

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. Congestive Heart Failure

Ch 7:

10. Blood Physiology



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1. Review of Heart Anatomy and Circulatory System

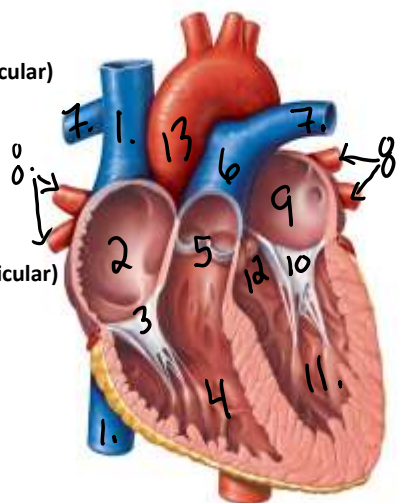
Page 138 in text

ANATOMY REVIEW!

Other vessels attached to heart:

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

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 (O₂-poor) to lungs
8. Pulmonary veins (O₂-rich)
9. Left atrium
 10. bicuspid valve (L atrioventricular)
11. Left ventricle
 12. aortic semilunar valve
13. Aorta
 - > ascending
 - > arch
 - > descending



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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 O₂-rich blood

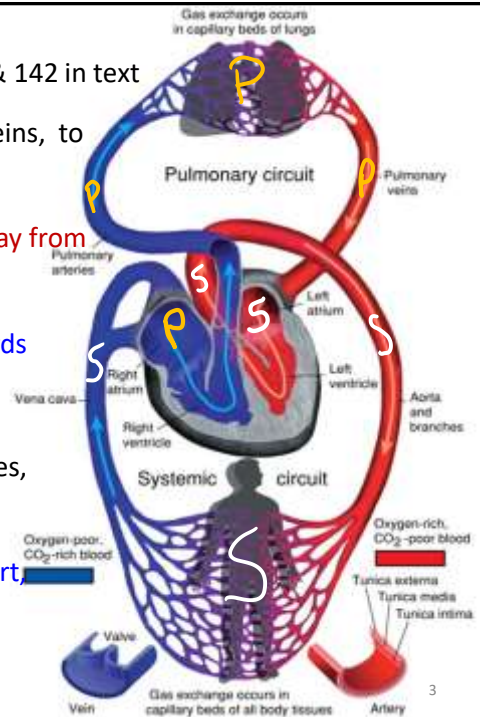
> **Systemic Veins** = always travel towards heart, carry O₂-poor blood.

Pulmonary Circuit

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

> **Pulmonary arteries** = away from heart, towards lungs w/O₂-poor blood.

> **Pulmonary veins** = towards heart, w/O₂-rich blood.



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

Ventricular systole =

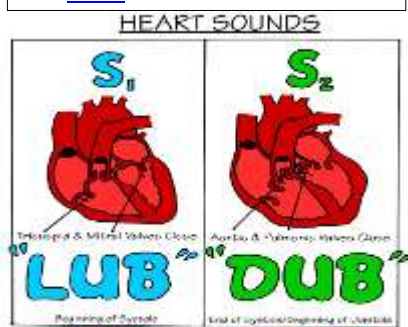
> Hear **"Lub"** or **S1 sound** with atrial valves closing.

Ventricular diastole =

> Hear **"Dub"** or **S2 sound** w/semilunar valves closing.

Asystole =

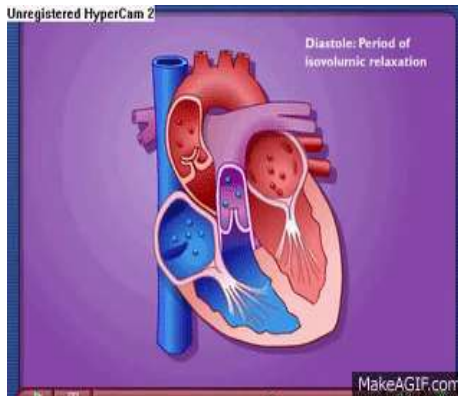
Click [HERE](#) for Normal heart sounds



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Ventricular Diastole

Ventricular Systole



[GIF](#)

5

5

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](#)

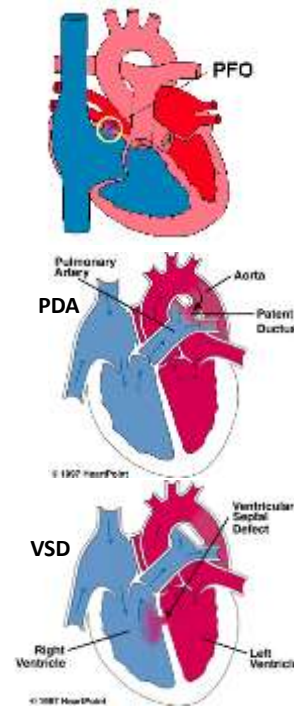
6

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Heart defects: Clinical App

Septal Defects:

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



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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

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3: The Heart's Conduction System & ECGs

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

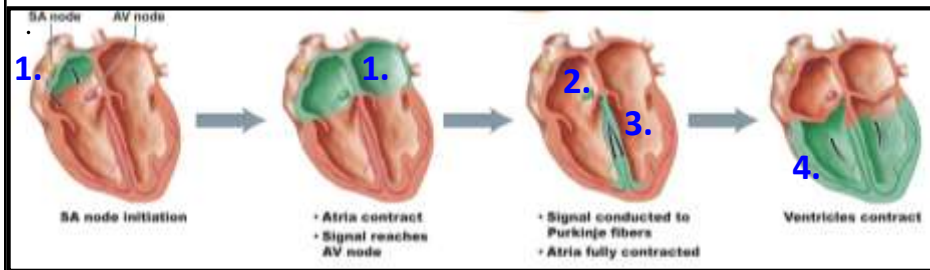
1. SA node =

2. AV node =

3. Bundle of HIS =

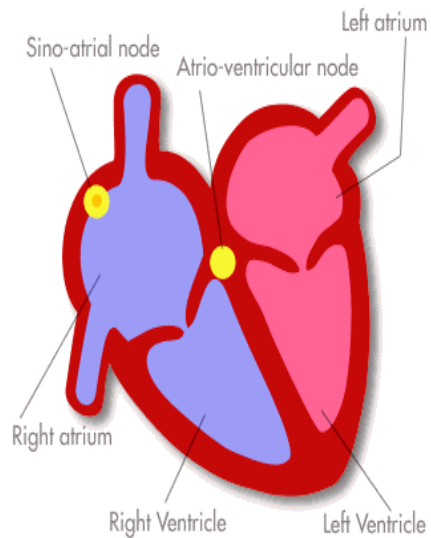
4. Purkinje fibers =

Page 146- 148 in text



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Heart Conduction System



[GIF](#)

[GIF](#)

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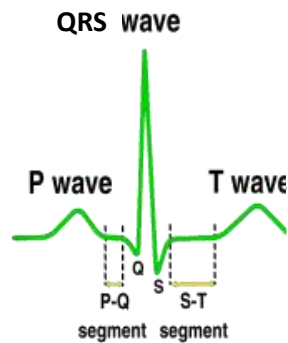
10

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 β_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⁺ channel blockers (*quinidine, lidocaine*)
- > Ca²⁺ channel blockers (*verapamil*)

> Beta blockers

ex. *General beta blocker =*

Review { *B1-specific blocker =*

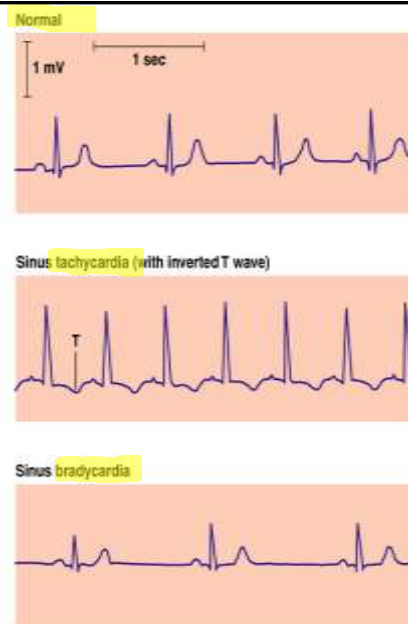
Bradycardia =

Treatments:

- > Digitalis – increases Ca²⁺ 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

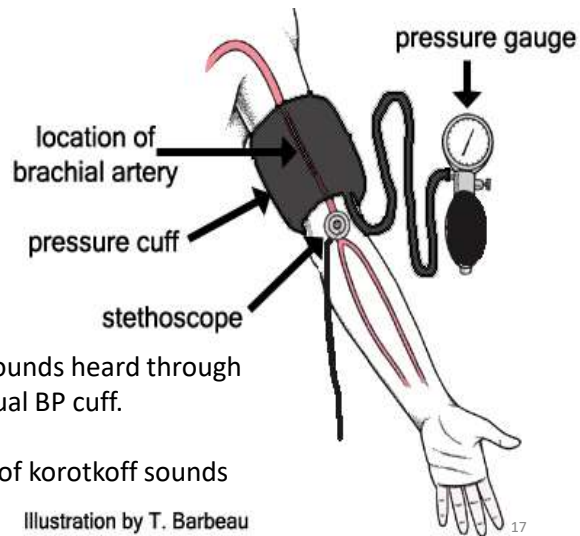
120 mmHg
80

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.

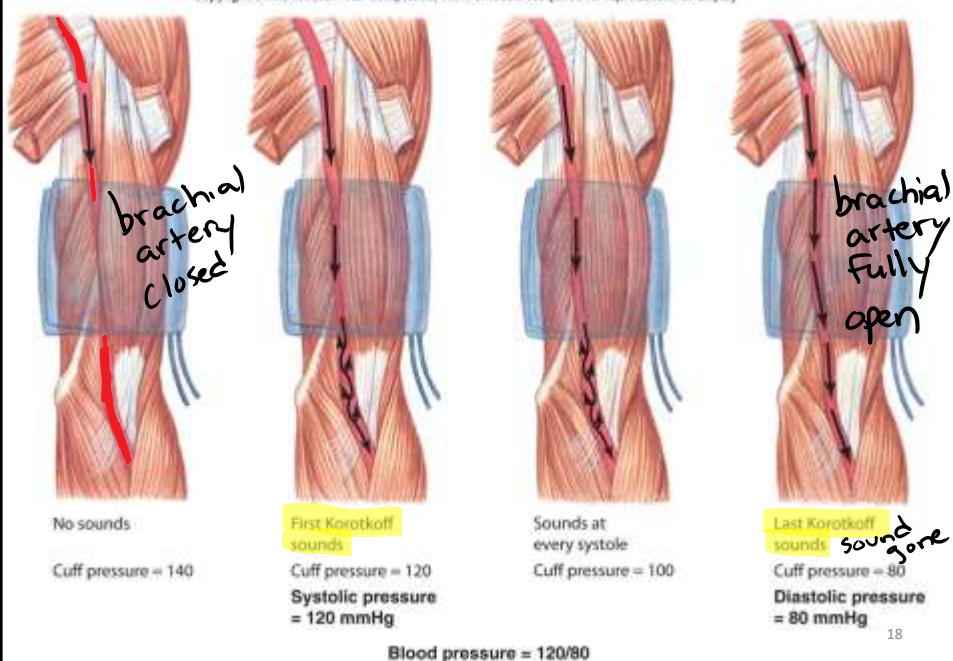
Click [HERE](#) for YouTube video of korotkoff sounds

Illustration by T. Barbeau

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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.



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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?

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Things That Influence Cardiac Output:

2. Stroke Volume:

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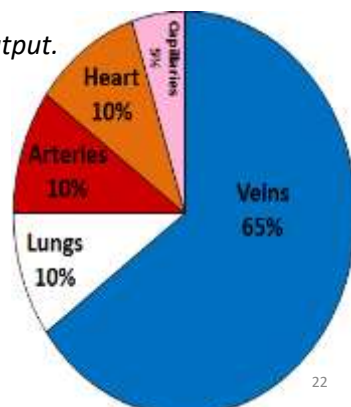
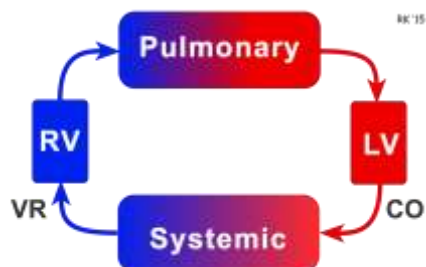
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Things That Influence Cardiac Output:

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 \uparrow , cardiac output \uparrow
 As HR \downarrow , cardiac output \downarrow

As stroke volume \uparrow , cardiac output \uparrow
 As stroke volume \downarrow , cardiac output \downarrow

As EDV \uparrow , cardiac output \uparrow
 As EDV \downarrow , cardiac output \downarrow

\uparrow ventricular stretch, \uparrow heart contractility, so cardiac output \uparrow
 \downarrow ventricular stretch, \downarrow heart contractility, so cardiac output \downarrow

And

As TPR \uparrow , stroke volume and cardiac output \downarrow
 As TPR \downarrow , stroke volume and cardiac output \uparrow

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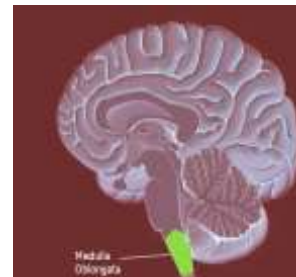
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1. Arterial baroreceptor reflex (the “Quick Fix”)

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

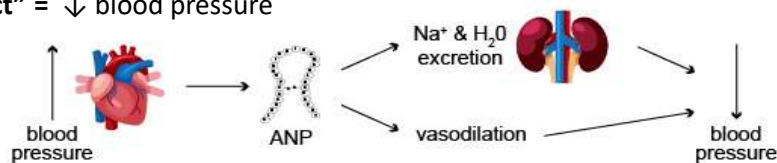
Cause arterial vasodilation

\uparrow filtration rate at kidneys, which

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

\downarrow blood volume

“effect” = \downarrow blood pressure



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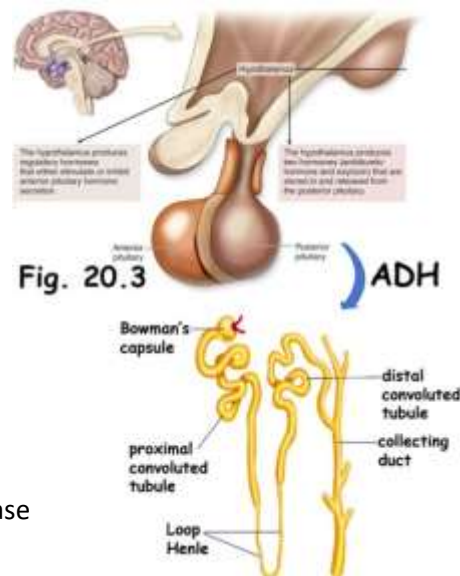
3. Hypothalamus – Blood Osmolarity Center setpoint of 280 – 290 mOsm

stimulus = \uparrow blood osmolarity

(above 290 mOsm)

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

effect of ADH =



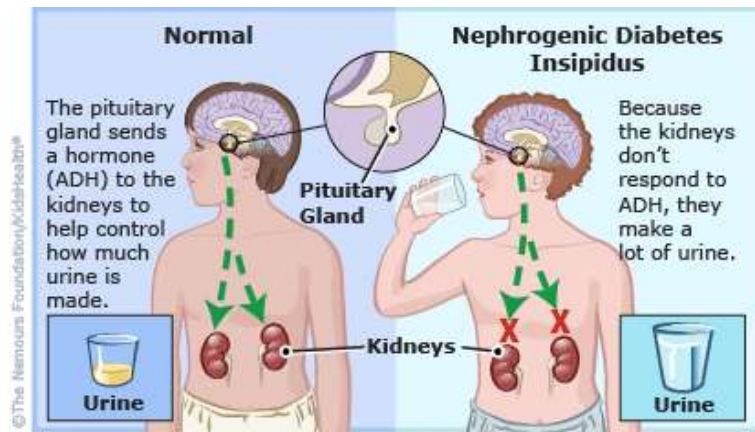
Stimulus = \downarrow blood osmolarity

hypothalamus inhibits ADH release
opposite things happen

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Diabetes insipidus = insufficient ADH release by hypothalamus.

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



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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

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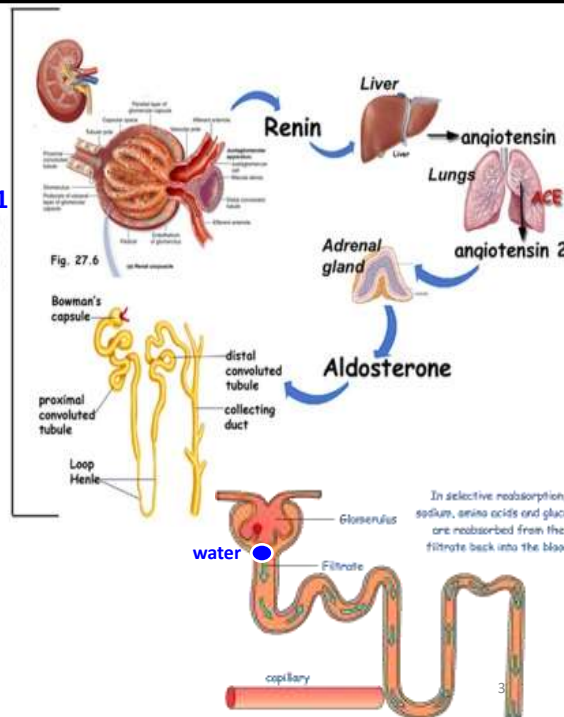
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IF blood volume & BP ↓:

- > 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



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The diagram is identical to the one on slide 35, showing the RAAS pathway and kidney anatomy. An inset image in the bottom left corner shows a bottle of 'ACE INHIBITORS TABLETS' and two red pills. The text 'ACE = angiotensin converting enzyme' is written in red below the diagram. The text 'ACE inhibitors – Clinical App' is written in blue below the text. The diagram is labeled 'Fig. 27.6' and '© 2011 Pearson Education, Inc.'

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

↓ salt (reabsorption by kidneys)

↓ water reabsorption by kidneys

Clinical presentation?

Hyponatremia =

Hyperkalemia =

Bradycardia =

Polyuria =

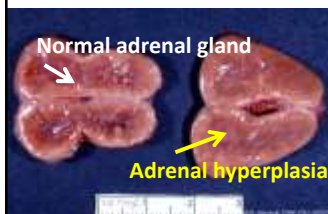
Hypovolemia=

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)

Possible 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. Hypovolemic shock = drop in blood volume and BP (blood loss, dehydration)

body response = ↑ heart rate (compensatory to ↑ BP)
= vasoconstriction (to ↑ BP)

2. Septic shock = drop in blood volume and BP from infection (sepsis). Caused by bacterial toxins in blood. Causes vasodilation & ↓ BP

body response = same as for #1.

3. Anaphylactic 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

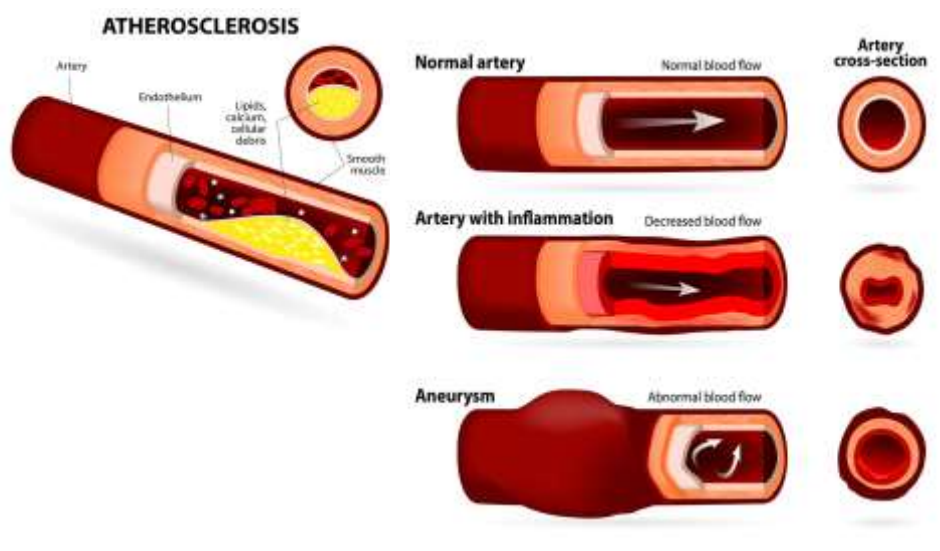
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Cardiac Diseases: Atherosclerosis

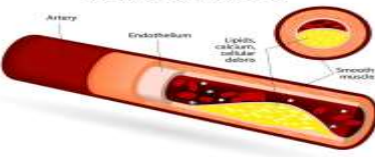
= buildup of cholesterol plaques within arterioles.

Can decrease blood flow and lead to:



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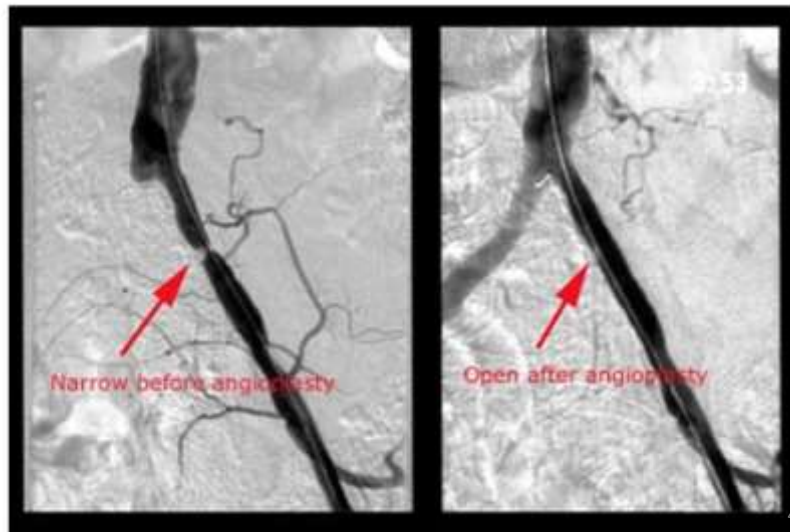
Click [HERE](#) for a brief YouTube diagrammatic explanation of an embolism.

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

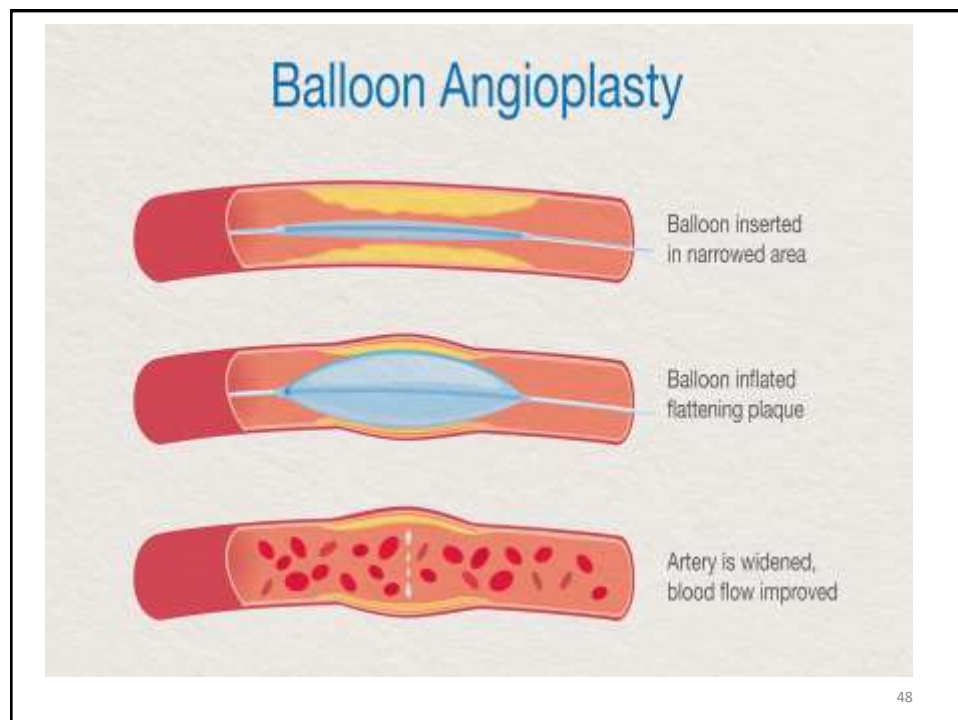
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Angiogram = diagnostic test that uses x-rays to take pictures of your blood vessels. A long flexible catheter is inserted through the blood stream to deliver dye (contrast agent) into the arteries making them visible on the x-ray



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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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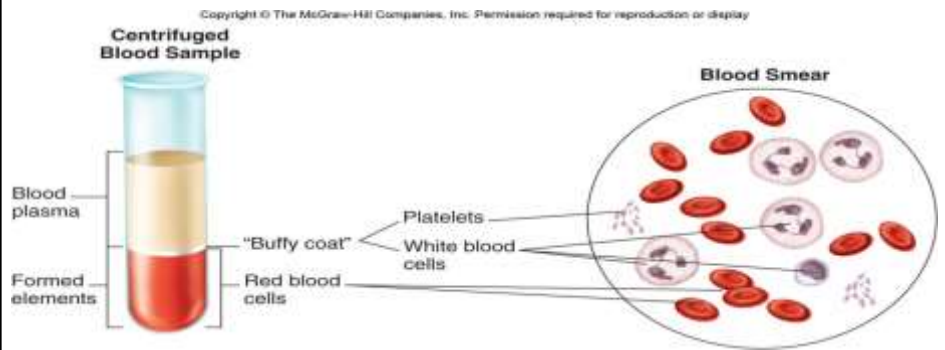
9: Blood Physiology

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.



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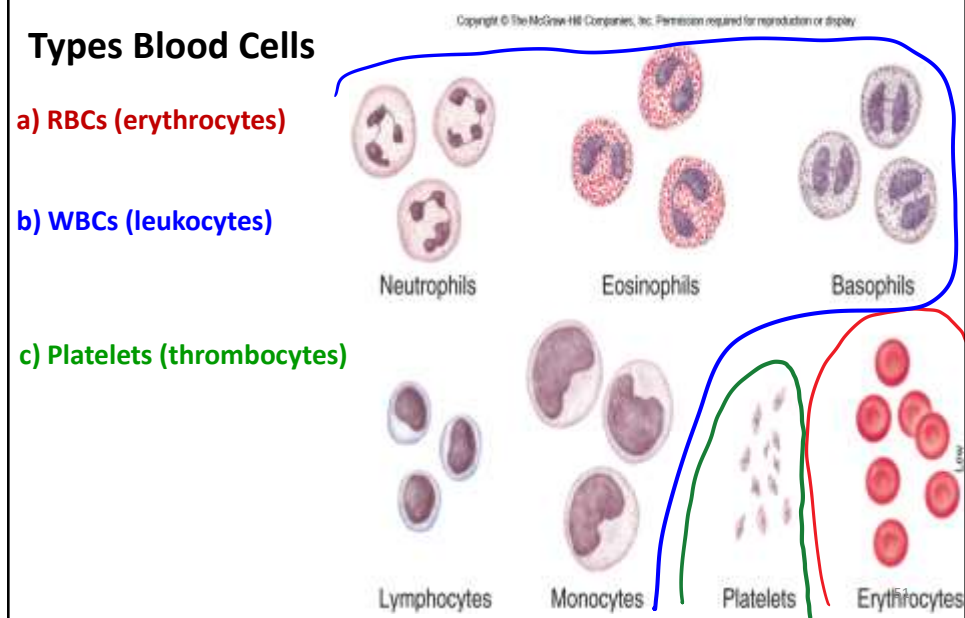
2: Blood Physiology

Types Blood Cells

a) RBCs (erythrocytes)

b) WBCs (leukocytes)

c) Platelets (thrombocytes)



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2: Blood Physiology

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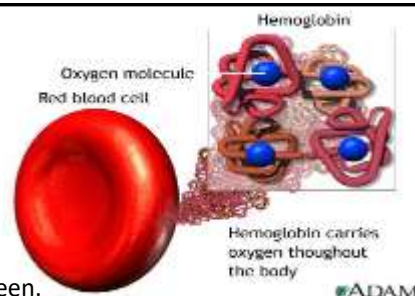
RBCs (Erythrocytes)

- > Carry O₂ 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** = yellowing of the skin & mucus membranes due to liver failure.



- **erythropoiesis** = process by which new RBCs are made (in bone marrow). Stimulated by hormone **erythropoietin** (released by liver & kidneys.)



ADAM

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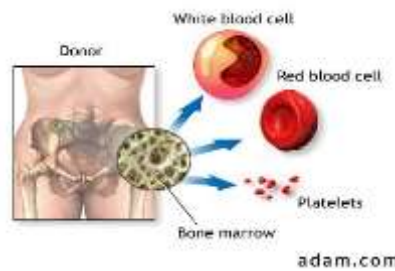
Polycythemia = higher than normal RBC count.

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)



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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)

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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**

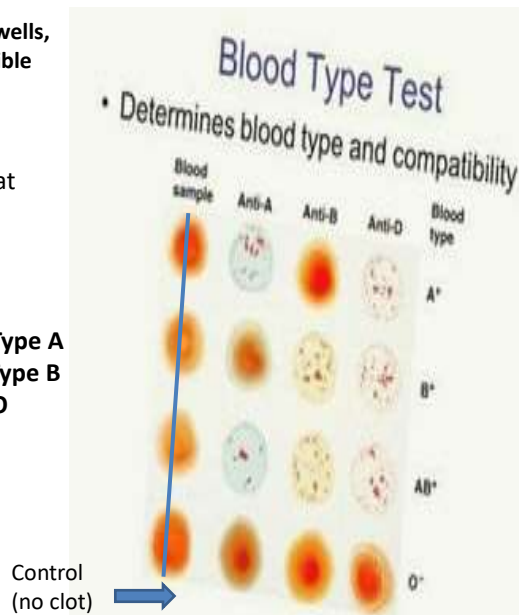
Add anti-B antibodies – if clots = **Type B**

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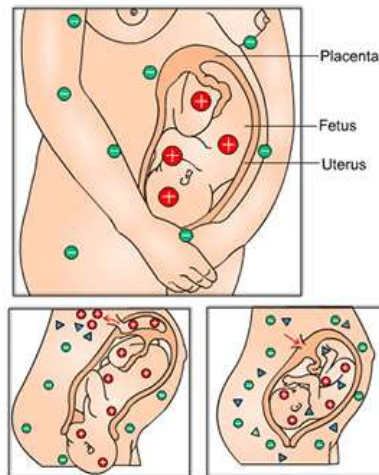
If Rh- woman pregnant from Rh+ man – 50% chance baby is Rh+

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](#)

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Rh incompatibility in pregnancy

Page 129 in text

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% - **Neutrophils** = 1st responders to infection/inflammation.

2-4% - **Eosinophils** = see w/chronic inflammation, infection, allergies, parasites

<1% - **Basophils** = non-phagocytes, produce histamine & heparin in allergic reaction.

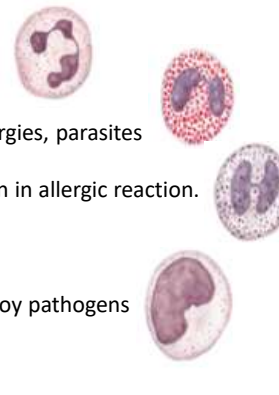
2) **Agranulocytes** = lack granules.

2-8% - **Monocytes** = phagocytes that seek out, engulf, & destroy pathogens

20-30% - **Lymphocytes** = defense from pathogens

> T cells =

> B cells = become plasma cells to produce antibodies.



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

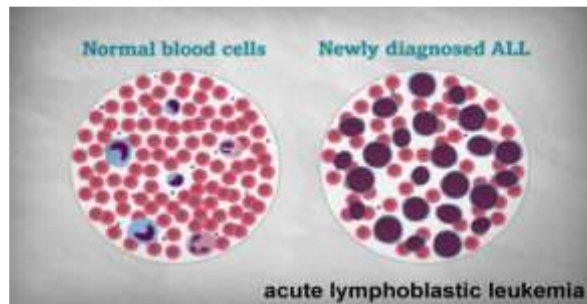
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Leukocyte disorders – Clinical App

> **Leukocytosis** = \uparrow WBC count (infections!)

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



> **Leukopenia** = \downarrow WBC count (with immunosuppression, radiation Tx)

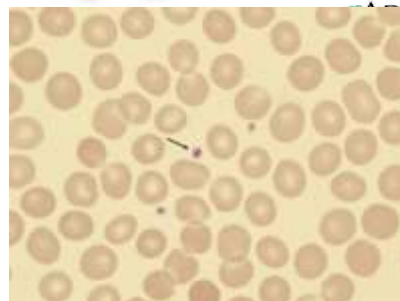
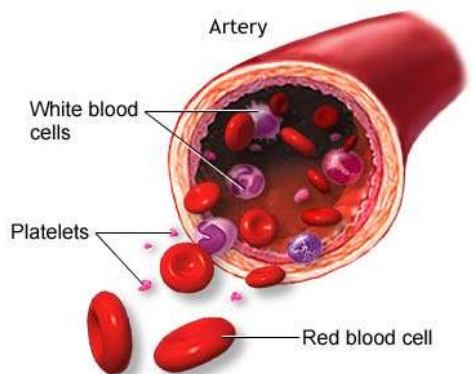
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c) Platelets (Thrombocytes)

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

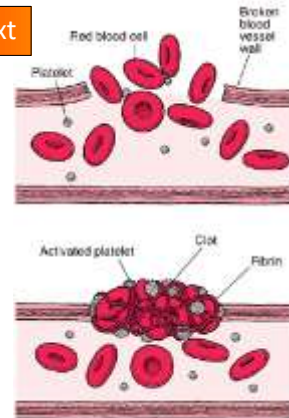


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3: Physiology of Blood Clotting Page 126 -127 in text

"Hemostasis" = stopping bleeding from damaged blood vessel

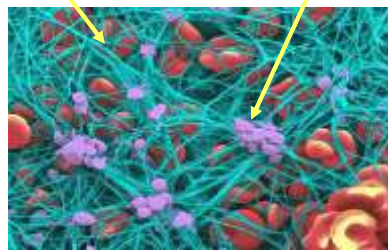
- > A blood vessel is damaged:
- > arterioles constrict
- > Platelets form a "plug"
 - Platelets convert **prothrombin** to **thrombin**
 - **Thrombin** activates **fibrinogen**
 - Fibrinogen converts into **fibrin threads**
 - fibrin threads "knit" the wound closed.



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

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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**

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