Objectives
Ch 8:
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

1. Review of Heart Anatomy and Circulatory System

ANATOMY REVIEW!

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

Other vessels attached to heart:
- Brachiocephalic a.
- L common carotid a.
- L subclavian a.
Circulatory Systems REVIEW!

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.

2: The Cardiac Cycle & Heart Sounds

Ventricular systole = sound of tricuspid & bicuspid valves closing simultaneously.
> Hear “Lub” or S1 sound with atrial valves closing. The ventricles have contracted & pushed blood into the pulmonary trunk and aorta.

Ventricular diastole = sound of the semilunar valves closing simultaneously.
The atria are now contracting & pushing blood into the ventricles.
> Hear “Dub” or S2 sound w/semilunar valves closing.

Asystole = no heart sounds (BAD)

Click HERE for Normal heart sounds

<table>
<thead>
<tr>
<th>HEART SOUNDS</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;LUB&quot;</td>
</tr>
<tr>
<td>&quot;DUB&quot;</td>
</tr>
</tbody>
</table>
Heart Murmurs:

- **Innocent murmur** = quiet whoosh between S1 and S2.
  - Normal in children, pathological if develop later in life.

- **Pathologic murmur** = loud whoosh between S1 and S2
  - could be **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](#)
Heart defects: Clinical App

Septal Defects:

1. Patent foramen ovale = fetal hole between L & R atria.


3. Ventricular septal defect = fetal hole in Interventricular septum.

Review

• The cardiovascular system
• Pulmonary & systemic circuits
• The heart and blood vessels
  – Arteries, arterioles, capillaries, veins, venules
  – The heart
    » The atria and the ventricles
    » The semilunar and atroventricular valves
• Cardiac cycle & heart sounds
• Heart defects
Heart is “autorhythmic” = starts its own signal for contraction.

1. **SA node** = “pacemaker” is where action potential (AP) starts in upper R atrium. AP jumps from right to left atria, and both atria contract simultaneously.

2. **AV node** = AP pauses briefly in lower R atrium, to allow ventricles time to fill with blood.

3. **Bundle of HIS** = AP travels down interventricular septum

4. **Purkinje fibers** = AP is within the ventricular walls, both ventricles contract simultaneously.

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**Heart Conduction System**

- Sino-atrial node
- Atrio-ventricular node
- Right atrium
- Left atrium
- Right Ventricle
- Left Ventricle

---

**GIF**
Electrocardiogram (ECG or EKG) =

P-wave = Atria contract simultaneously, push all blood into ventricles.

QRS wave = Tricuspid & bicuspid valves have shut already, & ventricles contract simultaneously (ventricular systole)

T-wave = Ventricles relax (are filling during diastole) Semilunar valves have shut just before this.

4: The Heart’s Pacemaker & Its Regulation

Regulated by cardiac center in medulla oblongata!

Sympathetic innervation with thoracic & cardiac nerves:

– Neurotransmitter =
– Receptor =
– Effect =

Parasympathetic innervation with vagus nerves:

– Neurotransmitter =
– Receptor =
– Effect =
4: The Heart’s Pacemaker & Its Regulation

**Spontaneous APs start in pacemaker myocardial cells**

1. **Heart pacemaker cell depolarization:**
   - Opening of Na+ & Ca²⁺ channels
   - Causes AP (or EPSP)
   - Myocardial cells contract! (Signal started!)

2. **Heart pacemaker cell repolarization:**
   - Opening of K+ channels
   - Myocardial cells relax!

Epinephrine binding to β₁ adrenergic receptors on pacemaker increases rate of depolarization

ACh binding to muscarinic cholinergic receptors on pacemaker decreases rate of depolarization

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

**Arrhythmias = abnormal heart rate**

*Clinical App*

**Tachycardia** = higher than normal HR

*Treatments:*
- Na+ channel blockers (quinidine, lidocaine)
- Ca²⁺ channel blockers (verapamil)
- Beta blockers
  - General beta blocker = propranolol
  - B1-specific blocker = atenolol

**Bradycardia** = lower than normal HR

*Treatments:*
- Digitalis – increases Ca²⁺ available to increase contractile strength.
- B1-agonist = dobutamine
- MAO-I A = an epinephrine agonist
Review

- Cardiac conduction system:
- Monitoring electrical activity of the heart (EKG)
  - P wave, QRS wave, S-T segment, T wave
- The Heart’s Pacemaker
  - parasympathetic and sympathetic regulation
- Arrhythmias

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

\[
\frac{120}{80} \text{ mmHg}
\]

Page 149 – 150 in text
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
Cardiac Output = Stroke Volume \times \text{Heart Rate}
\begin{align*}
\text{(ml/min)} & \quad \text{(ml/beat)} & \quad \text{(bpm)} \\
\end{align*}

Average HR varies (~60 – 80 bpm)
AVG stroke volume = 70 – 80 ml/beat
AVG cardiac output = 5500 ml/min (5.5L/min)

**Things That Influence Cardiac Output:**

1. **Heart Rate:**
   - as HR ↑, cardiac output ↑
   - as HR ↓, cardiac output ↓

   HR changes with sympathetic or parasympathetic stimulation.

   **Drugs that ↑ cardiac output by ↑ HR**
   - A) Epinephrine (thru \( \beta_1 \) adrenergic receptors).
   - B) Digitalis – increased Ca+2 available, increases heart contractility
   - C) MAO-I A – epinephrine agonist
   - D) Dobutamine – \( \beta_1 \) adrenergic receptor agonist

   **Drugs that ↓ cardiac output by ↓ HR**
   - A) General \( \beta \)-blockers (propranolol)
   - B) specific \( \beta_1 \)-blockers (atenolol)
   - C) Na+ channel blockers
   - D) Ca+2 channel blockers
**Things That Influence Cardiac Output:**

2. **Stroke Volume:**
   - As stroke volume ↑, cardiac output ↑
   - As stroke volume ↓, cardiac output ↓

**Stroke Volume, in turn, regulated by:**

**Heart “contractility”** = strength of heart contraction.
   - As contractility ↑, stroke volume ↑, so cardiac output ↑
   - As contractility ↓, stroke volume ↓, so cardiac output ↓

---

**Things That Influence Cardiac Output:**

REVIEW!

3. **End Diastolic Volume (EDV)** – = volume of blood that fills the ventricles in diastole of heart cycle. Is influenced by venous return to the heart.
   - As venous return (EDV) ↑, cardiac output ↑
   - As venous return (EDV) ↓, cardiac output ↓

Essentially, venous return must equal cardiac output.
Things That Influence Cardiac Output:

4. **Stretching of ventricles (and Contractility)**
   - $\uparrow$ EDV causes greater ventricular filling (ventricles stretch)

**Frank-Starling Law of the Heart**

So...
As EDV $\uparrow$, ventricle stretch $\uparrow$, contractility $\uparrow$, so cardiac output $\uparrow$

As EDV $\downarrow$, ventricle stretch $\downarrow$, contractility $\downarrow$, so cardiac output $\downarrow$

---

5) **Total peripheral resistance (TPR)** = resistance of blood flow in the arteries (depends on blood viscosity, vessel length and vessel diameter).

   - As TPR $\uparrow$ cardiac output $\downarrow$
   - As TPR $\downarrow$ cardiac output $\uparrow$
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 ↑

Review
Heart anatomy

Cardiac cycle and heart sounds (lub dub, or S1 and S2)

Heart defects

Conduction system of heart and EKG

Regulation of pacemaker depolarization

Arrhythmias

Blood pressure

Factors that influence cardiac output.
1. **Baroreceptor reflexes**
   “baroreceptors” = pressure receptors that detect stretching of arteries OR the heart chambers.

2. **Hypothalamus**

3. **Kidney regulation**

---

**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 into urine.

Kidneys affected by sympathetic and parasympathetic control, AND by hormones.
1. Baroreceptor Reflex

A) Artery baroreceptors (the “quick fix”)

“stimulus” = change in blood pressure
“sensor” = aortic arch & carotid artery baroreceptors
Integrating center = medulla cardiac & vasomotor center

A) If stimulus of ↓ BP (below 80mmHg)
Autonomic response is sympathetic
- Neurotransmitter = epinephrine
- Binds to β1 adrenergic receptors on heart. (HR ↑)
- Also have arterial vasoconstriction
- BP ↑

B) If stimulus of ↑ BP (above 160 mmHg)
Autonomic response is parasympathetic
- Neurotransmitter = ACh
- Binds to muscarinic cholinergic receptors on heart. (HR ↓)
- Also have arterial vasodilation
- BP ↓

1. Baroreceptor Reflex

B) Heart baroreceptors (hormonal response & “long fix”)

“stimulus” = ↑ blood pressure (stretch of heart)
“sensor” = heart baroreceptors

“response” = heart secretes ANP
ANP = atrial natriuretic peptide.
Functions to
↑ filtration rate at kidneys, which
↓ water reabsorption & ↑ urine output (pee more)
↓ blood volume

“effect” = ↓ blood pressure
2. Hypothalamus – Blood Osmolarity Center setpoint of 280 – 290 mOsm

A) Stimulus = ↑ blood osmolarity (above 290 mOsm)

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

effect of ADH =
↑ water reabsorption from kidneys
↓ urine output (pee less)
↑ blood volume (which will ↑ BP)
↓ blood osmolarity

B) Stimulus = ↓ blood osmolarity
hypothalamus inhibits ADH release
opposite things happen

Diabetes insipidus = insufficient ADH release by hypothalamus.

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

“stimulus” = low BP at renal artery of kidneys

“sensor, integration center, & effector” = kidney juxtaglomerular apparatus (JGA)

“motor response” = Renin release by juxtaglomerular apparatus

“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
Addison’s Disease = low aldosterone production by adrenal cortex.

↓ salt (reabsorption by kidneys)

↓ water reabsorption by kidneys

Clinical presentation?

Hyponatremia = low blood Na+

Hyperkalemia = high blood K+

Bradycardia = low heart rate

Polyuria = high urine output (peeing out body water)

Hypovolemia = low blood volume

Hypotension = low blood pressure

Skin bronzing

Conn’s Syndrome = “hyperaldosteronism” or excess aldosterone

Clinical presentation?

Hypernatremia = high blood Na+

Hypokalemia = low blood K+

Oliguria = low urine output (retain body water)

Hypervolemia = high blood volume

Hypertension = high blood pressure
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.

---

**Review**

- **Cardiac output** = stroke volume X cardiac rate
  - Is influenced by EDV, ventricular stretch, heart contractility, stroke volume, heart rate, and TPR.
- **Heart rate regulated by**
  - sympathetic & parasympathetic systems
- **Blood volume & blood pressure regulated by**
  - Heart baroreceptor response (ANP)
  - Medulla baroreceptor response
    - Low BP causes sympathetic arteriole vasoconstriction and ↑ in HR & BP
    - High BP causes parasympathetic arteriole vasodilation and ↓ in HR & BP
  - Hypothalamus (ADH release)
  - Kidney (renin-angiotensin-aldosterone system)
8: Abnormal Blood Pressure

**Hypotension** = low BP

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

### Table 14.8 | Blood Pressure Classification in Adults

<table>
<thead>
<tr>
<th>Blood Pressure Classification</th>
<th>Systolic Blood Pressure</th>
<th>Diastolic Blood Pressure</th>
<th>Drug Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Under 120 mmHg and</td>
<td>Under 80 mmHg</td>
<td>No drug therapy</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139 mmHg or 80-89 mmHg</td>
<td>Lifestyle modification; no antihypertensive drug indicated</td>
<td></td>
</tr>
<tr>
<td>Stage 1 Hypertension</td>
<td>140-159 mmHg or 90-99 mmHg</td>
<td>Lifestyle modification; antihypertensive drugs</td>
<td></td>
</tr>
<tr>
<td>Stage 2 Hypertension</td>
<td>160 mmHg or greater or 100 mmHg or greater</td>
<td>Lifestyle modification; antihypertensive drugs</td>
<td></td>
</tr>
</tbody>
</table>

*Note: Lifestyle modifications include weight reduction; reduction in dietary fat and increased consumption of vegetables and fruit; reduction in dietary sodium (salt); engaging in regular aerobic exercise, such as brisk walking for at least 30 minutes a day, most days of the week; and moderation of alcohol consumption.


---

2 types of Hypertension:

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

**Pausible causes of 2nd Hypertension**

- **Hypervolemia** = high blood volume.
  - 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.
Circulatory Shock = inadequate blood flow to all body tissues

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
Cardiac Diseases: Atherosclerosis

= buildup of cholesterol plaques within arterioles. Can decrease blood flow and lead to:

- **Formation of thrombus** = blood clot from chronic inflammation.

- **Embolism** = clot breaks free & floats in bloodstream.

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

- **Arteriosclerosis** = hardening in arteries due to chronic inflammation & scar tissue.

- **Aneurysm** = swelling of artery due to weakened arterial walls (chronic inflammation). Can lead to rupture artery.

Click HERE for a brief YouTube diagramatic explanation of an embolism.

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

9: Blood Physiology

Blood volume

Majority of total body water (67%) is intracellular fluid (ICF)

33% of total body water is plasma & interstitial fluid.

ECF 33%

Interstitial fluid (in between cells)

Plasma

ICF 67%

Interstitial fluid

Plasma
# Body Water balance

<table>
<thead>
<tr>
<th>Water Intake (+)</th>
<th>Water Loss (-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food &amp; drink – 1.5 -2.5 L in 24hr</td>
<td>Kidney filtration (urination) – 0.5 – 1.5 L</td>
</tr>
<tr>
<td><strong>Question: What hormone can</strong></td>
<td><strong>Skin (sweating) – 0.2 – 1 L</strong></td>
</tr>
<tr>
<td>A) Increase water retention by increasing salt retention, to increase blood volume &amp; BP?</td>
<td><strong>Lungs (exhalation of water vapor) – 0.4 L</strong></td>
</tr>
<tr>
<td><strong>aldosterone</strong></td>
<td><strong>Feces – 0.2 L</strong></td>
</tr>
<tr>
<td>B) Decrease water retention by increasing kidney filtration rate, so you pee out body water?</td>
<td></td>
</tr>
<tr>
<td><strong>ANP</strong></td>
<td></td>
</tr>
<tr>
<td>A) Increase water retention so that blood osmolarity can be reduced.</td>
<td></td>
</tr>
<tr>
<td><strong>ADH</strong></td>
<td></td>
</tr>
</tbody>
</table>

---

# Review

- **Blood volume distribution**
- **Total body water distribution**
  - 66% ICF + 33% ECF
- **Water balance regulation**
  - Input
  - Output
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.

---

**Calculate blood volume for a 70 kg man:**
What is his blood volume range?

______________________ml or ____________________ L

---

**TABLE 10.1 Some Normal Blood Values**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood volume</td>
<td>80–85 ml/kg body weight</td>
</tr>
<tr>
<td>Blood osmolarity</td>
<td>285–295 mOsM</td>
</tr>
<tr>
<td>Blood pH</td>
<td>7.38–7.44</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>Female: 36%–46%; Male: 41%–53%</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>Female: 13–16 g/100 ml; Male: 13.5–17.5 g/100 ml</td>
</tr>
<tr>
<td>Red blood cell count</td>
<td>4.50–5.90 million/mm³</td>
</tr>
<tr>
<td>White blood cell count</td>
<td>4.500–11,000/mm³</td>
</tr>
</tbody>
</table>
Types Blood Cells

a) RBCs (erythrocytes)

b) WBCs (leukocytes)

c) Platelets (thrombocytes)

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.)
**Erythropoiesis** stimulus is low blood O2.

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/radiation treatment).

- **Renal anemia** = low erythropoietin production by kidneys.

- **Autoimmune hemolytic anemia** = immune attack on RBCs (see with Rh disease)

**Clinical App**
2 Major RBC antigens:
1) ABO antigens
2) Rh antigen

1) ABO
Blood Type A – have ______ A ______ antigens & ___________ anti-B ________ antibodies
– receives type ___ A or O ______ blood

Blood Type B – has ______ B ______ antigens & ___________ anti-A ________ antibodies
– receives type ___ B or O ______ blood

Blood Type AB – has ______ A & B ______ antigens & ______ No ________ antibodies
– “universal recipient”, can receive ______ A, B, AB, or O ______ blood

Blood Type O – has ______ No ______ antigens, & ______ Anti-A and anti-B ________ antibodies
– “universal donor”, but can receive only type O blood

2) Rh factor
Rh+ = have Rh antigen on RBCs    Rh- = not have Rh antigen
(~85% of population)    (~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
Add anti-Rh (D) antibodies
– if clots = Rh+
– no clot = Rh-

Click HERE for YouTube video of me running a blood typing test.
Rh incompatibility in pregnancy

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

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.
b) WBCs (Leukocytes)  

**2 Groups:**

1) **Granulocytes** = WBCs with granules in cytoplasm
   - **Neutrophils** = 1st responders to infection/inflammation.  
     - 50-70%
   - **Eosinophils** = see w/chronic inflammation, infection, allergies, parasites
     - 2-4%
   - **Basophils** = non-phagocytes, produce histamine & heparin in allergic reaction.
     - <1%

2) **Agranulocytes** = lack granules.
   - **Monocytes** = phagocytes that seek out, engulf, & destroy pathogens
     - 2-8%
   - **Lymphocytes** = defense from pathogens
     - 20-30%
     - > **T cells** =
     - > **B cells** = become plasma cells to produce antibodies.

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

---

**Leukocyte disorders – Clinical App**

> **Leukocytosis** = ↑ WBC count (infections!)

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

> **Leukopenia** = ↓ WBC count (with immunosuppression, radiation Tx)
c) Platelets (Thrombocytes)

Page 126 in text

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

Hemostasis = stopping bleeding from damaged blood vessel
- A blood vessel is damaged:
  - arterioles contract
  - 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)

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.

> Green top = heparin anticoagulant. For blood chemistry panels.

Microhematocrit tube = used for quick measure of %RBCs in blood. Can be used to tell if patient dehydrated or has blood loss.

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