## Functions of Blood

1. **Transportation of dissolved gases, nutrients, hormones and metabolic wastes** (carries O₂ & CO₂, nutrients from digestion, etc.)

2. **Regulation of pH & electrolyte composition of interstitial fluids throughout the body** (absorbs & neutralizes acids)

3. **Restriction of fluid losses through damaged vessels** (blood clotting mechanism)

4. **Defense against toxins/pathogens** (antibodies/immune system)

5. **Stabilization of body temp.** (blood absorbs heat generated by skeletal muscles)

## Composition of Blood

**Blood is a fluid connective tissue**

The extracellular matrix of blood is called **Plasma**

- Plasma is made of plasma proteins and a ground substance called **serum**

- The plasma proteins are in solution so the plasma is more dense than water

**Formed elements are suspended in the plasma** 

These consist of:

- a. Blood cells
- b. Cell fragments

## There are two basic blood cell classes...

1. **RBC’s (erythrocytes)**

   - Most abundant blood cells

   - Essential for O₂ transport

2. **WBC’s (leukocytes)**

   - Involved with body’s immune system

   - 5 types
     
     - Neutrophils: 50-70%
     - Eosinophils: 2-4%
     - Basophils: < 1%
     - Lymphocytes: 10-30%
     - Monocytes: 2-8%
**Blood Collection & Analysis**

• Fresh whole blood is usually collected from a superficial vein (*median cubital v.*) in a process called **Venipuncture**

  **This is a common sampling technique because:**

  1. superficial veins are easy to locate
  2. walls of veins are thinner than arteries
  3. BP is relatively low in veins, so puncture wounds seal quickly

**Capillary puncture:** finger tip, ear lobe, big toe, heel produces a drop of blood

**Arterial puncture:** or “arterial stick” generally drawn from radial artery (wrist) or brachial artery (elbow)

**So why use different techniques**

• **Capillary puncture** used for a **blood smear**

• **Venipuncture** used for most common **clinical blood test**

• **Arterial puncture** used for testing **blood gases/efficiency of gas exchange**

**All blood drawn shares the following characteristics**

• Temp. ≈ 38° / 100.4°F

• 5x more viscous than H2O (stickier & resistant to flow)

• pH between 7.35 – 7.45 (avg. 7.4)
• Adult ♂ 5-6 L of whole blood

• Adult ♀ 4-5 L of whole blood

• Estimate blood volume 7% of body weight

• Hypovolemic, normovolemic, hypervolemic

<table>
<thead>
<tr>
<th>Plasma</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. 46-83% of volume of whole blood (water is 92% of plasma volume)</td>
</tr>
<tr>
<td>b. on avg. 7.6g of protein in 100 ml of plasma</td>
</tr>
</tbody>
</table>

3 classes of plasma proteins

1) albumins (≈ 60%) important transport proteins

2) globulins (≈ 35%) immunoglobulins are antibodies, transport very small molecules.

3) fibrinogen (≈ 4%) responsible for clotting

*other plasma proteins (≈ 1%)

Insulin, prolactin, thyroid stimulating hormone, Follicle stimulating hormone (FSH), luteinizing hormone

(ALL NORMALLY PRESENT IN THE BLOOD)

<table>
<thead>
<tr>
<th>Red Blood Cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Contain the iron bearing compound hemoglobin</td>
</tr>
<tr>
<td>• Transport O₂ &amp; CO₂</td>
</tr>
<tr>
<td>• Most Abundant blood cells</td>
</tr>
<tr>
<td>• Std. blood tests report # of RBC’s/μl of whole blood</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Formed Elements</th>
</tr>
</thead>
<tbody>
<tr>
<td>• The formed elements are produced in a process called hemopoiesis.</td>
</tr>
<tr>
<td>• In adults, the only site of red blood cell production, and the primary site of white blood cell formation, is the red bone marrow.</td>
</tr>
</tbody>
</table>

♂ 4.5 – 6.3 million
♀ 4.2 – 5.5 million

≥260 million/drop of whole blood and 25 trillion (10¹²) in avg. adult

Hematocrit: the % of whole blood occupied by cellular elements
Factors that can alter the hematocrit
1. ↑ during dehydration (because of ↓ in plasma)
2. ↑ after erythroprotein stimulation (hormonal byproduct)
3. ↓ due to internal bleeding
4. ↓ due to problems in RBC formation mechanism

Structure of RBC’s
1. RBC’s are bi-concave discs this shape helps RBC function in 3 ways
   1. Gives a relatively large SA that aids in gas exchange functions
   2. Can form packed stack= called rouleaux (rue-LOW) which move efficiently through small diameter capillaries
   3. This thin in the middle design allows the RBC’s to be very flexible and bend through tight spots as small as 4 µm wide

- Normal ♂ range (40-54) [46]
- Normal ♀ range (27–47)[42]
- Androgens (♂ hormones) stimulate RBC production
- Estrogens (♀ hormones) do not

• Normal ♂ range (40-54) [46]
Normal ♀ range (27–47)[42]

• Androgens (♂ hormones) stimulate RBC production

• Estrogens (♀ hormones) do not

Factors that can alter the hematocrit
1. ↑ during dehydration (because of ↓ in plasma)
2. ↑ after erythroprotein stimulation (hormonal byproduct)
3. ↓ due to internal bleeding
4. ↓ due to problems in RBC formation mechanism

Structure of RBC’s
1. RBC’s are bi-concave discs this shape helps RBC function in 3 ways
   1. Gives a relatively large SA that aids in gas exchange functions
   2. Can form packed stack= called rouleaux (rue-LOW) which move efficiently through small diameter capillaries
   3. This thin in the middle design allows the RBC’s to be very flexible and bend through tight spots as small as 4 µm wide

- Normal ♂ range (40-54) [46]
Normal ♀ range (27–47)[42]

- Androgens (♂ hormones) stimulate RBC production

- Estrogens (♀ hormones) do not
• RBC’s in humans have no nuclei (one way to tell animal blood from human blood)

• Most of a RBC (95%) is composed of molecules of hemoglobin (proteins associated with it’s 1st function)

• Hb (hemoglobin shorthand) content is reported in terms of grams of Hb/100 ml of whole blood (g/dl) normal ranges
  ♂ 14-18 g/dl
  ♀ 12-16 g/dl

• Each Hb molecule has a complex 4th structure two α chains & two β chains

• Each chain contains a heme molecule which is a porphyrin (metal assoc. organic molecule) which contains an iron molecule

• O₂ molecules bind to the Fe in the heme to form a compound called oxyhemoglobin

≈ 280 million Hb molecules/RBC and since each Hb can carry 4 Oxygen molecules, each RBC can potentially carry 1.1 billion oxygen molecules

If hematocrit is low, Hb conc. is low so a condition called anemia exists

• Interferes with O₂ delivery

• Affected individuals become
  a. lethargic
  b. weak
  c. confused (brain is effected)

• The round trip of 1 RBC from heart – peripheral tissues, back to the heart takes ≈1 minute

• So... RBC’s travel ≈700 miles in 120 days an wear out

≈ 3 million new erythrocytes enter the circulation each second

• Old heme units are stripped of Fe and converted to a green compound called biliverden. The biliverden is then converted to a orange/yellow compound called biliruben.

• Biliverden and Biliruben are collected by the liver and stored in the gallbladder until released into the digestive system

• If bile ducts are blocked → JAUNDICE
Blood Type

• Attached to the surface of RBC’s are proteins called antigens

• Of course these antigens are under genetic control. One particular gene will produce either an A form antigen, a B form antigen, or no antigen at all

• People are classified in ABO blood groups corresponding to these antigens

<table>
<thead>
<tr>
<th>Allelic Allocations</th>
<th>Phenotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genotype</td>
<td></td>
</tr>
<tr>
<td>Homozygous AA</td>
<td>A type blood</td>
</tr>
<tr>
<td>Heterozygous AO</td>
<td>A type blood</td>
</tr>
<tr>
<td>Homozygous BB</td>
<td>B type blood</td>
</tr>
<tr>
<td>Heterozygous BO</td>
<td>B type blood</td>
</tr>
<tr>
<td>Heterozygous AB</td>
<td>AB type blood</td>
</tr>
<tr>
<td>Homozygous OO</td>
<td>O type blood</td>
</tr>
</tbody>
</table>

A group has A antigens only
B group has B antigens only
AB group has both A & B antigens
O group has neither A or B antigens

So...A & B are codominant
In addition:
• A blood contains anti-B antibodies
• B blood contains anti-A antibodies
• AB blood contain no antibodies
• O contain both anti-A & anti-B antibodies
Transfusions

Type A blood can give to A, AB; can get from A, O

Type B blood can give to B, AB; can get from B, O

Type AB blood can give to AB; can get from A, B, AB, O

*universal recipient

Type O can give to A, B, AB, O; can get from O

*universal donor

---

Agglutination

---

**TABLE 19-2** Differences in Blood Group Distribution

<table>
<thead>
<tr>
<th>Population</th>
<th>Percentage with Each Blood Type</th>
<th>O</th>
<th>A</th>
<th>B</th>
<th>AB</th>
<th>Rh*</th>
</tr>
</thead>
<tbody>
<tr>
<td>U.S. (average)</td>
<td></td>
<td>46</td>
<td>40</td>
<td>10</td>
<td>4</td>
<td>85</td>
</tr>
<tr>
<td>Caucasian</td>
<td></td>
<td>45</td>
<td>40</td>
<td>11</td>
<td>4</td>
<td>85</td>
</tr>
<tr>
<td>African American</td>
<td></td>
<td>49</td>
<td>27</td>
<td>20</td>
<td>4</td>
<td>95</td>
</tr>
<tr>
<td>Chinese American</td>
<td></td>
<td>42</td>
<td>27</td>
<td>25</td>
<td>6</td>
<td>100</td>
</tr>
<tr>
<td>Japanese American</td>
<td></td>
<td>31</td>
<td>39</td>
<td>21</td>
<td>10</td>
<td>100</td>
</tr>
<tr>
<td>Korean American</td>
<td></td>
<td>32</td>
<td>28</td>
<td>30</td>
<td>10</td>
<td>100</td>
</tr>
<tr>
<td>Filipino American</td>
<td></td>
<td>44</td>
<td>22</td>
<td>29</td>
<td>6</td>
<td>100</td>
</tr>
<tr>
<td>Hawaiian</td>
<td></td>
<td>46</td>
<td>46</td>
<td>5</td>
<td>3</td>
<td>100</td>
</tr>
<tr>
<td>Native North American</td>
<td></td>
<td>79</td>
<td>16</td>
<td>4</td>
<td>&lt;1</td>
<td>100</td>
</tr>
<tr>
<td>Native South American</td>
<td></td>
<td>100</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>100</td>
</tr>
<tr>
<td>Australian Aborigines</td>
<td></td>
<td>44</td>
<td>56</td>
<td>0</td>
<td>0</td>
<td>100</td>
</tr>
</tbody>
</table>

Blood Type (cont.)

• Another surface antigen of importance is the Rhesus factor (originally found in Rhesus monkeys) or Rh factor

  - people that are Rh positive have the surface antigen

  - people who are Rh negative do not have the surface antigen

  • A+, O-, etc.
Rh+ carry no anti-Rh antibodies

Rh- normally carry no anti-Rh antibodies, but they can become sensitized if they are exposed to Rh+ blood (will develop anti-Rh antibodies)

Within 6 months of delivery 20% of Rh- mothers who carried Rh+ babies will have become sensitized to Rh+ and make anti-Rh antibodies

Subsequent Rh+ children will be in danger of having the anti-Rh antibodies cross the placenta and destroy the child’s RBCs

This puts a demand on the fetal system for RBCs so they leave the marrow, not fully formed and only slightly able to carry O2

This shows up most often during pregnancy

Rh+ mother can carry an Rh+ child w/no problems
Rh+ mother can carry an Rh- child w/no problems
Rh- mother can carry an Rh- child w/no problems
Rh- mother can carry the first Rh+ child w/no problems, but all subsequent Rh+ fetus’s are in danger

Thus these children have a very dangerous anemic condition called erythroblastis fetalis

These babies often die just before or just after birth

Modern Treatments Include

1. administration of RhoGam to mother to prevent production of anti-Rh antibodies
2. transfusion of neonate’s blood
3. transfusion of fetal blood, in utero
### White Blood Cells
- **aka leukocytes**
- They have nuclei and organelles, but lack hemoglobin.
- They defend the body against invasion by pathogens and remove toxins, wastes, and damaged cells.

Traditionally WBC's are divided into two groups by their staining characteristics:

#### Granulocytes (stain appears to have large granules)
- **a. neutrophils**
- **b. eosinophils**
- **c. basophils**

#### Agranulocytes (stain appears to have no granules)
- **d. monocytes**
- **e. lymphocytes**

Typically 1μl of blood contains 6,000-9,000 leukocytes.

At any one moment most leukocytes are in connective tissue and lymphatic organs.

Leukocytes can detect the chemical signs that accompany damage to tissue. When they do, they leave circulation, and lodge in the affected area.

Circulating Leukocytes share the following characteristics:

1. They are capable of amoeboid movement.
2. They can migrate out of the blood stream (slip between vascular endothelial cells [diapedesis]).
3. They are attracted to specific chemical stimuli (positive chemotaxis).
4. Neutrophils, Eosinophils, and Monocytes are capable of phagocytosis.
→ monocytes leave blood, convert to a cell type called macrophages and then become phagocytic

→ neutrophils & eosinophils are called microphages

**General Functions**

- Activated by a variety of stimuli
- All elicit the same responses
- Neutrophils, eosinophils, basophils and monocytes are involved

**Specific immunity**

- Mount an attack against a specific pathogen
- Lymphocytes are involved

**Platelets**

- *Megakaryocytes* in the bone marrow release packets of cytoplasm (platlets) into the circulating blood
- (1) transport chemicals important to clotting process; (2) forming a temporary patch in the wall of damaged blood vessels; and (3) contracting after clot has formed in order to reduce the size of the break in the vessel wall

**Hemostasis**

- The process of hemostasis prevents the loss of blood through the walls of damaged blood vessels
- The vascular phase is a period of local vasoconstriction resulting from vascular spasms at the injury site
- The platelet phase follows as platelets are activated, aggregate at the site, and adhere to the damaged surface
The coagulation phase occur as factors released by platelets and endothelial cells interact with clotting factors to form a blood clot. In this reaction sequence, suspended fibrinogen is converted to large insoluble fibers of fibrin.

During clot retraction, platelets contract and pull the torn edges of damaged vessels closer together.

During fibrinolysis, the clot gradually dissolves through the action of plasmin, the activated form of circulating plasminogen.

Additional clotting notes:
- Vitamin K is needed by the liver for the production of clotting factors.
- Aspirin reduces the formation of clots because it inhibits platelet aggregation.
- Intrinsic clots can be more complex than extrinsic pathways.
- An internal blood clot is called a thrombus. If the thrombus becomes dislodged it begins to flow along with the blood. It is now called an embolus. The result may be disastrous as in a pulmonary or cerebral embolism.

Manipulating Hemostasis
- Clotting may be prevented by administering drugs that depress the clotting response or dissolve existing clots.
- Important anti-coagulant drugs include heparin, coumadin, dicumarol, t-PA, streptokinase, urokinase, and aspirin.