I. Overview: Blood Composition and Functions (pp. 635–636; Fig. 17.1)
   A. Components (p. 635; Fig. 17.1)
      1. Blood is a specialized connective tissue consisting of living cells, called formed elements, suspended in a nonliving fluid matrix, blood plasma.
      3. The blood hematocrit represents the percentage of erythrocytes in whole blood.
   C. Functions (pp. 635–636)
      1. Blood is the medium for delivery of oxygen and nutrients, removal of metabolic wastes to elimination sites, and distribution of hormones.
      2. Blood aids in regulating body temperature, body fluid pH, and fluid volume within fluid compartments.
      3. Blood protects against excessive blood loss through the clotting mechanism, and from infection through the immune system.

II. Blood Plasma (p. 636; Table 17.1)
   A. Blood plasma consists of mostly water (90%), and solutes including nutrients, gases, hormones, wastes, products of cell activity, ions, and proteins (p. 636; Table 17.1).
   B. Plasma proteins account for 8% of plasma solutes, mostly albumin, which function as carriers (p. 636).

III. Formed Elements (pp. 637–649; Figs. 17.2–17.12; Table 17.2)
   A. Erythrocytes (pp. 637–643; Figs. 17.2–17.8)
      1. Erythrocytes, or red blood cells, are small cells that are biconcave in shape. They lack nuclei and most organelles, and contain mostly hemoglobin.
         a. Hemoglobin is an oxygen-binding pigment that is responsible for the transport of most of the oxygen in the blood.
         b. Hemoglobin is made up of the protein globin bound to the red heme pigment.
      2. Production of Erythrocytes
         a. Hematopoiesis, or blood cell formation, occurs in the red bone marrow.
         b. Erythropoiesis, the formation of erythrocytes, begins when a myeloid stem cell is transformed to a proerythroblast, which develops into mature erythrocytes.
         c. Erythrocyte production is controlled by the hormone erythropoietin.
         d. Dietary requirements for erythrocyte formation include iron, vitamin B₁₂, and folic acid, as well as proteins, lipids, and carbohydrates.
         e. Blood cells have a short life span due to the lack of nuclei and organelles; destruction of dead or dying blood cells is accomplished by macrophages.
      3. Erythrocyte Disorders
         a. Anemias are characterized by a deficiency in RBCs.
         b. Polycythemia is characterized by an abnormal excess of RBCs.
   B. Leukocytes (pp. 643–648; Figs. 17.9–17.11; Table 17.2)
      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.
      3. Granulocytes are a main group of leukocytes characterized as large cells with lobed nuclei and visibly staining granules; all are phagocytic.
         a. Neutrophils are the most numerous type of leukocyte. They are chemically attracted to sites of inflammation and are active phagocytes.
         b. Eosinophils are relatively uncommon and attack parasitic worms.
c. Basophils are the least numerous leukocyte and release histamine to promote inflammation.

4. Agranulocytes are a main group of lymphocytes that lack visibly staining granules.
   a. T lymphocytes directly attack virus-infected and tumor cells; B lymphocytes produce antibody cells.
   b. Monocytes become macrophages and activate T lymphocytes.

5. Production and Life Span of Leukocytes
   a. Leukopoiesis, the formation of white blood cells, is regulated by the production of interleukins and colony-stimulating factors (CSF).
   b. Leukopoiesis involves differentiation of hemocytoblasts along two pathways: lymphoid and myeloid stem cells.

6. Leukocyte Disorders
   a. Leukopenia is an abnormally low white blood cell count.
   b. Leukemias are clones of a single white blood cell that remain unspecialized and divide out of control.
   c. Infectious mononucleosis is a disease caused by the Epstein-Barr virus.

C. Platelets (pp. 648–649; Fig. 17.12)
   1. Platelets are not complete cells, but fragments of large cells called megakaryocytes.
   2. Platelets are critical to the clotting process, forming the temporary seal when a blood vessel breaks.
   3. Formation of platelets involves repeated mitoses of megakaryocytes without cytokinesis.

V. Transfusion and Blood Replacement (pp. 654–657; Fig. 17.15; Table 17.4)
   A. Transfusion of whole blood is routine when blood loss is substantial, or when treating thrombocytopenia (pp. 654–656; Fig. 17.15; Table 17.4).
      1. Humans have different blood types based on specific antigens on RBC membranes.
      2. ABO blood groups are based on the presence or absence of two types of agglutinogens.
      3. Preformed antibodies (agglutinins) are present in blood plasma and do not match the individual’s blood.
      4. The Rh factor is a group of RBC antigens that are either present in Rh+ blood, or absent in Rh− blood.
      5. A transfusion reaction occurs if the infused donor blood type is attacked by the recipient’s blood plasma agglutinins, resulting in agglutination and hemolysis of the donor cells.
   B. Plasma and blood volume expanders are given in cases of extremely low blood volume (pp. 656–657).

VI. Diagnostic Blood Tests (p. 657)
   A. Changes in some of the visual properties of blood can signal diseases such as anemia, heart disease, and diabetes (p. 657).
   B. Differential white blood cell counts are used to detect differences in relative amounts of specific blood cell types (p. 657).
   C. Prothrombin time, which measures the amount of prothrombin in the blood, and platelet counts evaluate the status of the hemostasis system (p. 657).
   D. SMAC, SMA12–60, and complete blood count (CBC) give comprehensive values of the condition of the blood (p. 657).
Ch 18: Heart Chapter Outline

I. Heart Anatomy (pp. 662–672; Figs. 18.1–18.10)

A. Size, Location, and Orientation (p. 663; Fig. 18.1)
   1. The heart is the size of a fist and weighs 250–300 grams.
   2. The heart is found in the mediastinum and two-thirds lies left of the midsternal line.
   3. The base is directed toward the right shoulder and the apex points toward the left hip.

B. Coverings of the Heart (p. 663; Fig. 18.2)
   1. The heart is enclosed in a doubled-walled sac called the pericardium.
   2. Deep to the pericardium is the serous pericardium.
   3. The parietal pericardium lines the inside of the pericardium.
   4. The visceral pericardium, or epicardium, covers the surface of the heart.

C. Layers of the Heart Wall (pp. 663–664; Fig. 18.3)
   1. The myocardium is composed mainly of cardiac muscle and forms the bulk of the heart.
   2. The endocardium lines the chambers of the heart.

D. Chambers and Associated Great Vessels (pp. 664–668; Fig. 18.4)
   1. The right and left atria are the receiving chambers of the heart.
   2. The right ventricle pumps blood into the pulmonary trunk; the left ventricle pumps blood into the aorta.

E. Pathway of Blood Through the Heart (pp. 668–669; Fig. 18.5)
   1. The right side of the heart pumps blood into the pulmonary circuit; the left side of the heart pumps blood into the systemic circuit.

F. Coronary Circulation (pp. 669–670; Fig. 18.7)
   1. The heart receives no nourishment from the blood as it passes through the chamber.
   2. The coronary circulation provides the blood supply for the heart cells.
   3. In a myocardial infarction, there is prolonged coronary blockage that leads to cell death.
   4. Regulation of Heart Rate
      a. Sympathetic stimulation of pacemaker cells increases heart rate and contractility, while parasympathetic inhibition of cardiac pacemaker cells decreases heart rate.
      b. Epinephrine, thyroxine, and calcium influence heart rate.
      c. Age, gender, exercise, and body temperature all influence heart rate.
   5. Homeostatic Imbalance of Cardiac Output
      a. Congestive heart failure occurs when the pumping efficiency of the heart is so low that blood circulation cannot meet tissue needs.
      b. Pulmonary congestion occurs when one side of the heart fails, resulting in pulmonary edema.
Ch 19: Vessels Chapter Outline

PART 1: OVERVIEW OF BLOOD VESSEL STRUCTURE AND FUNCTION (pp. 694–703; Figs. 19.1–19.5; Table 19.1)

I. Structure of Blood Vessel Walls (p. 695; Figs. 19.1–19.2; Table 19.1)
   A. The walls of all blood vessels except the smallest consist of three layers: the tunica intima, tunica media, and tunica externa (p. 695; Fig. 19.1).
   B. The tunica intima reduces friction between the vessel walls and blood; the tunica media controls vasoconstriction and vasodilation of the vessel; and the tunica externa protects, reinforces, and anchors the vessel to surrounding structures (p. 695; Fig. 19.2; Table 19.1).

VIII. Maintaining Blood Pressure (pp. 706–713; Figs. 19.8–19.12; Table 19.2)
   A. Blood pressure varies directly with changes in blood volume and cardiac output, which are determined primarily by venous return and neural and hormonal controls (p. 706; Fig. 19.8).
   B. Short-term neural controls of peripheral resistance alter blood distribution to meet specific tissue demands, and maintain adequate MAP by altering blood vessel diameter (pp. 706–709; Fig. 19.9).
      1. The vasomotor center is a cluster of sympathetic neurons in the medulla that controls changes in the diameter of blood vessels.
      2. Baroreceptors detect stretch and send impulses to the vasomotor center, inhibiting its activity and promoting vasodilation of arterioles and veins.
   C. Chemical controls influence blood pressure by acting on vascular smooth muscle or the vasomotor center (p. 709; Table 19.2).
      1. Norepinephrine and epinephrine promote an increase in cardiac output and generalized vasoconstriction.
      2. Atrial natriuretic peptide
      3. Antidiuretic hormone promotes vasoconstriction and water conservation by the kidneys, resulting in an increase in blood volume.
   D. Long-Term Mechanisms (p. 710; Figs. 19.10–19.11)
      1. The direct renal mechanism counteracts an increase in blood pressure by altering blood volume, which increases the rate of kidney filtration.
      2. The indirect renal mechanism is the renin-angiotensin mechanism, which counteracts a decline in arterial blood pressure by causing systemic vasoconstriction.
   E. Monitoring circulatory efficiency is accomplished by measuring pulse and blood pressure; these values together with respiratory rate and body temperature are called vital signs (pp. 710–712; Fig. 19.12).
      1. A pulse is generated by the alternating stretch and recoil of elastic arteries during each cardiac cycle.
      2. Systemic blood pressure is measured indirectly using the auscultatory method, which relies on the use of a blood pressure cuff to alternately stop and reopen blood flow into the brachial artery of the arm.
   F. Alterations in blood pressure may result in hypotension (low blood pressure) or transient or persistent hypertension (high blood pressure) (pp. 712–713).

PART 3: CIRCULATORY PATHWAYS: BLOOD VESSELS OF THE BODY (pp. 721–745; Figs. 19.19–19.30; Tables 19.3–19.13)

X. The Two Main Circulations of the Body (p. 721; Figs. 19.19–19.20; Table 19.3)
   A. Two distinct pathways travel to and from the heart: pulmonary circulation runs from the heart to the lungs and back to the heart; systemic circulation runs to all parts of the body before returning to the heart.