

## Ch 7 & 8: Blood and Cardiac Physiology

### Objectives

Ch 8:

1. Review: Heart Anatomy, and Systemic & Pulmonary circuits.
2. The Cardiac Cycle and Heart Sounds
3. The Heart's Conduction Cycle & the ECG
4. Regulation of Heart's Pacemaker (heart rate)
5. Blood Pressure
6. Cardiac output and its Regulation
7. Three Ways the Body Regulates Blood Pressure
8. Abnormal Blood Pressure
9. Ch 7: Blood Physiology



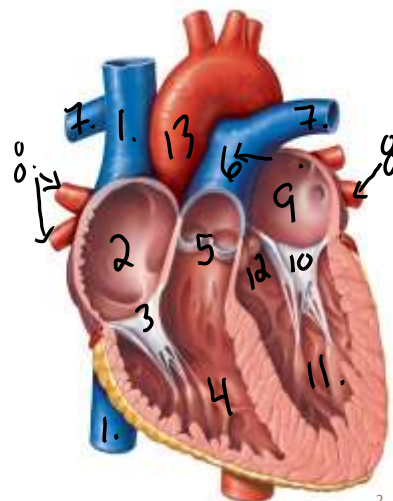
## 1. Review of Heart Anatomy and Circulatory System

**ANATOMY REVIEW!** Page 138 in text

1. Superior & inferior vena cava
2. Right atrium
3. tricuspid valve (R atrioventricular)
4. Right ventricle
5. pulmonary semilunar valve
6. Pulmonary trunk
7. Pulmonary arteries (**O<sub>2</sub>-poor**) to lungs
8. Pulmonary veins (**O<sub>2</sub>-rich**)
9. Left atrium
10. bicuspid valve (L atrioventricular)
11. Left ventricle
12. aortic semilunar valve
13. Aorta
  - > ascending
  - > arch
  - > descending

Other vessels attached to heart:

- Brachiocephalic a.
- L common carotid a.
- L subclavian a.



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## Circulatory Systems REVIEW!

Page 139 & 142 in text

### Systemic Circuit

> From left atrium, arteries, tissues, veins, to vena cava.

> **Systemic Arteries** = always travel away from heart, carry O<sub>2</sub>-rich blood

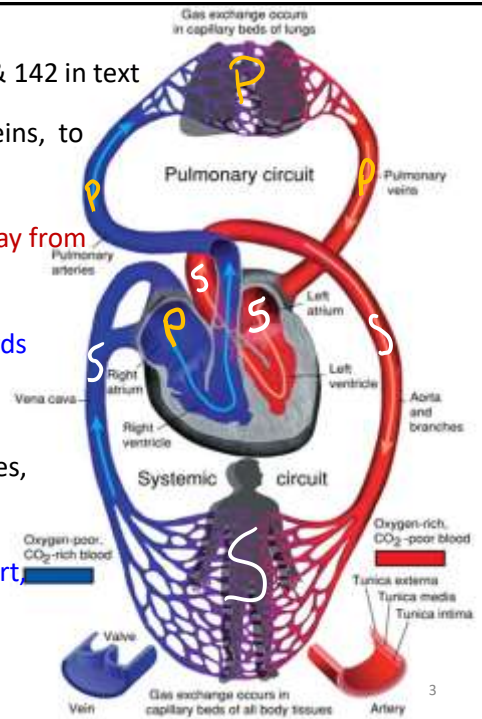
> **Systemic Veins** = always travel towards heart, carry O<sub>2</sub>-poor blood.

### Pulmonary Circuit

> From right atrium, pulmonary arteries, lungs, and pulmonary veins.

> **Pulmonary arteries** = away from heart, towards lungs w/O<sub>2</sub>-poor blood.

> **Pulmonary veins** = towards heart, w/O<sub>2</sub>-rich blood.



## 2: The Cardiac Cycle & Heart Sounds Page 144- 146 in text

**Ventricular systole** =

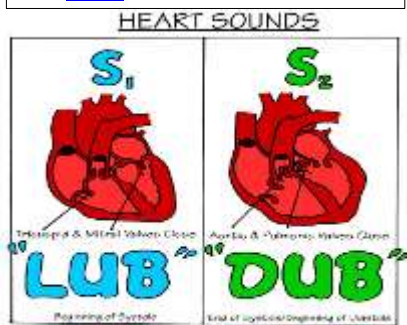
> Hear **"Lub"** or **S<sub>1</sub> sound** with atrial valves closing.

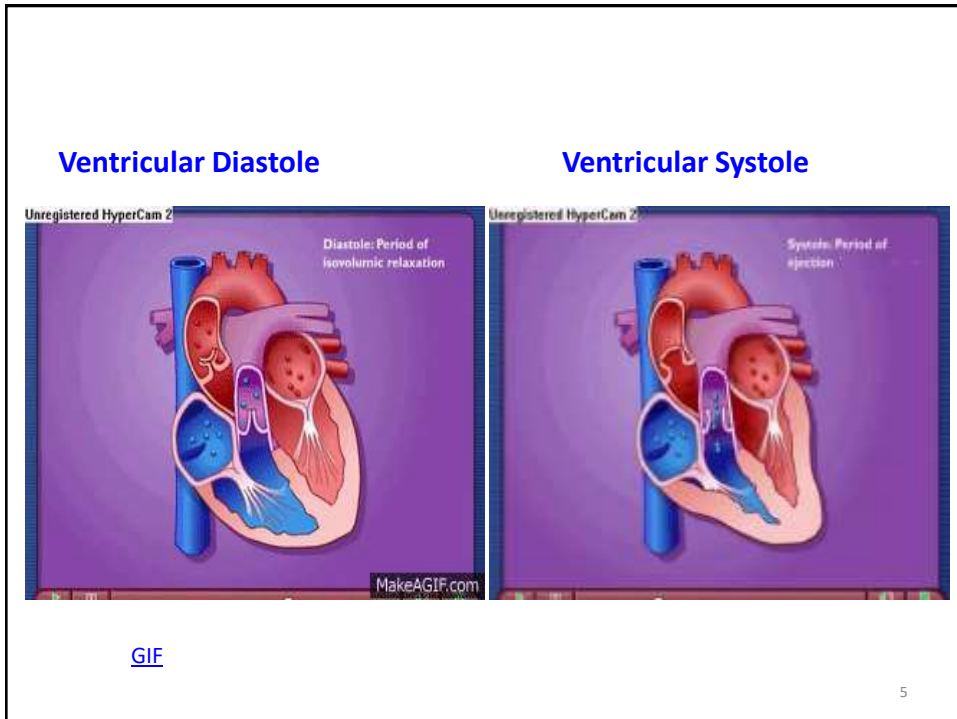
**Ventricular diastole** =

> Hear **"Dub"** or **S<sub>2</sub> sound** w/semilunar valves closing.

**Asystole** =

Click [HERE](#) for Normal heart sounds





Click [HERE](#) for Normal heart sounds

### Heart Murmurs:

> Innocent murmur =

> Pathologic murmur =

Murmurs typically caused by valve disease:

- Ex.
- **“regurgitant flow”** = backward flow through valves.
  - **aortic stenosis** = stiffening of aortic semilunar valve.
  - **rheumatic heart disease** = autoimmune attack on valves (bicuspid)

Click [HERE](#) for innocent (benign) murmur sound

Click [HERE](#) for aortic stenosis sound

Click [HERE](#) for split S2 (split dub) sound

Click [HERE](#) for bicuspid (mitral) valve regurgitant flow sound.

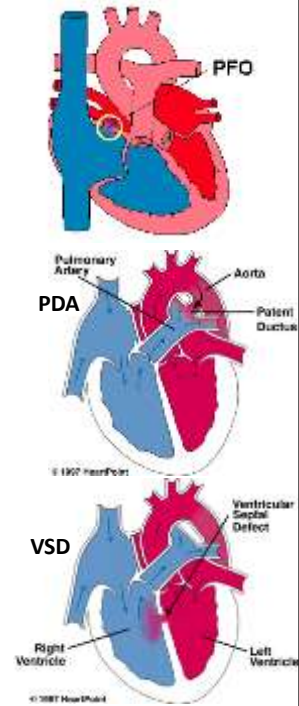
These, and more, heart sounds can be found [HERE](#)

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### Heart defects: Clinical App

#### Septal Defects:

1. Patent foramen ovale =
2. Patent ductus arteriosus =
3. Ventricular septal defect =



## Review

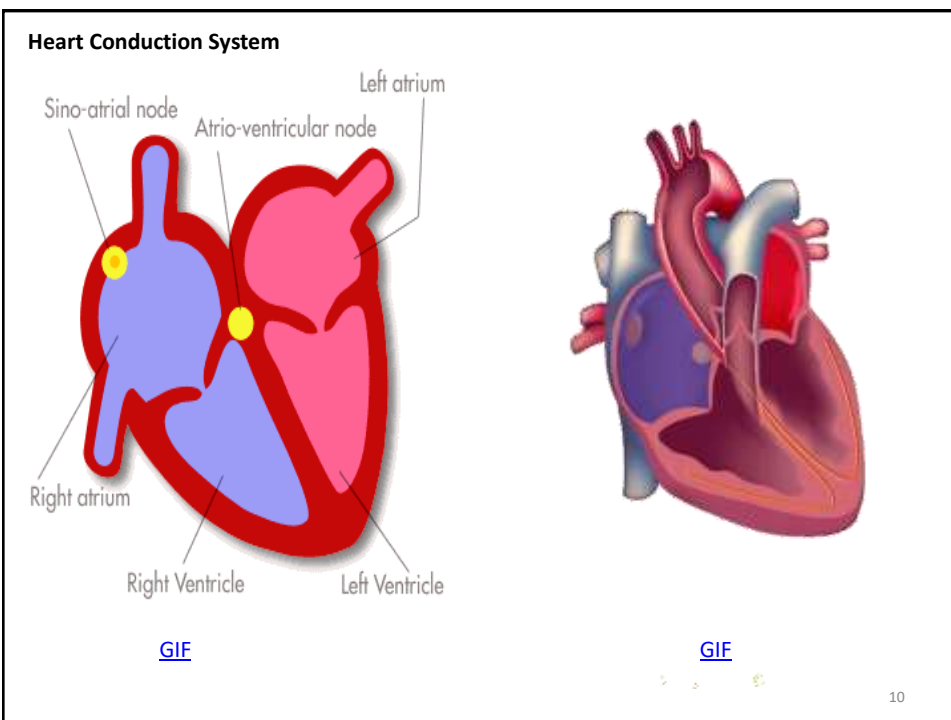
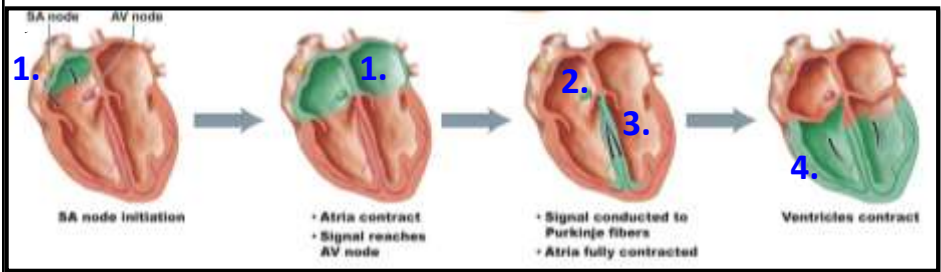
- The cardiovascular system (pulmonary & systemic circuits)
- Cardiac cycle & heart sounds
  - ventricular diastole Vs systole
  - Lub / Dub or S1 / S2 sounds (normal sounds & innocent murmur)
  - Abnormal heart sounds (pathological murmurs)
- Heart defects
  - Patent foramen ovale
  - Patent ductus arteriosus
  - Ventricular septal defect

### 3: The Heart's Conduction System & ECGs

Heart is "autorhythmic" = starts its own signal for contraction.

1. SA node =
2. AV node =
3. Bundle of HIS =
4. Purkinje fibers =

Page 146- 148 in text

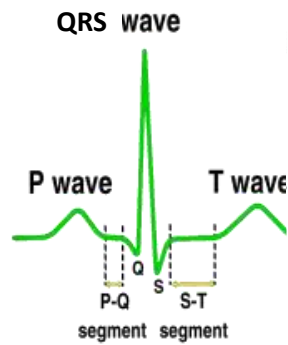


## Electrocardiogram (ECG or EKG) =

P-wave =

QRS wave =

T-wave =



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## 4: The Heart's Pacemaker & Its Regulation

*Regulated by cardiac center in medulla oblongata!*

Sympathetic innervation with thoracic & cardiac nerves:

- Neurotransmitter =
- Receptor =
- Heart rate

Parasympathetic innervation with vagus nerves:

- Neurotransmitter =
- Receptor =
- Heart rate

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## 4: The Heart's Pacemaker & Its Regulation

### Spontaneous APs start in pacemaker myocardial cells

- 1. Heart pacemaker cell depolarization (stimulation):** *Epinephrine binding to  $\beta_1$  adrenergic receptors on pacemaker increase rate of depolarization*
  - > opening of \_\_\_\_\_ & \_\_\_\_\_ channels
  - > causes AP (or EPSP)
  - > Myocardial cells contract! (Signal started!)
- 2. Heart pacemaker cell repolarization (rest):** *ACh binding to muscarinic cholinergic receptors on pacemaker decrease rate of depolarization*
  - > opening of \_\_\_\_\_ channels
  - > Myocardial cells relax!

APs started in SA node pacemaker muscle cells travel through rest of conduction system (i.e. AV node, Bundle of HIS, & Purkinje fibers).

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### Arrhythmias = abnormal heart rate

#### Clinical App

#### Tachycardia =

##### Treatments:

- > Na<sup>+</sup> channel blockers (*quinidine, lidocaine*)
- > Ca<sup>2+</sup> channel blockers (*verapamil*)

#### > Beta blockers

ex. *General beta blocker =*

*Review* } *B1-specific blocker =*

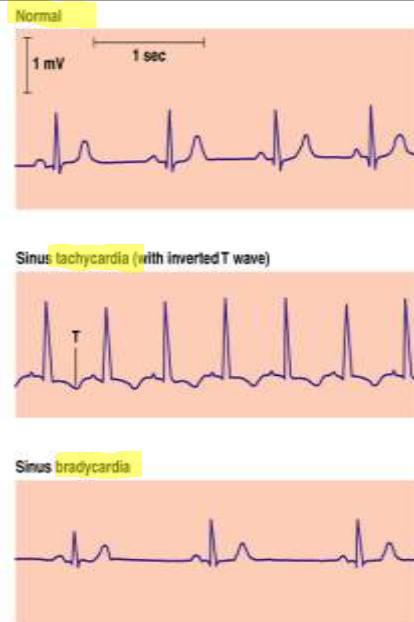
#### Bradycardia =

##### Treatments:

- > Digitalis – increases Ca<sup>2+</sup> available to increase contractile strength.

#### > B1-agonist =

#### > MAO-I A =



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# Review



- **Cardiac conduction system**
  - SA node, AV node, Bundle of HIS, Purkinje fibers
- **Monitoring electrical activity of the heart (EKG)**
  - P wave, QRS wave, T wave
- **The Heart's Pacemaker**
  - parasympathetic and sympathetic regulation
- **Arrhythmias (tachycardia & bradycardia)**
  - Drugs to treat arrhythmias

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## 5: Blood Pressure

**Blood Pressure** = pressure of arterial blood against vessel wall.

**Systolic BP** = pressure resulting from ventricular contraction.  
- always the higher number.

Systolic arterial BP normal range = 80 – 160 mmHg

**Diastolic BP** = pressure with ventricular relaxation.  
- always the lower number

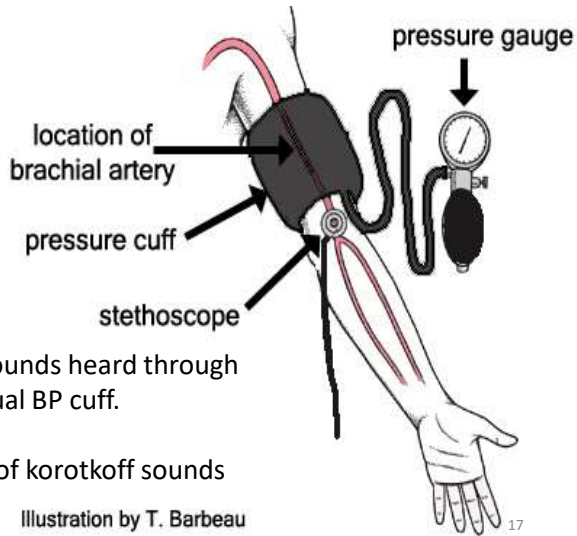
120 mmHg  
80

Page 149 – 150 in text

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### Measuring Blood Pressure (Systolic & Diastolic) using a sphygmomanometer



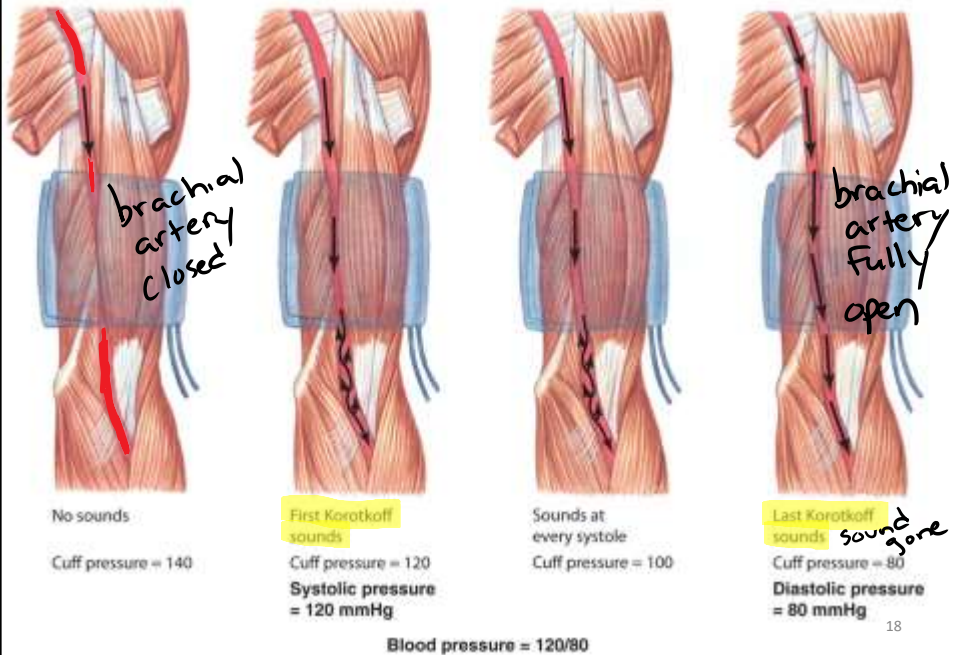
“**korotkoff sounds**” = blood sounds heard through stethoscope when using manual BP cuff.

Click [HERE](#) for YouTube video of korotkoff sounds

Illustration by T. Barbeau

### Measuring Blood Pressure (Systolic & Diastolic)

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## 6: Cardiac Output

$$\text{Cardiac Output} = \text{Stroke Volume} \times \text{Heart Rate}$$

(ml/min)                      (ml/beat)                      (bpm)

Average HR varies (~60 – 80 bpm)

AVG stroke volume = 70 – 80 ml/beat

AVG cardiac output = 5500 ml/min (5.5L/min)

Cardiac Output influenced by:

- 1.
- 2.
- 3.
- 4.
- 5.



## Things That Influence Cardiac Output:

### 1. Heart Rate:

HR changes with sympathetic or parasympathetic stimulation.

What are some drugs we've covered that can ↑ HR, thus will ↑ cardiac output?

What are some drugs we've covered that can ↓ HR, thus will ↓ cardiac output?

## Things That Influence Cardiac Output:

### 2. Stroke Volume:

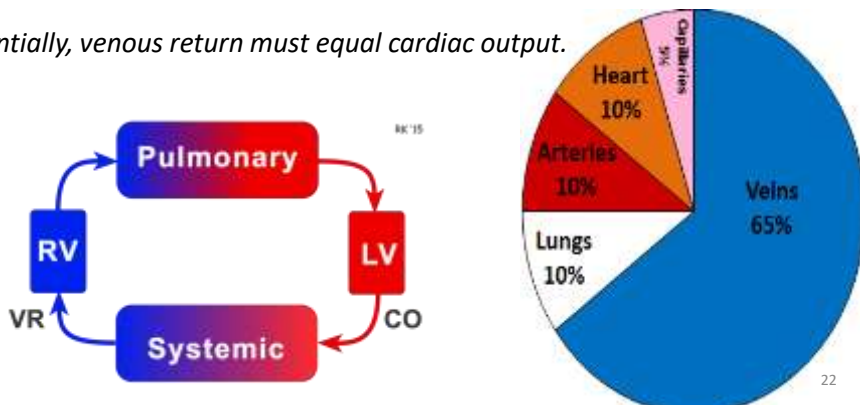
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## Things That Influence Cardiac Output:

REVIEW!

### 3. End Diastolic Volume (EDV) – =

*Essentially, venous return must equal cardiac output.*



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## Things That Influence Cardiac Output:

### 4. Stretching of ventricles (and heart contractility)

#### Frank-Starling Law of the Heart

So....

As EDV  $\uparrow$ ,

As EDV  $\downarrow$ ,

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## Things That Influence Cardiac Output:

### 5) Total peripheral resistance (TPR) =

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## Things That Influence Cardiac Output:

### In summary:

As HR ↑, cardiac output ↑

As HR ↓, cardiac output ↓

As stroke volume ↑, cardiac output ↑

As stroke volume ↓, cardiac output ↓

As EDV ↑, cardiac output ↑

As EDV ↓, cardiac output ↓

↑ ventricular stretch, ↑ heart contractility, so cardiac output ↑

↓ ventricular stretch, ↓ heart contractility, so cardiac output ↓

### And

As TPR ↑, stroke volume and cardiac output ↓

As TPR ↓, stroke volume and cardiac output ↑

### To re-cap:

Click [HERE](#) for a really good YouTube video explaining effect of HR, stroke volume, EDV, ventricular stretch, and TPR on cardiac output.

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## Review



- Blood pressure
- Factors that influence cardiac output:
  - there are MANY factors, but we covered:
  - Heart rate
  - Stroke volume
  - EDV
  - Stretching of ventricles (Frank Starling principle) and heart contractility.
  - TPR

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## 7: Three Ways the Body Regulates Blood Pressure

**Blood pressure is directly influenced by blood volume:**

**The kidneys** have the most important control of blood volume, by how much water they retain (keep in bloodstream) versus how much they excrete as urine.

Kidneys affected by sympathetic and parasympathetic control, AND by hormones.

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## 7: Three Ways the Body Regulates Blood Pressure

### **The Quick Fix to BP**

vs

### **The Slow Fix to BP**

Artery baroreceptors & medulla oblongata (cardiac and vasomotor centers)

*Fixes BP quickly, but does not last long*

Hormones that affect kidney water reabsorption.

*Takes more time to fix BP, but its effect lasts longer (hours or days)*

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## 7: Three Ways the Body Regulates Blood Pressure

### 1. Baroreceptor reflexes

“baroreceptors” = pressure receptors that detect stretching of arteries  
OR the heart chambers.

### 2. Hypothalamus

### 3. Kidney regulation

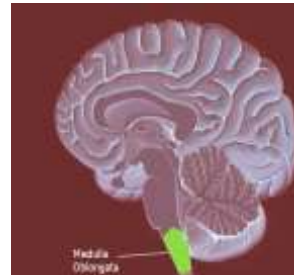
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### 1. Arterial baroreceptor reflex (the “Quick Fix”)

**Stimulus** = change in arterial blood pressure outside  
80 – 160 mmHg

**Sensor** = aortic arch & carotid artery baroreceptors

**Integrating center** = medulla cardiac & vasomotor centers



#### A) If stimulus of ↓ BP (below 80mmHg)

**Autonomic response is sympathetic**

- Neurotransmitter = \_\_\_\_\_
- Pacemaker muscle receptors = \_\_\_\_\_, HR ↑
- arterial vasoconstriction
- BP ↑

#### B) If stimulus of ↑ BP (above 160 mmHg)

**Autonomic response is parasympathetic**

- neurotransmitter = \_\_\_\_\_
- pacemaker muscle receptors = \_\_\_\_\_, HR ↓
- arterial vasodilation
- BP ↓

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## 2. Heart baroreceptors reflex (hormonal response & “Long Fix”)

**Stimulus** =  $\uparrow$  blood pressure (heart chamber stretched)

**Sensor, integrating center, and effector** = heart

Heart secretes \_\_\_\_\_



**ANP** functions to

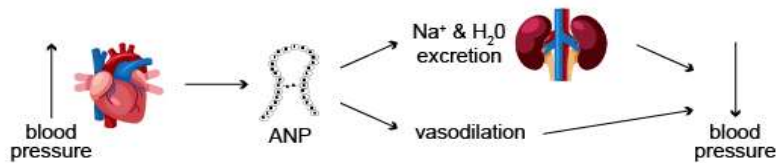
Cause arterial vasodilation

\_\_\_\_\_ filtration rate at kidneys, which

\_\_\_\_\_ salt & water reabsorption &  $\uparrow$  urine output (pee more)

\_\_\_\_\_ blood volume

**“effect”** =  $\downarrow$  blood pressure



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## 3. Hypothalamus – Blood Osmolarity Center setpoint of 280 – 290 mOsm

**stimulus** =  $\uparrow$  blood osmolarity

(above 290 mOsm)

**sensor & integrating center, & effector**  
= hypothalamus, which releases **ADH**.

**effect of ADH** =

\_\_\_\_\_ water reabsorption at kidneys

\_\_\_\_\_ blood volume & blood pressure

\_\_\_\_\_ **blood osmolarity**

**Stimulus** =  $\downarrow$  blood osmolarity

hypothalamus inhibits ADH release

opposite things happen

\_\_\_\_\_ water reabsorption at kidneys

\_\_\_\_\_ blood volume & blood pressure

\_\_\_\_\_ **blood osmolarity**

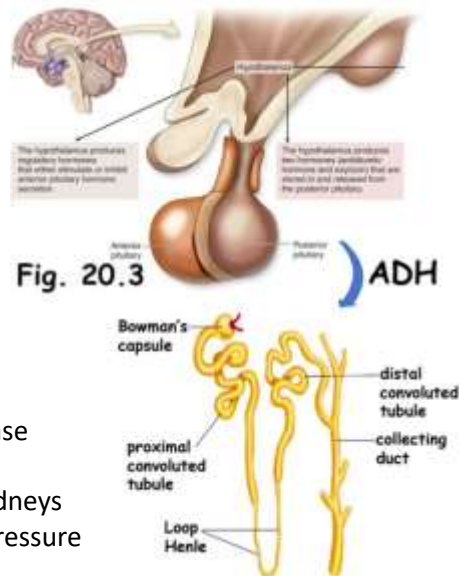
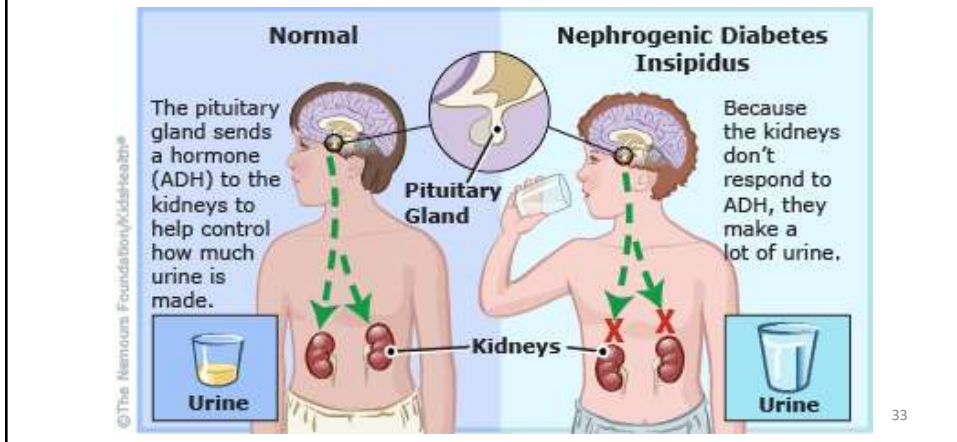


Fig. 20.3



**Diabetes insipidus** = insufficient ADH release by hypothalamus.

- ↓ water reabsorption at kidneys
- ↑ urine output (pee more)
- ↓ blood volume and BP
- become chronically dehydrated



#### 4. Kidney –renin-angiotensin-aldosterone system (“long fix”)

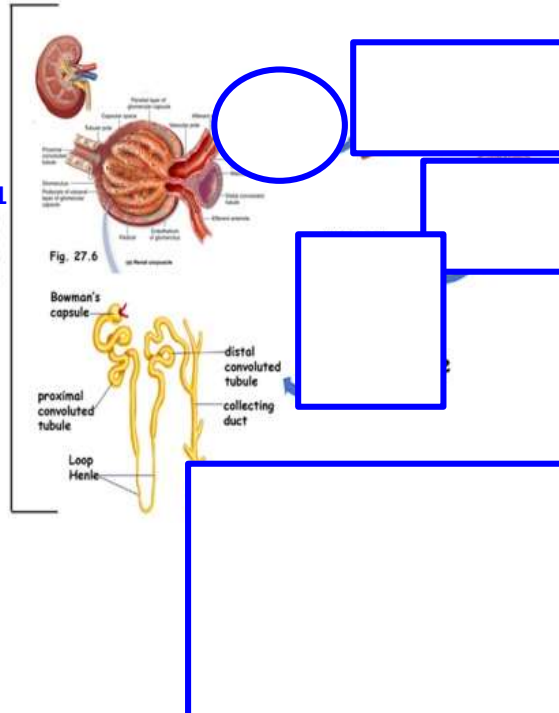
**Stimulus** = low BP at renal artery of kidneys

**Sensor, integration center, & effector** = kidney juxtaglomerular apparatus (JGA)

**“effect”** = ultimately, an increase in BP

**IF blood volume & BP ↓:**

- > Sensed by **JGA**
- > JGA releases **Renin**
- > Renin causes liver to convert **angiotensinogen** → **angiotensin 1**
- > Angiotensin 1 → **angiotensin 2** by **ACE** in lungs
- > Angiotensin 2 stimulates Adrenal cortex make **aldosterone**
- > **Aldosterone** ↑ salt reabsorption
- > ↑ Water reabsorbed w/salt
- > ↑ blood volume, ↑ **BP**



**IF blood volume & BP ↑:**

- > Renin release is inhibited

**ACE = angiotensin converting enzyme**

**ACE inhibitors – Clinical App**

**Addison's Disease** = low aldosterone production by adrenal cortex.

- ↓ salt (reabsorption by kidneys)
- ↓ water reabsorption by kidneys

### Clinical presentation?

Hyponatremia =

Hyperkalemia =

Bradycardia =

Polyuria =

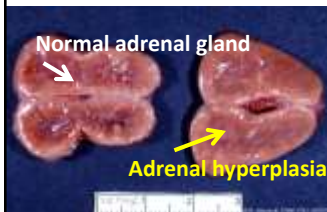
Hypovolemia=

Hypotension =

Skin bronzing

**Conn's Syndrome** = "hyperaldosteronism" or excess aldosterone

### Clinical presentation?



Hypernatremia =

Hypokalemia =

Oliguria =

Hypervolemia=

Hypertension =

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## See blank and key flow diagrams on the online syllabus!

For the quick fix and long fix for **LOW BP** and the **KEY**

(This will involve a **quick fix** by the medulla, and a **long fix** by the renin angiotensin aldosterone system)

For the quick fix and long fix for **HIGH BP** and the **KEY**

(This will involve a **quick fix** by the medulla, and a **long fix** by the heart secreting ANP)

For the fix of **HIGH BLOOD OSMOLARITY** and the **KEY**

(This involves the hypothalamus and ADH)

And click [HERE](#) for a PDF outlining which systems engage when BP is too low or too high.

These will each involve a **quick fix** by the medulla, and a **long fix** by a hormone. Also, this goes over blood osmolarity regulation by the hypothalamus and ADH.

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## Review



- Blood volume & blood pressure regulated by
  - Medulla baroreceptor response (Quick fix)
    - Low BP causes sympathetic arteriole vasoconstriction and  $\uparrow$  in HR & BP
    - High BP causes parasympathetic arteriole vasodilation and  $\downarrow$  in HR & BP

### Slow fixes

- Heart baroreceptor response (ANP)
- Hypothalamus (ADH release)
- Kidney (renin-angiotensin-aldosterone system)

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## 8: Abnormal Blood Pressure

**Hypotension** = low BP

**Hypertension** = high BP. Can be due to MANY factors.

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## **2 types of Hypertension:**

1. Primary (idiopathic) Hypertension = exact cause unknown.
2. Secondary Hypertension = result of disease (i.e. kidney or cardiac problem)

### **Pausable causes of 2° Hypertension**

- **Hypervolemia** = high blood volume.

Can occur with:

- Excess ADH secretion
  - “Conn’s syndrome” (hyperaldosteronism) = Excess aldosterone secretion
- **Stress**
  - **Pheochromocytoma** = high epinephrine from adrenal medulla
  - **Atherosclerosis** – narrowing of arteries from cholesterol deposits
  - **Renal artery disease** (↑ renin or increased angiotensin 2 secretion)
  - **Pre-eclampsia (gestational hypertension)** = vasoconstriction of maternal arteries or problems with placenta. Causes still largely unknown.

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## **Hypertension Drugs**

Can you think of any drugs (especially ones we’ve covered) that can lower BP?

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## Circulatory Shock = inadequate blood flow to all body tissues

Page 155 in text

### Many types:

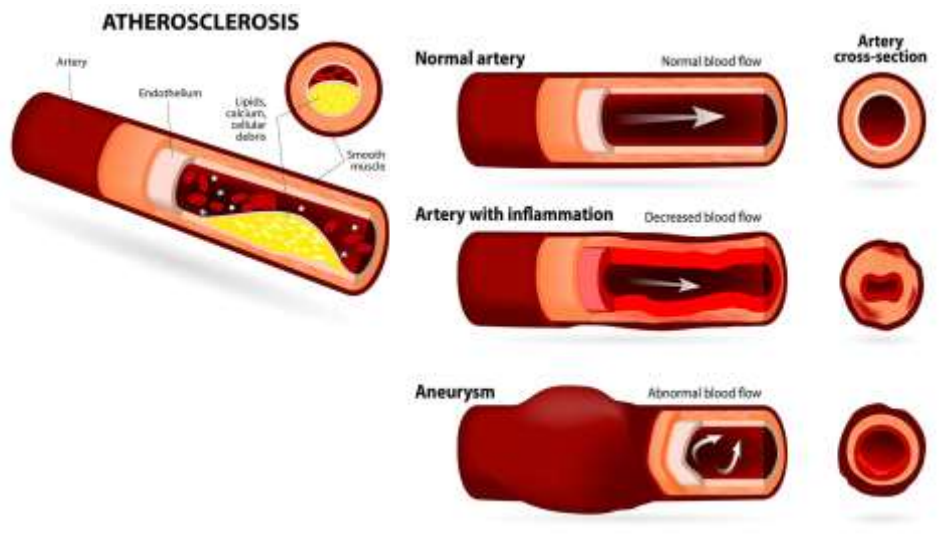
1. \_\_\_\_\_ shock = drop in blood volume and BP (blood loss, dehydration)  
**body response** = ↑ heart rate (compensatory to ↑ BP)  
 = vasoconstriction (to ↑ BP)
2. \_\_\_\_\_ shock = drop in blood volume and BP from infection. Caused by bacterial toxins in blood. Causes vasodilation & ↓ BP  
**body response** = same as for #1.
3. \_\_\_\_\_ shock = drop in blood volume and BP due to massive histamine release which causes vasodilation and ↓ BP.  
**body response** = same as #1
4. **Congestive heart failure** = drop in blood volume and BP due to heart not working.  
**Body response** = same as #1

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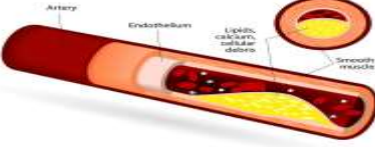
## Cardiac Diseases: Atherosclerosis

= buildup of cholesterol plaques within arterioles.

Can decrease blood flow and lead to:



**ATHEROSCLEROSIS**




Formation of **thrombus** =

**Embolism** =

**Ischemia** =

Ex. **Stroke** = blocked blood flow to brain.  
Ex. **Heart attack** = blocked blood flow to heart.



**Arteriosclerosis** =

**Aneurysm** =

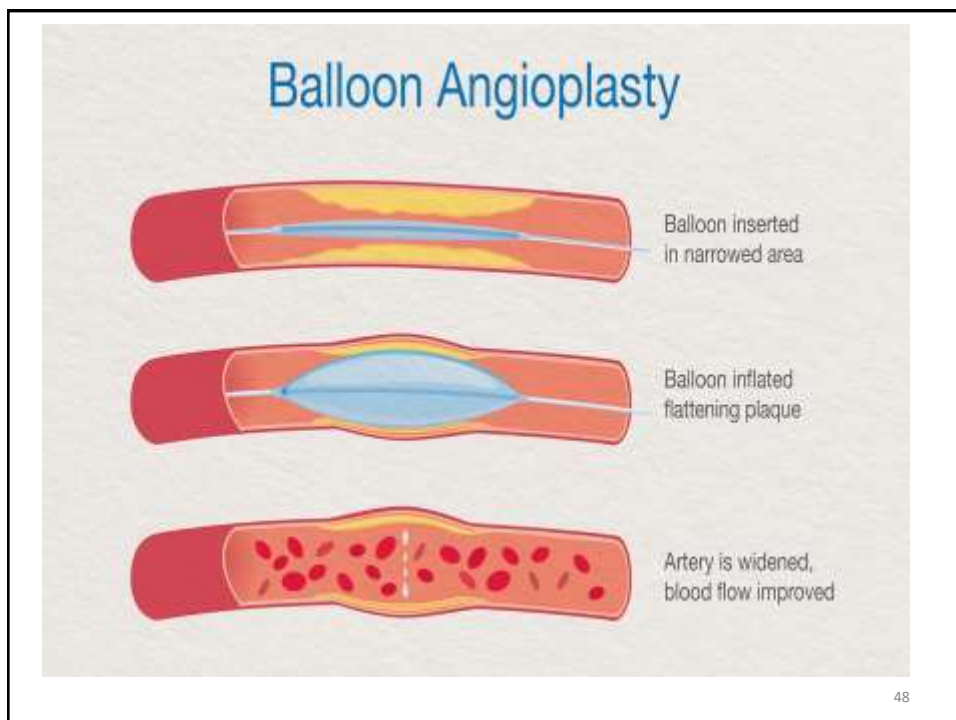
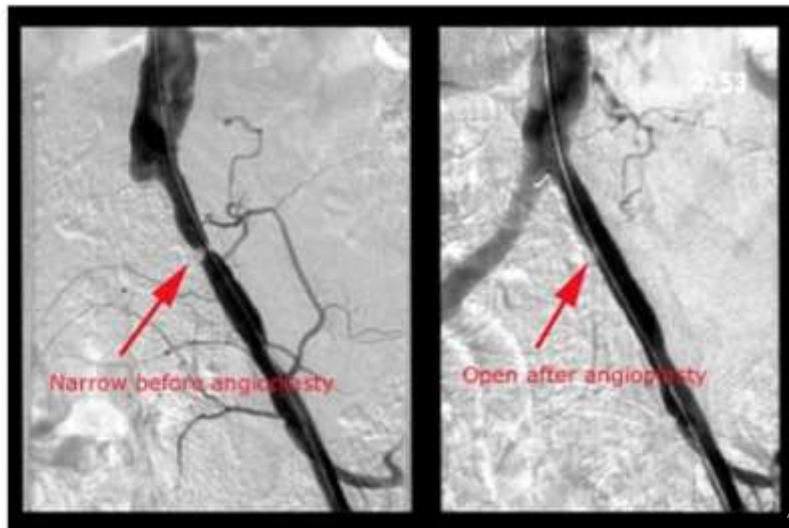
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Click [HERE](#) for a brief YouTube diagrammatic explanation of an embolism.

Click [HERE](#) for YouTube video of an actual surgical removal of an embolism. (*Warning: graphic content*)

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**Angiogram** = diagnostic test that uses x-rays to take pictures of your blood vessels. A long flexible catheter is inserted through the blood stream to deliver dye (contrast agent) into the arteries making them visible on the x-ray





## Review

- **Abnormal blood pressure**
  - Hypotension
  - Hypertension (1° and 2°)
  - Some causes of 2° hypertension
- **Circulatory shock**
  - Hypovolemic shock
  - Septic shock
  - Body's response to shock
- **Atherosclerosis leads to many other circulatory problems.**

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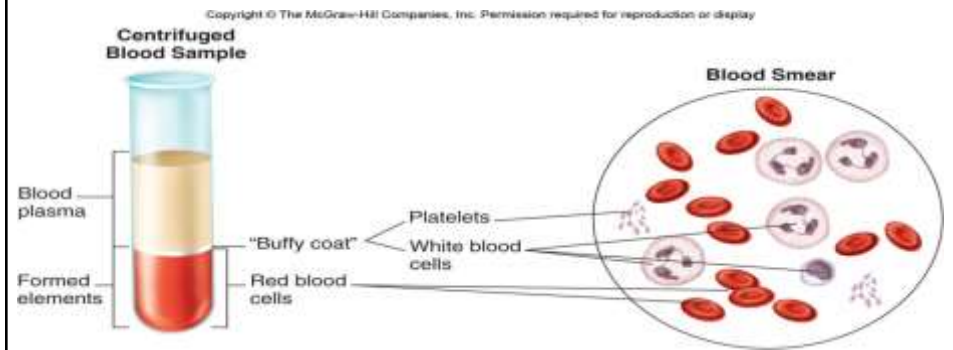
## 9: Blood Physiology

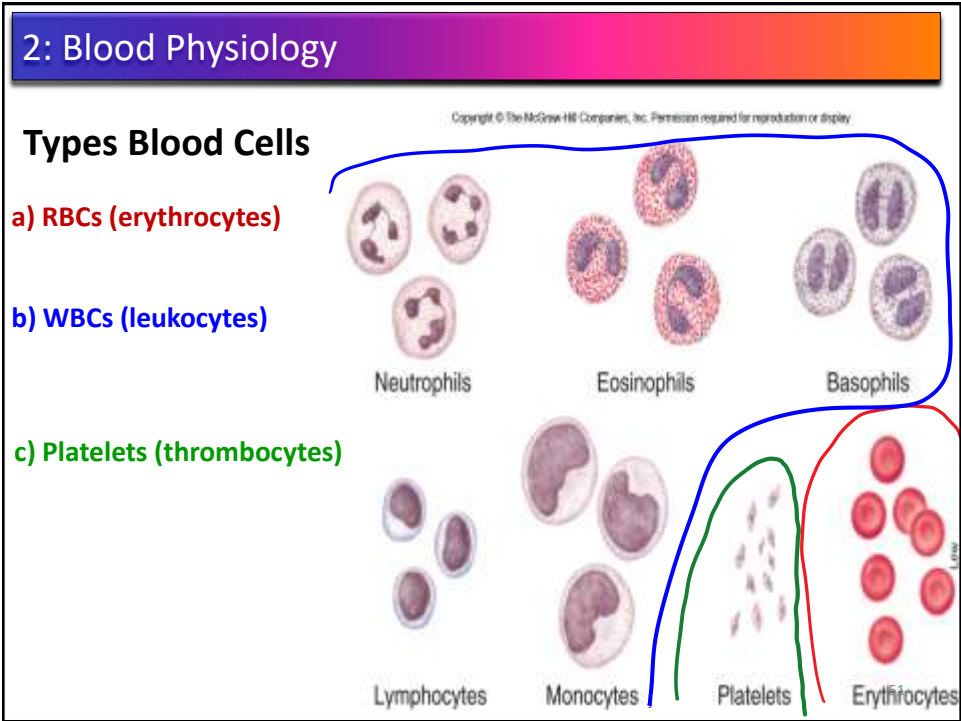
### **Blood Composition: Page 123 in text**

**Whole blood** = liquid portion of blood along with the cells.

**Plasma** = liquid portion of blood (with things dissolved in it like salts, hormones, proteins, etc...)

**Cellular portion** = RBCs, WBCs, and platelets.






**2: Blood Physiology**

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**RBCs (Erythrocytes)**

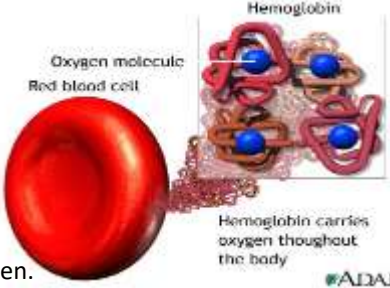
- > Carry O<sub>2</sub> bound to hemoglobin (heme + iron).
- > ~500 million new RBC each day!
- > RBCs last ~120 days then removed by liver & spleen.
  - Heme broken into bilirubin (yellow pigment), which liver removes.
  - Iron in hemoglobin re-used in new RBCs.

- **jaundice** = \_\_\_\_\_.



- **erythropoiesis** = \_\_\_\_\_.

Stimulated by hormone **erythropoietin** (released by liver & kidneys.)



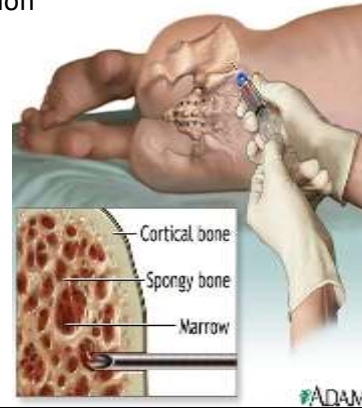
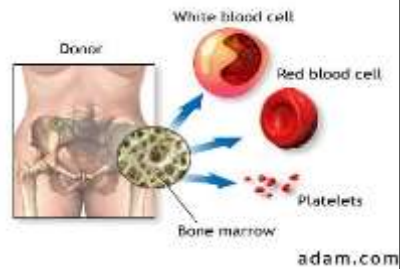
**Polycythemia** = higher than normal RBC count.

**Anemia** = lower than normal RBC count.

[Clinical App](#)

Can be due to many factors:

- **Iron deficiency anemia** = low iron in diet.
- **Pernicious anemia** = poor vitamin B12 absorption (need to make RBCs)
- **Aplastic anemia** = bone marrow defect (often from chemotherapy treatment).
- **Renal anemia** = low erythropoietin production by kidneys.
- **Autoimmune hemolytic anemia** = immune attack on RBCs (see with Rh disease)



## Blood Typing

### 2 Major RBC antigens:

- 1) ABO antigens
- 2) Rh antigen

#### 1) ABO

**Blood Type A** – have \_\_\_\_\_ antigens & \_\_\_\_\_ antibodies  
– receives type \_\_\_\_\_ blood

**Blood Type B** – has \_\_\_\_\_ antigens & \_\_\_\_\_ antibodies  
– receives type \_\_\_\_\_ blood

**Blood Type AB** – has \_\_\_\_\_ antigens & \_\_\_\_\_ antibodies  
– “universal recipient”, can receive \_\_\_\_\_ blood

**Blood Type O** – has \_\_\_\_\_ antigens, & \_\_\_\_\_ antibodies  
– “universal donor”, but can receive only type O blood

#### 2) Rh factor

**Rh+** = have Rh antigen on RBCs (~85% of population)      **Rh-** = not have Rh antigen (~15% of population)

## Blood Type Test

You put blood sample into each of 3 wells, then add antibodies against the possible Antigens.

If see clotting (**agglutination**)  
The RBCs must have antigen to that antibody.

Add patient's blood to test card:

Add anti-A antibodies - if clots = **Type A**

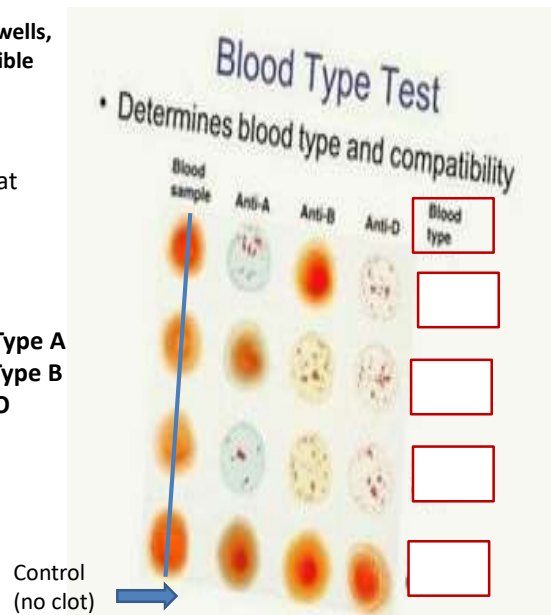
Add anti-B antibodies – if clots = **Type B**

If no clots in either A or B = **Type O**

Add anti-Rh (D) antibodies

– if clots = Rh+

– no clot = Rh-



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## Rh incompatibility in pregnancy

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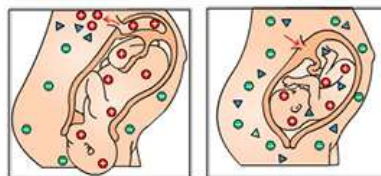
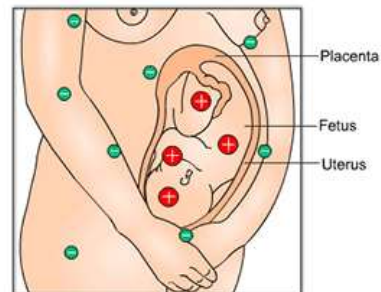
If Rh- woman pregnant from Rh+ man – 50% chance baby is Rh+

Risk of exposure of mom's blood stream to fetal RBCs with Rh+ antigens.

(Ex. During miscarriage or tissue tearing during birth or C-section)

Mom's immune system would develop anti-Rh antibodies within 2 weeks of exposure.

- During her next pregnancy if baby Rh+, maternal antibodies cross placenta
- Maternal antibodies attack (hemolyze) fetal RBCs
- “**autoimmune hemolytic anemia**” = immune destruction of RBCs in baby from mom's antibodies



[Clinical App](#)

## Rh incompatibility in pregnancy

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### Prevention:

> If doctor suspects exposure to Rh+ blood in mom's first pregnancy.

> Give injection of anti-Rh antibodies to mom

> antibodies destroy and fetal Rh+ fetal RBCs in mom's body BEFORE her immune system detects & makes own antibodies.



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## b) WBCs (Leukocytes)

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### 2 Groups:

1) **Granulocytes** = WBCs with granules in cytoplasm

50-70% - \_\_\_\_\_ = 1<sup>st</sup> responders to infection/inflammation.

2-4% - \_\_\_\_\_ = see w/chronic inflammation, infection, allergies, parasites

<1% - \_\_\_\_\_ = non-phagocytes, produce histamine & heparin in allergic reactio

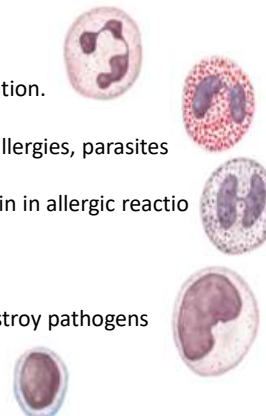
2) **Agranulocytes** = lack granules.

2-8% - \_\_\_\_\_ = phagocytes that seek out, engulf, & destroy pathogens

20-30% - \_\_\_\_\_ = defense from pathogens

> T cells =

> B cells = become plasma cells to produce antibodies.



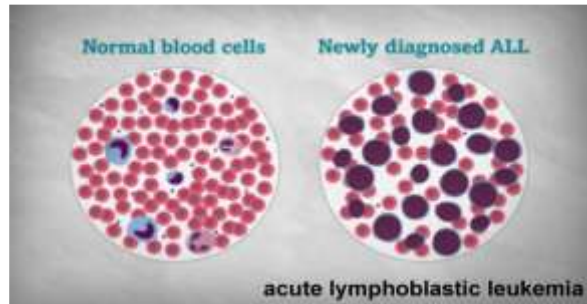
**Never Let Monkeys Eat Bananas** (neutrophils, lymphocytes, monocytes, eosinophils, basophils)

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## Leukocyte disorders – Clinical App

> \_\_\_\_\_ =  $\uparrow$  WBC count (infections!)

> \_\_\_\_\_ =  $\uparrow$  in immature numbers of WBCs, especially lymphocytes. (immature cells not protective)



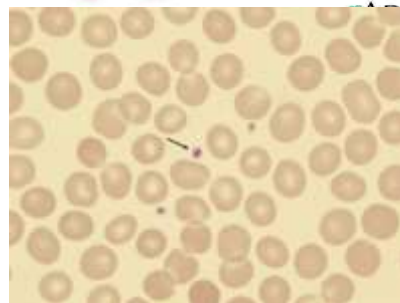
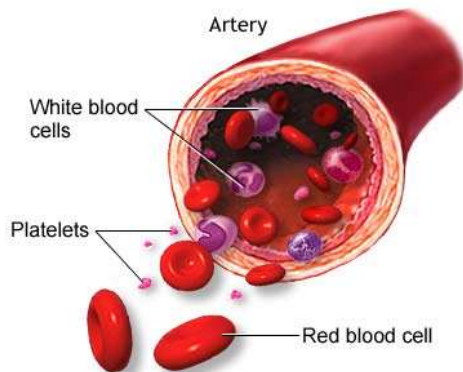
> \_\_\_\_\_ =  $\downarrow$  WBC count (with immunosuppression, chemotherapy Tx)

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### c) Platelets (Thrombocytes)

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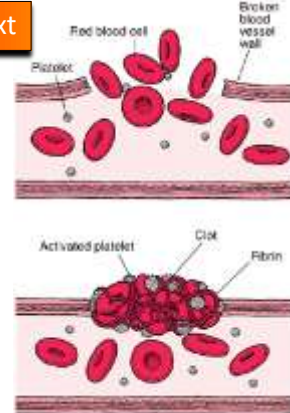
- Circulate  $\sim$  5-9 days
- Function to start clot formation
- “**thrombopoietin**” = hormone from liver & kidney that stimulates platelet production by bone marrow.



### 3: Physiology of Blood Clotting Page 126 -127 in text

**"Hemostasis"** = stopping bleeding from damaged blood vessel

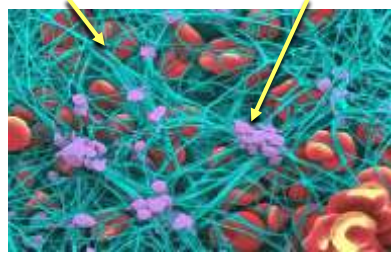
- > A blood vessel is damaged:
- > arterioles constrict
- > Platelets form a "plug"
  - Platelets convert **prothrombin to thrombin**
  - **Thrombin** activates **fibrinogen**
  - Fibrinogen converts into **fibrin threads**
  - fibrin threads "knit" the wound closed.



#### Clotting Disorder Tests:

1. **Bleeding time** w/skin prick (< 1 – 3 min)
2. **Prothrombin time** – treat blood plasma w/citrate and thromboplastin, and add Ca+2 then measure time to clot (< than 12 sec OK, but longer = prothrombin deficiency)

Fibrin threads



#### Clotting and Anticoagulants

##### [Clinical App](#)

### Collecting & Examining Blood Components:

**Vacutainer tubes** = use vacuum to draw blood into tube.

- > **Red top** = no anticoagulant. After spin get serum as fluid portion (use in serological tests)
- > **Purple top** = has EDTA anticoagulant. After spin get "plasma". No spin use for blood counts, disease testing.
- > **Light blue** = sodium citrate (for clotting tests)



Click [HERE](#) for Wikipedia description & uses of various vacutainer tubes.

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# Review



- **The 3 ways the body regulates blood volume and blood pressure**
  - Heart baroreceptors: heart secretes ANP when BP is too high (a long fix)
  - Arterial baroreceptors and the medulla's cardiac and vasomotor center (provides a quick fix to BP that is either too low or too high)
  - Hypothalamic ADH secretion when blood osmolarity rises too high.
  - Renin angiotensin aldosterone system, which engages when BP is too low
- **Blood composition**
  - Plasma
  - Erythrocytes, leukocytes, platelets
    - Granulocytes (basophils, eosinophils, neutrophils)
    - Agranulocytes (lymphocytes, monocytes)
- **Blood Typing**
- **Blood clotting with platelets**
- **Techniques for Collecting & Examining Blood**