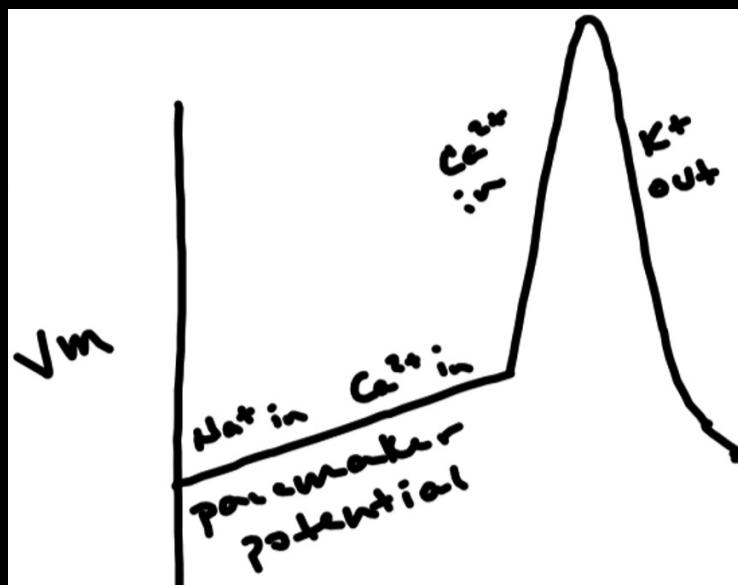
- .)      Calcium      Enters
- .) Voltage-Gated      Potassium      Channels Close



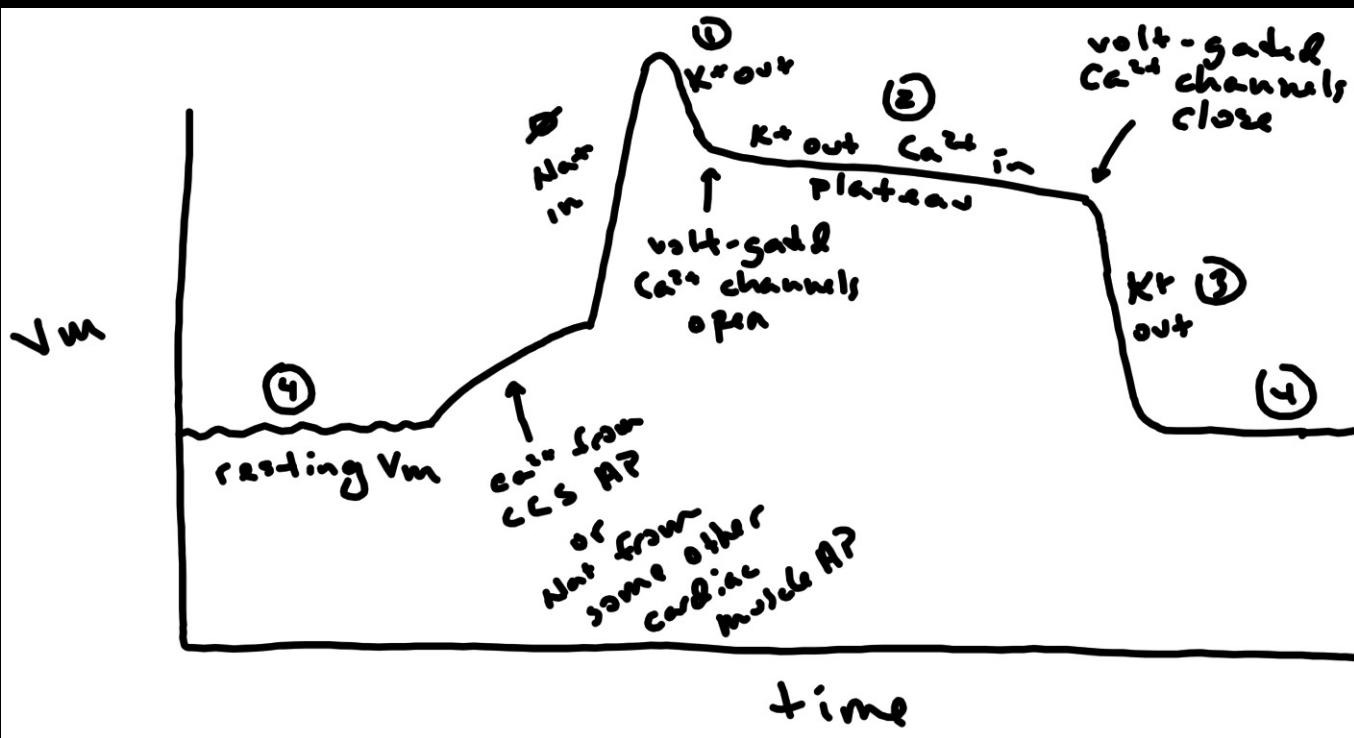
# Sinoatrial Node- Action Potential- Pacemaker

## 3.) Repolarization Phase

- .) Voltage-Gated Calcium Channels Close
- .) Voltage-Gated Potassium Channels Open
- .) Less Potassium  $\therefore$  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

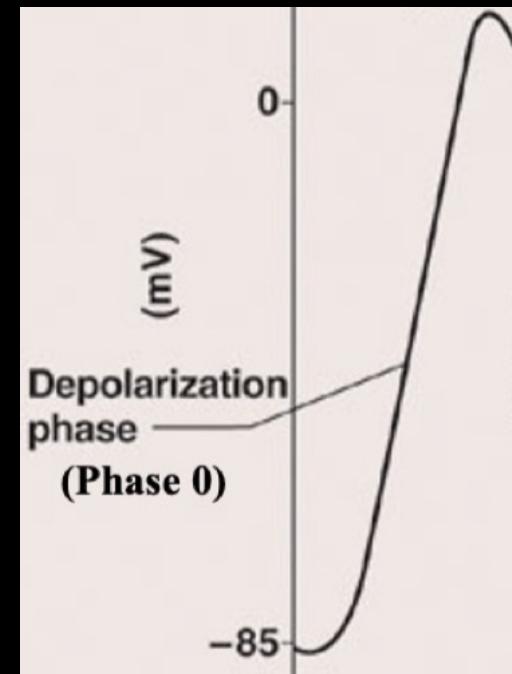
Threshold reached via:

Action potential from cardiac conduction system

Action potential from adjacent cardiac muscle cell

Phase 0 - fast depolarization phase

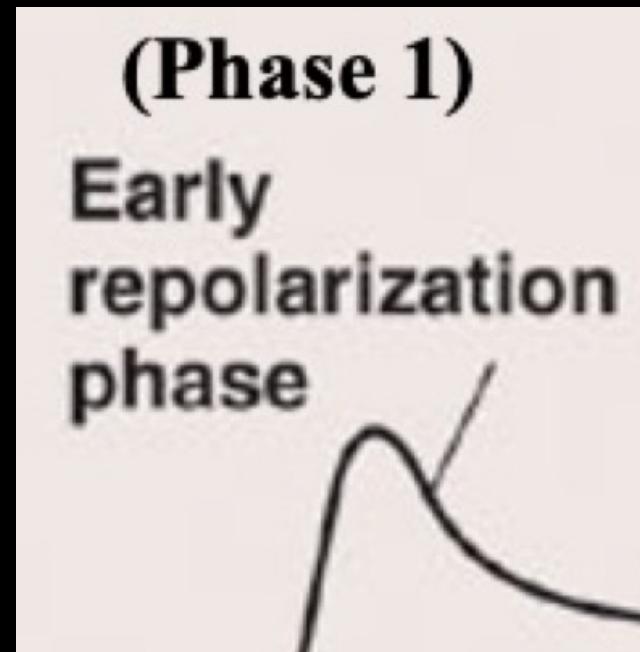
- 1.) Voltage-Gated Sodium Channels Open
- 2.) Sodium Enters



# Cardiac Muscle - Action Potential

Phase 1 - early fast repolarization

- 3.) Voltage-Gated Potassium Channels Open
- 4.) Potassium Enters



# Cardiac Muscle- Action Potential

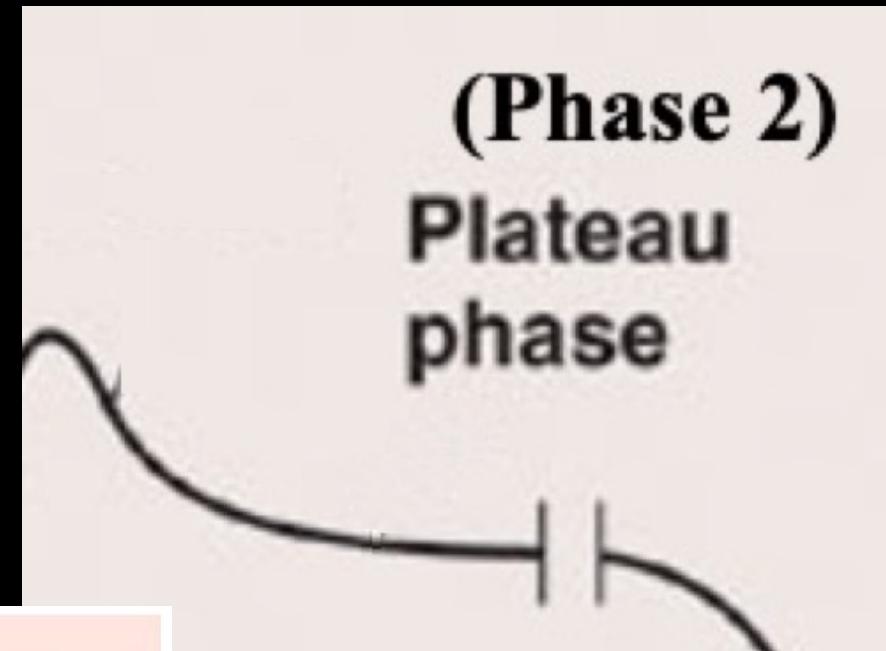
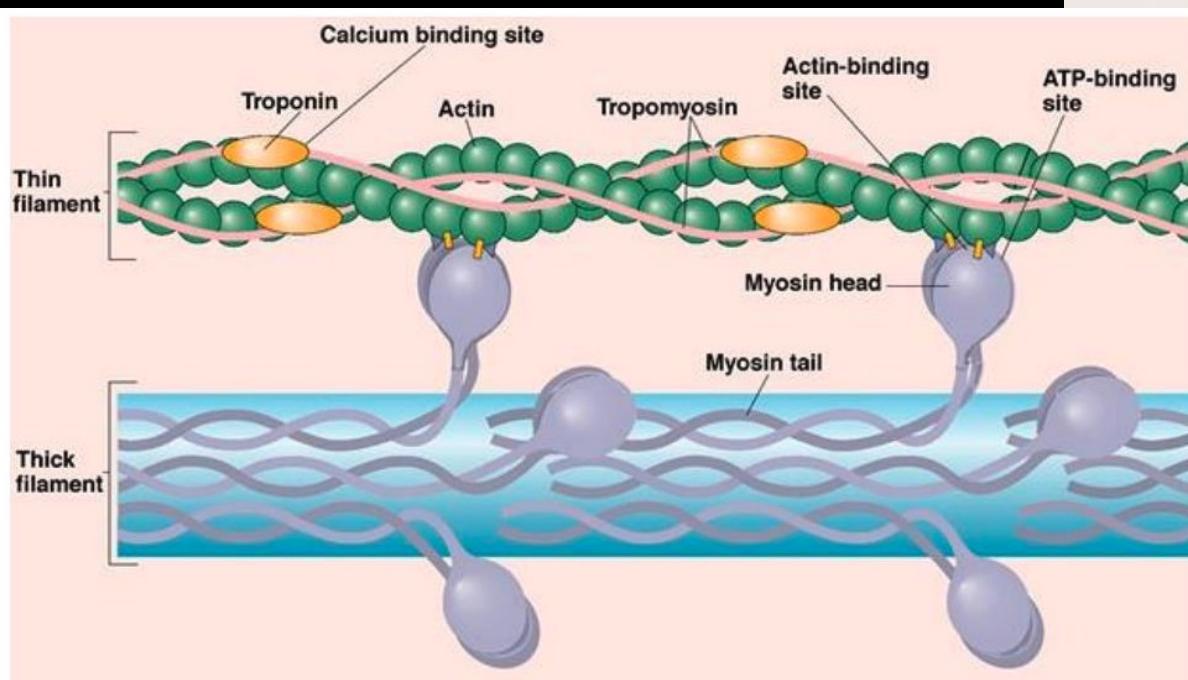
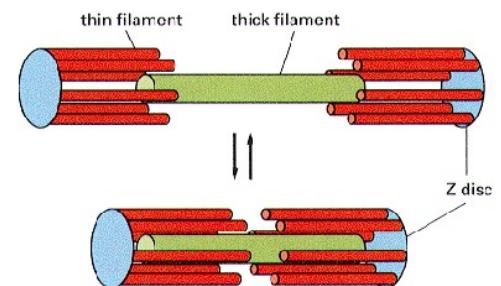
Phase 2 - Plateau Phase

5.) Voltage-Gated Calcium Channels Open

Potassium is still leaving

6.) Calcium Enters

Called Trigger Calcium

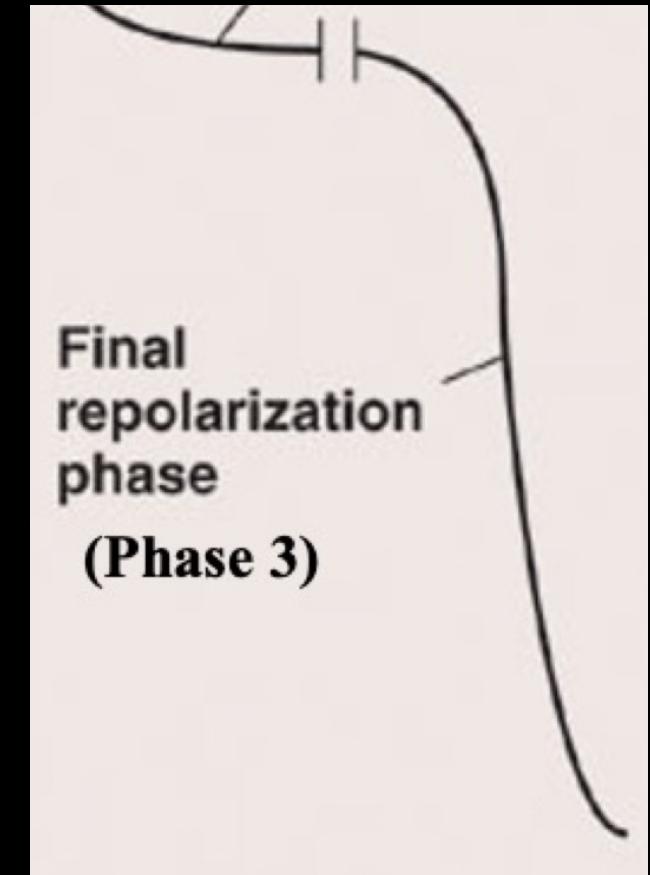
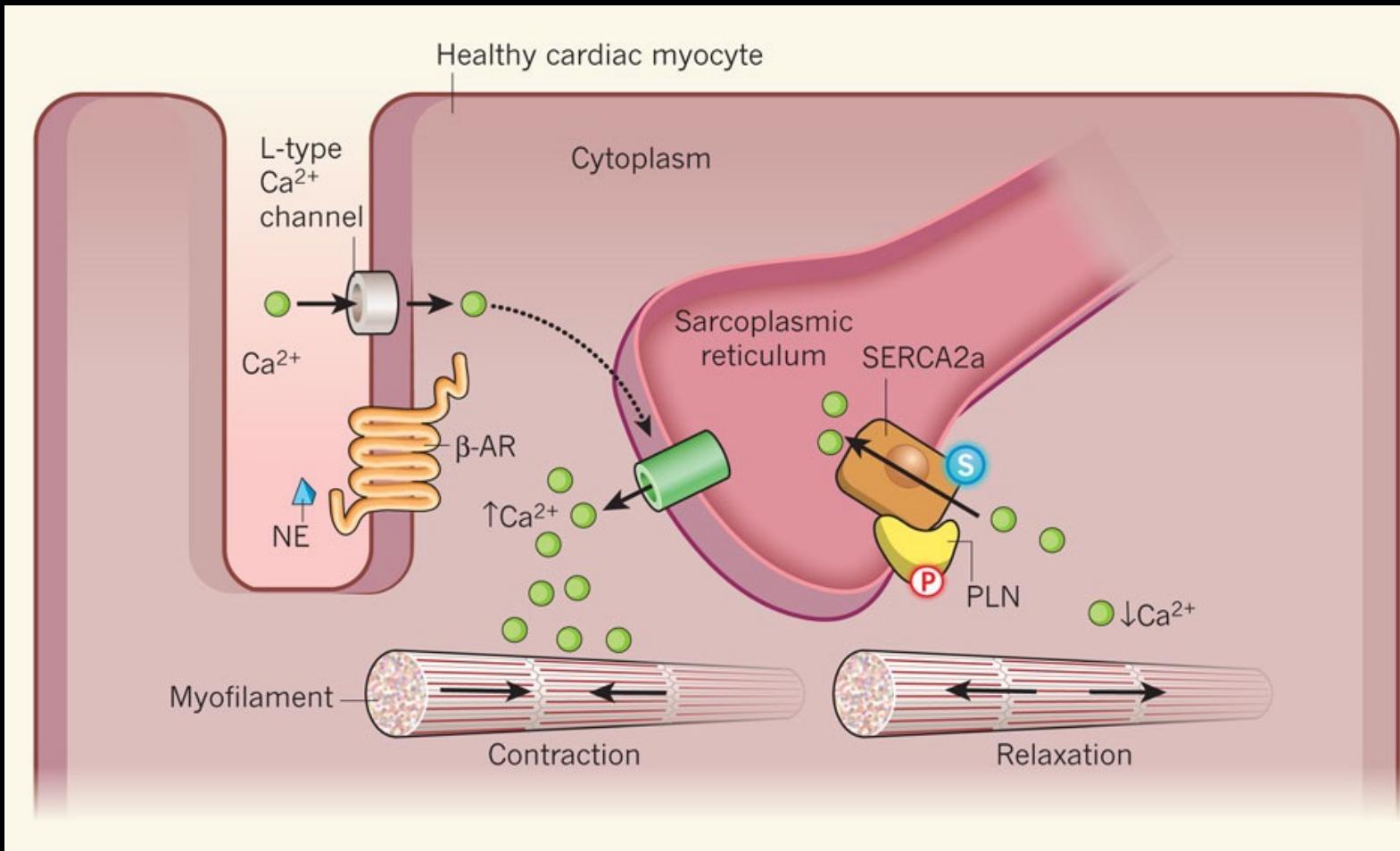


# Cardiac Muscle - Action Potential

Phase 3 - Final Fast Repolarization Phase

7.) Potassium Leaves

8.) Voltage-Gated Calcium Channels Close

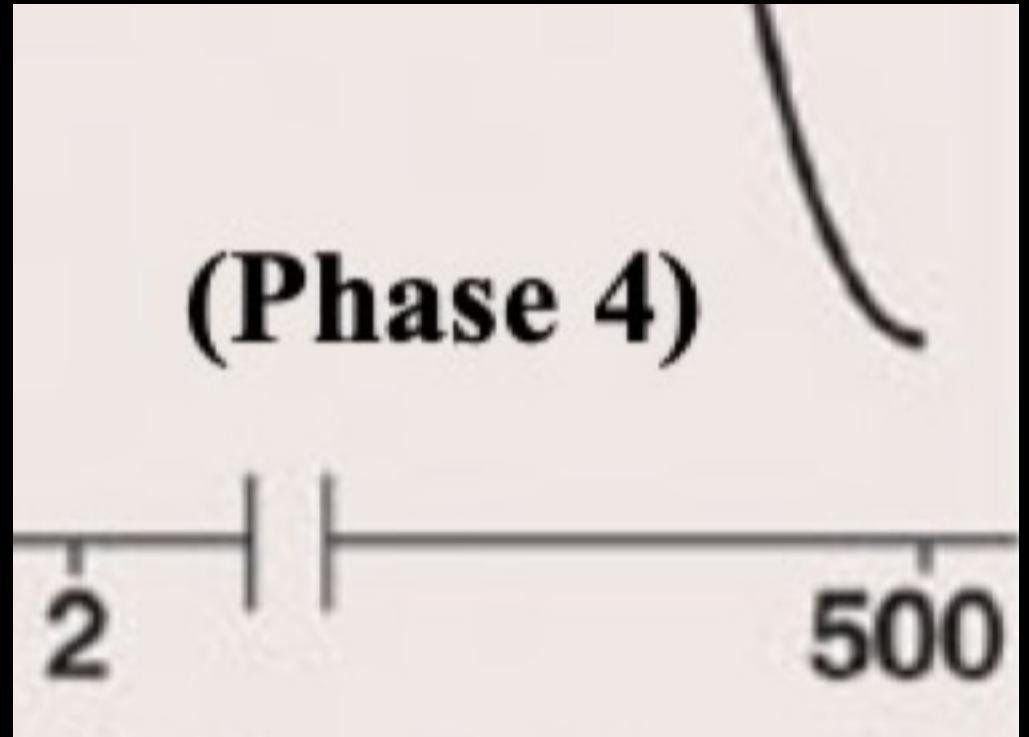


Contraction stops when calcium is pumped back into the sarcoplasmic reticulum

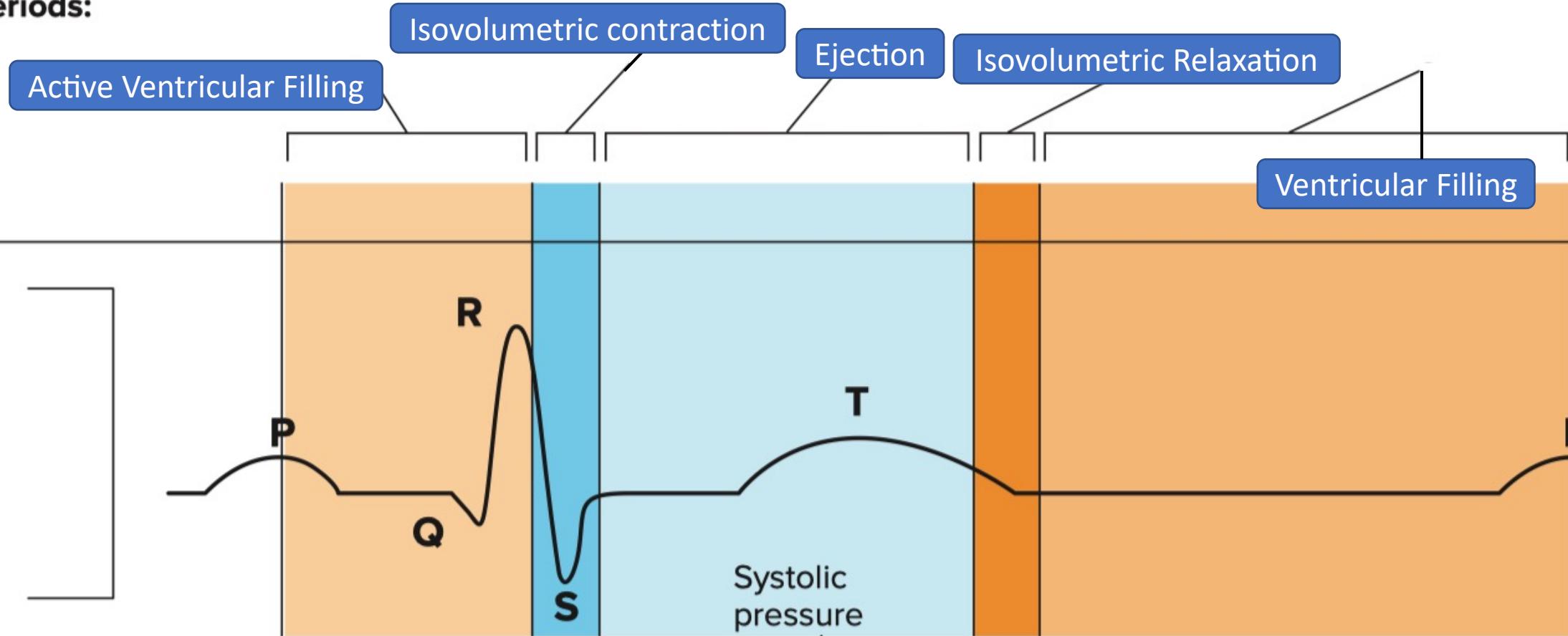
# Cardiac Muscle- Action Potential

Phase 4 - resting  $V_m$

- 9.) Potassium Channels Eventually Close
- 10.) Resting  $V_m$  is established

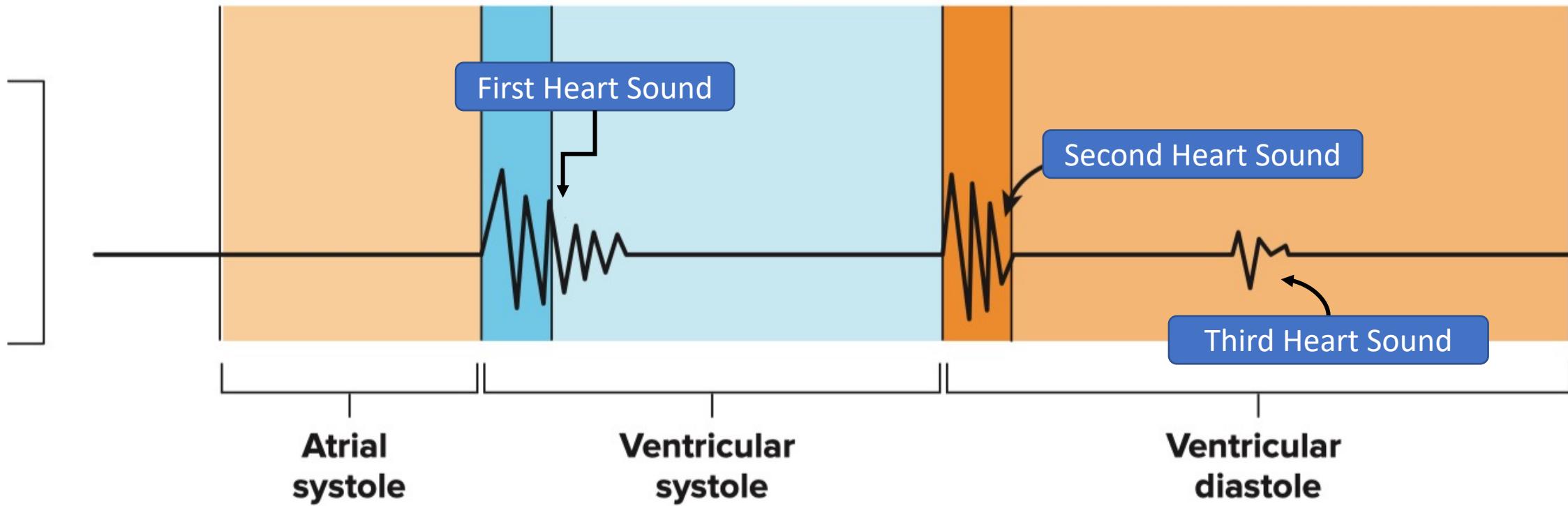


**Time periods:**

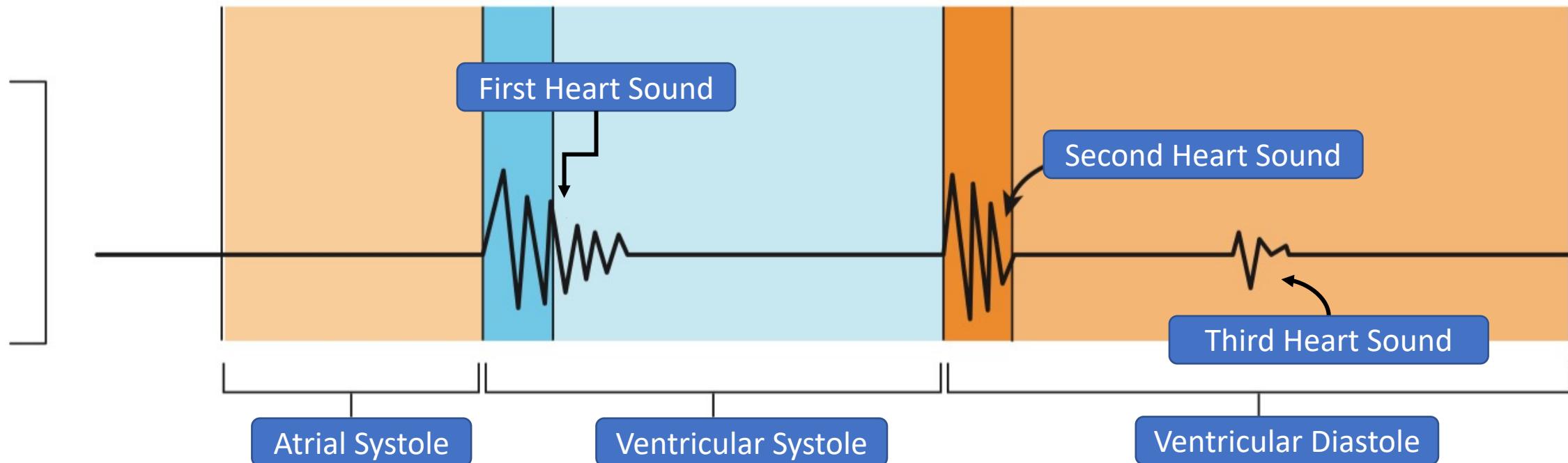


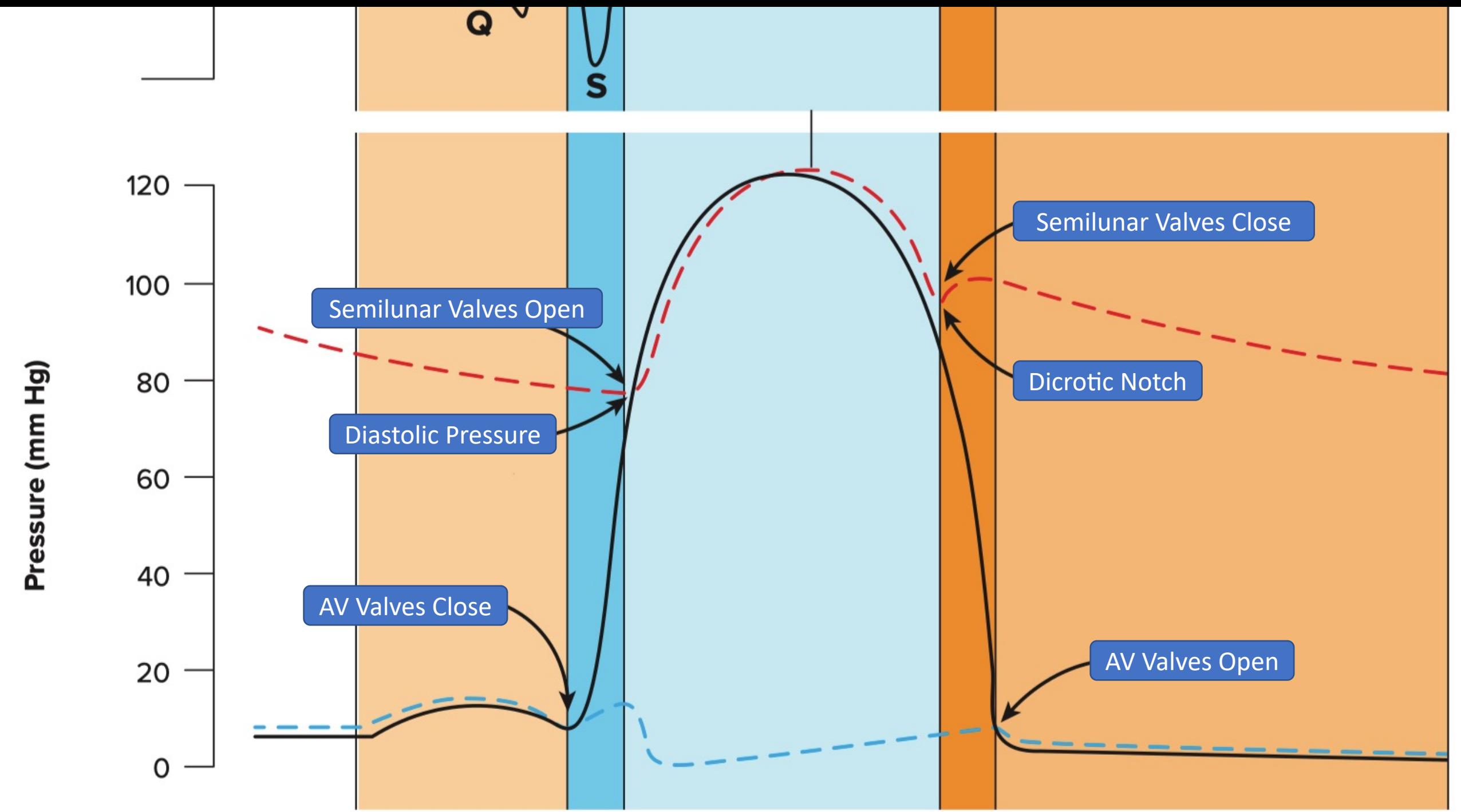
Panel 1

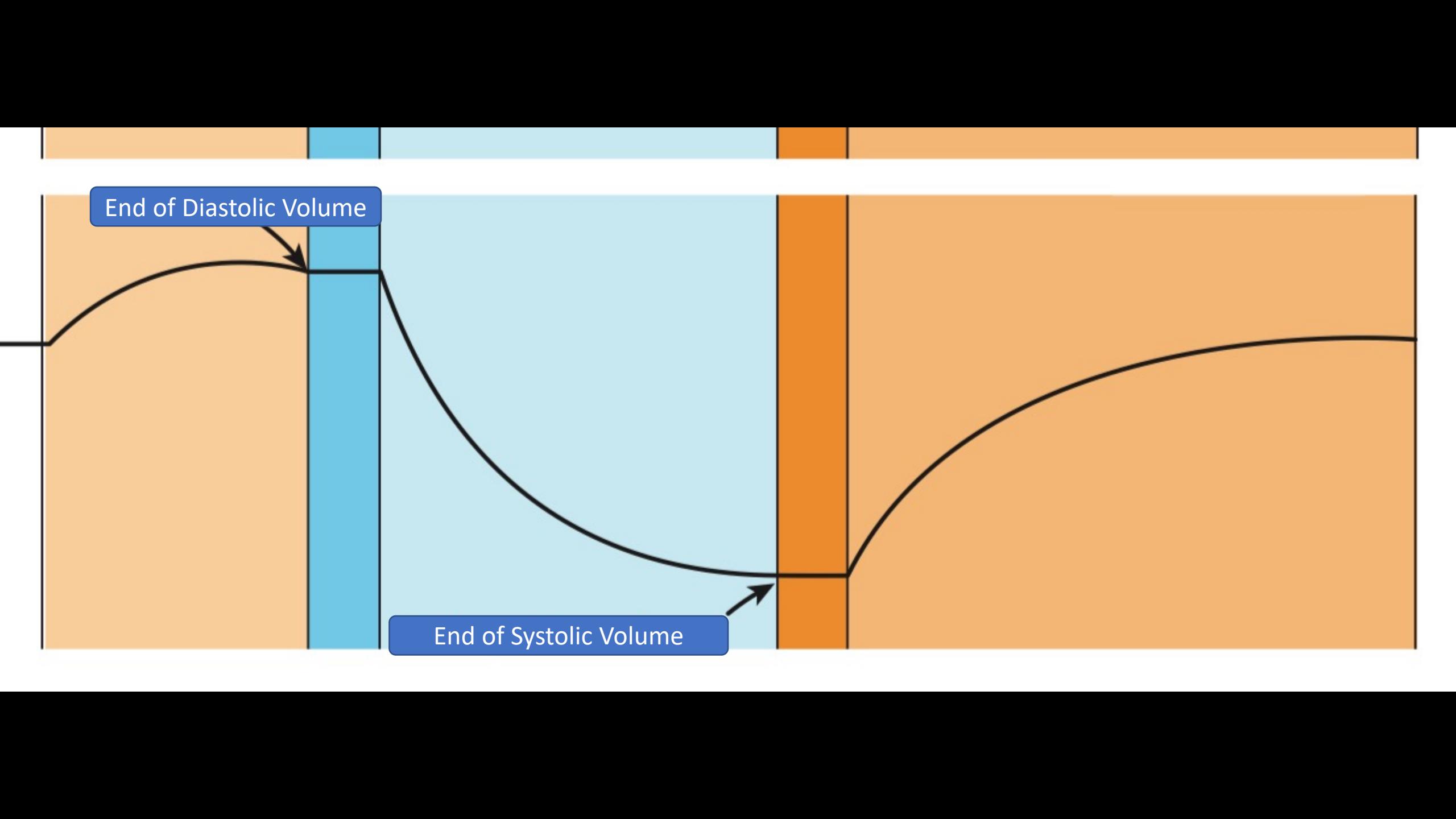
"Sound" frequency  
(cycles/second)



"Sound" frequency  
(cycles/second)



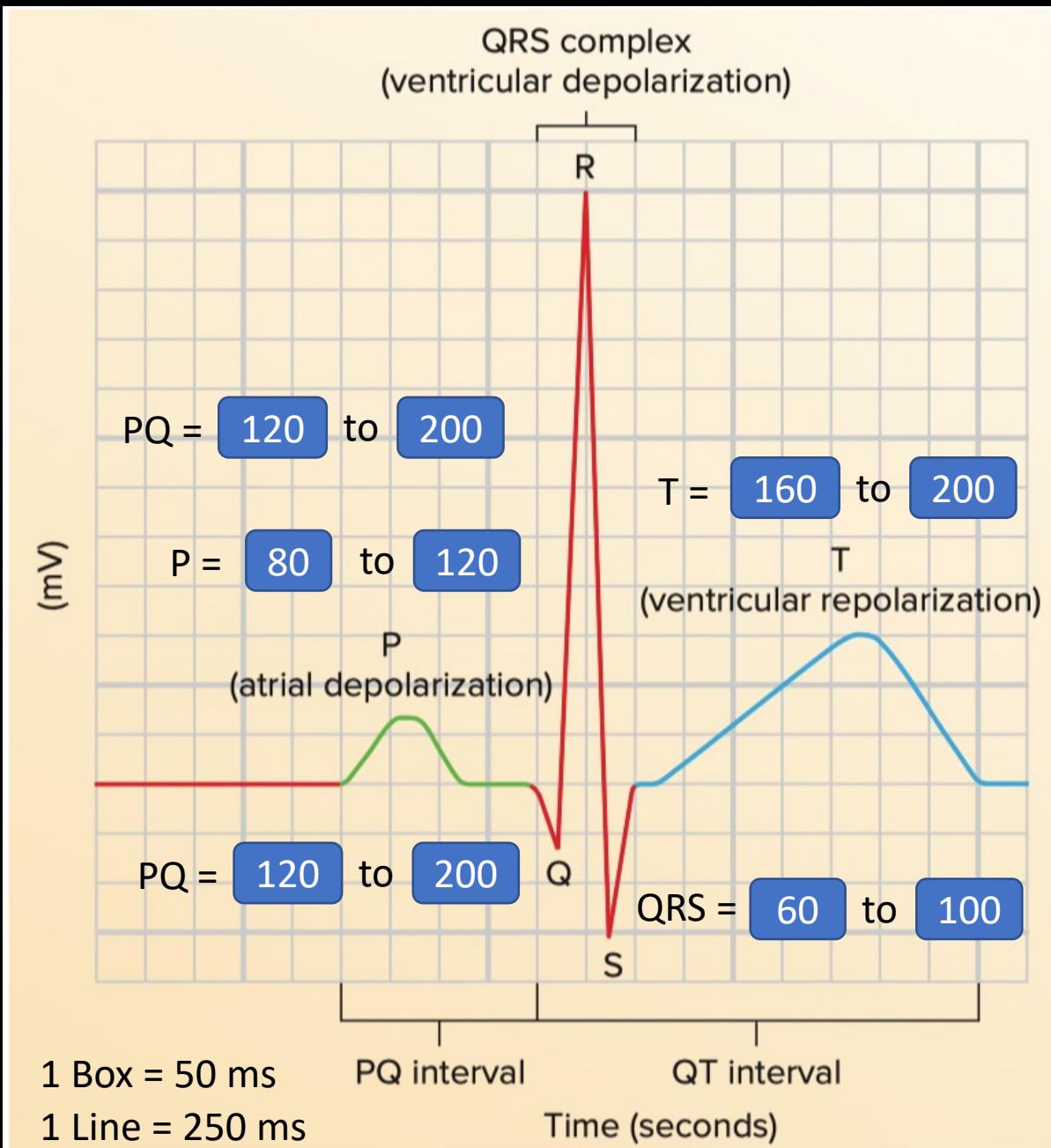




End of Diastolic Volume

End of Systolic Volume

# Trace



## **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 40 to 60 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  $0^\circ$  angle
- Lead II: Right Arm ( - ) to Left Leg ( + )
  - Measures electrical activity across the heart at a  $+60^\circ$  angle
- Lead III: Left Arm ( - ) to Left Leg ( + )
  - Measures electrical activity across the heart at a  $+120^\circ$  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  $-150^\circ$  angle
- aVL: Left Arm ( + ) to central terminal ground lead ( joining of left leg and right arm )
  - Measures electrical activity across the heart at a  $-30^\circ$  angle
- aVF: Left Leg ( + ) to central terminal ground lead ( joining of right arm and left arm )
  - Measures electrical activity across the heart at a  $+90^\circ$  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

# Limb Leads – Standard Bipolar

- **Standard bipolar limb leads**

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  - Measures electrical activity across the heart at a  $+60^\circ$  angle
- **Lead 3** : left arm ( - ) to left leg ( + )
  - Measures electrical activity across the heart at a  $+120^\circ$  angle

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

- **Chest leads / Precordial leads**

- Measure the electrical activity of the heart in a transverse plane
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# Limb Leads – Standard Bipolar

- **Standard bipolar limb leads**

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  - 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

# Limb Leads – Standard Bipolar

- **Standard bipolar limb leads**

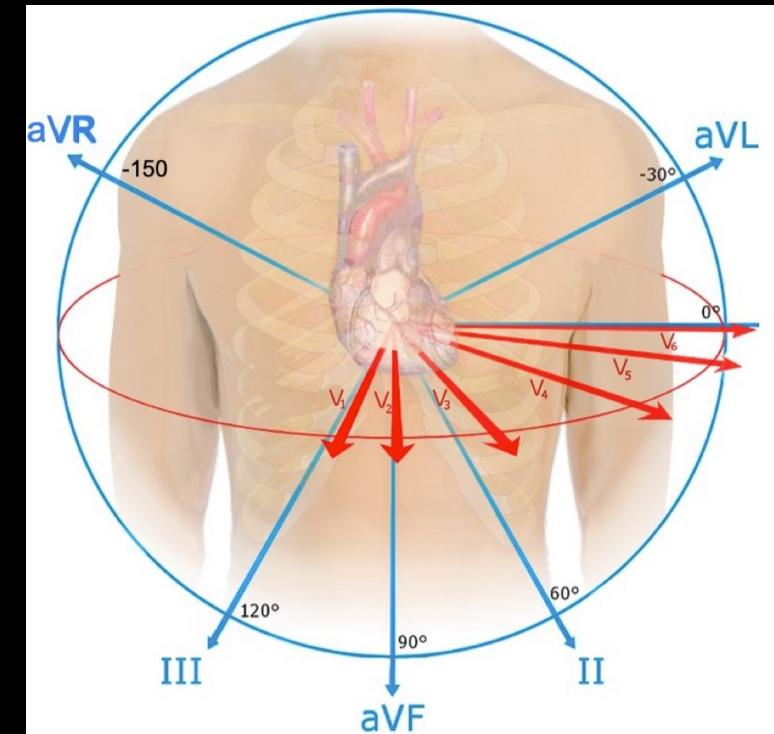
- Measure the electrical activity of the heart in a **Frontal Plane**
- **Lead 1** : Right Arm ( - ) to Left Arm ( + )
  - Measures electrical activity across the heart at a  $0^\circ$  angle
- **Lead 2** : Right Arm ( - ) to Left Leg ( + )
  - Measures electrical activity across the heart at a  $+60^\circ$  angle
- **Lead 3** : Left Arm ( - ) to Left Leg ( + )
  - Measures electrical activity across the heart at a  $+120^\circ$  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  $-150^\circ$  angle
- **aVL** : Left Arm ( + ) to central terminal ground lead ( joining of left leg and right arm )
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- **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 )