Cardiac Conduction System

Group of specialized, highly excitable cells that control heart rate

Spontaneously generates and conducts action potentials

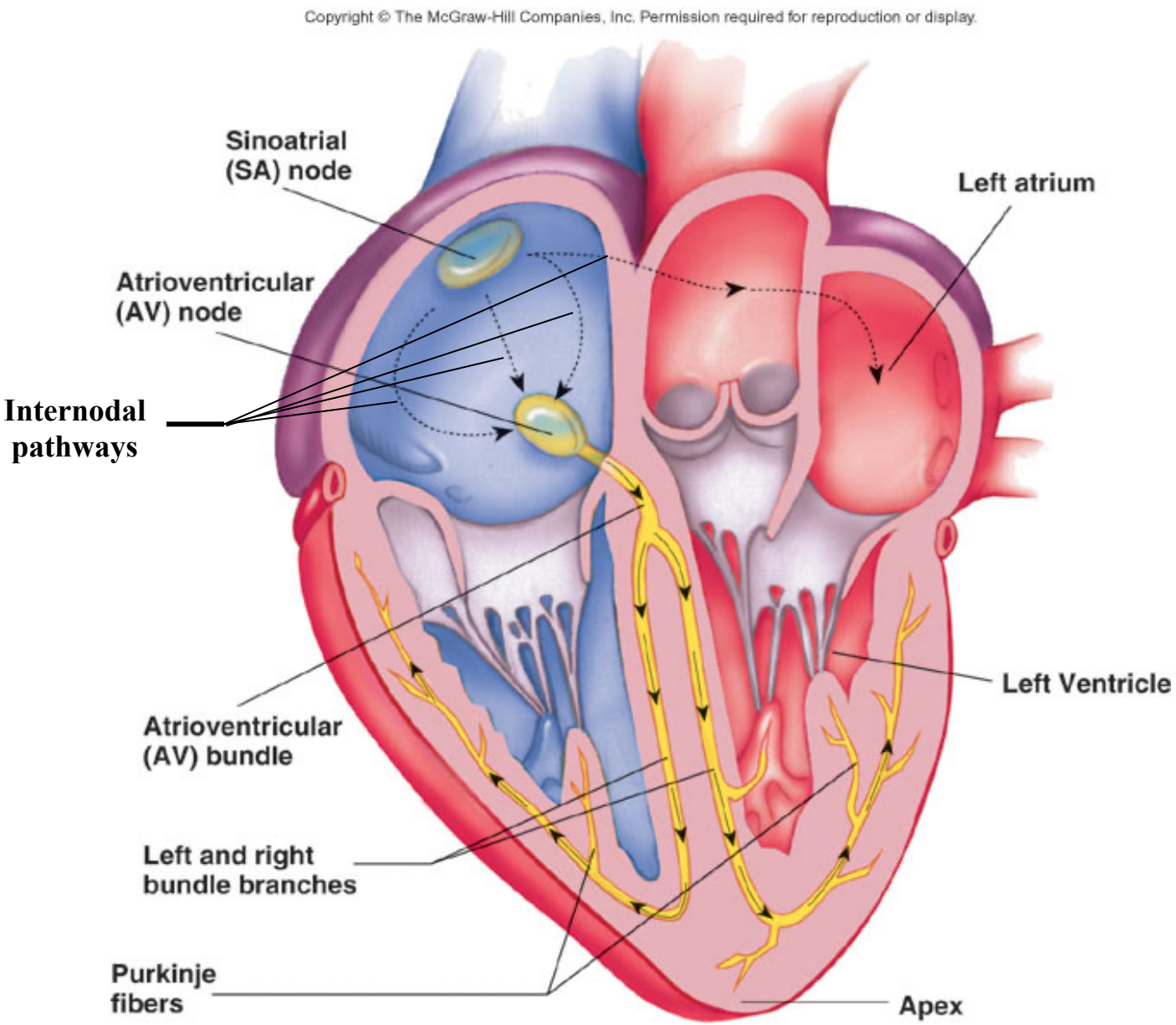
Sinoatrial node (SA node)

Atrioventricular node (AV node)

AV bundle / Bundle of His

Purkinje fibers

Cardiac Conduction System



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

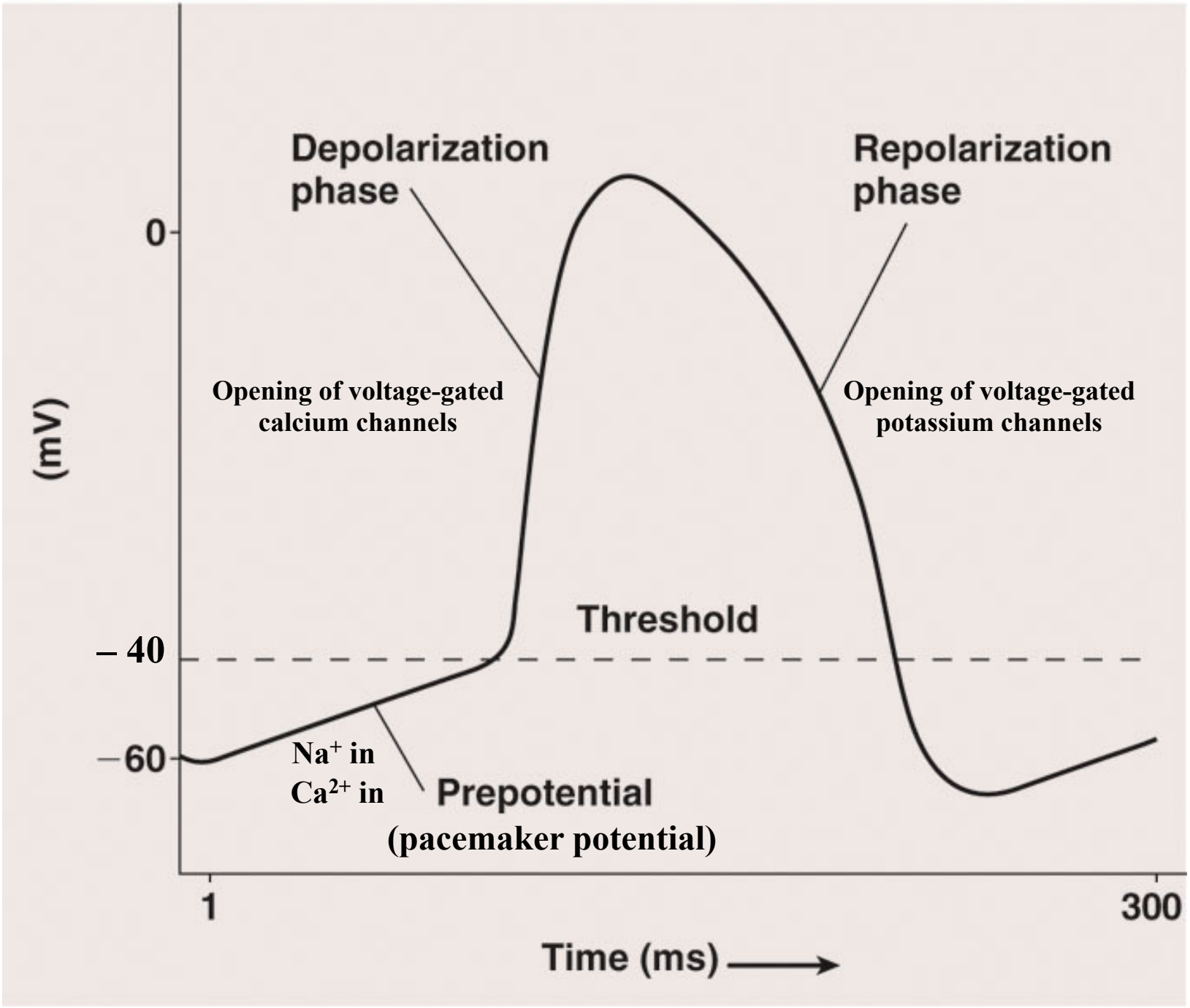
- Opening of voltage-gated Ca^{2+} channels
  - Ca^{2+} transported inward

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



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

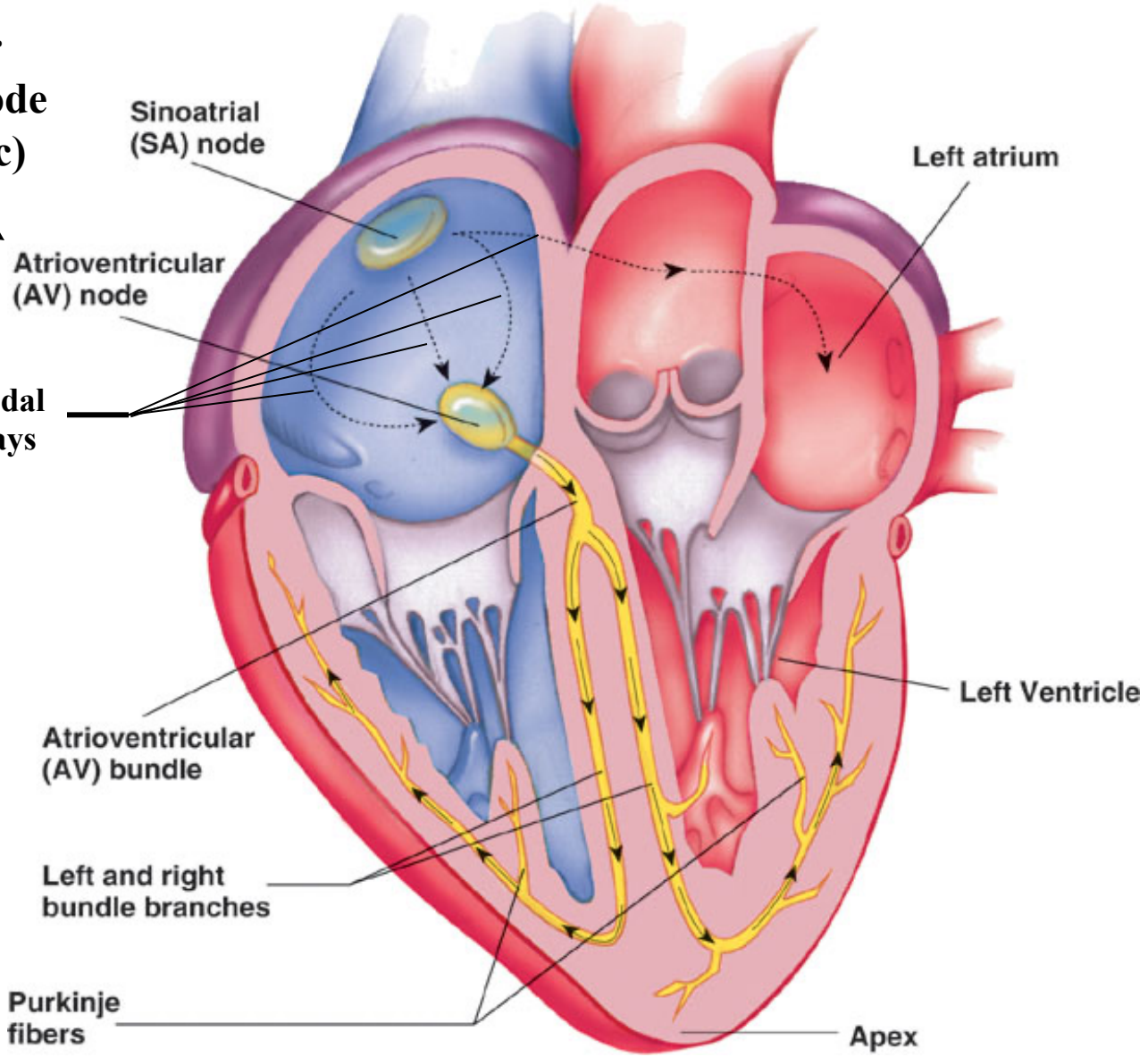
Purkinje fibers

Receive action potentials from the AV bundle / Bundle of His

Conduct action potentials to the cardiac muscle of the ventricles

Cardiac Conduction System

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



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 as the ectopic pacemaker
  - Would slow heart rate to approximately 25 to 45 beats / min

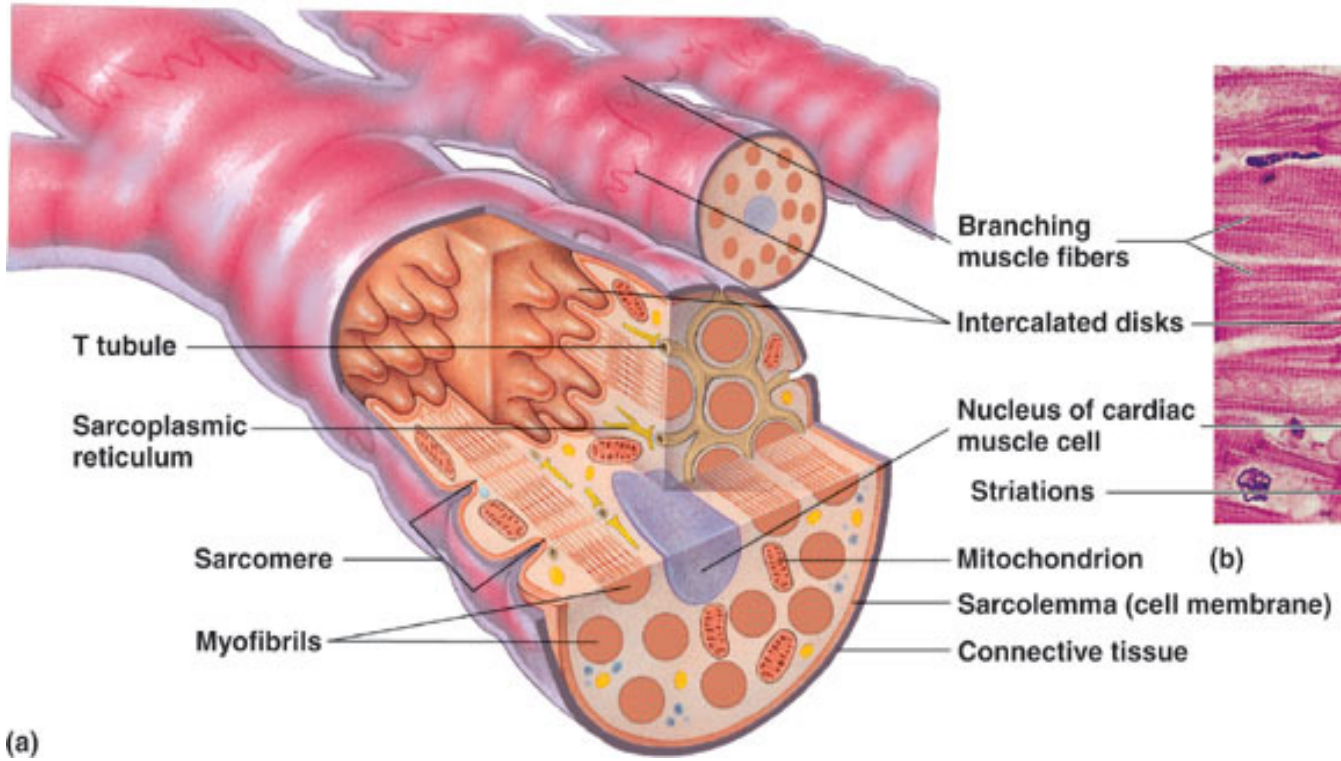
Ectopic pacemakers can either slow down or speed up heart rate

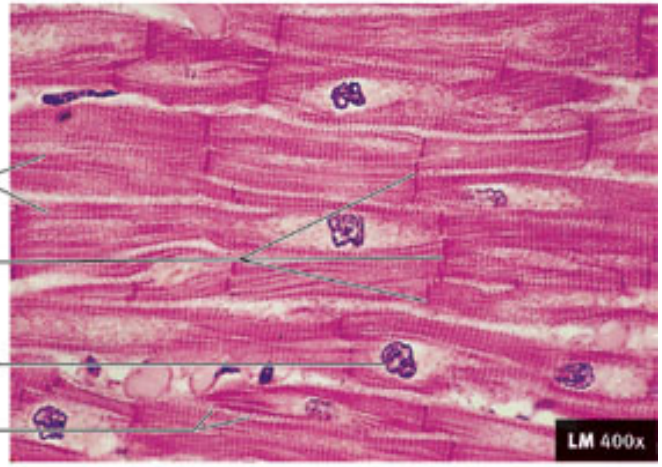
Cardiac Muscle Fiber/Cell

Cardiac muscle cells (myocardium) are arranged in a functional syncitium

Cardiac muscle makes a bulk of the atrial and ventricular walls

Myocardium is a Syncitium





Cardiac Muscle Cell Action Potential

Phase 4 (resting Vm)

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

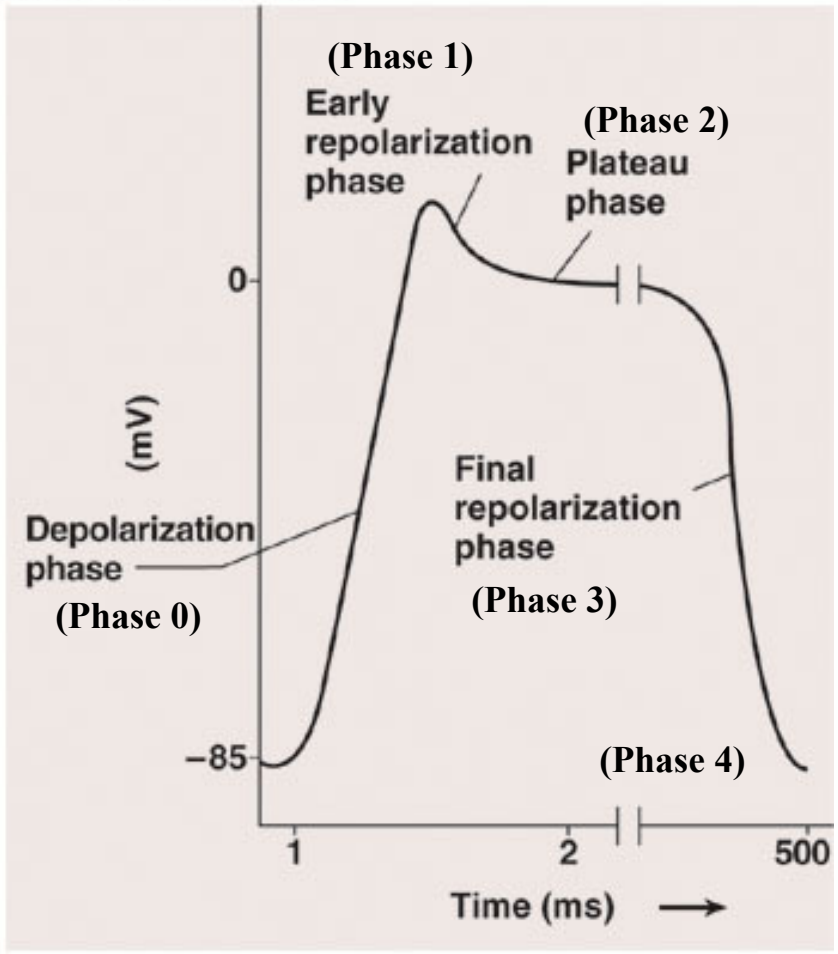
Repolarization occurs slowly at this time

Phase 3 (final fast repolarization phase)

Outward transport of K^+ continues while voltage-gated Ca^{2+} channels close

Causes fast repolarization

Action Potential in a Cardiac Muscle Fiber



(b)

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**

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)

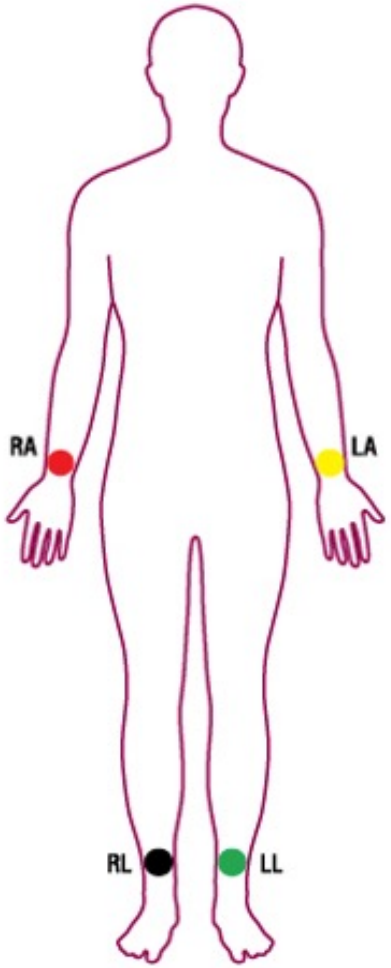
Measures electrical activity across the heart at a +90° angle

Chest leads / Precordial leads

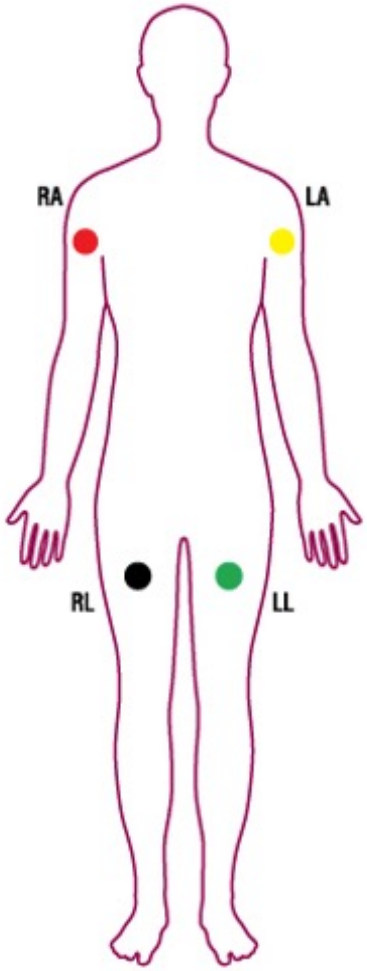
Measure the electrical activity of the heart in a transverse plane

V_1, V_2, V_3, V_4, V_5, V_6 leads arranged across the chest

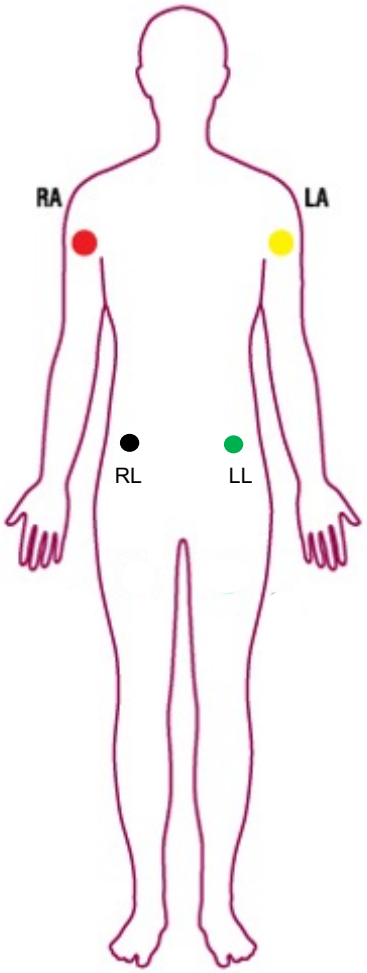
Limb Lead Electrode Placement



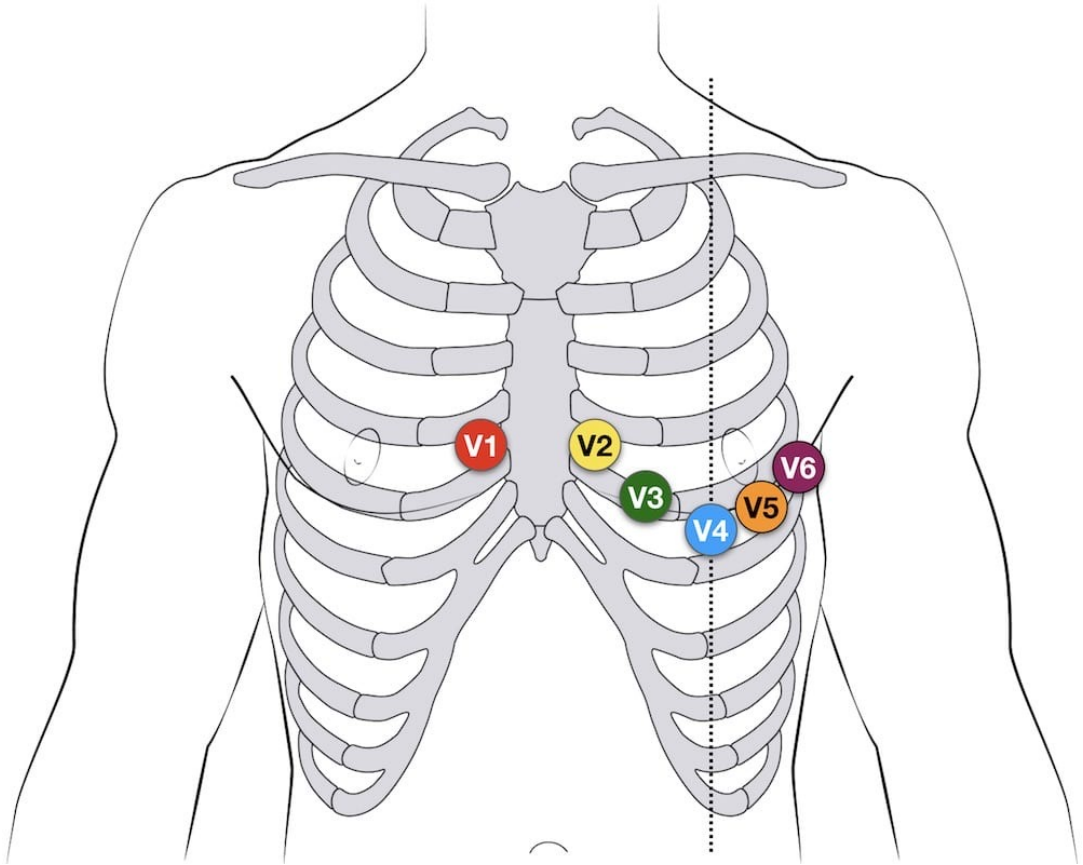
OR

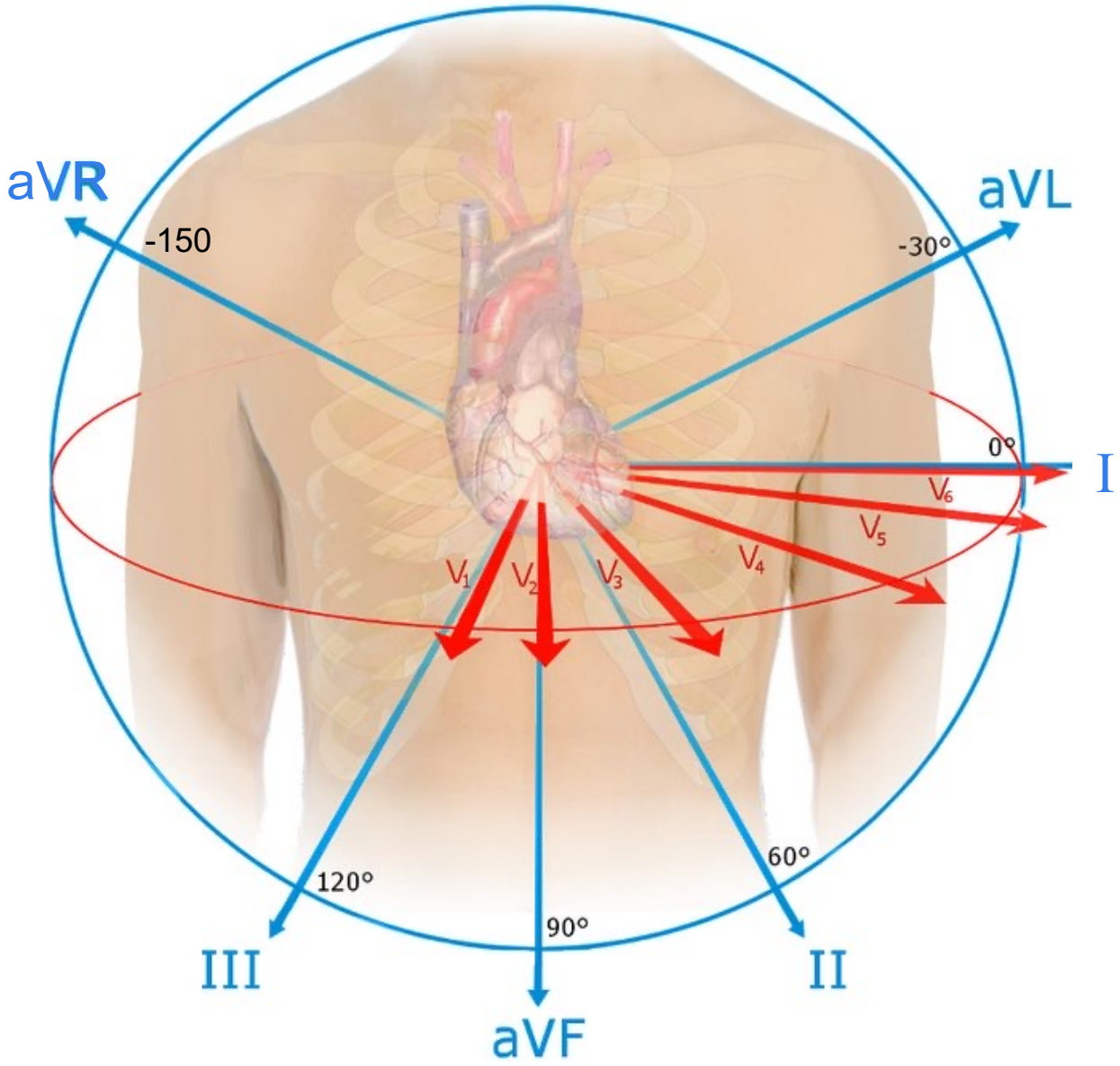


OR



Chest Lead / Precordial Lead Electrode Placement

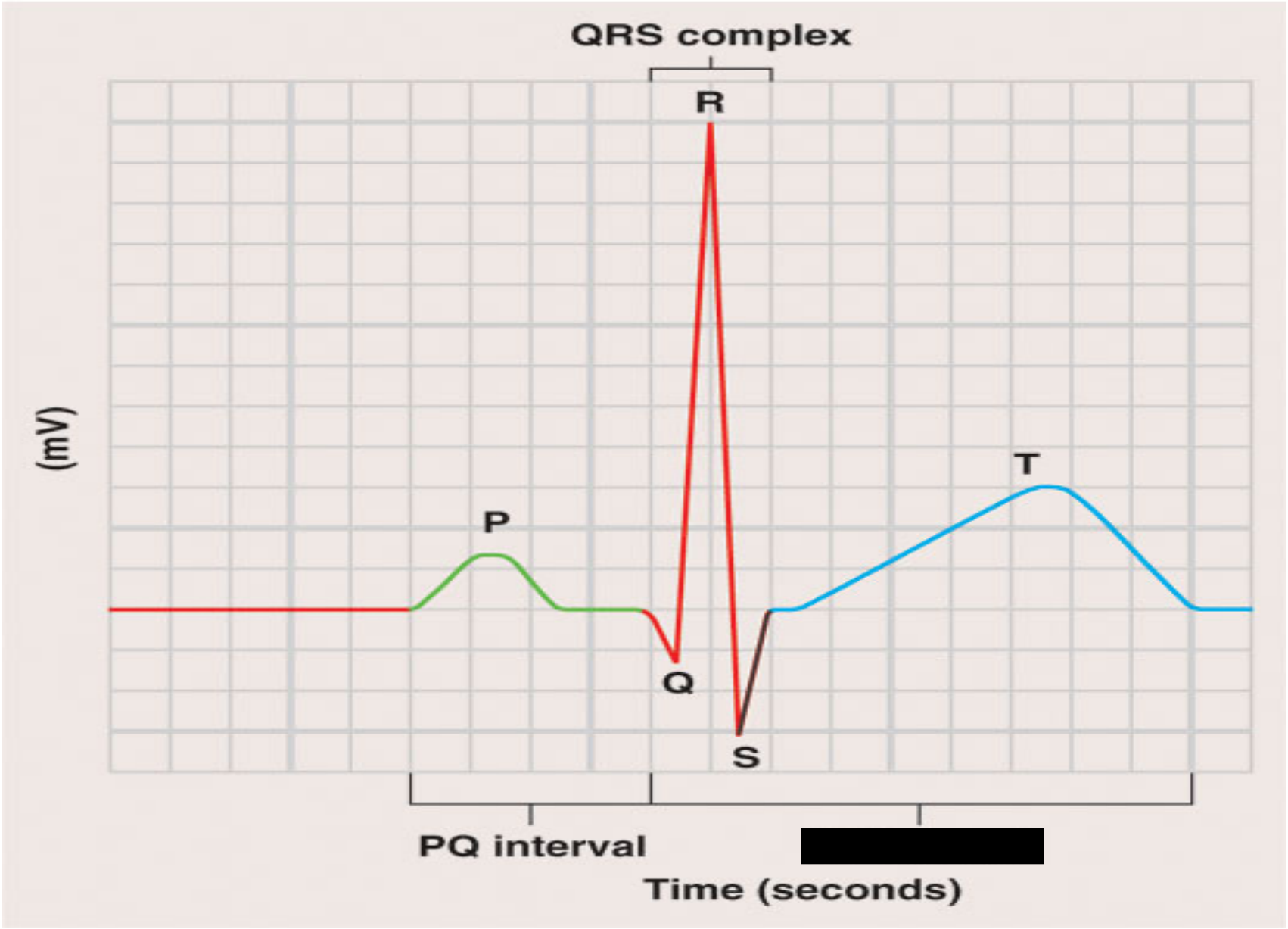




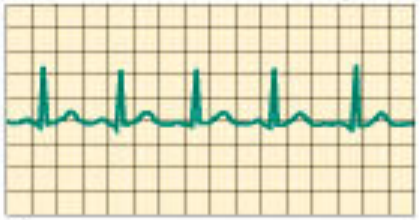
Limb Leads: measure the electrical activity of the heart in a frontal plane

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

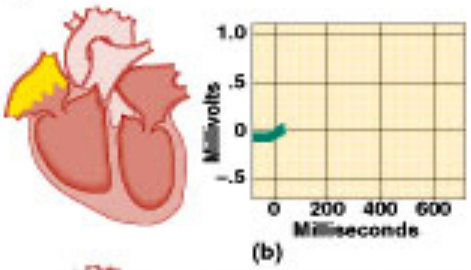
EKG Trace



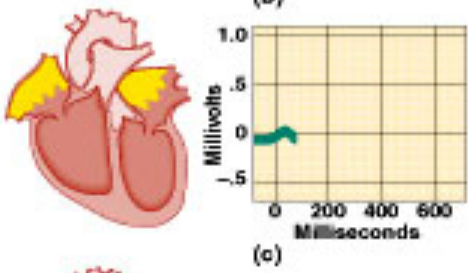
Electrocardiogram (ECG) / Elektrokardiogram (EKG)



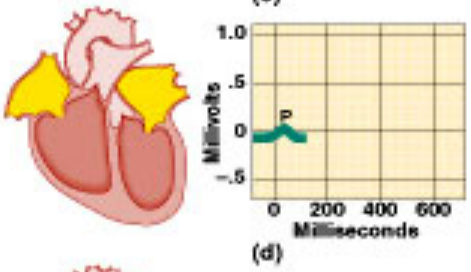
(a)



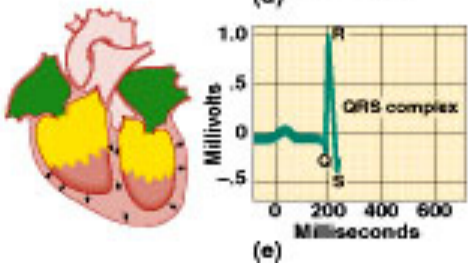
(b)



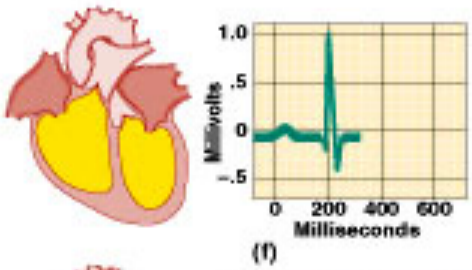
(c)



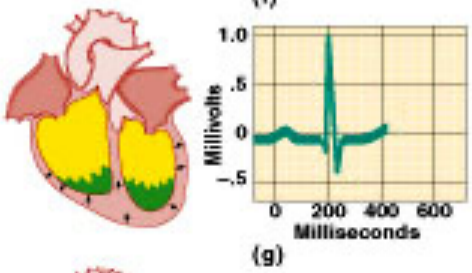
(d)



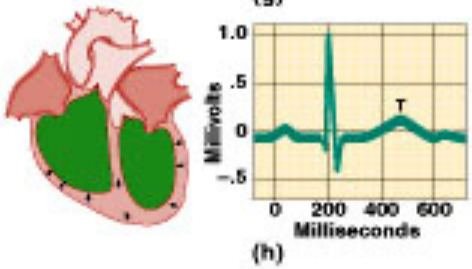
(e)



(f)



(g)



(h)

EKG 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

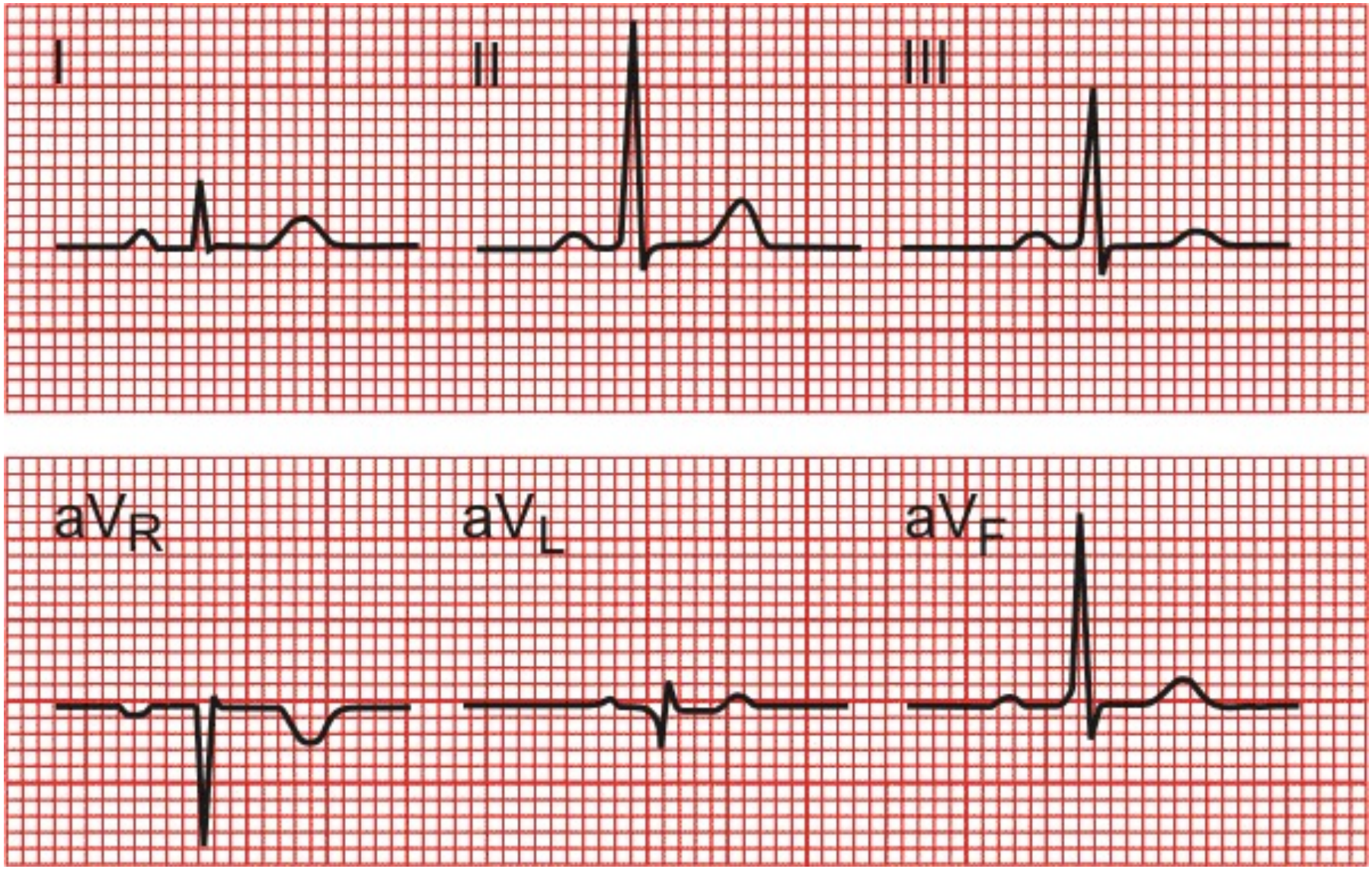
From the beginning of the P wave to the beginning of the Q wave

Time from onset of atrial depolarization to onset of ventricular depolarization

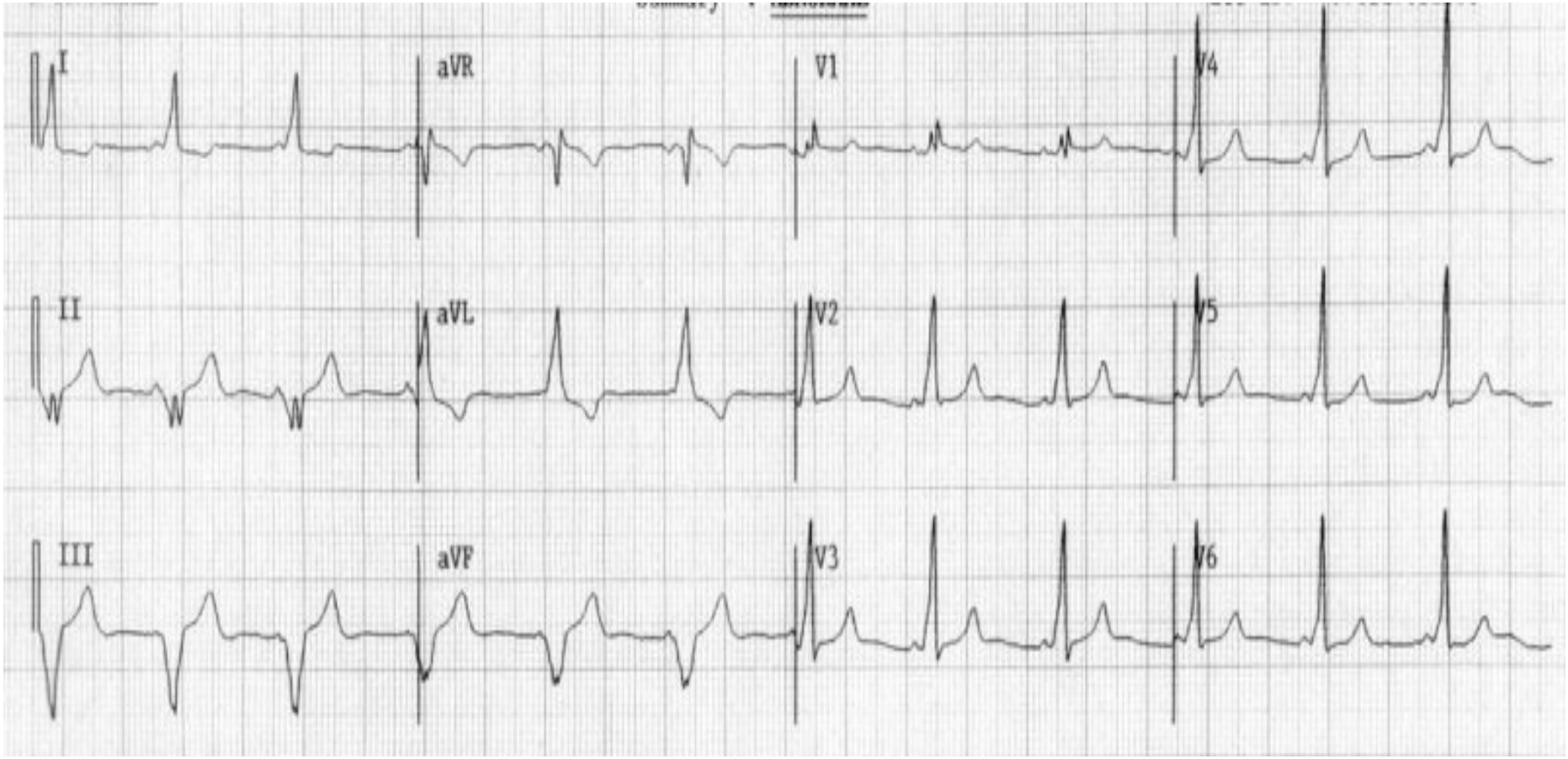
Time it takes action potentials to conduct through cardiac conduction system

Normally 120 to 200 msec in duration

EKG Trace of Limb Leads

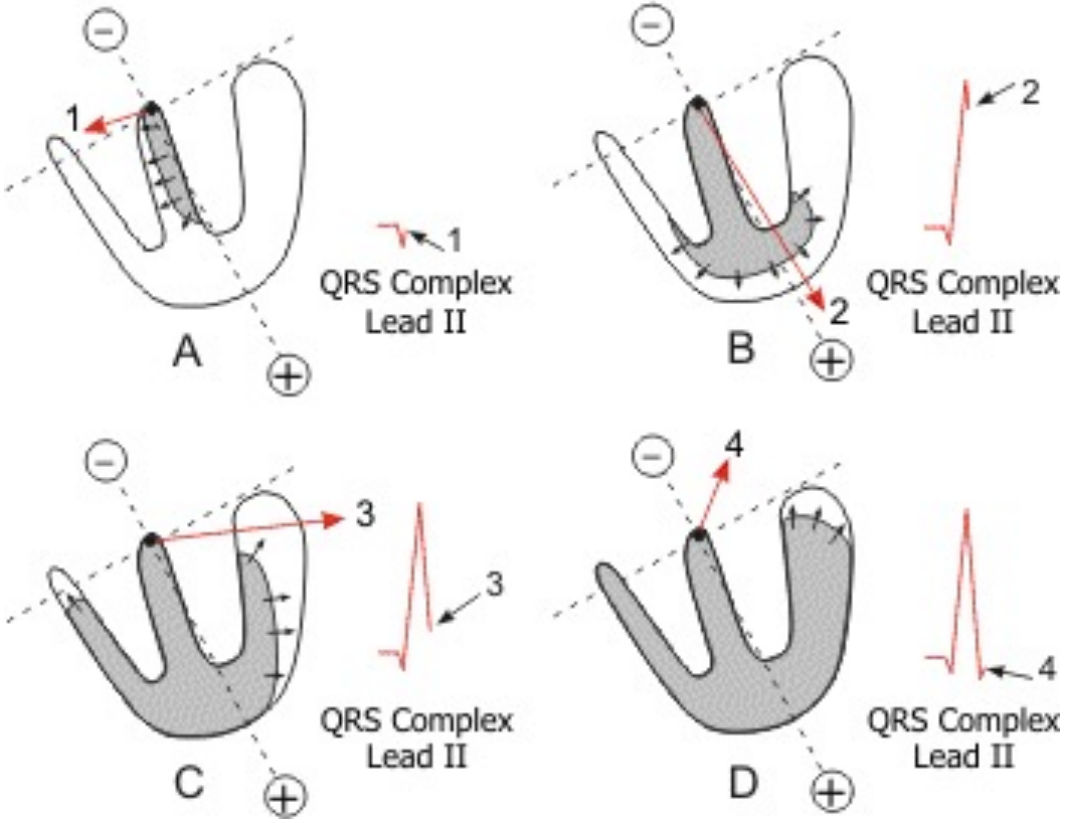


12 Lead EKG Trace



~~Mean Electrical Axis~~

~~average of all the instantaneous mean electrical vectors occurring sequentially during depolarization of the ventricles~~



~~Based on the QRS complexes of limb leads I, II & III~~

~~Normal value is +59º (-30º to +90º is normal range)~~

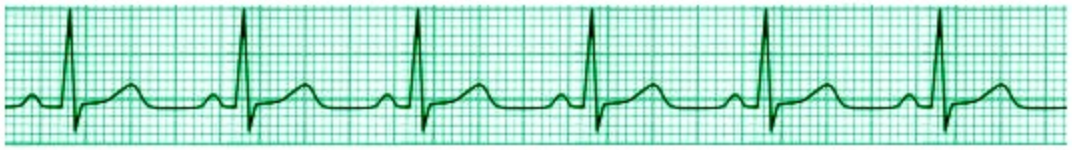
Arrhythmia / Dysrhythmia

Condition where the electrical activity is irregular

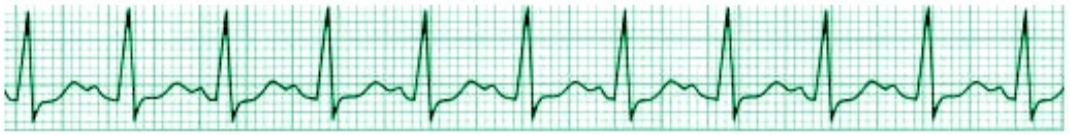
Tachycardia – heart rate above 100 beats per minute

e.g. high sympathetic tone; hyperthyroidism, ectopic pacemaker

normal



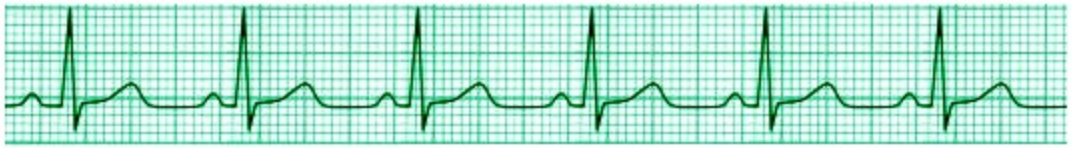
tachycardia



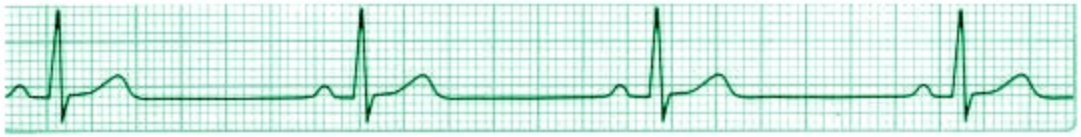
Bradycardia – heart rate below 60 beats per minute

e.g high parasympathetic tone, hypothyroidism; ectopic pacemaker, athletes

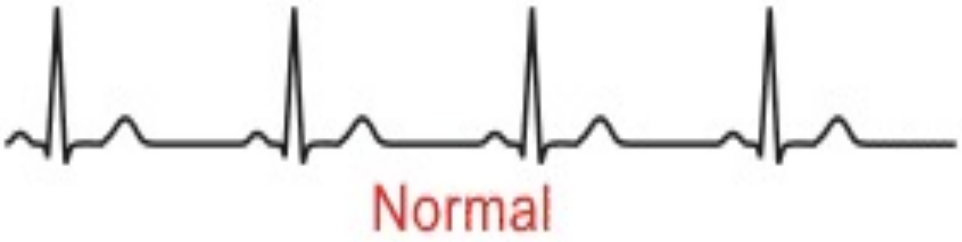
normal



bradycardia

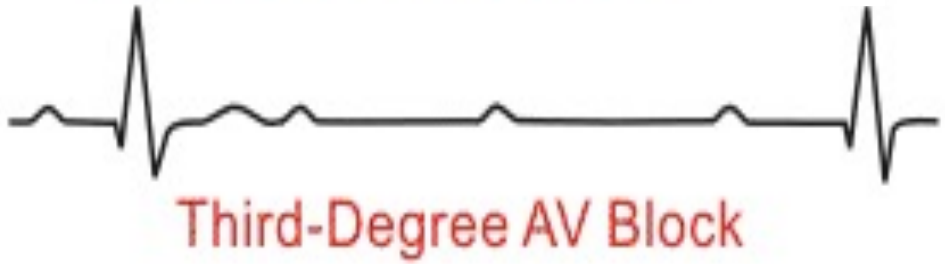


Heart Block



Conduction of action potentials is slowed
Not serious . . . needs no medical attention

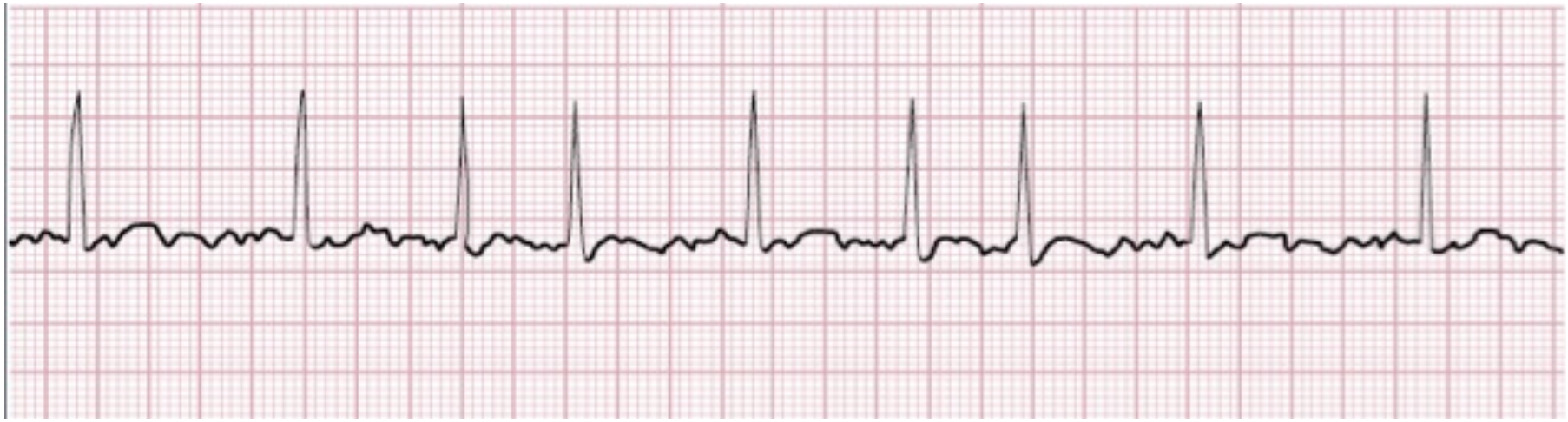
Type I / Wenckebach
Conduction of action potentials is slowed more
Not too serious but dizziness can occur
Type II / Mobitz
Conduction of action potentials
is slowed more and blocked
Most will require an **artificial pacemaker**



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

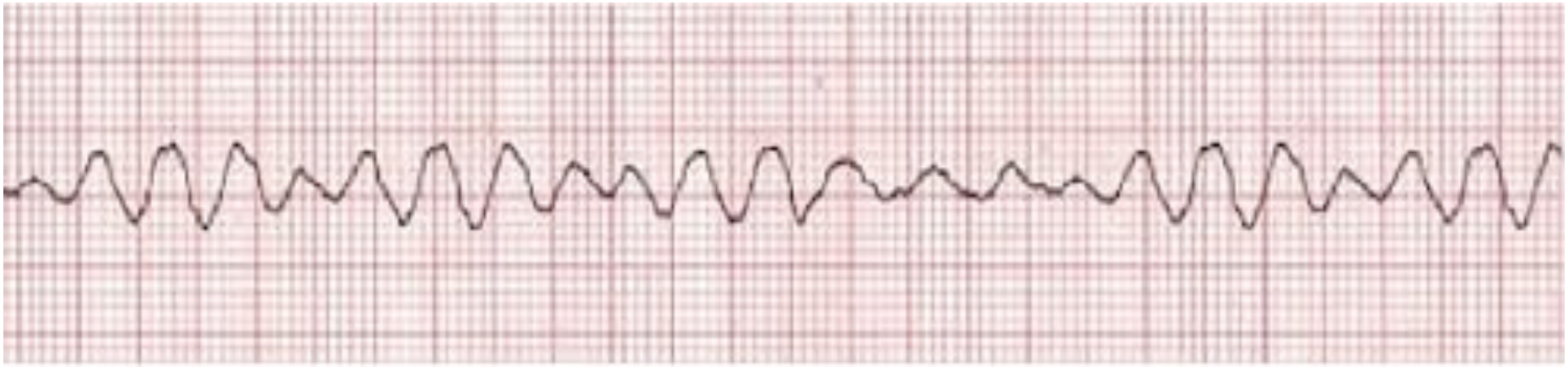
Atrial Fibrillation

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



Ventricular Fibrilation

Conduction system is disordered
No clear waves are seen
Fluttering of ventricles,
Ineffective pumping of blood from heart
Loss of consciousness followed by death if not remedied



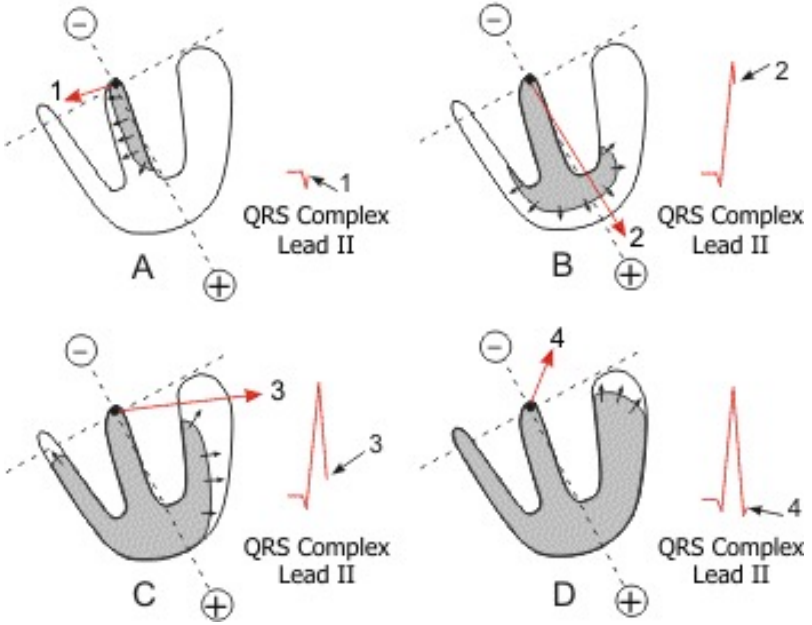
Mean Electrical Axis Deviations

Right axis deviation (+90º to +180º)

- Hypertrophy of the right side of the heart or damage to left side of the heart
- Pulmonary hypertension or pulmonary valve dysfunction
- Heart attack that damages the left side of the heart

Left axis deviation (-30º to -90º)

- Hypertrophy of the left side of the heart
- Systemic hypertension or aortic valve dysfunction
- Heart attack that damages the right side of the heart
- Athletic heart



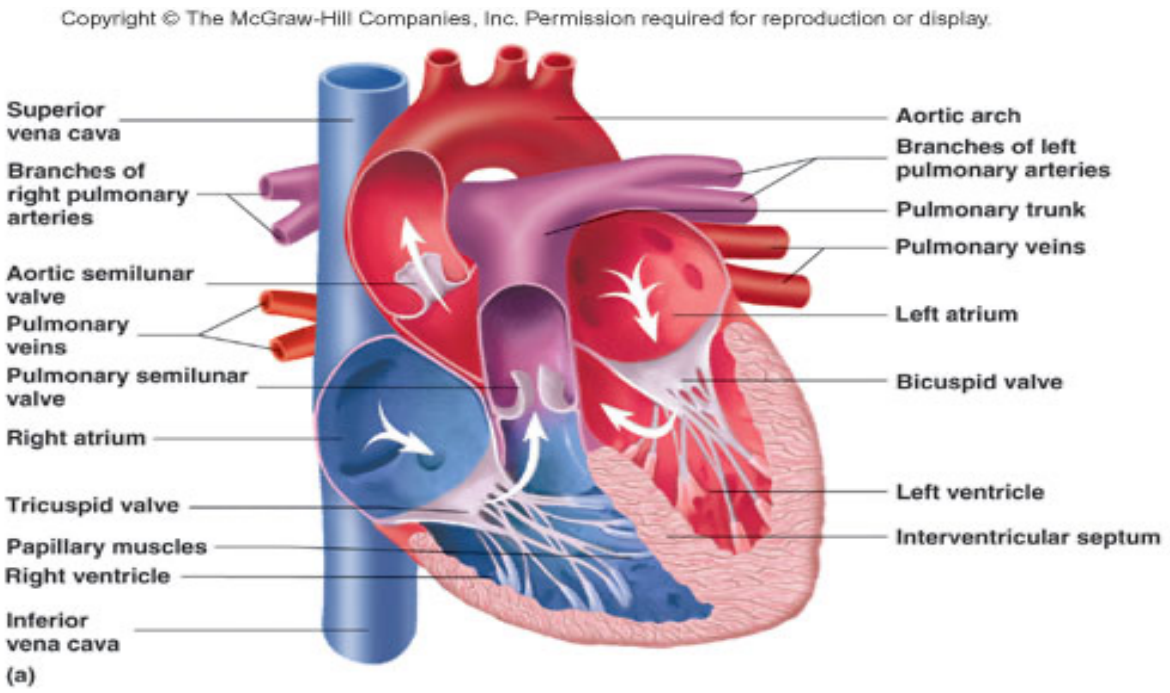
Opening and Closing of Heart Valves

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 and Closing of Heart Valves



Heart Murmurs

Indicates turbulent blood flow

Septal defects (hole in the septum) and **increased blood flow** thru 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

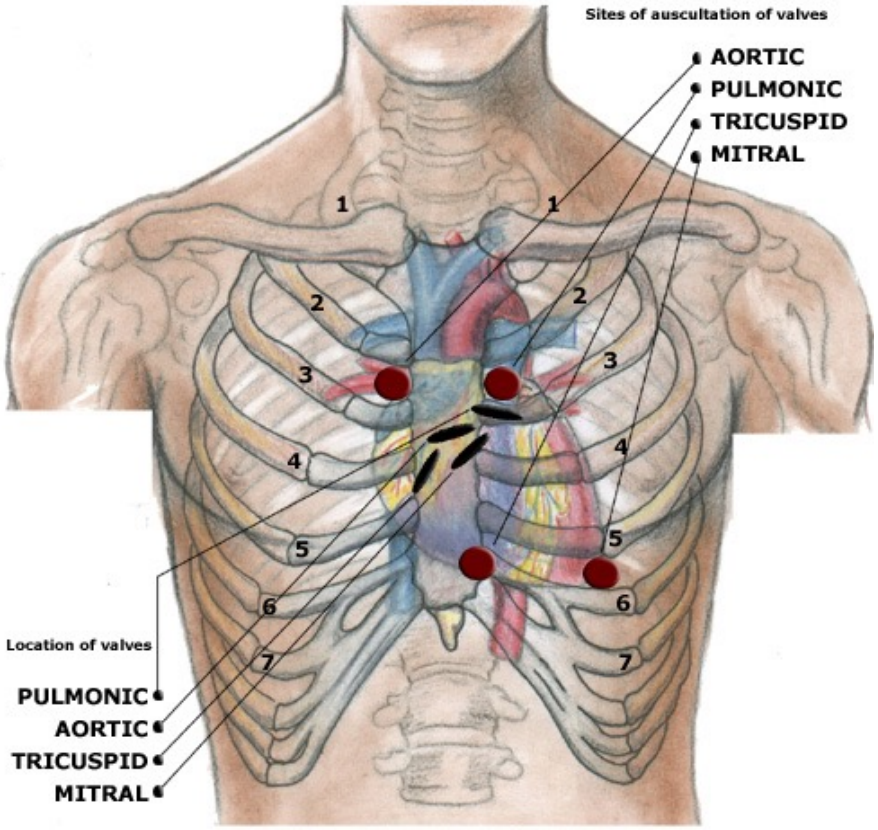
Systolic murmur (*between S_1 and S_2*)

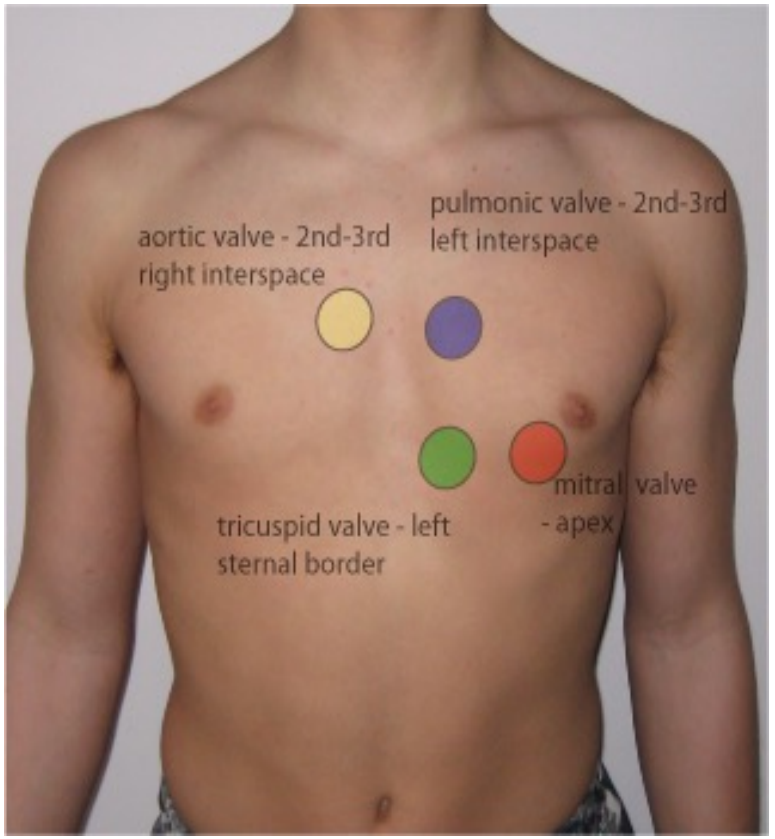
- AV regurgitation
- Semilunar stenosis

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

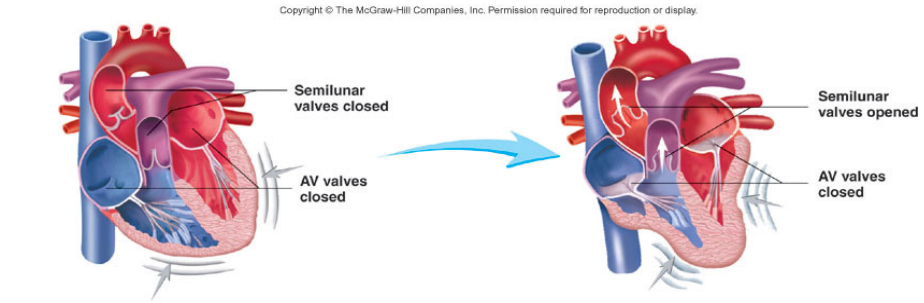
Isovolumic relaxation

Passive ventricular filling

Active ventricular filling

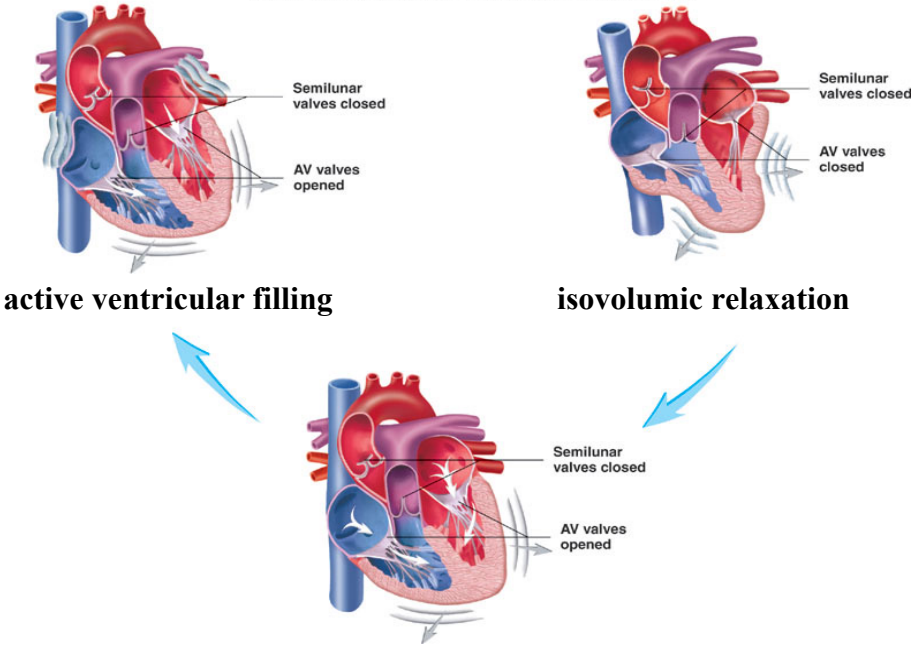
Cardiac Cycle

Systole



isovolumic contraction **period of ejection**

Diastole



active ventricular filling **isovolumic relaxation**

passive ventricular filling

Cardiac Cycle (Systole)

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

Cardiac Cycle (Systole)

Period of ejection (*part of systole*)

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

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 (S_2)

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)

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

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 of the medulla by way of the autonomic nervous system

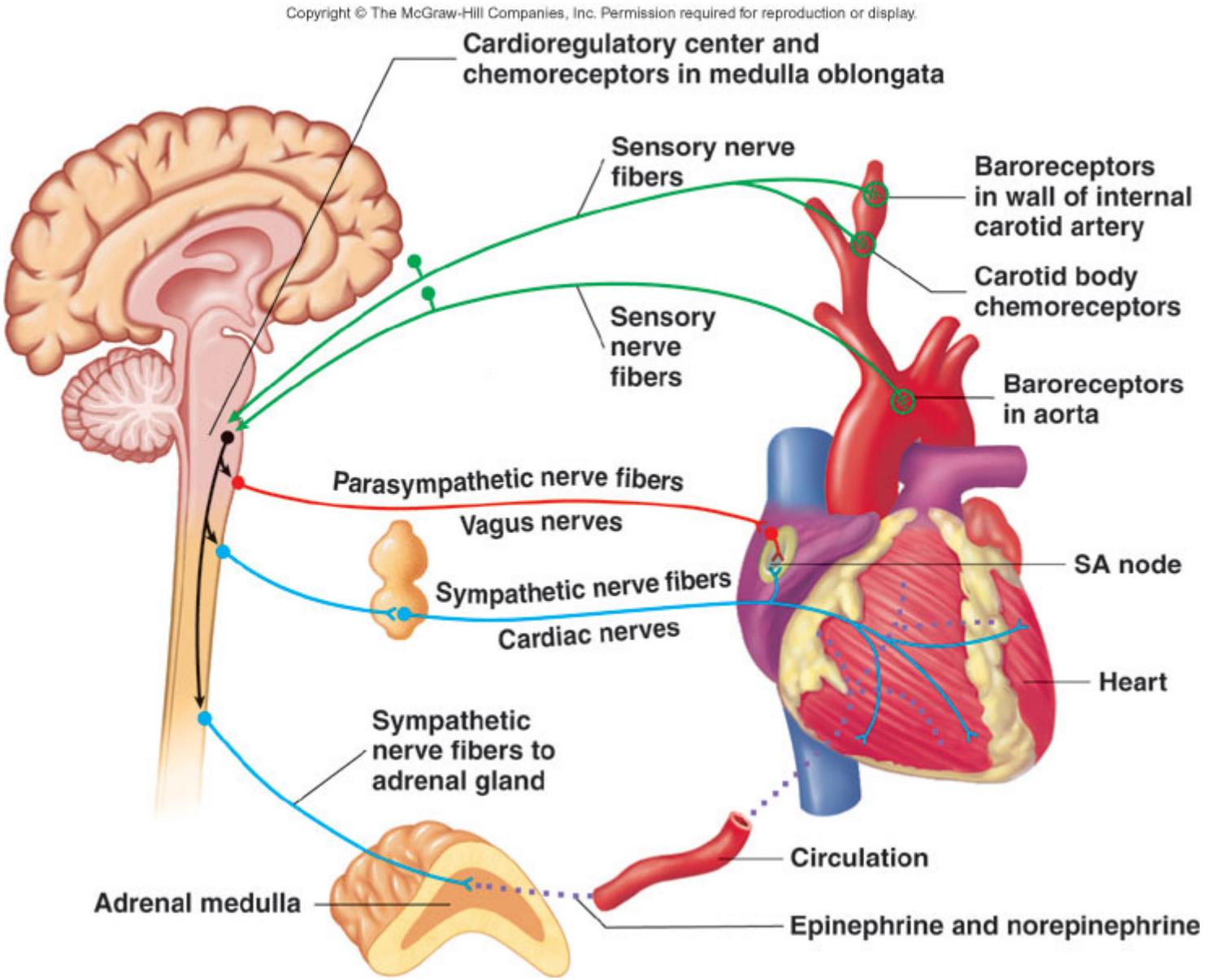
- Parasympathetic via the vagus nerve
  - Primary controller of resting heart rate
  - Innervates SA node, AV node, atrial myocardium
  - Has an inhibitory influence on the heart
    - Decreases heart rate
      - Negative chronotropic effect

Extrinsic Factors

Cardiac control center of the medulla by way of the autonomic nervous system

- Sympathetic via **cardiac nerve**
  - Primary controller of heart contractility
  - Contributes very little to resting heart rate
  - Can cause large changes in heart rate and 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 inotropic effect

Extrinsic Factors



Extrinsic Factors

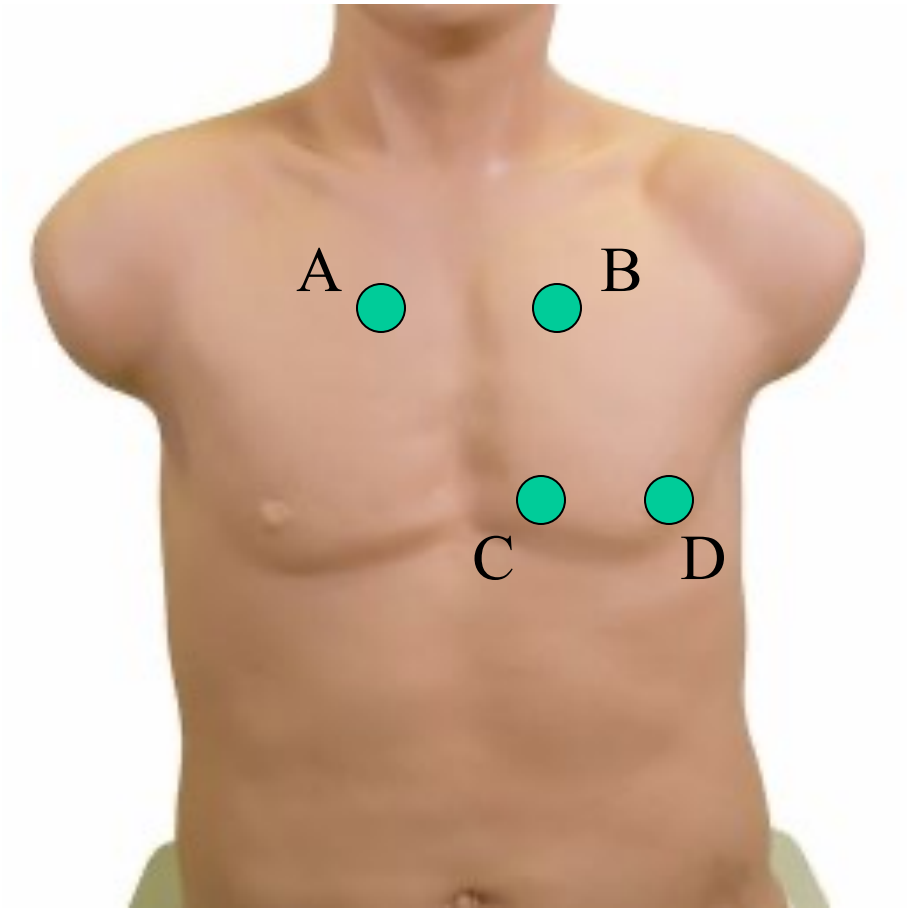
Hormones

- 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 1ºC 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

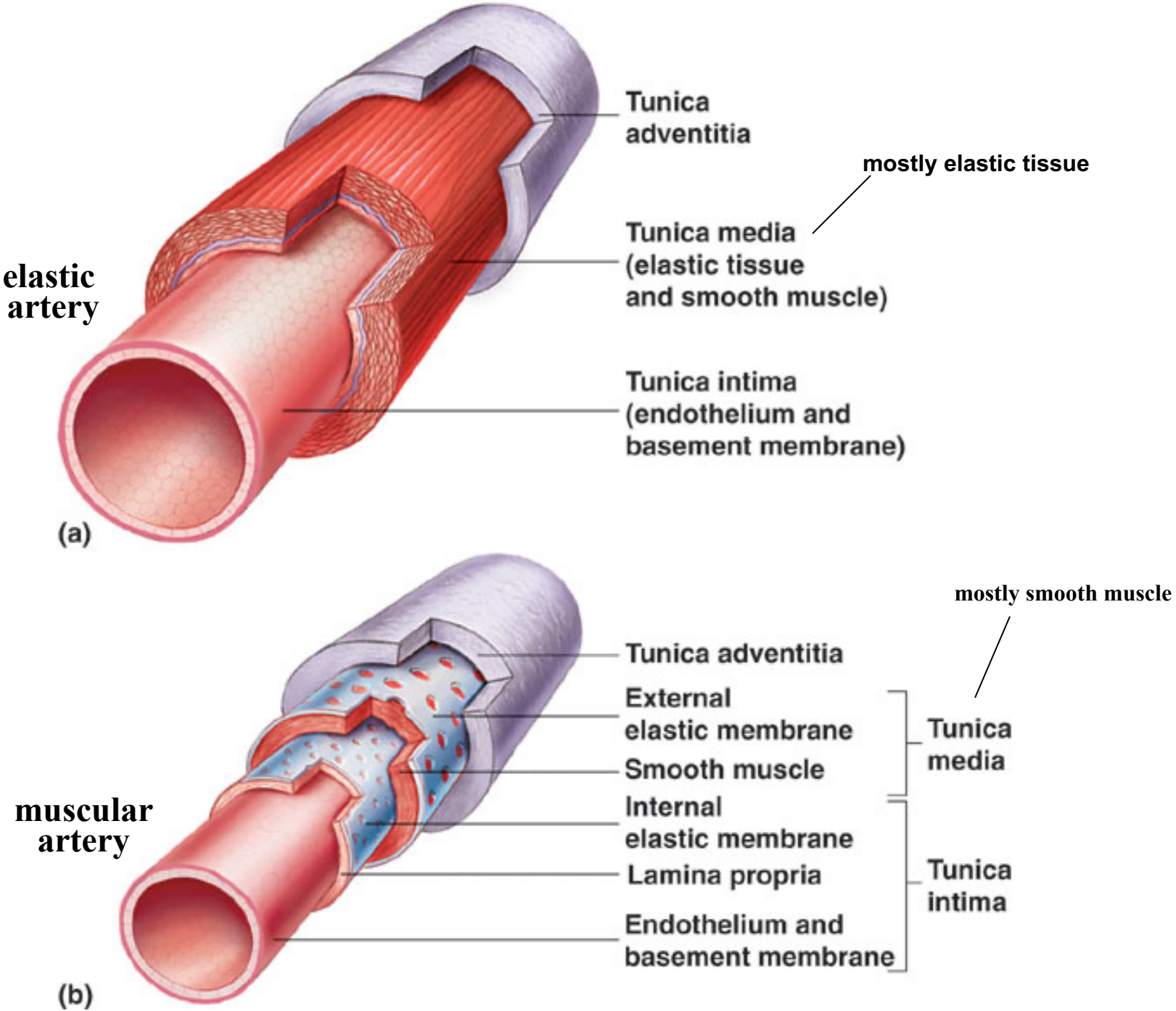
Blood Vessels

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



Blood Vessels

Venules and veins

Highly **compliant** vessels

Small changes in pressure cause large changes in blood volume

Carry blood back to the heart

Lowest resistance to blood flow

Influenced by sympathetics and local factors

Veins merge from **venules** (smallest branches of a vein)

Contain approximately 65% of total blood volume (venules: 25%; veins: 40%)

Act as reservoirs for blood

Vital during hemorrhage when blood is needed in arterial system

Venous return to the heart

Blood pressure gradients in systemic and pulmonary venous systems

e.g. Peripheral venous pressure (~ 10 mm Hg)

e.g. Right atrial pressure (0 to 5 mm Hg)

***Respiratory pump** is a minor contributor*

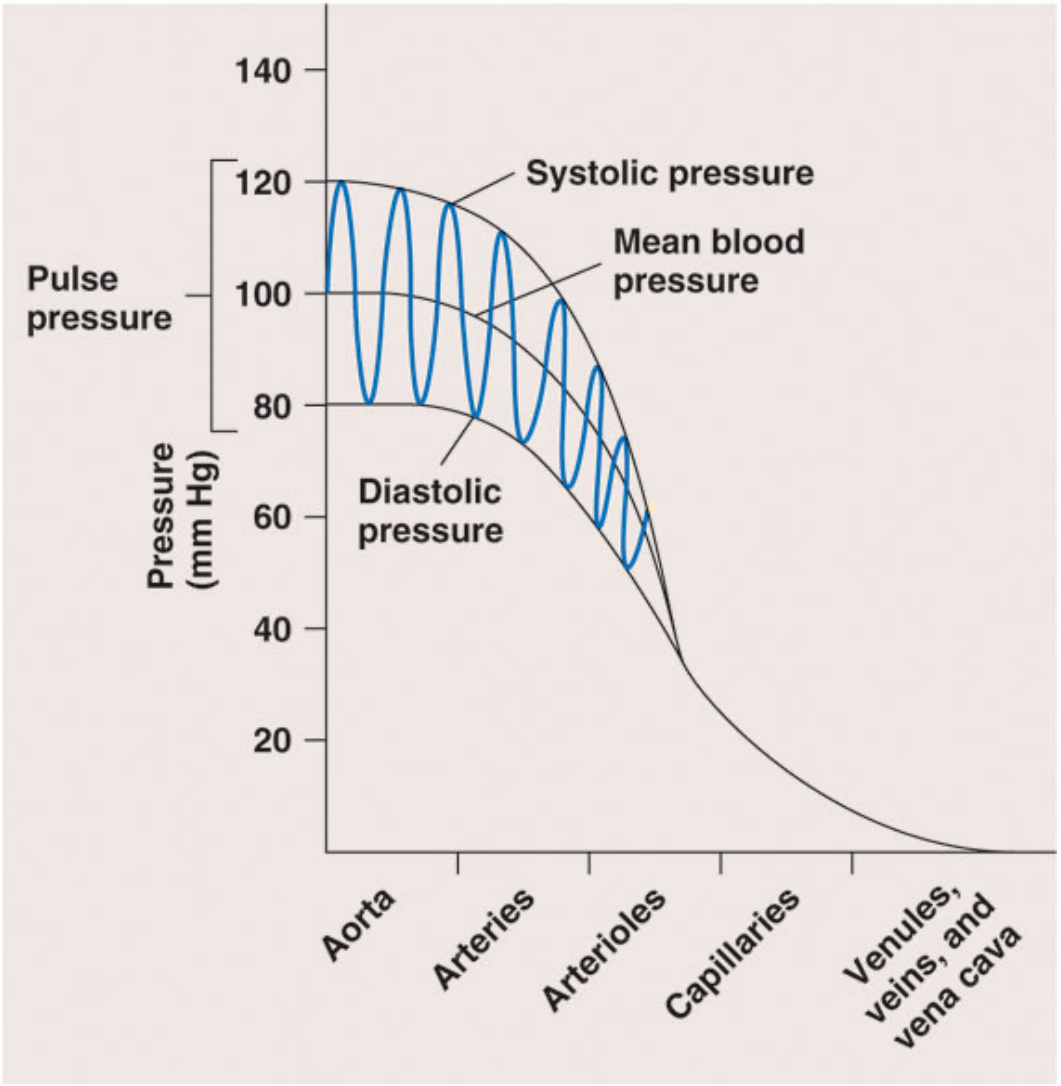
Expansion of thoracic cavity during inhalation

Increases venous return by lowering thoracic cavity pressure

***Skeletal muscle contraction** is the major contributor*

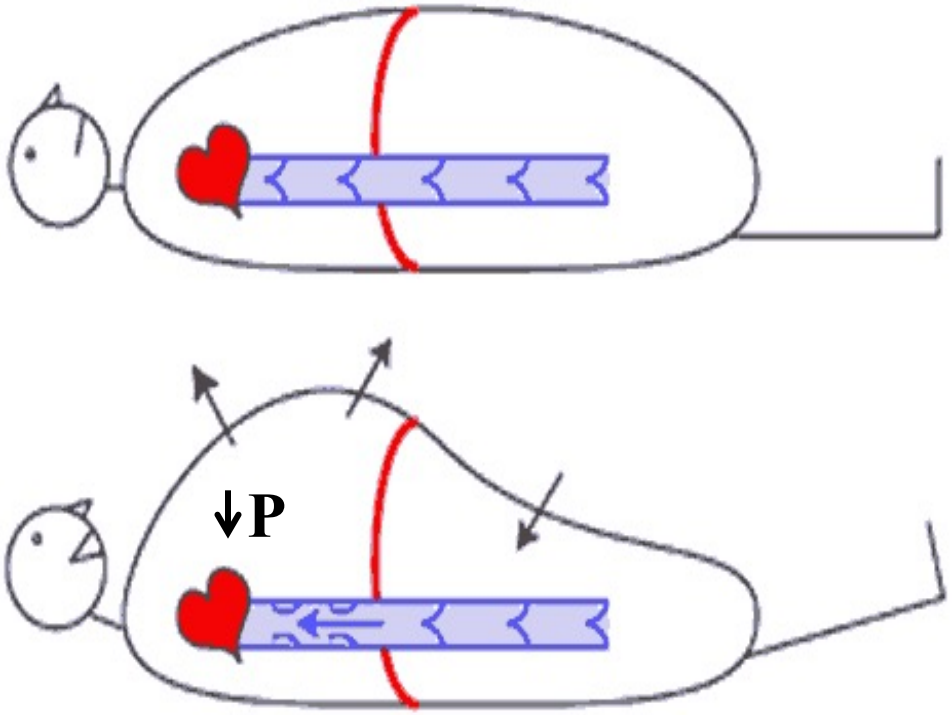
Contraction squeezes veins and pushes blood towards the heart

Factors Contributing to Venous Return



Factors Contributing to Venous Return

Respiratory Pump

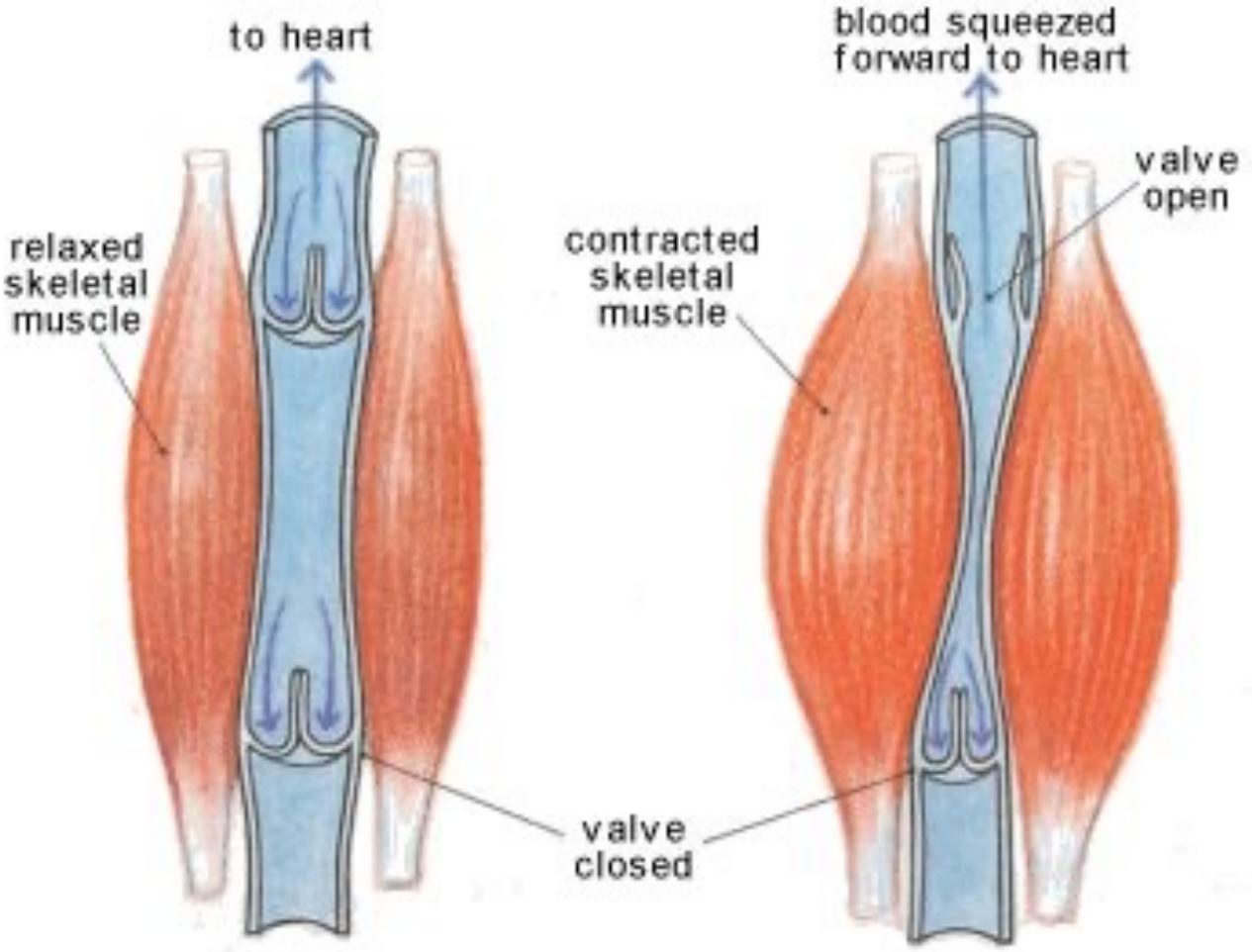


Inhale

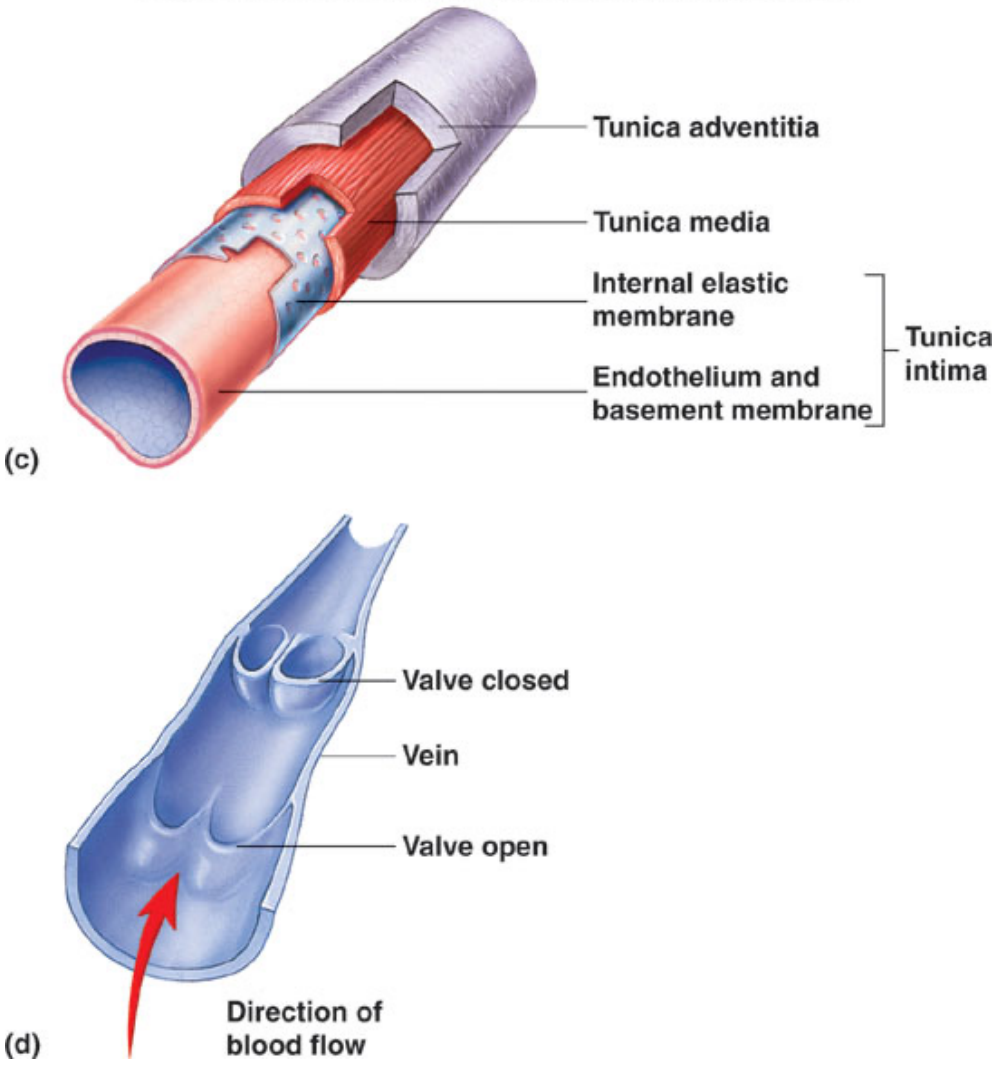
greater venous return

Factors Contributing to Venous Return

Muscle Contraction



Veins and Venules

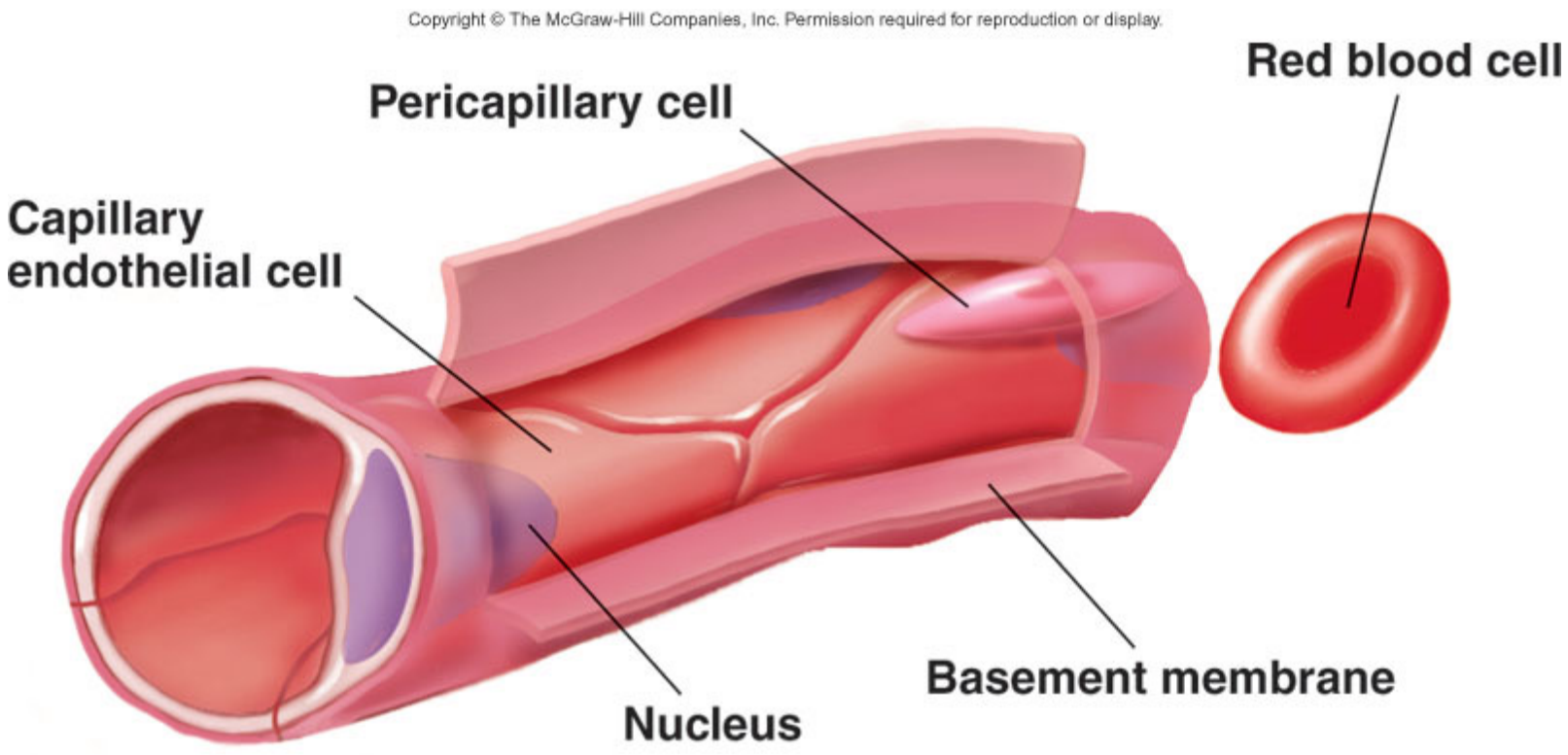


Blood Vessels

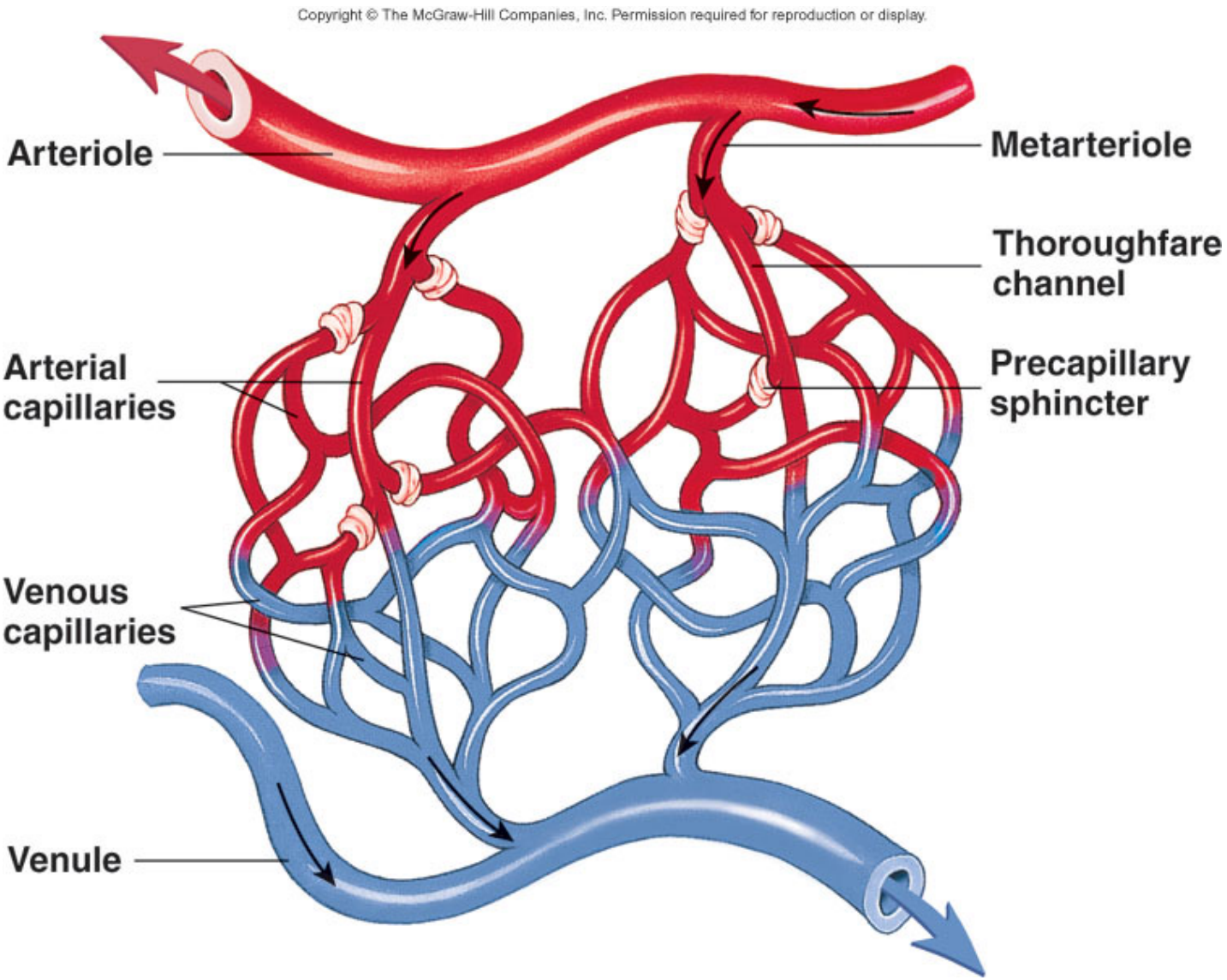
Capillaries

- 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

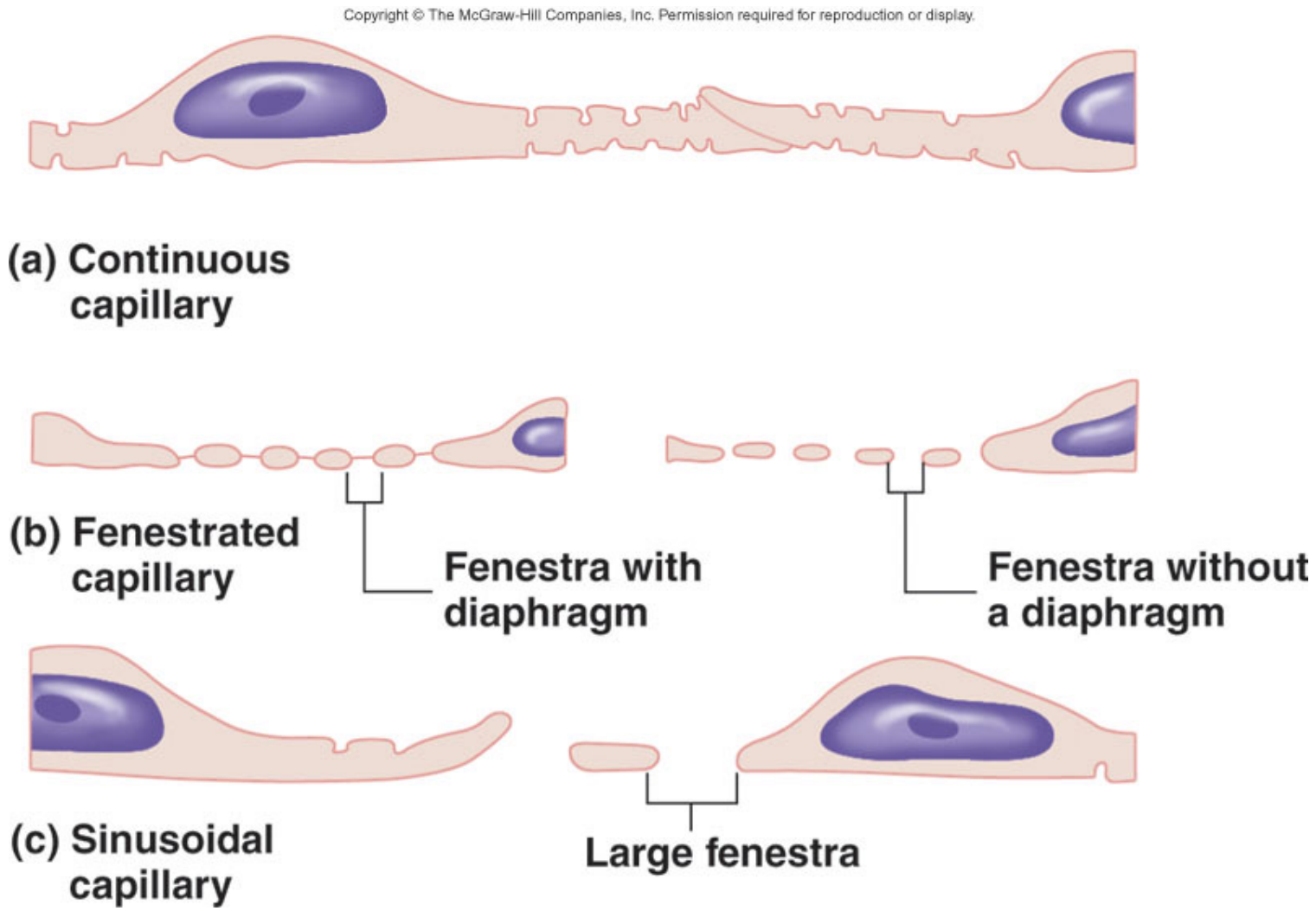
Capillary


Copyright © The McGraw-Hill Companies, Inc. Permission required for reproduction or display.
Red blood cell
Pericapillary cell
Capillary endothelial cell
Basement membrane
Nucleus

Capillary Network



Permeability of Capillaries



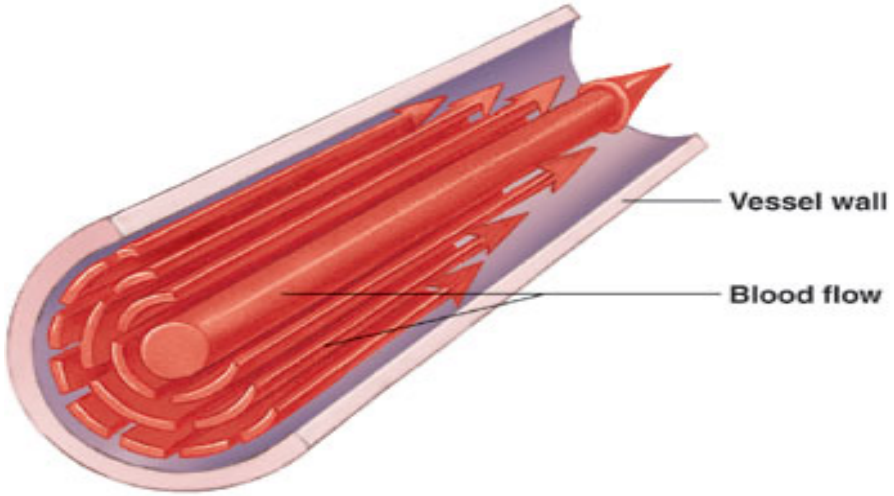
Blood Flow

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

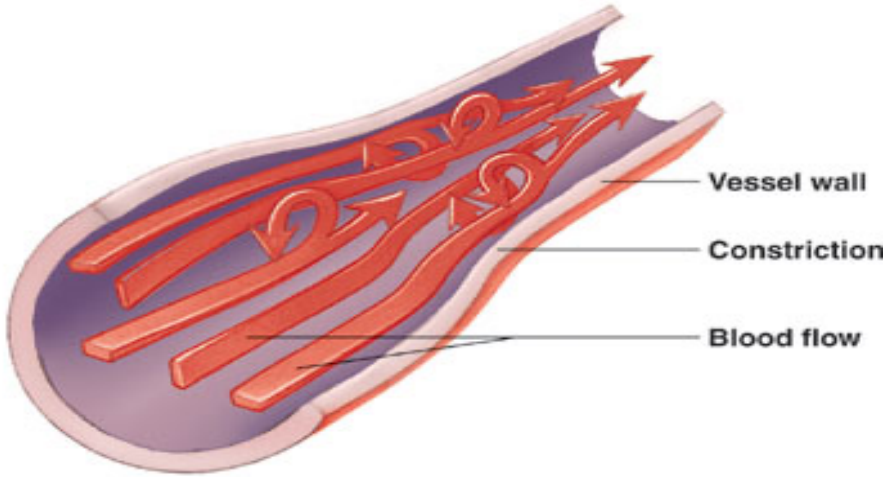
Blood Flow

Laminar



(a)

Turbulent



(b)

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
P_2 = pressure at point two
R = resistance to flow

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

ν = 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 and flows through systemic and pulmonary circulations and 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

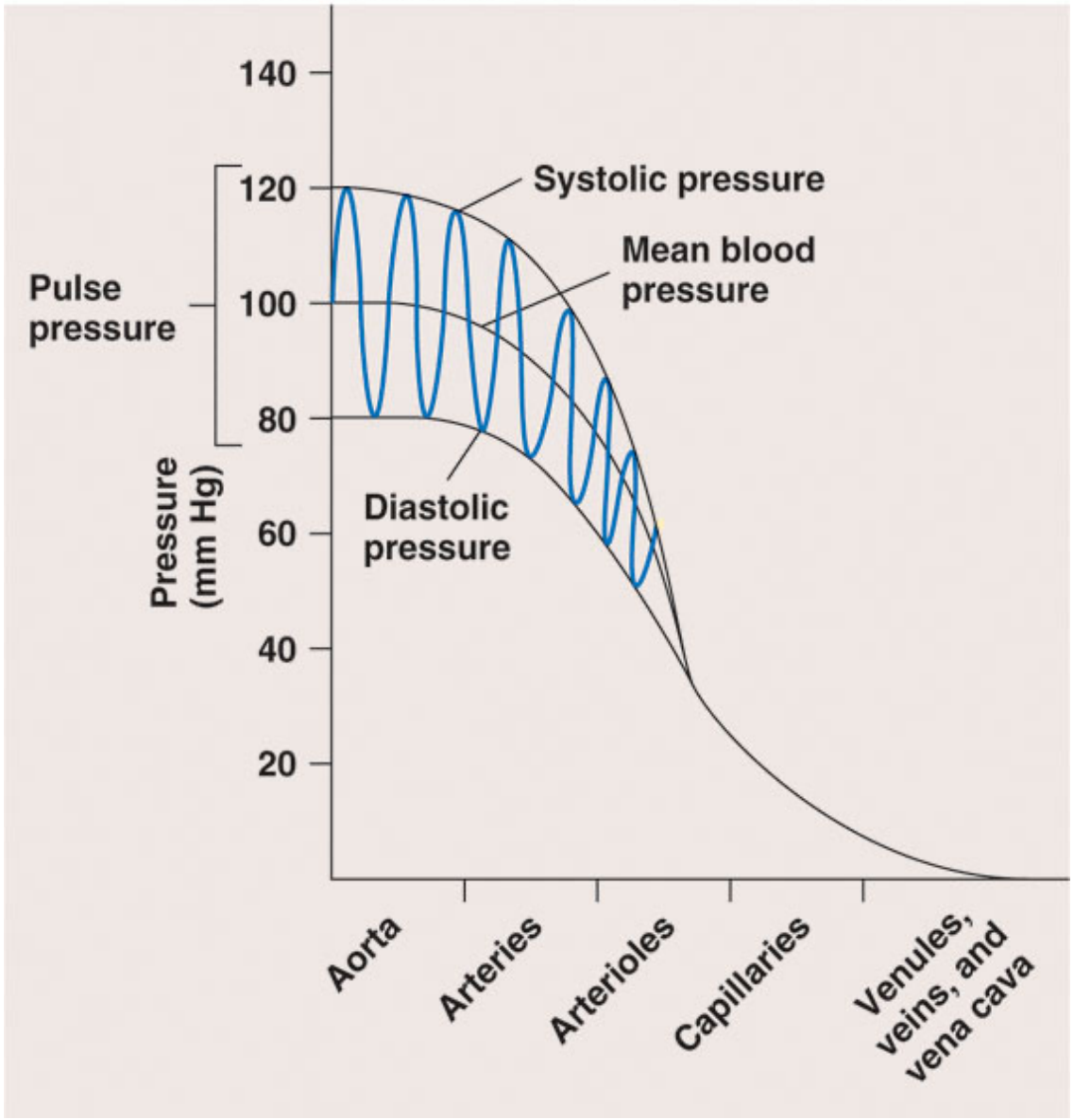
Greatest effect on resistance to blood flow

Arterial radius affected by sympathetic tone and local factors

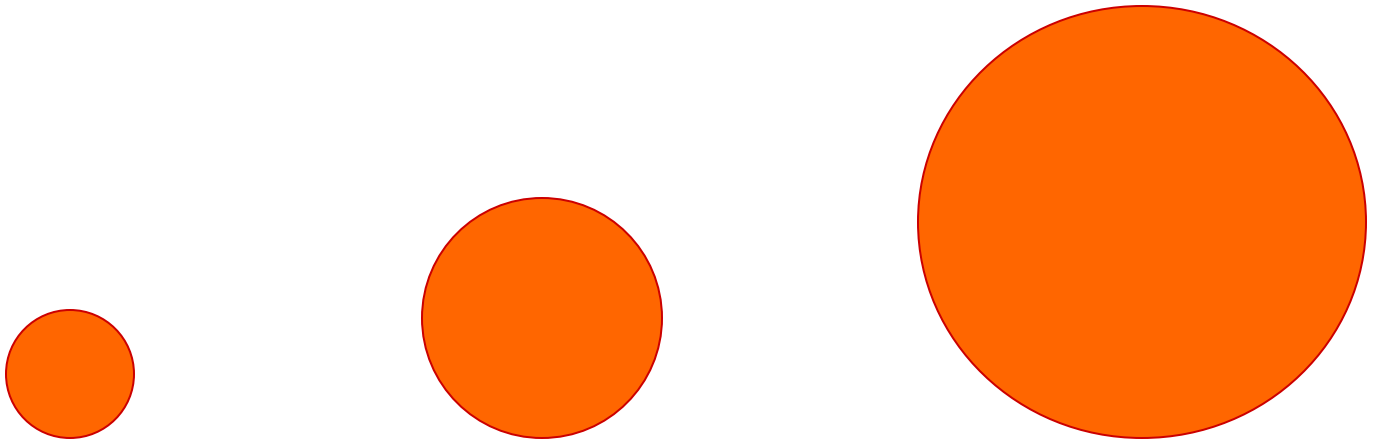
Vasoconstriction = decrease blood vessel radius

Vasodilation = increase blood vessel radius

Blood Pressure Gradient from Aorta to Right Atrium



Effect of Radius on Blood Flow



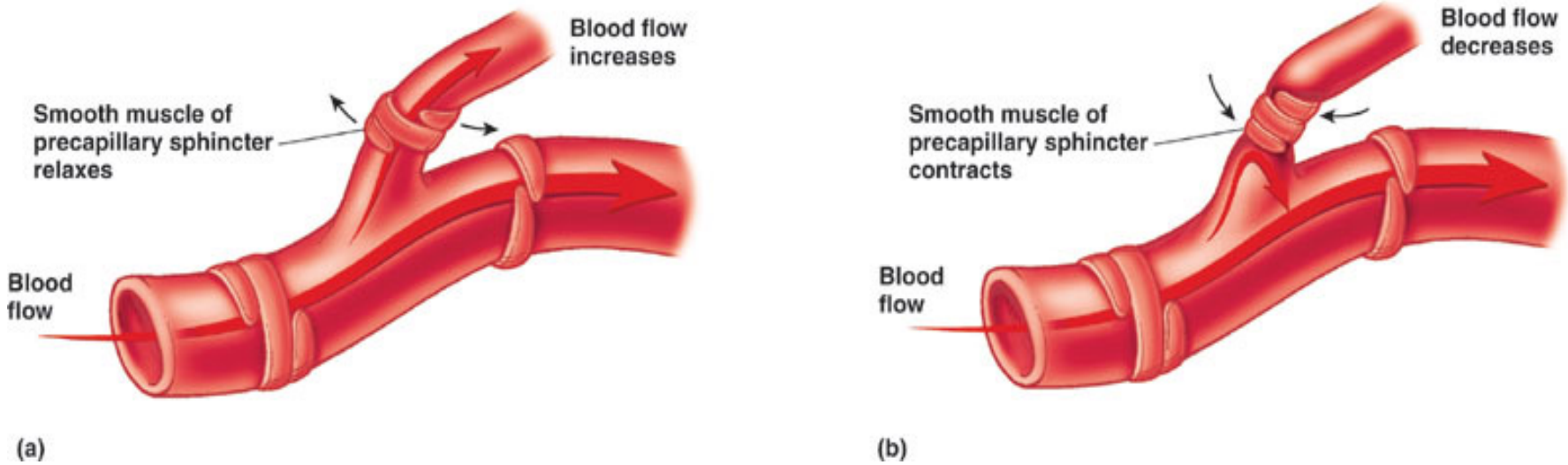
decrease radius / increase resistance

increase radius / decrease resistance

decrease blood flow tremendously

increase blood flow tremendously

Effect of Radius on 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

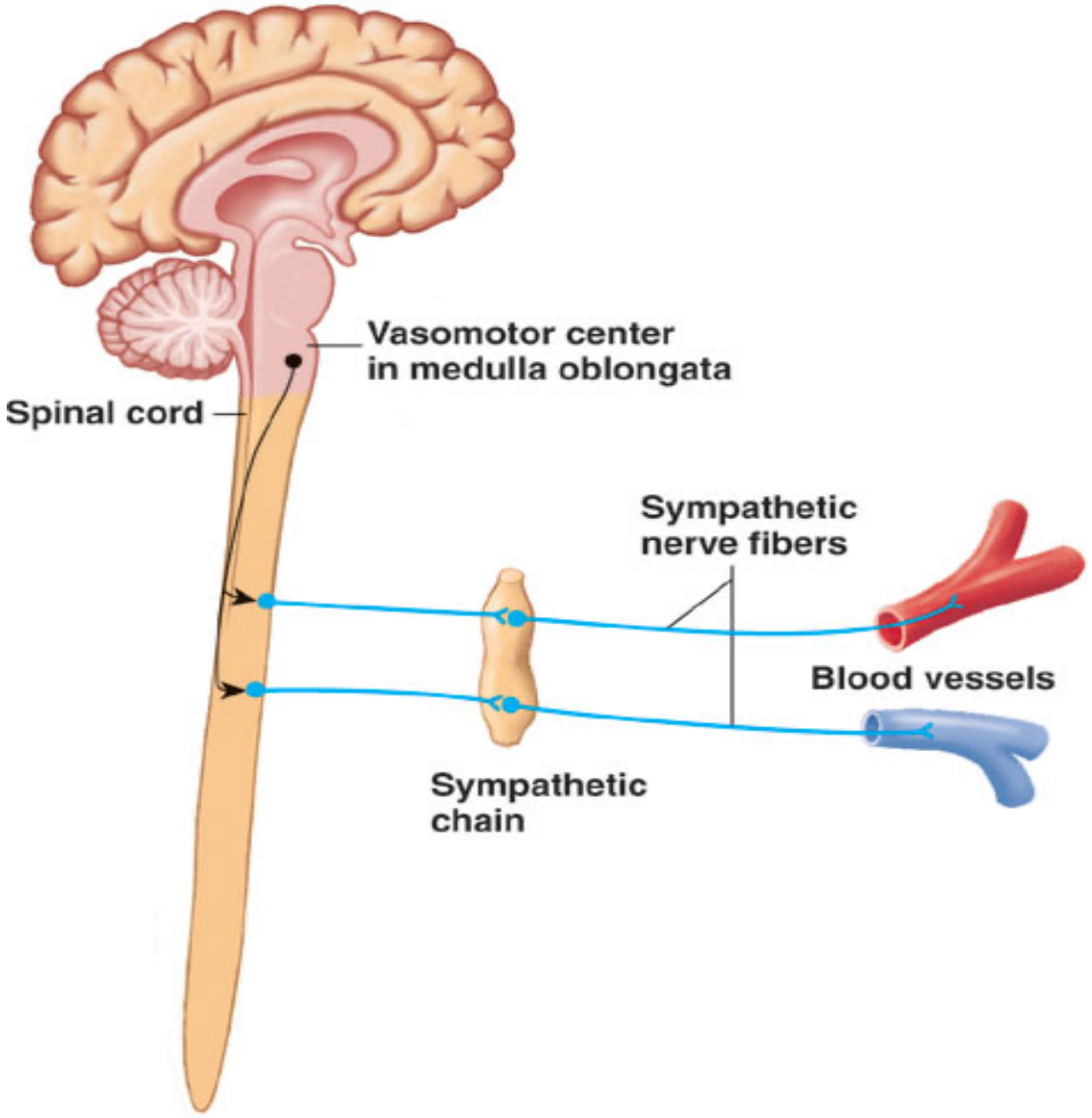
Control of Blood Flow

controlled via smooth muscle tone

Vasomotor Centers

Local Factors

Vasomotors Centers of the Brainstem

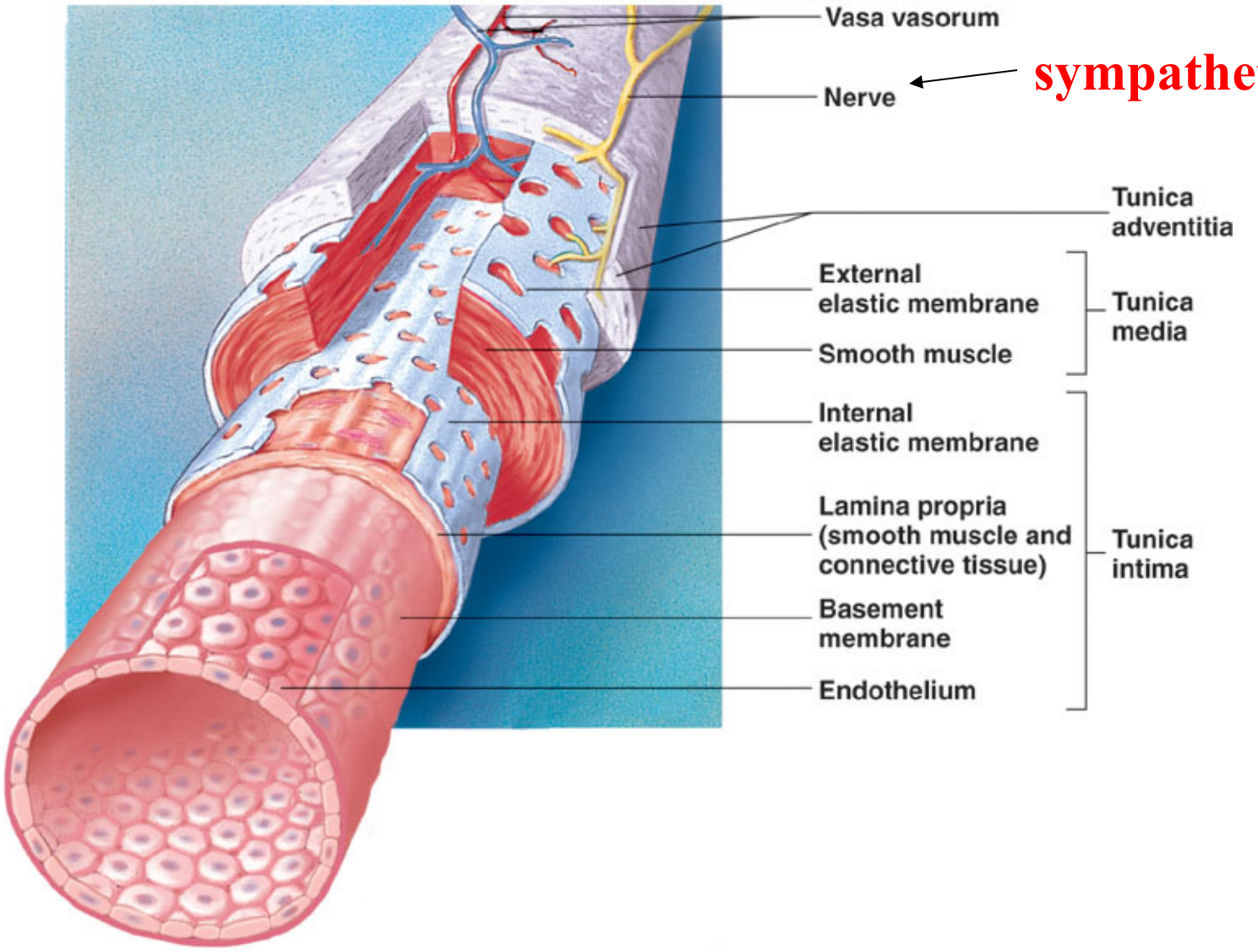


Vasomotors Centers of the Brainstem

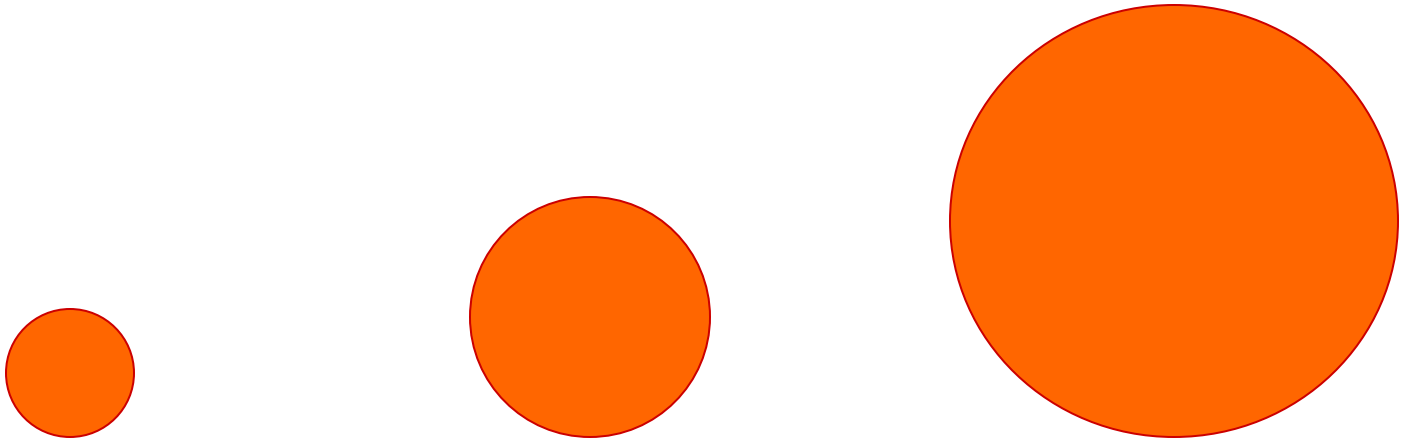
Vasomotor centers in the brainstem

- 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

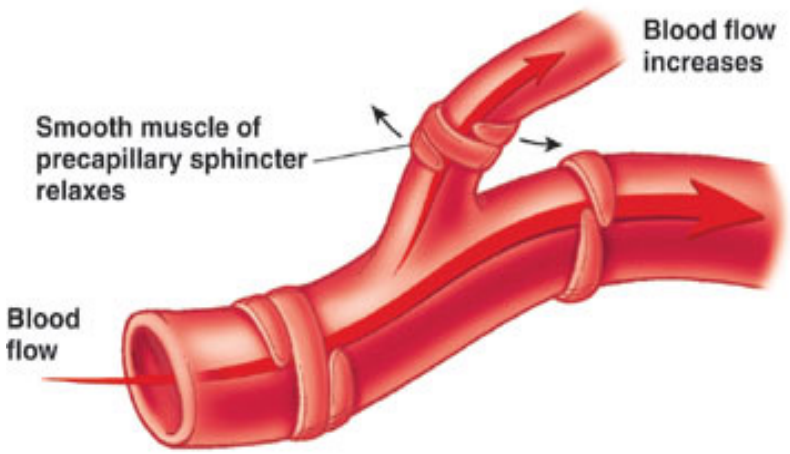
Sympathetic Innervation of Vascular Smooth Muscle

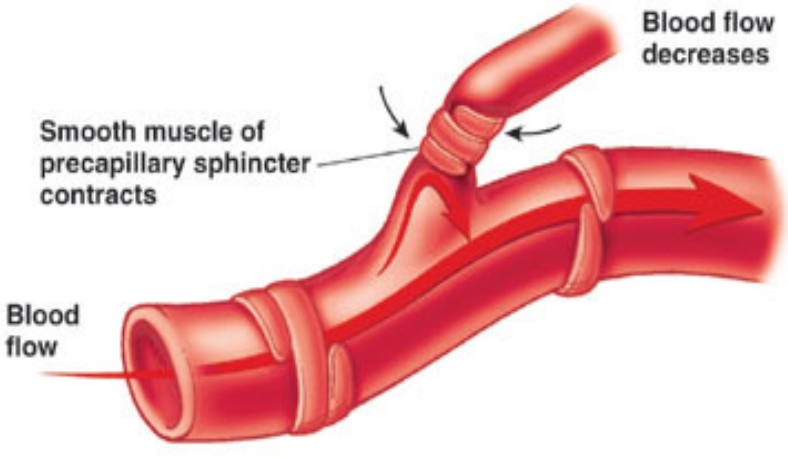


Vasodilation and Vasoconstriction



vasoconstriction **vasodilation**





Local Factors

Cause smooth muscle relaxation (vasodilation) → increase blood flow

- Nitric oxide
- Histamine (part of inflammatory response and allergic reactions)
- 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)

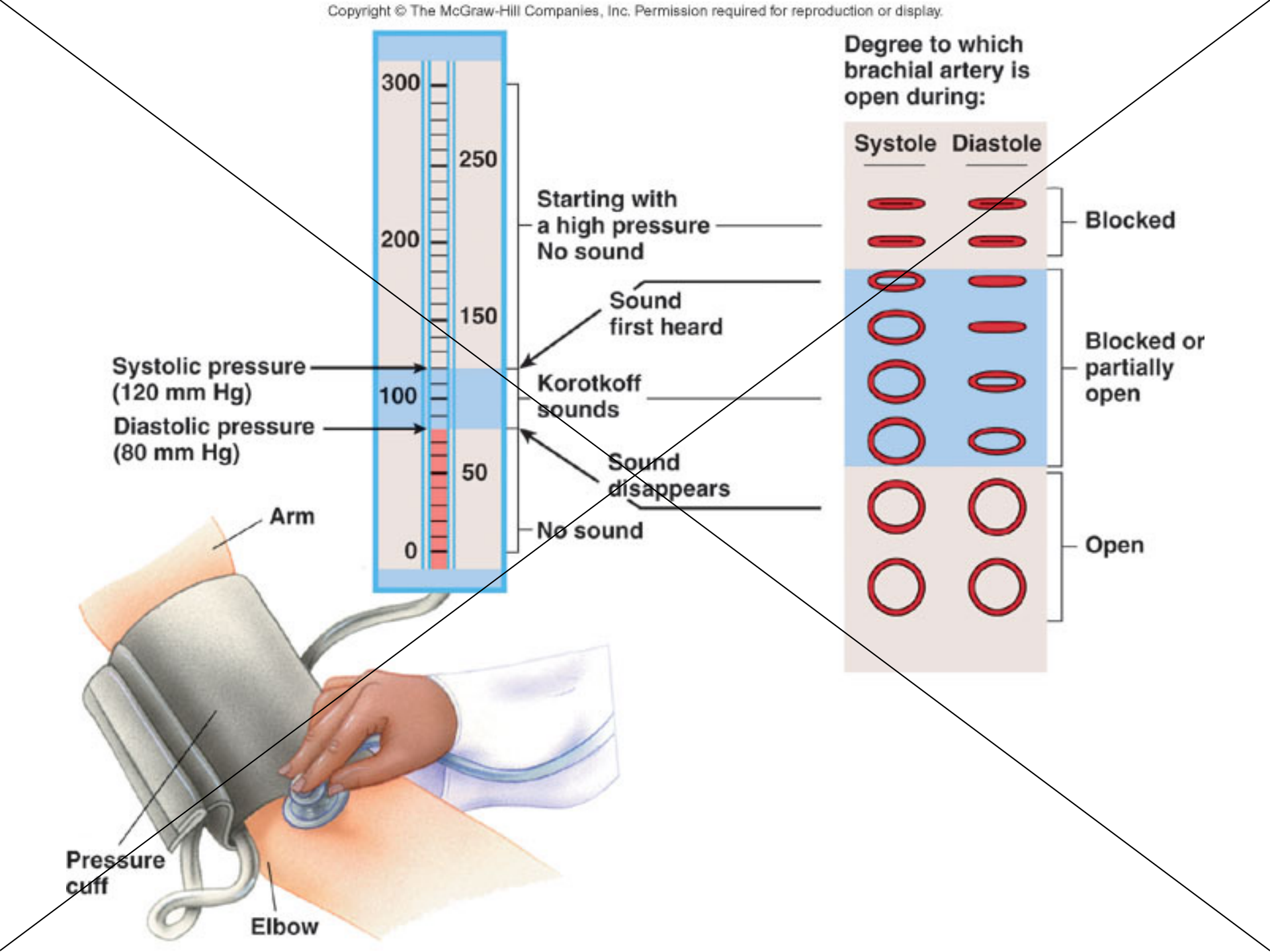
Systolic pressure: blood pressure during systole (i.e. the "top" number)

Diastolic pressure: blood pressure during diastole (i.e. the "bottom" number)

Pulse pressure: difference between systolic pressure and diastolic pressure

~~Systemic blood pressure measured with a **sphygmomanometer**~~

- ~~Wrapped around arm just above elbow~~
  - ~~Stethoscope placed over the brachial artery~~
- ~~Inflated to increase pressure around the arm~~
  - ~~Collapses the brachial artery~~
    - ~~No blood flow through artery, therefore no sound heard~~
- ~~Air is slowly released~~
  - ~~Slowly decreases pressure in the sphygmomanometer~~
- ~~Listen for **Korotkoff sound**~~
  - ~~When sound is first heard, signifies systolic pressure~~
    - ~~Pressure overcomes partial constriction of brachial artery~~
    - ~~Can be heard because of turbulent blood flow~~
- ~~Continuation of the release of pressure until there is an absence of sound~~
  - ~~Signifies diastolic pressure~~
    - ~~Lowest pressure that allows full distension of brachial artery~~
  - ~~No sound heard because of laminar blood flow~~



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:

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

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:

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

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

$\approx 84 \text{ mm Hg}$

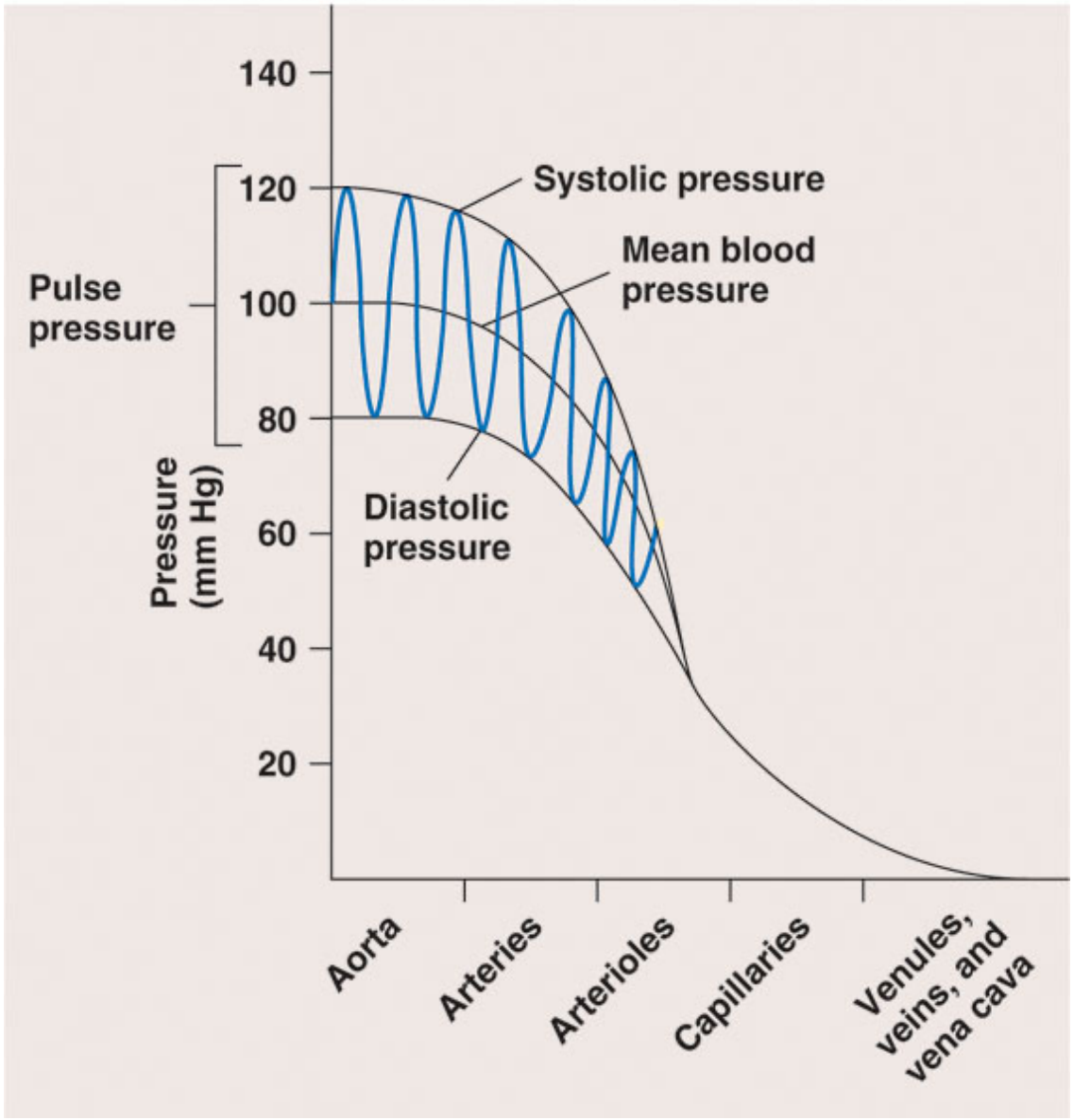
OR

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

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

$\approx 84 \text{ mm Hg}$

Blood Pressure Gradient from Aorta to Right Atrium



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

Antiduretic 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

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

Atrial 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 (i.e. Hypotension)

Antidiuretic hormone (ADH) / Vasopressin

Increases blood pressure via:

- Increased blood volume
- Vasoconstriction

Epinephrine and norepinephrine

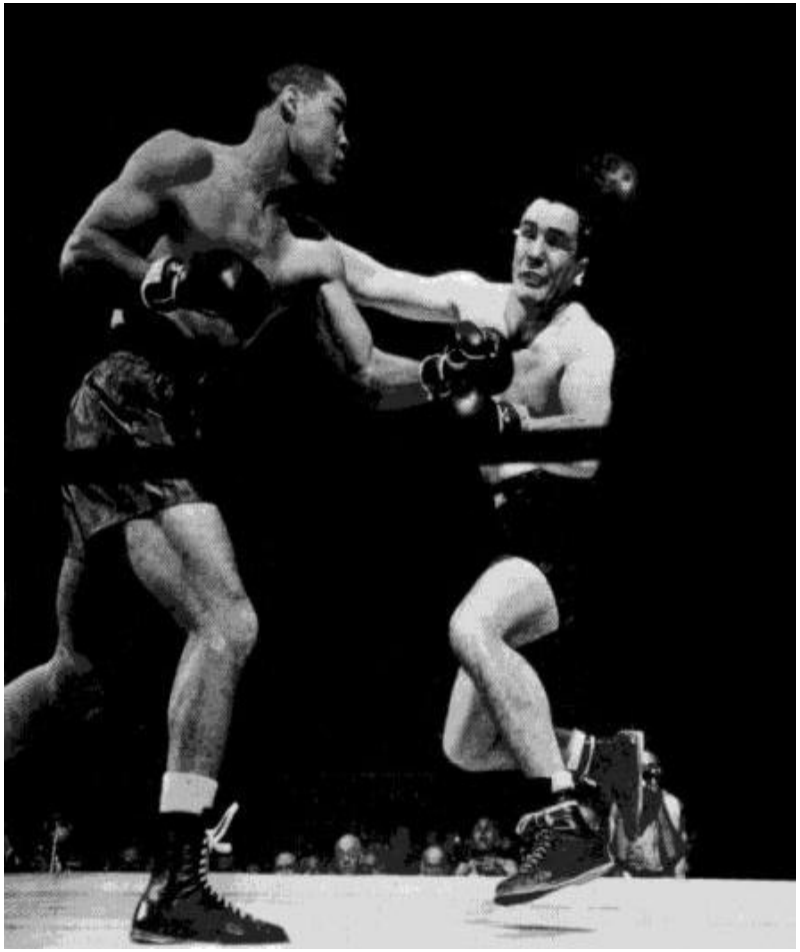
Increases blood pressure via:

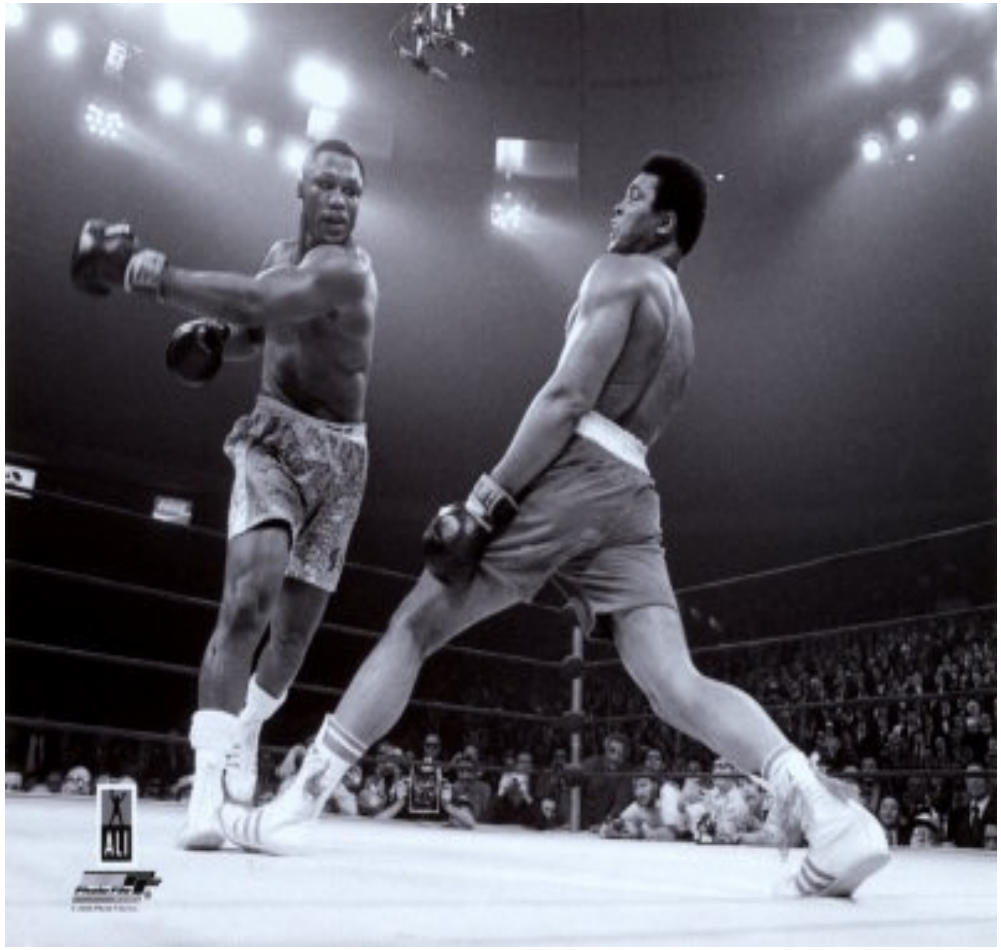
- Increased heart rate
- Increased heart contractility and thus increased stroke volume
- Vasoconstriction

Factors That Affect Blood Pressure

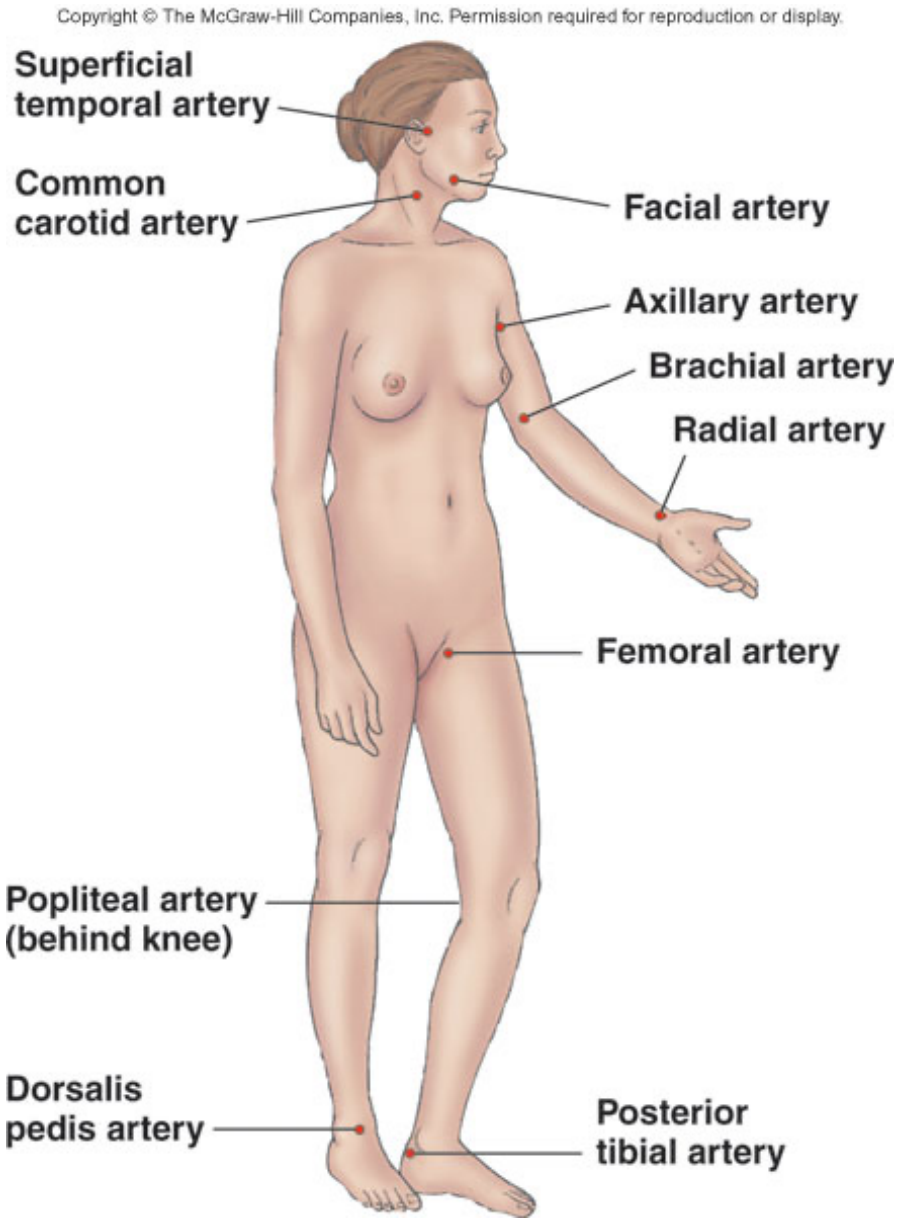
Compliance

- 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




ALI

Arterial Compliance and Pulse



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

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

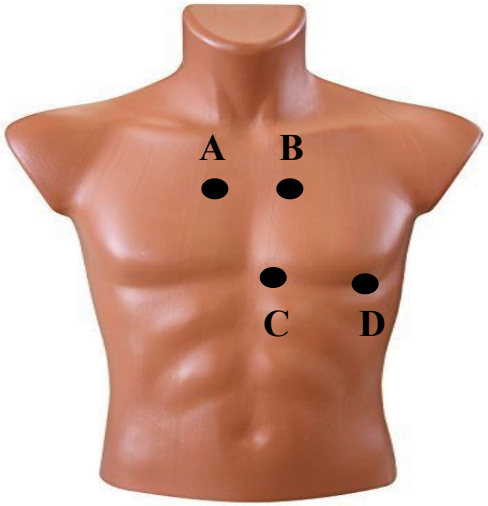
Calcium channel blockers
- Inhibit calcium channels of conduction system and cardiac muscle
  - Decrease contractility (i.e. SV) and heart rate
- Inhibit calcium channels of vascular smooth muscle
  - Decrease peripheral resistance
    - Vasodilation

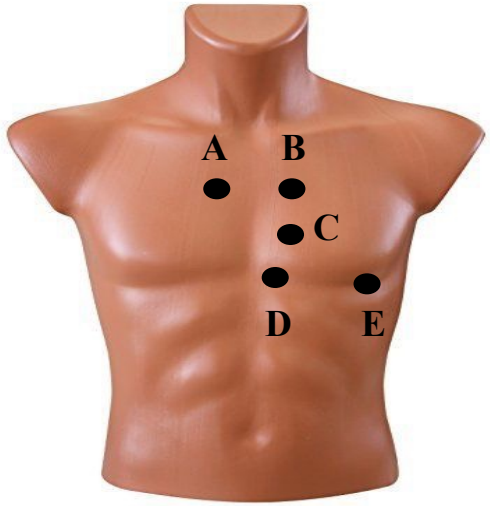
Diuretics
- Cause the excretion of fluids from the body
  - Decrease blood volume

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