Blood Outline

17.1 The functions of blood are transport, regulation, and protection (p. 636)
- A. Transport functions include delivery of oxygen and nutrients, transport of metabolic wastes for elimination, and transport of hormones. (p. 636)
- B. Regulatory functions include maintaining body temperature, pH, and fluid balance. (p. 636)
- C. Protective functions include preventing blood loss and infection. (p. 636)

17.2 Blood consists of plasma and formed elements (pp. 636–638; Fig. 17.1; Table 17.1)
- A. Blood is a specialized connective tissue consisting of living cells, called formed elements, suspended in a nonliving fluid matrix, blood plasma. (p. 632)
- B. Blood that has been centrifuged separates into three layers: erythrocytes, the buffy coat, and plasma. (pp. 636–637; Fig. 7.1)
- C. The blood hematocrit represents the percentage of erythrocytes in whole blood. (p. 637)
- D. Physical Characteristics and Volume (p. 637)
  1. Blood is a slightly basic (pH = 7.35–7.45) fluid that has a higher density and viscosity than water, due to the presence of formed elements.
  2. Normal blood volume in males is 5–6 liters, and 4–5 liters for females.
- E. Blood plasma consists of mostly water (90%) and solutes including nutrients, gases, hormones, wastes, products of cell activity, ions, and proteins. (p. 637; Table 17.1)
  1. Eight percent of plasma solutes are proteins: albumin constitutes roughly 60% of plasma proteins and functions as a carrier, a pH buffer, and an osmoregulating protein.
- F. Formed elements of the blood are erythrocytes, leucocytes, and platelets, and have special features (pp. 637–638; Fig. 17.2):
  1. Of the three, only leucocytes are complete cells: erythrocytes have no nucleus, and platelets are cell fragments.
  2. Most survive in the blood for only a few days.
  3. Instead of dividing, blood cells are replaced by stem cells located in red bone marrow.

Elements (pp. 634–646; Figs. 17.2–17.12; Table 17.2)

17.3 Erythrocytes play a crucial role in oxygen and carbon dioxide transport (pp. 638–644; Figs. 17.2–17.8; Table 17.2)
- A. Erythrocytes, or red blood cells, are small cells that are biconcave in shape, lack nuclei and most organelles, and contain mostly hemoglobin. (pp. 638–639; Fig. 17.3; Table 17.2)
  1. The size and shape of erythrocytes provide a larger surface area for gas exchange.
  2. Not considering water content, an erythrocyte is over 97% hemoglobin.
  3. The lack of organelles and anaerobic ATP synthesis means that erythrocytes do not consume any oxygen they carry.
- B. Erythrocytes function to transport respiratory gases in the blood on hemoglobin. (pp. 639–640; Fig. 17.4)

1. The normal range for hemoglobin in the blood is 13–18 g/100 ml.
2. Hemoglobin is a protein consisting of four polypeptide chains, globin proteins, each with a ring-like heme.
   a. Each heme contains an atom of iron that serves as the binding site for a molecule of oxygen.
   b. Oxygen diffuses into the blood in the lungs and binds to hemoglobin, forming bright red oxyhemoglobin.
   c. At body tissues, oxygen detaches from iron, forming dark red deoxyhemoglobin.
   d. About 20% of the carbon dioxide carried in the blood is bound to amino acids on the globins, forming carbaminohemoglobin.

C. Production of Erythrocytes (p. 640; Fig. 17.5)
1. Hematopoiesis, or blood cell formation, occurs in the red bone marrow.
2. All blood cells form from a common hematopoietic stem cell, the hemocytoblast.
3. Erythropoiesis, the formation of erythrocytes, begins when a myeloid stem cell is transformed to a proerythroblast, which progresses through several successive stages.
   a. During the first two phases of development, hemoglobin is synthesized, and iron accumulates.
   b. After accumulating all its hemoglobin, the reticulocyte (immature erythrocyte) ejects most organelles, the nucleus degenerates, and the cell assumes its biconcave shape.
4. The hematopoietic process takes about 15 days, at which time the reticulocyte enters the bloodstream, becoming a fully mature, oxygen-carrying cell within two days.

D. Regulation and Requirements for Erythropoiesis (pp. 641–642; Fig. 17.6)
1. Erythrocyte production is controlled by the hormone erythropoietin (EPO), produced mostly by the kidneys when certain kidney cells become hypoxic.
   a. Erythropoietin production is triggered by excessive loss of RBCs, insufficient hemoglobin, or reduced availability of oxygen, and may be enhanced by testosterone.
2. Dietary requirements for erythrocyte formation include iron, vitamin B_{12}, and folic acid, as well as proteins, lipids, and carbohydrates.
3. Blood cells have a life span of 100–120 days due to the lack of nuclei and organelles.

E. Destruction of dead or dying blood cells is accomplished by macrophages in the spleen. (p. 642; Fig. 17.7)
1. Heme is split from globin: globin is broken down to amino acids, and the iron from heme is salvaged.
2. What remains of the heme is degraded to bilirubin, which is ultimately secreted in bile to the intestine for removal from the body.

F. Erythrocyte Disorders (pp. 642–644; Fig. 17.8)
1. Anemias are characterized by a deficiency in RBCs that may originate from three main causes:
   a. Blood loss: hemorrhagic anemia.
   b. Not enough red blood cells produced: iron-deficiency anemia, pernicious anemia (lack of vitamin B12), renal anemia (low EPO), or aplastic anemia (destruction of red bone marrow).

c. Too many red blood cells destroyed: hemoglobin abnormalities (thalassemias and sickle-cell anemia), transfusion mismatch, or bacterial or parasitic infections.

2. Polycythemia is characterized by an excess of RBCs due to oxygen deficiency or disease, which may increase blood viscosity, causing poor blood flow and oxygen delivery.

17.4 Leukocytes defend the body (pp. 644–650; Figs. 17.9–17.12; Table 17.2)

A. General Structural and Functional Characteristics (pp. 644–645; Fig. 17.9)

1. Leukocytes, or white blood cells, are the only formed elements that are complete cells and make up less than 1% of total blood volume.
2. Leukocytes are critical to our defense against disease, and can leave the blood to enter the tissues, a process called diapedesis, and move through the tissue by amoeboid movement.
   a. Leukocytes exhibit positive chemotaxis, following chemical trails of molecules from damaged cells, to migrate toward areas of tissue damage and infection.
   b. Leukocytosis, a white blood cell count of over 11,000, is characteristic as a consequence of an infection.

B. Granulocytes are a main group of leukocytes characterized as large cells with lobed nuclei and visibly staining granules; all are phagocytic. (pp. 645–646; Fig. 17.10; Table 17.2)

1. Neutrophils, 50–70% of all leukocytes, are chemically attracted to sites of inflammation and are active phagocytes.
2. Eosinophils account for 2–4% of all erythrocytes, attack parasitic worms in loose connective tissues, and have a role in asthma and allergies.
3. Basophils are the least numerous leukocyte, 0.5–1% of all WBCs, and release histamine to promote inflammation.

C. Agranulocytes are lymphocytes and monocytes that lack visibly staining granules. (pp. 646–647; Table 17.2)

1. Lymphocytes comprise 25%+ of all WBCs and are found throughout the body—but relatively few are found in the blood.
   a. T lymphocytes directly attack virus-infected and tumor cells; B lymphocytes produce antibodies.
   b. B lymphocytes give rise to plasma cells, which produce antibodies.
2. Monocytes make up 3–8% of all WBCs, become actively phagocytotic macrophages as they enter tissues, and activate lymphocytes.

D. Production and Life Span of Leukocytes (pp. 647–649; Fig. 17.11)

1. Leukopoiesis, the formation of white blood cells, is regulated by the production of interleukins and colony-stimulating factors (CSF).
2. Leukopoiesis involves differentiation of hemocytoblasts along two pathways: lymphoid stem cells that give rise to lymphocytes and myeloid stem cells that give rise to all other WBCs.
3. Monocytes have a life span of a few months, while lymphocytes live for months to years.

E. Leukocyte Disorders

1. Leukopenia is an abnormally low white blood cell count, possibly due to drugs, such as glucocorticoids or anticancer drugs.
2. Leukemias are cancerous conditions in which clones of a single white blood cell remain unspecialized and divide out of control.

3. Infectious mononucleosis is a disease caused by the Epstein-Barr virus, characterized by excessive numbers of agranulocytes.

17.5 Platelets are cell fragments that help stop bleeding (p. 650; Fig. 17.12)

A. Platelets are not complete cells, but fragments of large cells called megakaryocytes, and have a short life span of around 10 days. (p. 650)
   1. Platelets are critical to the clotting process, forming the temporary seal when a blood vessel breaks.
   2. Thrombopoietin is a hormone that regulates the formation of platelets, which takes place by repeated mitoses of megakaryocytes without cytokinesis.
   3. Platelets enter the blood when a megakaryocyte sends cytoplasmic extensions through a sinusoid wall, ruptures, and releases platelets.

17.6 Hemostasis prevents blood loss (pp. 650–656; Figs. 17.13–17.15; Table 17.3)

A. Three steps occur in rapid sequence during hemostasis: vascular spasm, platelet plug formation, and coagulation. (p. 650)
B. Vascular spasms are the immediate vasoconstriction response to blood vessel injury and become more efficient with increased tissue damage. (p. 651)
C. Platelet Plug Formation (p. 651; Fig. 17.13)
   1. When endothelium is damaged, platelets become sticky and spiky, adhering to each other and the damaged vessel wall.
   2. Once attached, other platelets are attracted to the site of injury, activating a positive feedback loop for clot formation.
D. Coagulation, or blood clotting, is a multistep process in which blood is transformed from a liquid to a gel. (pp. 651–652; Figs. 17.13–17.15; Table 17.3)
   1. Factors that promote clotting are called clotting factors, or procoagulants; those that inhibit clot formation are called anticoagulants.
   2. The clotting process involves three phases: formation of prothrombin activator, conversion of prothrombin to thrombin, and the formation of fibrin mesh from fibrinogen in the plasma.
   3. There are two pathways to the formation of prothrombin activator:
      a. The intrinsic pathway, so named because all factors necessary are present within the blood, is a slower clotting pathway and may be triggered by negatively charged surfaces, such as activated platelets, collagen, or glass.
      b. The extrinsic pathway is triggered through an endothelium-derived protein factor, called tissue factor (TF) or factor III, and can occur very rapidly.
   4. Thrombin catalyzes the reactions that convert fibrinogen to fibrin, which forms strands that form the structure of a clot.
E. Clot Retraction and Fibrinolysis (pp. 653–654)
   1. Clot retraction is a process in which the contractile proteins within platelets contract and pull on neighboring fibrin strands, squeezing plasma from the clot and pulling damaged tissue edges together.
   2. Repair is stimulated by platelet-derived growth factor (PDGF).
   3. Fibrinolysis removes unneeded clots through the action of the fibrin-digesting enzyme, plasmin.

F. Factors Limiting Clot Growth or Formation (pp. 654–655)
   1. Two mechanisms limit the size of clots as they form:
      a. Rapidly moving blood disseminates clotting factors before they can initiate a clotting cascade.
      b. Activated clotting factors are inhibited.
   2. Thrombin that is not bound to fibrin is inactivated by antithrombin III and protein C, as well as heparin.
   3. As long as the vascular endothelium is smooth and intact, platelets are prevented from clotting.

G. Disorders of Hemostasis (pp. 655–656)
   1. Thromboembolic disorders result from conditions that cause undesirable clotting, such as roughening of vessel endothelium, slow-flowing blood, or blood stasis.
      a. A clot that forms and persists in an unbroken vessel is called a thrombus and, if large enough, may block blood flow to tissues.
      b. A thrombus that breaks away from a vessel wall is called an embolus, which may become lodged in a small diameter vessel, also restricting blood flow.
   2. Anticoagulant drugs, such as aspirin, heparin, and warfarin, are used clinically to prevent undesirable clotting.
   3. Bleeding disorders arise from abnormalities that prevent normal clot formation.
      a. Thrombocytopenia is a deficiency in circulating platelets and may result from any condition that suppresses or destroys red bone marrow.
      b. Impaired liver function results in a lack of synthesis of procoagulants, which may be due to a shortage of vitamin K, or diseases such as hepatitis or cirrhosis.
      c. Hemophilia is a genetic condition that results in a deficiency of factors VIII (antihemophilic factor), IX, or XI.
   4. Disseminated intravascular coagulation is a situation leading to widespread clotting and severe bleeding, and may occur as a complication of pregnancy, septicemia, or incompatible blood transfusions.

17.7 Transfusion can replace lost blood (pp. 656–658; Fig. 17.16; Table 17.4)

A. Transfusion of whole blood is routine only when blood loss is substantial or when treating thrombocytopenia; most of the time, packed red blood cells are used. (pp. 656–658)
   1. Humans have different blood types based on specific antigens, called agglutinogens, on RBC membranes.
   2. At least 30 groups of RBC antigens occur in humans, but the ABO and Rh antigens cause strong transfusion reactions.
      a. ABO blood groups are based on the presence or absence of two types of heritable agglutinogens: type A, and type B. The type O blood group has neither agglutinogen.
      b. Preformed antibodies (agglutinins) are present in blood plasma and are of the opposite type as the individual's blood: Since type AB blood has both A and B agglutinogens, it has no anti-A or anti-B antibodies.
   3. The Rh factor is a group of RBC antigens that are either present in Rh^+ blood or absent in Rh^- blood.

a. Rh antibodies form in Rh^- individuals only after exposure to the Rh antigen.

4. A transfusion reaction occurs if the agglutinogens in the donor blood type are attacked by the recipient's blood plasma agglutinins, resulting in agglutination and hemolysis of the donor cells.
   a. Group O blood is the universal donor type; the AB blood group has neither A nor B antibodies in the plasma and can potentially receive any ABO blood type—the universal recipient.
5. Blood typing involves determination of possible transfusion reactions prior to transfusion between the donor and recipient blood types.

B. Blood volume expanders are given in cases of extremely low blood volume. (p. 658)
   1. Isotonic salt solutions, such as Ringer's solution, mimic the normal electrolyte concentrations of plasma.
   2. Plasma expanders mimic the osmotic properties of albumin in the blood, but they cannot replace the oxygen-carrying properties of hemoglobin.

17.8 Blood tests give insights into a patient's health (p. 659)

A. Changes in some of the visual properties of blood can signal diseases such as anemia, heart disease, diabetes, and infections. (p. 659)
B. Differential white blood cell counts are used to detect differences in relative amounts of specific blood cell types. (p. 659)
C. Prothrombin time, which measures the amount of prothrombin in the blood, and platelet counts evaluate the status of the hemostasis system. (p. 659)
D. A comprehensive medical panel (CMP), and a complete blood count (CBC) give overall values of the condition of the blood. (p. 659)

Developmental Aspects of Blood (p. 659)

A. Prior to birth, blood cell formation occurs within the fetal yolk sac, liver, and spleen, but by the seventh month, red bone marrow is the primary site of hematopoiesis. (p. 659)
B. Fetal blood cells form hemoglobin F, which has a higher affinity for oxygen than adult hemoglobin, hemoglobin A. (p. 659)