Physiology and Anatomy of Blood

Prepared by

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Disclosure

- The material and the illustrations are adopted from the textbook “Human Anatomy and Physiology / Ninth edition/ Eliane N. Marieb 2013”
Blood

Artery

White blood cells

Platelets

Red blood cells

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Functions of Blood

- Deliver $O_2$
- Remove metabolic wastes
- Maintain temperature, pH, and fluid volume
- Protection from blood loss - platelets
- Prevent infection - antibodies and WBC
- Transport hormones
Blood Composition

- Hematocrit: % of erythrocytes volume to the total blood volume.
- Normal hematocrit values in healthy males is 47% ± 5% and in females it is 42% ± 5%
- Normal pH between 7.35 and 7.45
Approximately 8% of body weight
Average volume in healthy adult males is 5-6 L, but 4-5 L in healthy adult females
Blood Plasma Components (55%)

- 90% Water
- Proteins 8% w/v
  - Albumin (60%):
    - Produced by the liver
    - Maintain osmotic pressure
    - Transport hormones and enzymes
Blood Plasma Components (55%)

- **Proteins 8% w/v**
  - **Globulins (36%)** :
    - Alpha and Beta Globulins: produced by the liver, transport lipids, metals and fat soluble vitamins
    - Gamma Globulins: Antibodies released by plasma cells in response to immune response
  - **Fibrinogens (4%)**:
    - Produced by the liver, form fibrin fibers of blood clots
Blood Plasma Components (55%)

- Gas
- Electrolytes:
  - Na+, K+, Ca2+, Mg2+, Cl-, SO4-, HCO3
  - Maintain plasma osmotic pressure and pH
Blood Plasma Components (55%)

- **Organic Nutrients**
  - Carbohydrates
  - Amino Acids
  - Lipids
  - Vitamins

- **Hormones**: Steroid and thyroid hormones are carried by plasma proteins

- **Metabolic waste**
  - CO2, urea, uric acid, creatinin, ammonium salts
Buffy Coat < 1%

- Platelets
- Leukocytes
Formed Elements of the Blood - 45%

- Erythrocytes (red blood cells)
- Leukocytes (white blood cells)
- Platelets (thrombocytes)
Erythrocytes
Erythrocytes

- Diameter: 7.5 μm
- Morphology: biconcave discs—flattened discs with depressed centers
- Anucleate and have essentially no organelles
- Contain 97% hemoglobin
- Contain antioxidant enzymes that rid the body of harmful oxygen radicals
- Function: transport respiratory gases

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Erythrocytes

- Hemoglobin- quaternary structure, 2 $\alpha$ chains and 2 $\beta$ chains
- Lack mitochondria, because they generate ATP by anaerobic mechanisms, therefore they do not consume any of the oxygen they carry
- An RBC contains 280 million hemoglobin molecules
- Life span 100-120 days and then are destroyed in spleen (RBC graveyard)
Erythrocytes count

- Men: 4.7–6.1 million cells/ microliter
- Women: 4.2- 5.4 million cells/ microliter
- Erythrocytes are the major factor contributing to blood viscosity.
- When the number of Erythrocytes increases beyond the normal range, blood becomes more viscous and flows more slowly.
- When the number of Erythrocytes drops below the lower end of the range, the blood thins and flows more rapidly.
Hemoglobin

(a) Hemoglobin consists of globin (two alpha and two beta polypeptide chains) and four heme groups.

(b) Iron-containing heme pigment.
Hemoglobin

• Normal values for hemoglobin are 13–18 g/100 ml in adult males, and 12–16 g/100 ml in adult females

• Hemoglobin is made up of the red heme pigment bound to the protein globin.

• Globin consists of four polypeptide chains: two alpha (α) and two beta (β)—each binding a ring-like heme

• Each heme group bears an atom of iron in its center
Hemoglobin

• A hemoglobin molecule can transport four molecules of oxygen because each iron atom can combine reversibly with one molecule of oxygen

• A single RBC contains about 250 million hemoglobin molecules, so each RBC can carry about 1 billion molecules of oxygen
Why Hemoglobin does not exist free in the plasma?

1) To protect hemoglobin from breaking into fragments that would leak out of the bloodstream (through porous capillary walls)

2) To prevent hemoglobin from making blood more viscous and raising osmotic pressure
Hematopoiesis

- Hematopoiesis (hemopoiesis): blood cell formation
- Occurs in red bone marrow of axial skeleton, girdles and proximal epiphyses of humerus and femur
- On average, the marrow turns out an 29.5 ml of new blood containing 100 billion new cells every day
Hematopoiesis

- Hemocytoblasts (hematopoietic stem cells)
- Give rise to all formed elements
- Hormones and growth factors push the cell toward a specific pathway of blood cell development
Erythropoiesis: formation of red blood cells

Reticulocytes are released into the bloodstream.
Erythopoiesis

- Erythopoiesis: red blood cell production

- A hematopoietic stem cell descendant called a myeloid stem cell transforms into a proerythroblast

- Proerythroblasts develop into basophilic erythroblasts
Erythropoiesis: formation of red blood cells

Reticulocytes are released into the bloodstream
Erythropoiesis

- Hemoglobin is synthesized and iron accumulates as the basophilic erythroblast transforms into a polychromatic erythroblast and then an orthochromatic erythroblast

- Reticulocytes (account for 1–2% of all erythrocytes) are formed after ejection all of the organelles and the nuclei

- Reticulocytes fully mature to erythrocytes within two days of release as their ribosomes are degraded by intracellular enzymes
Erythopoiesis: formation of red blood cells

Reticulocytes are released into the bloodstream

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Regulation of Erythropoiesis

- Too few RBCs leads to tissue hypoxia

- Too many RBCs increases blood viscosity

- Balance between RBC production and destruction depends on
  - Hormonal controls: Erythropoietin (EPO)
  - Adequate supplies of iron, amino acids, and B vitamins
Hormonal Control of Erythropoiesis

- Erythropoietin (EPO)
  - Direct stimulus for erythropoiesis
  - Released by the kidneys in response to hypoxia
Hormonal Control of Erythropoiesis

• Causes of hypoxia
  – Hemorrhage or increased RBC destruction reduces RBC numbers
  – Insufficient hemoglobin per RBC (e.g., iron deficiency)
  – Reduced availability of O₂ (e.g., high altitudes) or during pneumonia
Hormonal Control of Erythropoiesis

- Effects of EPO
  - More rapid maturation of committed bone marrow cells
- Increased circulating reticulocyte count in 1–2 days after erythropoietin levels rise in the blood

- hypoxia does not activate the bone marrow directly. Instead it stimulates the kidneys to provide EPO

- Testosterone also enhances EPO production, resulting in higher RBC counts in males
Imbalance of EPO

- Kidneys of renal dialysis patients fail to produce enough EPO to support normal erythropoiesis.

- Consequently, their RBC counts becomes less than half those of healthy individuals.

- Treatment: Genetically engineered (recombinant) EPO is frequently administered
Dietary Requirements

• Iron is essential for hemoglobin synthesis.

• Iron is available from the diet, and it is absorbed by the intestinal cells.

• Approximately 65% of the body’s iron supply (about 4000 mg) is in hemoglobin
Dietary Requirements

• Free iron ions (Fe²⁺, Fe³⁺) are toxic, so iron is stored inside cells as protein-iron complexes such as ferritin and hemosiderin

• In blood, iron is transported loosely bound to a transport protein called transferrin

• Developing erythrocytes take up iron as needed to form hemoglobin
Dietary Requirements

- Small amounts of iron are lost each day in feces, urine, and perspiration.
- The average daily loss of iron is 1.7 mg in women and 0.9 mg in men.
- In women, the menstrual flow accounts for the additional losses.
Dietary Requirements

• Vitamin B12 and folic acid are necessary for normal DNA synthesis.

• Even slight deficits endanger rapidly dividing cell populations, such as developing erythrocytes.
Fate and Destruction of Erythrocytes

• Life span of RBC ranges from 100 to 120 days

• Erythrocytes become “old” as they lose their flexibility, become increasingly rigid and fragile, and their hemoglobin begins to degenerate.

• They are trapped and fragmented by the spleen “red blood cell graveyard.”
Fate and Destruction of Erythrocytes

- Macrophages engulf and destroy dying erythrocytes.
- The heme is separated from globin.
- Its core of iron is salvaged, bound to protein (as ferritin or hemosiderin), and stored for reuse.
Fate and Destruction of Erythrocytes

1. Senescent red cells are a major source of hemeproteins.

2. Breakdown of heme to bilirubin occurs in macrophages of the reticuloendothelial system (tissue macrophages, spleen, and liver).

3. Unconjugated bilirubin is transported through the blood (complexed to albumin) to the liver.

4. Bilirubin is taken up via facilitated diffusion by the liver and conjugated with glucuronic acid.

5. Conjugated bilirubin is actively secreted into bile and then the intestine.

6. In the intestine, glucuronic acid is removed by bacteria. The resulting bilirubin is converted to urobilinogen.

7. Some of the urobilinogen is reabsorbed from the gut and enters the portal blood.

8. A portion of this urobilinogen participates in the enterohepatic urobilinogen cycle.

9. The remainder of the urobilinogen is transported by the blood to the kidney, where it is converted to yellow urobilin and excreted, giving urine its characteristic color.

10. Urobilinogen is oxidized by intestinal bacteria to the brown stercobilin.

To feces

To urine
Formation & Destruction of RBCs

1. Red blood cell death and phagocytosis
2. Heme
3. Amino acids reused for protein synthesis
4. Globin
5. Ferritin
6. Bilirubin
7. Fe³⁺ - Transferrin
8. Erythropoiesis in red bone marrow
9. Biliverdin
10. Liver
11. Small intestine
12. Stercobilin
13. Urobilinogen
14. Large intestine

Key:
- Red in blood
- Green in bile

Macrophage in spleen, liver, or red bone marrow

Circulation for about 120 days
Fate and Destruction of Erythrocytes

• Most of this degraded pigment leaves the body in feces, as a brown pigment called stercobilin.

• Some of urobilinogen (coloreless) is reabsorbed from the intestine, and converted into urobilin (yellow color) that is excreted by the kidneys.

• The protein (globin) part of hemoglobin is metabolized or broken down to amino acids, which are released to the circulation.
Erythrocyte Disorders
Blood Cell Production

Stem cells are located in red bone marrow

Stem cells multiply and become specialized

Mature blood cells

- Erythroblast
- Myeloblast
- Monoblast
- Lymphoblast
- Megakaryoblast
- Nucleus lost
- Erythrocyte (red blood cell)
- Neutrophil
- Eosinophil
- Basophil
- Monocyte
- Lymphocyte
- Megakaryocyte
- Platelets

Granular leukocytes

Agranular leukocytes

White blood cells

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Anemia

- It is a condition in which the blood’s oxygen-carrying capacity is too low to support normal metabolism.

- It is a sign of some disorder rather than a disease in itself.

- Anemic individuals are fatigued, often pale, short of breath, and chilled.

- The causes of anemia can be divided into three groups:
  - Blood loss
  - Not enough red blood cells produced
  - or too many of them destroyed
Blood loss (Hemorrhagic anemia)

- Acute hemorrhagic anemia: blood loss is rapid

- Persistent blood loss: due to hemorrhoids, an undiagnosed bleeding ulcer or malignancies
Iron-deficiency anemia

• Cause:
  ➢ A secondary result of hemorrhagic anemia
  ➢ Inadequate intake of iron-containing foods and impaired iron absorption.

• Erythrocytes produced, called microcytes, are small and pale because they cannot synthesize their normal complement of hemoglobin
Pernicious anemia

- Autoimmune disease most often affects the elderly.

- The immune system destroys cells in the stomach mucosa that produce a substance called intrinsic factor (important for vitamin B12 absorption) by intestinal cells.

- Without vitamin B12, the developing erythrocytes grow but cannot divide, and large, pale cells called macrocytes result (megaloblastic anemia).

- Treatment involves regular intramuscular injections of vitamin B12 or as sublingual tablets.

- Lack of vitamin B12 in the diet also leads to anemia.
Renal anemia

- Frequently accompanies renal disease because damaged or diseased kidneys cannot produce enough EPO.

- It can be treated with synthetic EPO.
Aplastic anemia

• Results from destruction or inhibition of the red marrow by certain drugs and chemicals, ionizing radiation, or viruses.

• In most cases, though, the cause is unknown.

• Because marrow destruction impairs formation of all formed elements, anemia is just one of its signs.
Aplastic anemia

• Defects in blood clotting and immunity are also present.

• Blood transfusions provide a temporal treatment until stem cells harvested from a donor’s blood, bone marrow, or umbilical cord blood can be transplanted.
Hemolytic anemias

• Erythrocytes rupture, or lyse, prematurely and with high rate

• **Common causes:**
  - Hemoglobin abnormalities: thalassemia and sickle-cell anemia
  - Transfusion of mismatched blood
  - Certain bacterial and parasitic infections
Thalassemias

• Occur in people of Mediterranean ancestry

• One of the globin chains is absent or faulty, and the erythrocytes are thin, delicate, and deficient in hemoglobin

• Severity of the diseased varies according to the associated mutation
Sickle-cell anemia

• Caused by the abnormal hemoglobin, hemoglobin S (HbS)

• Results from a change in just one of the 146 amino acids in a beta chain of the globin molecule!

• This alteration causes the beta chains to link together under low-oxygen conditions, forming stiff rods so that hemoglobin S becomes spiky and sharp
Genetics of Sickle Cell Anemia

![Diagram showing normal and mutant hemoglobin DNA and mRNA, with a comparison of normal and sickle-cell hemoglobin structures.](image)
Sickle-cell anemia

• This causes the red blood cells to become crescent shaped when they unload oxygen molecules or when the oxygen content of the blood is lower than normal, as during vigorous exercise and other activities that increase metabolic rate.

• The stiff, deformed erythrocytes rupture easily and tend to occlude small blood vessels.
Sickle-cell anemia

- This causes organ ischemia, leaving the victims gasping for air and in extreme pain.
Polycythemia

• Abnormal excess of erythrocytes that increases blood viscosity
• Risk of stroke and heart failure due to high hematocrit and high blood viscosity
• Types:
  ➢ Polycythemia vera
  ➢ Secondary polycythemia
Polycythemia vera

• Bone marrow cancer, characterized by dizziness and an exceptionally high RBC count (8–11 million cells/µl).

• The hematocrit may be as high as 80% and blood volume may double, causing the vascular system to become engorged with blood and severely impairing circulation.
Secondary polycythemias

- Due to high level of EPO secretion
- Detected in smokers and individuals living at high altitudes
Leukocytes (White Blood Cells)
Leukocytes (White Blood Cells)

• On average, there are 4800–10,800 WBCs/μl of blood

• Leukocytes are grouped into two major categories on the basis of structural and chemical characteristics.
  ➢ Granulocytes: contain obvious membrane-bound cytoplasmic granules, and
  ➢ Agranulocytes: lack obvious granules
Granulocytes

- Include: neutrophils, eosinophils, and basophils
- They have lobed nuclei
- Functionally, all granulocytes are phagocytes to some degree
Neutrophils

- Also called Polymorphonuclear leukocytes (PMNs)
- Account for 50–70% of the WBC population
- About twice as large as erythrocytes
- Their numbers increase explosively during acute bacterial infections
Neutrophils

• Some of their granules contain hydrolytic enzymes, and are regarded as lysosomes.

• Others, especially the smaller granules, contain a potent “brew” of antimicrobial proteins, called defensins that pierce holes in the membrane of the ingested pathogen.

• They also kill pathogens by respiratory burst (the cells metabolize oxygen to produce potent germ-killer oxidizing substances such as bleach and hydrogen peroxide.

• Neutrophils are chemically attracted to sites of inflammation and are active phagocytes
Eosinophils

- Account for 2–4% of all leukocytes

- Their granules are lysosome-like and filled with a unique variety of digestive enzymes.

- They lack enzymes that specifically digest bacteria.
Eosinophils

- The most important role is to lead the counterattack against parasitic worms, such as flatworms (tapeworms and flukes) and roundworms (pinworms and hookworms) that are too large to be phagocytized.

- They release the enzymes from their cytoplasmic granules onto the parasite’s surface to digesting it.

- Eosinophils have complex roles in many other diseases including allergies and asthma.
Basophils

• The rarest white blood cells, accounting for only 0.5–1% of the leukocyte population

• Their cytoplasm contains large, coarse, histamine-containing granules

• Histamine is an inflammatory chemical that acts as a vasodilator (makes blood vessels dilate) and attracts other white blood cells to the inflamed site

• Granulated cells similar to basophils, called mast cells, are found in connective tissues. They also release histamine
Agranulocytes

- Include lymphocytes and monocytes
- Lack visible cytoplasmic granules
Lymphocytes

• Accounting for 25% of the WBC population
• Few are found in the bloodstream, as they are closely associated with lymphoid tissues (lymph nodes, spleen, etc.), where they play a crucial role in immunity.
• **T lymphocytes (T cells)** function in the immune response by acting directly against virus-infected cells and tumor cells.
• **B lymphocytes (B cells)** give rise to plasma cells, which produce antibodies
Monocytes

• Account for 3–8% of WBCs

• When circulating monocytes leave the bloodstream and enter the tissues, they differentiate into highly mobile macrophages.

• Macrophages are actively phagocytic, and they are crucial in the body’s defense against viruses, certain intracellular bacterial parasites, and chronic infections such as tuberculosis.
Leukocyte Disorders

- **Leukemia and infectious mononucleosis**: overproduction of abnormal leukocytes

- **Leukopenia**: is an abnormally low white blood cell count, commonly induced by drugs, particularly glucocorticoids and anticancer agents.
Leukemia

- a group of cancerous conditions involving overproduction of abnormal white blood cells

- The diseased leukocytes are members of a single clone (descendants of a single cell) that remain unspecialized and proliferate out of control, impairing normal red bone marrow function.
Leukemia

- The leukemias are named according to the cell type primarily involved.

- For example, myeloid leukemia involves myeloblast descendants, whereas lymphocytic leukemia involves the lymphocytes.

- Leukemia is acute (quickly advancing) if it derives from stem cells, and chronic (slowly advancing) if it involves proliferation of later cell stages.
Leukemia

• The more serious acute forms primarily affect children.

• Chronic leukemia occurs more often in elderly people.

• Without therapy all leukemias are fatal

• Cancerous leukocytes fill the red bone marrow and immature WBCs flood into the bloodstream.
Leukemia

• The other blood cell lines are crowded out, so severe anemia and bleeding problems result.

• Other symptoms include fever, weight loss, and bone pain.

• Although tremendous numbers of leukocytes are produced, they are nonfunctional and cannot defend the body in the usual way.

• The most common causes of death are internal hemorrhage and overwhelming infections.
Infectious Mononucleosis

• Highly contagious viral disease

• Caused by the Epstein-Barr virus

• Its hallmark is excessive numbers of agranulocytes

• The affected individual complains of being tired and achy, and has a chronic sore throat and a low-grade fever.

• There is no cure, but with rest the condition typically runs its course to recovery in a few weeks.
Platelets

• They are not cells in the strict sense

• they are cytoplasmic fragments of extraordinarily large cells called **megakaryocytes**

• Their granules contain lots of chemicals that act in the clotting process, (including serotonin, Ca2+, a variety of enzymes, ADP, and platelet derived growth factor (PDGF)).
Hemostasis

Step 1: Vascular spasm
Smooth muscle contracts causing vasoconstriction
direct injury to vascular smooth muscle, chemicals released by endothelial cells and platelets, and reflexes initiated by local pain receptors

Step 2:
- Platelet plug formation
  Injury to lining of vessel exposing collagen fibers, platelets adhere.
- Platelets release chemicals that make nearby platelets sticky, platelet plug forms

Step 3: Coagulation
- Fibrin forms a mesh that traps RBCs & platelets, forming the clot.
Platelets

- As a rule, platelets do not stick to each other or to the smooth endothelial linings of blood vessels.

- Intact endothelial cells release nitric oxide and a prostaglandin called prostacyclin (or PGI2).

- Both chemicals prevent platelet aggregation in undamaged tissue and restrict aggregation to the site of injury.
Platelets

- However, when the endothelium is damaged and the underlying collagen fibers are exposed, platelets adhere tenaciously to the collagen fibers.

- A large plasma protein called von Willebrand factor stabilizes bound platelets by forming a bridge between collagen and platelets.
Platelets

- Platelets swell, form spiked processes, become stickier, and release chemical messengers including the following:

  - Adenosine diphosphate (ADP)—a potent aggregating agent that causes more platelets to stick to the area and release their contents

  - Serotonin and Thromboxane A2 messengers that enhance vascular spasm and platelet aggregation
Coagulation (blood clotting)

- Reinforces the platelet plug with fibrin threads that act as a “molecular glue” for the aggregated platelets

- Blood is transformed from a liquid to a gel in a multistep process that involves a series of substances called clotting factors (procoagulants)

- Most clotting factors are plasma proteins synthesized by the liver. They are numbered I to XIII according to the order of their discovery
Coagulation (blood clotting)

• Vitamin K (fat-soluble vitamin) is required for synthesizing four of the clotting factors

• In most cases, activation turns clotting factors into enzymes, except factor IV (Ca2+) and I (Fibrinogen)
Two Pathways to Prothrombin Activator

• Coagulation may be initiated by either the intrinsic or the extrinsic pathway

• In the body, the same tissue-damaging events usually trigger both pathways.

• Outside the body (such as in a test tube), only the intrinsic pathway initiates blood clotting.
Blood coagulation

**Intrinsic pathway**
- Vascular injury
- Expose collagen to blood
- **XII** → **XIIa**
- **XI** → **XIa**
- **IX** → **IXa**
- **X** → **Xa**
- Prothrombin (II) → Thrombin (IIa)
- Fibrinogen (I) → Fibrin

**Extrinsic pathway**
- Vascular injury
- Expose Tissue Factor (TF) (Thromboplastin)
- **Clotting Factors**
- **XII** → **XIIa**
- **XI** → **XIa**
- **IX** → **IXa**
- **VII** → **VIIa**
- **X** → **Xa**
- Thrombin (IIa)
- Fibrinogen (I) → Fibrin
Blood coagulation

Figure 17.15 Scanning electron micrograph of erythrocytes trapped in a fibrin mesh. (2700×).
Blood Clot

- Platelet
- Fibrin thread
- RBC
Disorders of Hemostasis

- Thromboembolytic disorders: undesirable clot formation
- Bleeding disorders: abnormalities that prevent normal clot formation
Thromboembolytic Conditions

• Thrombus: clot that develops and persists in an unbroken blood vessel
  – May block circulation, leading to tissue death
• Embolus: a thrombus freely floating in the blood stream
  – Pulmonary emboli impair the ability of the body to obtain oxygen
  – Cerebral emboli can cause strokes
Thromboembolytic Conditions

Prevented by

– Aspirin
  • Antiprostaglandin that inhibits thromboxane A2
– Heparin
  • Anticoagulant used clinically for pre- and postoperative cardiac care
– Warfarin
  • Used for those prone to atrial fibrillation
Bleeding disorders

**Thrombocytosis** - too many platelets due to inflammation, infection or cancer

**Thrombocytopenia** - too few platelets

- causes spontaneous bleeding
- due to suppression or destruction of bone marrow (e.g., malignancy, radiation)
  - Platelet count <50,000/mm$^3$ is diagnostic
  - Treated with transfusion of concentrated platelets
Bleeding disorders

• Impaired liver function
  – Inability to synthesize procoagulants
  – Causes include vitamin K deficiency, hepatitis, and cirrhosis
  – Liver disease can also prevent the liver from producing bile, impairing fat and vitamin K absorption
Bleeding disorders

- Hemophiliias include several similar hereditary bleeding disorders
- Symptoms include prolonged bleeding, especially into joint cavities
- Treated with plasma transfusions and injection of missing factors
Blood types

- Type A
- Type B
- Type AB
- Type O
Blood types

Blood type is based on the presence of 2 major antigens in RBC membranes-- A and B

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<th>Blood type</th>
<th>Antigen</th>
<th>Antibody</th>
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<td>Anti-B</td>
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<tr>
<td>B</td>
<td>B</td>
<td>Anti-A</td>
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<tr>
<td>A &amp; B</td>
<td>AB</td>
<td>no antibody</td>
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<tr>
<td>Neither A or B</td>
<td>O</td>
<td>anti-A and anti-B</td>
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</table>

**Antigen** - protein on the surface of a RBC membrane

**Antibody** - proteins made by lymphocytes in plasma which are made in response to the presence of antigens. They attack foreign antigens, which result in clumping (agglutination)
Type A

A antigen

Red blood

Blood type A

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Type B

Blood type B

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Type O

Blood type O
Universal donor

Dr. Naim Kittana, PhD
Type AB

Blood type AB
Universal recipient
# Table 17.4 ABO Blood Groups

<table>
<thead>
<tr>
<th>BLOOD GROUP</th>
<th>RBC ANTIGENS (AGGLUTINOGENS)</th>
<th>ILLUSTRATION</th>
<th>PLASMA ANTIBODIES (AGGLUTININS)</th>
<th>BLOOD THAT CAN BE RECEIVED</th>
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<td>A, B, AB, O “Universal recipient”</td>
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<td><img src="image4" alt="Illustration" /></td>
<td>Anti-A (a) Anti-B (b)</td>
<td>O “Universal donor”</td>
</tr>
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Rh Factor and Pregnancy

RH+ indicates protein

RH- indicates no protein
Rh Factor and Pregnancy

Rh⁺ mother w/Rh⁻ baby— no problem
Rh⁻ mother w/Rh⁺ baby— problem
Rh⁻ mother w/Rh⁻ father— no problem
Rh⁻ mother w/Rh⁻ baby-- no problem
Rho(D) immune globulin (anti-RhD)

- IgG anti-D antibodies

- Given at the 28th week of pregnancy as IM for pregnant women who are Rh-negative if the father is Rh-positive

- Target fetal RBCs that leak to the maternal blood circulation before the maternal immune system reacts to it

- Prevents Rh disease (hemolytic disease of new born) in the subsequent pregnancies
Type **AB**- universal recipients

Type **O**- universal donor

**Rh factor:**

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<th>Rh factor</th>
<th>Frequency</th>
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<td>Rh+</td>
<td>85% dominant in pop</td>
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<tr>
<td>Rh-</td>
<td>15% recessive</td>
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<table>
<thead>
<tr>
<th>Blood Type</th>
<th>Clumping</th>
<th>Antibody</th>
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<tr>
<td><strong>A</strong></td>
<td>antigen A</td>
<td>anti-A serum</td>
</tr>
<tr>
<td><strong>B</strong></td>
<td>antigen B</td>
<td>anti-B serum</td>
</tr>
<tr>
<td><strong>AB</strong></td>
<td>antigen A &amp; B</td>
<td>anti A &amp; B serum</td>
</tr>
<tr>
<td><strong>O</strong></td>
<td>neither A or B</td>
<td>no clumping w/ either anti A or B</td>
</tr>
</tbody>
</table>

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Blood test

Type AB (contains agglutinogens A and B; agglutinates with both sera)

Type A (contains agglutinogen A; agglutinates with anti-A)

Type B (contains agglutinogen B; agglutinates with anti-B)

Type O (contains no agglutinogens; does not agglutinate with either serum)