10

Blood

FUNCTION PREVIEW

- Blood serves as a vehicle for distributing body heat and for transporting nutrients, respiratory gases, and other substances throughout the body.

Blood is the "river of life" that surges within us. **Blood** transports everything that must be carried from one place to another within the body—nutrients, hormones, wastes (headed for elimination from the body), and body heat—through blood vessels. Long before modern medicine, blood was viewed as magical because when it drained from the body, life departed as well.

In this chapter, we consider the composition and function of this life-sustaining fluid. We discuss the means by which it is propelled throughout the body in Chapter 11.

Composition and Functions of Blood

- ✓ Describe the composition and volume of whole blood.
- ✓ Describe the composition of plasma, and discuss its importance in the body.

Blood is unique: It is the only *fuid* tissue in the body. Although blood appears to be a thick, homogeneous liquid, the microscope reveals that it has both solid and liquid components.

Components

Essentially, blood is a complex connective tissue in which living blood cells, the **formed elements,** are suspended in a nonliving fluid matrix called **plasma** (plaz′muh). The collagen and elastin fibers typical of other connective tissues are absent from blood, but dissolved proteins become visible as fibrin strands during blood clotting.

If a sample of blood is spun in a centrifuge, the formed elements, being heavier, are packed down by centrifugal force and the plasma rises to the top (**Figure 10.1**). Most of the reddish mass at the bottom of the tube is *erythrocytes* (eh-rith′ro-sīts; *erythro* = red), or red blood cells, the formed elements that function in oxygen transport. Although it is barely visible in Figure 10.1, there is a thin, whitish layer called the **buffy coat** at the junction between the erythrocytes and the plasma. This layer contains the remaining formed elements, *leukocytes* (lu′ko-sīts; *leuko* = white), the white blood cells that act in various ways to protect the body, and *platelets,* cell fragments that help stop bleeding. Erythrocytes normally account for about 45 percent of the total volume of a blood sample, a percentage known as the **hematocrit** ("blood fraction"). White blood cells and platelets contribute less than 1 percent, and plasma makes up most of the remaining 55 percent of whole blood.

Physical Characteristics and Volume

Blood is a sticky, opaque fluid with a characteristic metallic taste. As children, we discover its saltiness the first time we stick a cut finger into our mouth. Depending on the amount of oxygen it is carrying, the color of blood varies from scarlet (oxygen-rich) to a dull red (oxygen-poor). Blood is heavier than water and about five times thicker, or more viscous, largely because of its formed elements. Blood is slightly alkaline, with a pH between 7.35 and 7.45. Its temperature (38°C, or 100.4°F) is always slightly higher than body temperature.

Blood accounts for approximately 8 percent of body weight, and its volume in healthy men is 5 to 6 liters, or about 6 quarts.

Plasma

Plasma, which is approximately 90 percent water, is the liquid part of the blood. Over 100 different substances are dissolved in this straw-colored fluid. Examples of dissolved substances include nutrients, salts (electrolytes), respiratory gases, hormones, plasma proteins, and various wastes and products of cell metabolism. Others are listed in Figure 10.1.

Plasma proteins are the most abundant solutes in plasma. Except for antibodies and protein-based hormones, most plasma proteins are made by the liver. The plasma proteins serve a variety of functions. For instance, **albumin** (al-bu′min) acts as a carrier to shuttle certain molecules through the circulation, is an important blood buffer, and contributes to the osmotic pressure of blood, which acts to keep water in the bloodstream. Clotting proteins help stem blood loss when a blood vessel is injured, and antibodies help protect the body from pathogens. Plasma proteins are *not* taken up by cells to be used as food fuels or metabolic nutrients, as are other solutes such as glucose, fatty acids, and oxygen.

The composition of plasma varies continuously as cells remove or add substances to the blood. Assuming a healthy diet, however, the composition of plasma is kept relatively constant by various homeostatic mechanisms of the body. For example, when blood proteins drop to undesirable levels, the liver is stimulated to make more proteins, and when the blood starts to become too acid (*acidosis*) or too basic (*alkalosis*), both the respiratory system and the kidneys are called into action to restore it to its normal, slightly alkaline pH range of 7.35 to 7.45. Various body organs make dozens of adjustments day in and day out to maintain the many plasma solutes at life-sustaining levels. Besides transporting various substances around the body, plasma helps to distribute body heat, a by-product of cellular metabolism, evenly throughout the body.

DID YOU GET IT?

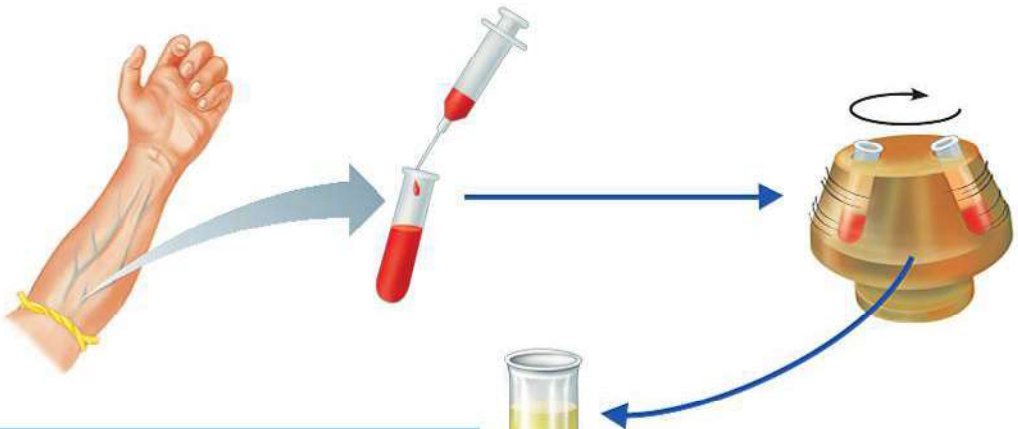
1. Which body organ plays the main role in producing plasma proteins?
2. What are the three major categories of formed elements?
3. What determines whether blood is bright red (scarlet) or dull red?

For answers, see Appendix D.

Formed Elements

- ✓ List the cell types making up the formed elements, and describe the major functions of each type.
- ✓ Define *anemia, polycythemia, leukopenia,* and *leukocytosis,* and list possible causes for each condition.

Q: *How would a decrease in the amount of plasma proteins affect plasma volume?*



Plasma 55%	
Constituent	**Major Functions**
Water	90% of plasma volume; solvent for carrying other substances; absorbs heat
Salts (electrolytes)	
Sodium, Potassium, Calcium, Magnesium, Chloride, Bicarbonate	Osmotic balance, pH buffering, regulation of membrane permeability
Plasma proteins	
Albumin	Osmotic balance, pH buffering
Fibrinogen	Clotting of blood
Globulins	Defense (antibodies) and lipid transport
Substances transported by blood	
Nutrients (glucose, fatty acids, amino acids, vitamins)	
Waste products of metabolism (urea, uric acid)	
Respiratory gases (O_2 and CO_2)	
Hormones (steroids and thyroid hormone are carried by plasma proteins)	

Formed elements (cells) 45%		
Cell Type	**Number (per mm^3 of blood)**	**Functions**
Erythrocytes (red blood cells)	4–6 million	Transport oxygen and help transport carbon dioxide
Leukocytes (white blood cells)	4,800–10,800	Defense and immunity
Basophil		
Eosinophil		Lymphocyte
Neutrophil		Monocyte
Platelets	250,000–400,000	Blood clotting

Figure 10.1 **The composition of blood.**

A: *Plasma proteins create the osmotic pressure that helps to maintain plasma volume and draws leaked fluid back into the circulation. Hence, a decrease in the amount of plasma proteins would result in a reduced plasma volume.*

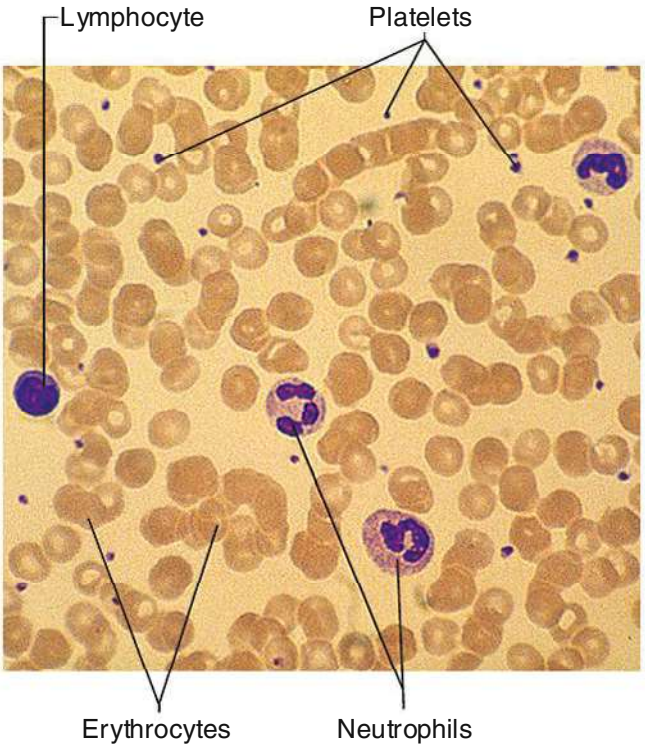


Figure 10.2 Photomicrograph of a blood smear. Most of the cells in this view are erythrocytes (red blood cells). Two kinds of leukocytes (white blood cells) are also present: lymphocytes and neutrophils. Also note the platelets.

If you observe a stained smear of human blood under a light microscope, you will see disc-shaped red blood cells, a variety of gaudily stained spherical white blood cells, and some scattered platelets that look like debris (**Figure 10.2**). However, erythrocytes vastly outnumber the other types of formed elements. Table 10.2 on p. 343 provides a summary of the important characteristics of the various formed elements.

Erythrocytes

Erythrocytes, or **red blood cells (RBCs),** function primarily to ferry oxygen in blood to all cells of the body. They are superb examples of the "fit" between cell structure and function. RBCs differ from other blood cells because they are *anucleate* (a-nu′kle-at); that is, they lack a nucleus. They also contain very few organelles. In fact, mature RBCs circulating in the blood are literally "bags" of hemoglobin molecules. **Hemoglobin** (he″mo-glo′bin) **(Hb),** an iron-bearing protein, transports the bulk of the oxygen that is carried in the blood.

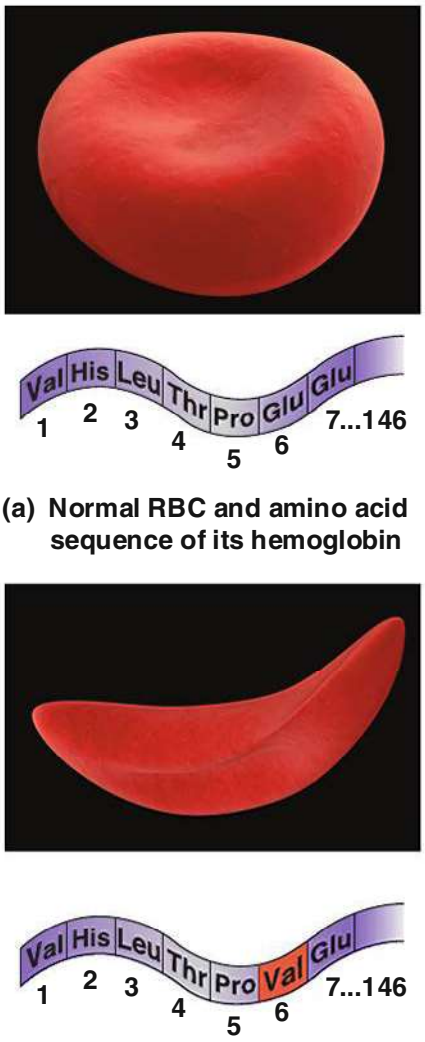


Figure 10.3 Comparison of (a) normal erythrocyte to a (b) sickled erythrocyte (5000×).

(It also binds with a small amount of carbon dioxide.) Moreover, because erythrocytes lack mitochondria and make ATP by anaerobic mechanisms, they do not use up any of the oxygen they are transporting, making them very efficient oxygen transporters indeed.

Erythrocytes are small, flexible cells shaped like biconcave discs—flattened discs with depressed centers on both sides (Figure 10.2 and **Figure 10.3a**). Because of their thinner centers, they look like miniature doughnuts when viewed with a microscope. Their small size and peculiar shape provide a large surface area relative to their volume, making them ideally suited for gas exchange.

Table 10.1 Types of Anemia

Direct cause	Resulting from	Leading to
Decrease in RBC number	Sudden hemorrhage	Hemorrhagic anemia
	Lysis of RBCs as a result of bacterial infections	Hemolytic (he″mo-lit′ik) anemia
	Lack of vitamin B_{12} (usually due to lack of intrinsic factor required for absorption of the vitamin; intrinsic factor is formed by stomach mucosa cells)	Pernicious (per-nish′us) anemia
	Depression/destruction of bone marrow by cancer, radiation, or certain medications	Aplastic anemia
Inadequate hemoglobin content in RBCs	Lack of iron in diet or slow/prolonged bleeding (such as heavy menstrual flow or bleeding ulcer), which depletes iron reserves needed to make hemoglobin; RBCs are small and pale because they lack hemoglobin	Iron-deficiency anemia
Abnormal hemoglobin in RBCs	Genetic defect leads to abnormal hemoglobin, which becomes sharp and sickle-shaped under conditions of increased oxygen use by body; occurs mainly in people of African descent	Sickle cell anemia

RBCs outnumber white blood cells by about 1,000 to 1 and are the major factor contributing to blood viscosity. Although the numbers of RBCs in the circulation do vary, there are normally about 5 million cells per cubic millimeter of blood. (A cubic millimeter [mm^3] is a very tiny drop of blood, almost not enough to be seen.) When the number of RBC/mm^3 increases, blood viscosity increases. Similarly, as the number of RBCs decreases, blood thins and flows more rapidly. However, let's not get carried away talking about RBC *numbers*. Although their numbers are important, it is the amount of hemoglobin in the bloodstream at any time that really determines how well the erythrocytes are performing their role of oxygen transport.

The more hemoglobin molecules the RBCs contain, the more oxygen they will be able to carry. So, perhaps the most accurate way of measuring the oxygen-carrying capacity of the blood is to determine how much hemoglobin it contains. A single red blood cell contains about 250 million hemoglobin molecules, each capable of binding 4 molecules of oxygen, so each of these tiny cells can carry about 1 billion molecules of oxygen! This information is astounding but not very practical. Much more important clinically is the fact that normal blood contains 12–18 grams (g) of hemoglobin per 100 milliliters (ml) of blood. The hemoglobin content is slightly higher in men (13–18 g/ml) than in women (12–16 g/ml).

HOMEOSTATIC IMBALANCE

A decrease in the oxygen-carrying ability of the blood, whatever the reason, is **anemia** (ah-ne′me-ah; "lacking blood"). Anemia may be the result of (1) a lower-than-normal *number* of RBCs or (2) abnormal or deficient *hemoglobin content* in the RBCs. Several types of anemia are classified and described briefly in **Table 10.1**, but one of these, *sickle cell anemia,* deserves a little more attention because people with this genetic disorder are frequently seen in hospital emergency rooms.

In **sickle cell anemia (SCA),** the abnormal hemoglobin formed becomes spiky and sharp (Figure 10.3b) when the RBCs unload oxygen molecules or when the oxygen content of the blood is lower than normal, as during vigorous exercise, anxiety, or other stressful situations. The stiff,

deformed (crescent-shaped) erythrocytes rupture easily and dam up in small blood vessels. These events interfere with oxygen delivery (leaving victims gasping for air) and cause extreme pain. It is amazing that this havoc results from a change in just *one* of the amino acids in two of the four polypeptide chains of the hemoglobin molecule!

Sickle cell anemia occurs chiefly in black people who live in the malaria belt of Africa and among their descendants. Apparently, the same gene that causes sickling makes red blood cells infected by the malaria-causing parasite stick to the capillary walls and then lose potassium, an essential nutrient for survival of the parasite. Hence, the malaria-causing parasite is prevented from multiplying within the red blood cells, and individuals with the sickle cell gene have a better chance of surviving where malaria is prevalent. Only those carrying two copies of the defective gene have sickle cell anemia. Those carrying just one sickling gene have **sickle cell trait (SCT);** they generally do not display the symptoms but can pass on the sickling gene to their offspring.

An excessive or abnormal increase in the number of erythrocytes is **polycythemia** pol″e-si-the′me-ah). Polycythemia may result from bone marrow cancer (*polycythemia vera*). It may also be a normal physiologic (homeostatic) response to living at high altitudes, where the air is thinner and less oxygen is available (*secondary polycythemia*). The major problem that results from excessive numbers of RBCs is increased blood viscosity, which causes blood to flow sluggishly in the body and impairs circulation.

Leukocytes

Although **leukocytes,** or **white blood cells (WBCs),** are far less numerous than red blood cells, they are crucial to body defense against disease. On average, there are 4,800 to 10,800 WBC/mm^3, and they account for less than 1 percent of total blood volume. White blood cells are the only complete cells in blood; that is, they contain nuclei and the usual organelles.

Leukocytes form a protective, movable army that helps defend the body against damage by bacteria, viruses, parasites, and tumor cells. As such, they have some very special characteristics. Red blood cells are confined to the bloodstream and carry out their functions in the blood. White blood cells, by contrast, are able to slip into and out of the blood vessels—a process called **diapedesis** (di″ah-peh-de′sis; "leaping across"). The circulatory system is simply their means of transportation to areas of the body where their services are needed for inflammatory or immune responses (as described in Chapter 12).

In addition, WBCs can locate areas of tissue damage and infection in the body by responding to certain chemicals that diffuse from the damaged cells. This capability is called **positive chemotaxis** (ke″mo-tax′is). Once they have "caught the scent," the WBCs move through the tissue spaces by **amoeboid** (ah-me′boid) **motion** (they form flowing cytoplasmic extensions that help move them along). By following the diffusion gradient, they pinpoint areas of tissue damage and rally round in large numbers to destroy microorganisms and dispose of dead cells.

Whenever WBCs mobilize for action, the body speeds up their production, and as many as twice the normal number of WBCs may appear in the blood within a few hours. A total WBC count above 11,000 cells/mm^3 is referred to as **leukocytosis** (lu″ko-si-to′sis). Leukocytosis generally indicates that a bacterial or viral infection is stewing in the body. The opposite condition, **leukopenia** (lu″ko-pe′ne-ah), is an abnormally low WBC count. It is commonly caused by certain drugs, such as corticosteroids and anticancer agents.

HOMEOSTATIC IMBALANCE

Leukocytosis is a normal and desirable response to infectious threats to the body. By contrast, the excessive production of abnormal WBCs that occurs in infectious mononucleosis and leukemia is distinctly pathological. In **leukemia** (lu-ke′me-ah), literally "white blood," the bone marrow becomes cancerous, and huge numbers of WBCs are turned out rapidly. Although this might not appear to present a problem, the "newborn" WBCs are immature and incapable of carrying out their normal protective functions. Consequently, the body becomes the easy prey of disease-causing bacteria and viruses. Additionally, because other blood cell lines are crowded out, severe anemia and bleeding problems result.

WBCs are classified into two major groups—granulocytes and agranulocytes—depending on whether or not they contain visible granules in their cytoplasm. Specific characteristics and microscopic views of the leukocytes can be seen in **Table 10.2**.

Table 10.2 Characteristics of Formed Elements of the Blood

Cell type	Occurrence in blood (per mm^3)	Cell anatomy*	Function
ERYTHROCYTES (red blood cells) 	4–6 million	Salmon-colored biconcave disks; anucleate; literally, sacs of hemoglobin; most organelles have been ejected	Transport oxygen bound to hemoglobin molecules; also transport small amount of carbon dioxide
LEUKOCYTES (white blood cells)	4,800–10,800		
Granulocytes			
• Neutrophils 	3,000–7,000 (40–70% of WBCs)	Cytoplasm stains pale pink and contains fine granules, which are difficult to see; deep purple nucleus consists of three to seven lobes connected by thin strands of nucleoplasm	Active phagocytes; number increases rapidly during short-term or acute infections
• Eosinophils 	100–400 (1–4% of WBCs)	Red coarse cytoplasmic granules; figure-8 or bilobed nucleus stains blue-red	Kill parasitic worms by deluging them with digestive enzymes; play a complex role in allergy attacks
• Basophils 	20–50 (0–1% of WBCs)	Cytoplasm has a few large blue-purple granules; U- or S-shaped nucleus with constrictions, stains dark blue	Release histamine (vasodilator chemical), at sites of inflammation; contain heparin, an anticoagulant
Agranulocytes			
• Lymphocytes 	1,500–3,000 (20–45% of WBCs)	Cytoplasm pale blue and appears as thin rim around nucleus; spherical (or slightly indented) dark purple-blue nucleus	Part of immune system; one group (B lymphocytes) produces antibodies; other group (T lymphocytes) involved in graft rejection, fighting tumors and viruses, via direct cell attack
• Monocytes 	100–700 (4–8% of WBCs)	Abundant gray-blue cytoplasm; dark blue-purple nucleus often kidney-shaped	Active phagocytes that become macrophages in the tissues; long-term "cleanup team"; increase in number during chronic infections such as tuberculosis
PLATELETS 	150,000–400,000	Essentially irregularly shaped cell fragments; stain deep purple	Needed for normal blood clotting; initiate clotting cascade by clinging to torn area

*Appearance when stained with Wright's stain.

Granulocytes (gran′u-lo-sīts″) are granule-containing WBCs. They have lobed nuclei, which typically consist of several rounded nuclear areas connected by thin strands of nuclear material. The granules in their cytoplasm stain specifically with Wright's stain. The granulocytes include the neutrophils (nu′tro-filz), eosinophils (e″o-sin′o-filz), and basophils (ba′so-filz).

1. **Neutrophils** are the most numerous of the WBCs. They have a multilobed nucleus and very fine granules that respond to both acidic and basic stains. Consequently, the cytoplasm as a whole stains pink. Neutrophils are avid phagocytes at sites of acute infection. They are particularly partial to bacteria and fungi, which they kill during a *respiratory burst* that deluges the phagocytized invaders with a potent brew of oxidizing substances (bleach, hydrogen peroxide, and others).
2. **Eosinophils** have a blue-red nucleus that resembles an old-fashioned telephone receiver and sport coarse, lysosome-like, brick-red cytoplasmic granules. Their number increases rapidly during infections by parasitic worms (flatworms, tapeworms, etc.) ingested in food (raw fish) or entering via the skin. When eosinophils encounter a parasitic worm prey, they gather around and release enzymes from their cytoplasmic granules onto the parasite's surface, digesting it away.
3. **Basophils,** the rarest of the WBCs, have large histamine-containing granules that stain dark blue. **Histamine** is an inflammatory chemical that makes blood vessels leaky and attracts other WBCs to the inflammatory site.

The second group of WBCs, **agranulocytes,** lack *visible* cytoplasmic granules. Their nuclei are closer to the norm—that is, they are spherical, oval, or kidney-shaped. The agranulocytes include lymphocytes (lim′fo-sīts) and monocytes (mon′o-sīts).

1. **Lymphocytes** have a large, dark purple nucleus that occupies most of the cell volume. Only slightly larger than RBCs, lymphocytes tend to take up residence in lymphatic tissues, where they play an important role in the immune response. They are the second most numerous leukocytes in the blood.
2. **Monocytes** are the largest of the WBCs. Except for their more abundant cytoplasm and distinctive U- or kidney-shaped nucleus, they resemble large lymphocytes. When they migrate into the tissues, they change into macrophages with huge appetites. Macrophages are important in fighting chronic infections, such as tuberculosis.

Students are often asked to list the WBCs in order of relative abundance in the blood—from most to least. The following phrase may help you with this task: **N**ever **l**et **m**onkeys **e**at **b**ananas (neutrophils, lymphocytes, monocytes, eosinophils, basophils).

Platelets

Platelets are not cells in the strict sense. They are fragments of bizarre multinucleate cells called **megakaryocytes** (meg″ah-kar′e-o-sīts), which pinch off thousands of anucleate platelet "pieces" that quickly seal themselves off from the surrounding fluids. The platelets appear as darkly staining, irregularly shaped bodies scattered among the other blood cells. The normal platelet count in blood is about 300,000/mm^3. As indicated in Table 10.2, platelets are needed for the clotting process that occurs in plasma when blood vessels are ruptured or broken. (We explain this process on pp. 346–348.)

DID YOU GET IT?

4. What is the role of hemoglobin in the red blood cell?
5. Which white blood cells are most important in body immunity?
6. If you had a severe infection, would you expect your WBC count to be closest to 5,000, 10,000, or 15,000/mm^3?
7. Little Lisa is pale and listless. What disorder of erythrocytes might she be suffering from?

For answers, see Appendix D.

Hematopoiesis (Blood Cell Formation)

✓ Explain the role of the hemocytoblast.

Blood cell formation, or **hematopoiesis** (hem″ah-to-poi-e′sis), occurs in red bone marrow, or *myeloid* tissue. In adults, this tissue is found chiefly in the flat bones of the skull and pelvis, the ribs, sternum, and proximal epiphyses of the humerus and femur. Each type of blood cell is produced in different numbers in response to changing body needs and different stimuli. After they mature, they are discharged into the blood

vessels surrounding the area. On average, the red marrow turns out, each and every day, an ounce of new blood containing 100 billion new cells.

All the formed elements arise from a common type of *stem cell,* the **hemocytoblast** (he″mo-si′to-blast; "blood cell former"), which resides in the red bone marrow. Their development differs, however, and once a cell is committed to a specific blood pathway, it cannot change. As indicated in the flowchart in **Figure 10.4**, the hemocytoblast forms two types of descendants—the *lymphoid stem cell,* which produces lymphocytes, and the *myeloid stem cell,* which can produce all other classes of formed elements.

Formation of Red Blood Cells

Because they are anucleate, RBCs are unable to synthesize proteins, grow, or divide. As they age, RBCs become more rigid and begin to fragment, or fall apart, in 100 to 120 days. Their remains are eliminated by phagocytes in the spleen, liver, and other body tissues. Lost cells are replaced more or less continuously by the division of hemocytoblasts in the red bone marrow. The developing RBCs divide many times and then begin synthesizing huge amounts of hemoglobin. Suddenly, when enough hemoglobin has been accumulated, the nucleus and most organelles are ejected and the cell collapses inward. The result is the young RBC, called a *reticulocyte* (rĕ-tik′u-lo-sīt) because it still contains some rough endoplasmic reticulum (ER). The reticulocytes enter the bloodstream to begin their task of transporting oxygen. Within 2 days of release, they have ejected the remaining ER and have become fully functioning erythrocytes. The entire developmental process from hemocytoblast to mature RBC takes 3 to 5 days.

The rate of erythrocyte production is controlled by a hormone called **erythropoietin** (ĕ-rith″ro-poi-e′tin). Normally a small amount of erythropoietin circulates in the blood at all times, and red blood cells are formed at a fairly constant rate. Although the liver produces some, the kidneys play the major role in producing this hormone. When blood levels of oxygen begin to decline for any reason, the kidneys step up their release of erythropoietin. Erythropoietin targets the bone marrow, prodding it into "high gear" to turn out more RBCs. Follow this sequence of events in **Figure 10.5**.

As you might expect, an overabundance of erythrocytes, or an excessive amount of oxygen in the bloodstream, depresses erythropoietin release and red blood cell production. An important point to remember is that it is *not* the relative number of RBCs in the blood that controls RBC production. Control is based on their ability to transport enough oxygen to meet the body's demands.

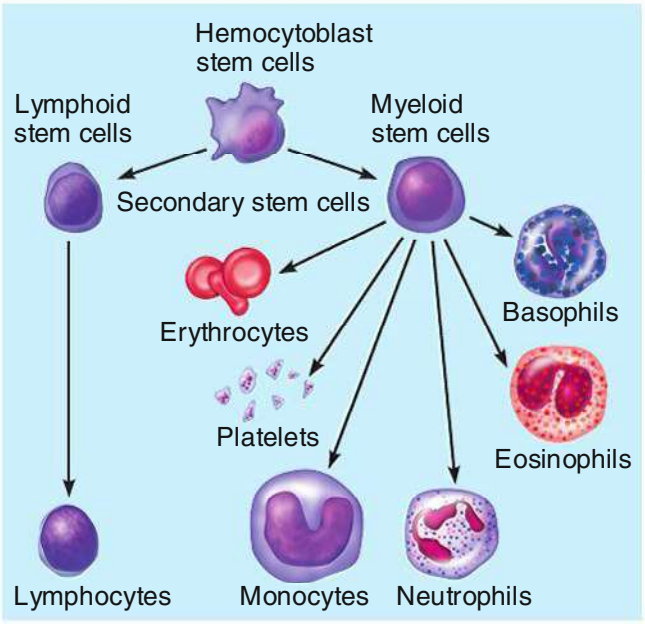


Figure 10.4 The development of blood cells. All blood cells differentiate from hemocytoblast stem cells in red bone marrow. The population of stem cells renews itself by mitosis. Some daughter cells become lymphoid stem cells, which develop into two classes of lymphocytes that function in the immune response. All other blood cells differentiate from myeloid stem cells.

Formation of White Blood Cells and Platelets

Like erythrocyte production, the formation of leukocytes and platelets is stimulated by hormones. These *colony stimulating factors (CSFs)* and *interleukins* not only prompt red bone marrow to turn out leukocytes, but also marshal up an army of WBCs to ward off attacks by enhancing the ability of mature leukocytes to protect the body. Apparently, they are released in response to specific chemical signals in the environment, such as inflammatory chemicals and certain bacteria or their toxins. The hormone *thrombopoietin* accelerates the production of platelets from megakaryocytes, but little is known about how that process is regulated.

When bone marrow problems or disease conditions such as aplastic anemia or leukemia are

Q: *Why do many people with advanced kidney disease become anemic?*

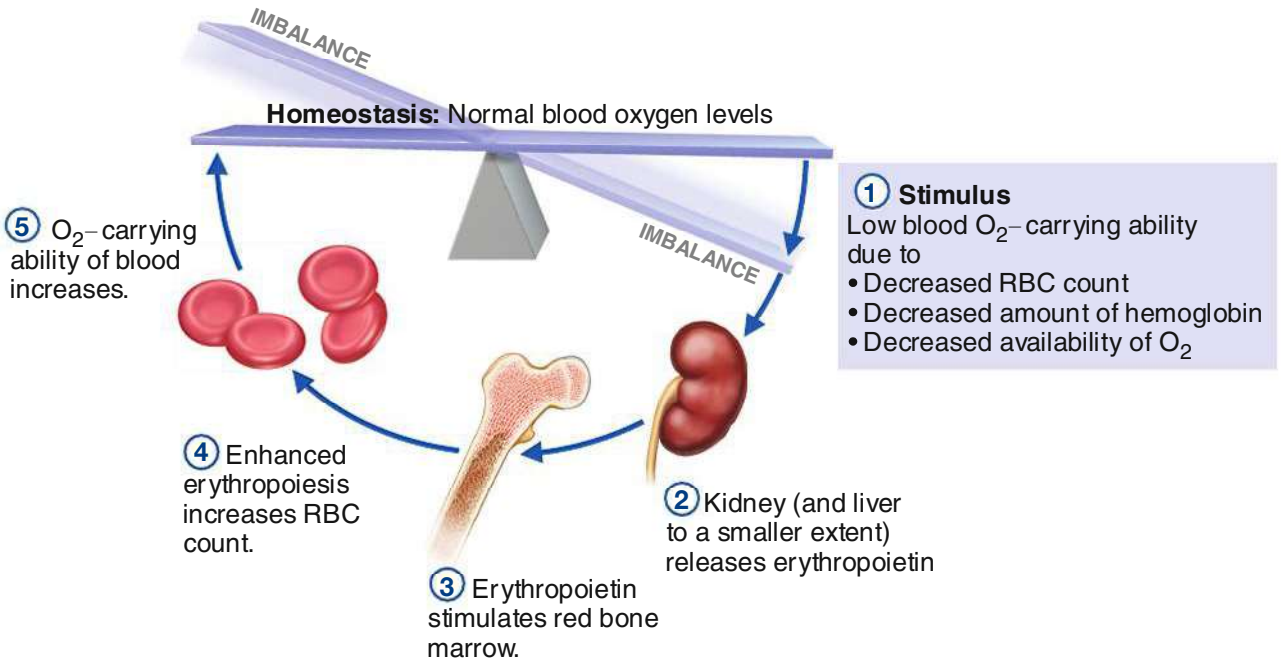


Figure 10.5 **Mechanism for regulating the rate of RBC production.**

suspected, a special needle is used to withdraw a small sample of red marrow from one of the flat bones (ilium or sternum) close to the body surface. This procedure provides cells for a microscopic examination called a *bone marrow biopsy.*

DID YOU GET IT?

8. What is the name of the stem cell that gives rise to all formed elements?

9. What property of RBCs dooms them to a limited life span of about 120 days?

10. How is the production of platelets different from that of all other formed elements?

For answers, see Appendix D.

Hemostasis

✓ Describe the blood-clotting process.

✓ Name some factors that may inhibit or enhance the blood-clotting process.

Normally, blood flows smoothly past the intact lining (endothelium) of blood vessel walls. But if a blood vessel wall breaks, a series of reactions is set in motion to accomplish **hemostasis** (*hem* = blood; *stasis* = standing still), or stoppage of bleeding. This response, which is fast and localized, involves many substances normally present in plasma, as well as some that are released by platelets and injured tissue cells.

Hemostasis involves three major phases, which occur in rapid sequence: **vascular spasms, platelet plug formation,** and **coagulation,** or **blood clotting.** Blood loss at the site is permanently prevented when fibrous tissue grows into the clot and seals the hole in the blood vessel.

Basically, hemostasis occurs as follows (**Figure 10.6**):

① **Vascular spasms occur.** The immediate response to blood vessel injury is vasoconstriction, which causes that blood vessel to go into spasms. The spasms narrow the blood vessel at

A: *The kidneys produce most of the erythropoietin that stimulates red blood cell production by the bone marrow.*

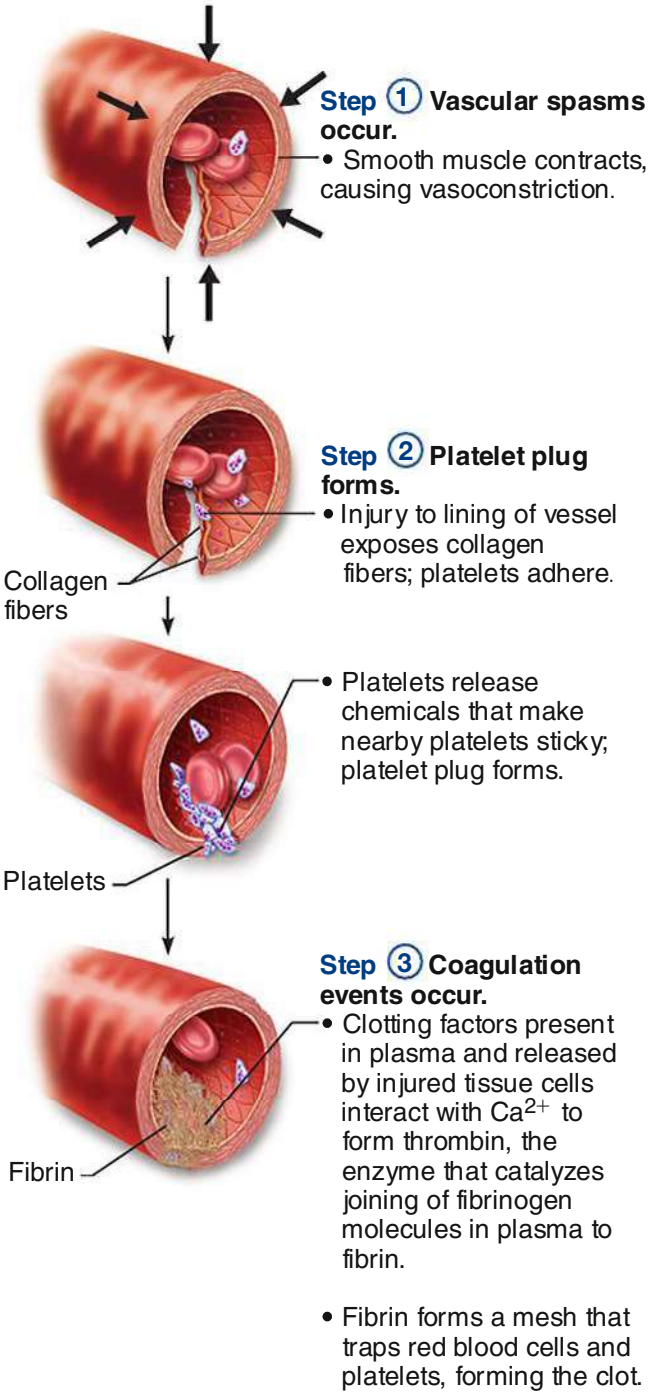


Figure 10.6 **Events of hemostasis.**

Figure 10.7 **Fibrin clot.** Scanning electron micrograph (artificially colored) of red blood cells trapped in a mesh of fibrin threads.

that point, decreasing blood loss until clotting can occur. (Other factors causing vessel spasms include direct injury to the smooth muscle cells, stimulation of local pain receptors, and release of serotonin by anchored platelets.)

② **Platelet plug forms.** Platelets are repelled by an intact endothelium, but when it is broken so that the underlying collagen fibers are exposed, the platelets become "sticky" and cling to the damaged site. Anchored platelets release chemicals that enhance the vascular spasms and attract more platelets to the site. As more and more platelets pile up, a small mass called a *platelet plug,* or *white thrombus,* forms.

③ **Coagulation events occur.** At the same time, the injured tissues are releasing **tissue factor (TF),** which interacts with **PF_3**, a phospholipid that coats the surfaces of the platelets. This combination interacts with other blood protein clotting factors and calcium ions (Ca^{2+}) to form an activator that leads to the formation of **thrombin,** an enzyme. Thrombin then joins soluble **fibrinogen** (fi-brin′o-jen) proteins into long, hairlike molecules of insoluble **fibrin**. Fibrin forms a meshwork that traps the RBCs and forms the basis of the clot (**Figure 10.7**). Within the hour, the clot begins to retract, squeezing **serum** (plasma minus the clotting proteins) from the mass and pulling the ruptured edges of the blood vessel closer together.

Normally, blood clots within 3 to 6 minutes. As a rule, once the clotting cascade has started, the triggering factors are rapidly inactivated to prevent widespread clotting ("solid blood"). Eventually, the endothelium regenerates, and the clot is broken down. Once these events of the clotting cascade were understood, it became clear that placing a sterile gauze over a cut or applying pressure to a wound would speed up the clotting process. The

gauze provides a rough surface to which the platelets can adhere, and the pressure fractures cells, increasing the release of tissue factor locally.

Disorders of Hemostasis

HOMEOSTATIC IMBALANCE

The two major disorders of hemostasis—undesirable clot formation and bleeding disorders—are at opposite poles.

Undesirable Clotting

Despite the body's safeguards against abnormal clotting, undesirable clots sometimes form in intact (unbroken) blood vessels, particularly in the legs. A clot that develops and persists in an unbroken blood vessel is called a **thrombus** (throm′bus). If the thrombus is large enough, it may prevent blood from flowing to the cells beyond the blockage. For example, if the blockage forms in the blood vessels serving the heart (*coronary thrombosis*), the consequences may be death of heart muscle and a fatal heart attack. If a thrombus breaks away from the vessel wall and floats freely in the bloodstream, it becomes an **embolus** (em′bo-lus; plural, *emboli*). An embolus is usually no problem unless or until it lodges in a blood vessel too narrow for it to pass through. For example, a *cerebral embolus* may cause a *stroke.*

Undesirable clotting may be caused by anything that roughens the endothelium of a blood vessel and encourages clinging of platelets, such as severe burns, physical blows, or an accumulation of fatty material. Slowly flowing blood, or blood pooling, is another risk factor, especially in immobilized patients. In this case, clotting factors are not washed away as usual and accumulate so that clot formation becomes possible. A number of anticoagulants, the most important of which are aspirin, heparin, and dicumarol, are used clinically for thrombus-prone patients.

Bleeding Disorders

The most common causes of abnormal bleeding are platelet deficiency (thrombocytopenia) and deficits of some of the clotting factors, such as might result from impaired liver function or certain genetic conditions.

Thrombocytopenia results from an insufficient number of circulating platelets. Even normal movements cause spontaneous bleeding from small blood vessels. This is evidenced by many small purplish blotches, called **petechiae** (pe-te′ke-e), on the skin. It can arise from any condition that suppresses the bone marrow, such as bone marrow cancer, radiation, or certain drugs.

When the liver is unable to synthesize its usual supply of clotting factors, abnormal and often severe bleeding episodes occur. If vitamin K (needed by the liver cells to produce the clotting factors) is deficient, the problem is easily corrected with vitamin K supplements. However, when liver function is severely impaired (as in hepatitis and cirrhosis), only whole blood transfusions are helpful. Transfusions of concentrated platelets provide temporary relief from bleeding.

The term **hemophilia** (he″mo-fil′e-ah) applies to several different hereditary bleeding disorders that result from a lack of any of the factors needed for clotting. Commonly called "bleeder's disease," the hemophilias have similar signs and symptoms that begin early in life. Even minor tissue trauma results in prolonged bleeding and can be life-threatening. Repeated bleeding into joints causes them to become disabled and painful.

When a bleeding episode occurs, hemophiliacs are given a transfusion of fresh plasma or injections of the purified clotting factor they lack. Because hemophiliacs are absolutely dependent on one or the other of these therapies, some have become the victims of blood-transmitted viral diseases such as hepatitis and AIDS. (AIDS, acquired immune deficiency syndrome, is a condition of depressed immunity and is described in Chapter 12.) These problems have been largely resolved because of the availability of genetically engineered clotting factors and hepatitis vaccines.

DID YOU GET IT?

11. What factors enhance the risk of thrombus formation in intact blood vessels?

For the answer, see Appendix D.

Blood Groups and Transfusions

- ✓ Describe the ABO and Rh blood groups.
- ✓ Explain the basis for a transfusion reaction.

As we have seen, blood is vital for transporting substances through the body. When blood is lost,

Phlebotomy Technician

Phlebotomists must know where all the arteries and veins are located in the body.

"Phlebotomy is the most important procedure done for a medical laboratory," says Michael Coté, who supervises the phlebotomy staff at Palo Alto Veterans Administration Hospital in California. "To make accurate diagnosis and effective treatment possible, it's vital to draw a good blood sample, place it in a sterile container, and process it accurately in the lab. Without a high-quality specimen, none of this can happen."

Phlebotomy is not exactly a household word. It derives from the Greek terms for "vein" and "to cut." A phlebotomy technician, or phlebotomist, is trained to collect and process blood samples that will be subjected to laboratory analysis.

Coté appreciates how important it is for phlebotomists to understand anatomy and physiology. "Anatomy is a key requirement in phlebotomy training," he says, "because you have to learn where all the arteries and veins are located in the body. Some patients' veins are easy to find, but others have veins that are practically invisible. You need to know the right place to insert that needle. Although 90 percent of the blood we draw comes from the antecubital region inside the elbow, we may also draw blood from the cephalic vein in the forearm, or from veins in the hands."

Coté notes that knowledge of physiology is also important. "I have to be able to assess patients' overall health and physical condition, because this affects their ability to give an adequate blood sample and may demand that a different needle size be used to draw the blood sample. People who are dehydrated can be difficult because their blood pressure is lower and venous return is impaired. Patients with poor circulation are also harder to work with. The blood tends to stay in the body trunk rather than flowing freely into the extremities because they're cold, and it's difficult to get a good return from veins in the arms. Cancer patients often show increased sensitivity to pain, so we have to be very gentle and use the smallest needle possible." Patients with a history of drug abuse pose other challenges. "Frequent 'sticking' with needles causes scar tissue to form. You can tell people who have used intravenous drugs—their veins feel rock-hard and are much more difficult to penetrate with a needle."

Coté says that a good phlebotomist must also possess effective interpersonal skills: "People are apprehensive about being stuck with needles, so you have to be patient and be able to put them at ease. Above all, you have to be confident. If the phlebotomist is nervous, the patient will sense it and get nervous too."

To become certified, a phlebotomy technician must be a high school graduate, complete a phlebotomy training program or acquire equivalent experience, and pass a certification exam offered by the American Society for Clinical Pathologists.

Anatomy is a key requirement in phlebotomy training.

Accreditation procedures for phlebotomists vary from state to state. For more information, contact the American Society for Clinical Pathologists at

33 West Monroe Street,
Suite 1600
Chicago, IL 60603
(312) 541-4999
http://www.ascp.org

For additional information on this career, click the Focus on Careers link at **www.anatomyandphysiology.com.**

the blood vessels constrict and the bone marrow steps up blood cell formation in an attempt to keep the circulation going. However, the body can compensate for a loss of blood volume only up to a certain limit. Losses of 15 to 30 percent lead to pallor and weakness. Loss of over 30 percent causes severe shock, which can be fatal.

Whole blood transfusions are routinely given to replace substantial blood loss and to treat severe anemia or thrombocytopenia. The usual blood bank procedure involves collecting blood from a donor and mixing it with an anticoagulant to prevent clotting. The treated blood can be stored (refrigerated at 4°C, or 39.2°F) for about 35 days until needed.

Human Blood Groups

Although whole blood transfusion can save lives, people have different blood groups, and transfusing incompatible or mismatched blood can be fatal. How so? The plasma membranes of RBCs, like those of all other body cells, bear genetically determined proteins (antigens), which identify each person as unique. An **antigen** (an′tĭ-jen) is a substance that the body recognizes as foreign; it stimulates the immune system to release antibodies or use other means to mount a defense against it. Most antigens are foreign proteins, such as those that are part of viruses or bacteria that have managed to invade the body. Although each of us tolerates our own cellular (self) antigens, one person's RBC proteins will be recognized as foreign if transfused into another person with different RBC antigens. The "recognizers" are **antibodies** present in the plasma that attach to RBCs bearing surface antigens different from those on the patient's (blood recipient's) RBCs. Binding of the antibodies causes the foreign RBCs to clump, a phenomenon called **agglutination*** (ah-gloo″tĭ-na′shun), which leads to the clogging of small blood vessels throughout the body. During the next few hours, the foreign RBCs are lysed (ruptured), and their hemoglobin is released into the bloodstream.

Although the transfused blood is unable to deliver the increased oxygen-carrying capacity hoped for and some tissue areas may be deprived of blood, the most devastating consequence of severe transfusion reactions is that the freed hemoglobin molecules may block the kidney tubules, causing kidney failure and death. Transfusion reactions can also cause fever, chills, nausea, and vomiting, but in the absence of kidney shutdown these reactions are rarely fatal. Treatment is aimed at preventing kidney damage by infusing fluids to dilute and dissolve the hemoglobin and diuretics to flush it out of the body in urine.

There are over 30 common RBC antigens in humans, so each person's blood cells can be classified into several different blood groups. However, it is the antigens of the ABO and Rh blood groups that cause the most vigorous transfusion reactions. We describe these two blood groups here.

As shown in **Table 10.3**, the **ABO blood groups** are based on which of two antigens, type A or type B, a person inherits. Absence of both antigens results in type O blood, presence of both antigens leads to type AB, and the presence of either A or B antigen yields type A or B blood, respectively. In the ABO blood group, antibodies form during infancy against the ABO antigens *not* present on your own RBCs. As shown in the table, a baby with neither the A nor the B antigen (group O) forms both anti-A and anti-B antibodies; those with type A antigens (group A) form anti-B antibodies, and so on.

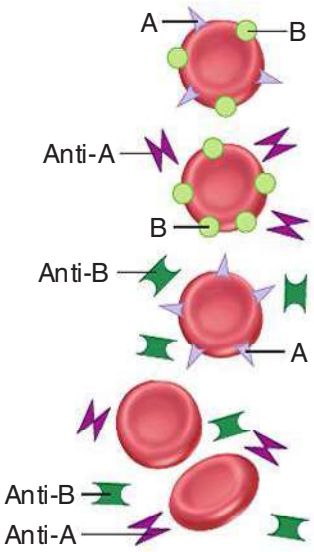
The **Rh blood groups** are so named because one of the eight Rh antigens (agglutinogen D) was originally identified in **Rh**esus monkeys. Later the same antigen was discovered in humans. Most Americans are Rh^+ (Rh positive), meaning that their RBCs carry the Rh antigen. Unlike the antibodies of the ABO system, anti-Rh antibodies are *not* automatically formed and present in the blood of Rh^- (Rh negative) individuals. However, if an Rh^- person receives mismatched blood (that is, Rh^+), shortly after the transfusion his or her immune system becomes sensitized and begins producing antibodies (anti-Rh^+ antibodies) against the foreign blood type.

Hemolysis (rupture of RBCs) does not occur with the first transfusion because it takes time for the body to react and start making antibodies. However, the second time and every time thereafter, a typical transfusion reaction occurs in which the patient's antibodies attack and rupture the donor's Rh^+ RBCs.

An important Rh-related problem occurs in pregnant Rh^- women who are carrying Rh^+ babies.

*The RBC antigens that promote this clumping are sometimes called **agglutinogens** (ag″loo-tin′o-jenz), and the antibodies that bind them together are called **agglutinins** (ag″loo′tĭ-ninz).

Table 10.3 ABO Blood Groups

Blood group	Frequency (% U.S. population) White	Black	Asian	Native American	RBC antigens (agglutinogens)	Illustration	Plasma antibodies (agglutinins)	Blood that can be received
AB	4	4	5	<1	A B		None	A, B, AB, O Universal recipient
B	11	20	27	4	B		Anti-A	B, O
A	40	27	28	16	A		Anti-B	A, O
O	45	49	40	79	None		Anti-A Anti-B	O Universal donor

The *first* such pregnancy usually results in the delivery of a healthy baby. But because the mother is sensitized by Rh^+ antigens that have passed through the placenta into her bloodstream, she will form anti-Rh^+ antibodies unless treated with RhoGAM shortly after giving birth. RhoGAM is an immune serum that prevents this sensitization and her subsequent immune response. If she is not treated and becomes pregnant again with an Rh^+ baby, her antibodies will cross through the placenta and destroy the baby's RBCs, producing a condition known as *hemolytic disease of the newborn*. The baby is anemic and becomes hypoxic and cyanotic (the skin takes on a blue cast). Brain damage and even death may result unless fetal transfusions are done before birth to provide more RBCs for oxygen transport.

Blood Typing

The importance of determining the blood group of both the donor and the recipient *before* blood is transfused is glaringly obvious. The general procedure for determining ABO blood type is briefly outlined in **Figure 10.8**. Essentially, it involves testing the blood by mixing it with two different types of immune serum—anti-A and anti-B. Agglutination occurs when RBCs of a group A person are mixed with the anti-A serum but not when they are mixed with the anti-B serum. Likewise, RBCs of type B blood are clumped by anti-B serum but not by anti-A serum. Because it is critical that blood groups be compatible, cross matching is also done. *Cross matching* involves testing for agglutination of donor RBCs by the recipient's serum and of the recipient's RBCs by the donor serum. Typing for the Rh factors is done in the same manner as ABO blood typing.

DID YOU GET IT?

12. What are the classes of human blood groups based on?

13. What is the probable result of infusing mismatched blood?

14. Cary is bleeding profusely after being hit by a truck as he was pedaling his bike home. At the hospital, the nurse asked him whether he knew his blood type. He told her that he "had the same blood as most other people." What is his ABO blood type?

15. What is the difference between an antigen and an antibody?

For answers, see Appendix D.

Q: *Which blood types can be transfused into a person with type B blood?*

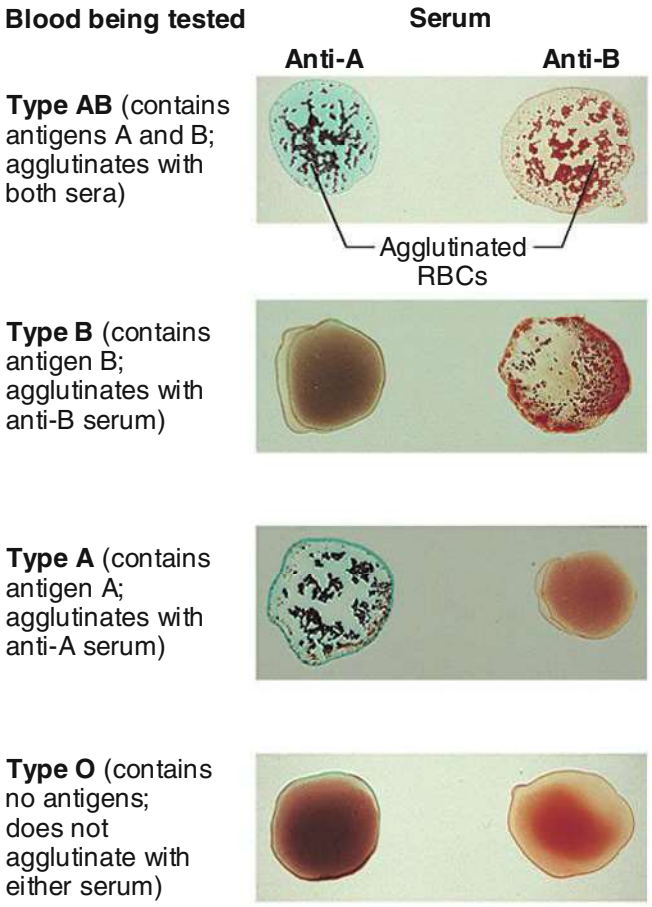


Figure 10.8 Blood typing of ABO blood groups. When serum containing anti-A or anti-B antibodies is added to a blood sample diluted with saline, agglutination will occur between the antibody and the corresponding antigen (if present).

Developmental Aspects of Blood

- ✓ Explain the basis of physiologic jaundice seen in some newborn babies.
- ✓ Indicate blood disorders that increase in frequency in the aged.

In the young embryo, the entire circulatory system develops early. Before birth, there are many sites of blood cell formation—the fetal liver and spleen, among others—but by the seventh month of development, the fetus's red marrow has become the chief site of hematopoiesis, and it remains so throughout life. Generally, embryonic blood cells are circulating in the newly formed blood vessels by day 28 of development. Fetal hemoglobin (HbF) differs from the hemoglobin formed after birth. It has a greater ability to pick up oxygen, a characteristic that is highly desirable because fetal blood is less oxygen rich than that of the mother. After birth, fetal blood cells are gradually replaced by RBCs that contain the more typical hemoglobin A (HbA). In situations in which the fetal RBCs are destroyed at such a rapid rate that the immature infant liver cannot rid the body of hemoglobin breakdown products in the bile fast enough, the infant becomes *jaundiced* (jawn′dist). This type of jaundice generally causes no major problems and is referred to as **physiologic jaundice,** to distinguish it from more serious disease conditions that result in jaundiced, or yellowed, tissues.

HOMEOSTATIC IMBALANCE

Various congenital diseases result from genetic factors (such as hemophilia and sickle cell anemia) and from interactions with maternal blood factors (such as hemolytic disease of the newborn). Dietary factors can lead to abnormalities in blood cell formation as well as hemoglobin production. Iron-deficiency anemia is especially common in women because of their monthly blood loss during menses. The young and the old are particularly at risk for leukemia.

With increasing age, chronic types of leukemias, anemias, and diseases involving undesirable clot formation are more prevalent. However, these are usually secondary to disorders of the heart, blood vessels, or immune system. The elderly are particularly at risk for pernicious anemia because the stomach mucosa (which produces intrinsic factor) atrophies with age.

DID YOU GET IT?

16. How does fetal hemoglobin differ from that of the adult?

17. What blood-related disorders are particularly common in the elderly?

For answers, see Appendix D.

A: Types B and O.

Summary

Access more review material and fun learning activities online—visit **www.anatomyandphysiology.com** and select Essentials of Human Anatomy & Physiology, 10th edition. In addition, references to Interactive Physiology are included below.

iP = Interactive Physiology

Composition and Functions of Blood (pp. 337–346)

1. Blood is composed of a nonliving fluid matrix (plasma) and formed elements. It is scarlet to dull red, depending on the amount of oxygen carried. Normal adult blood volume is 5 to 6 liters.
2. Dissolved in plasma (primarily water) are nutrients, gases, hormones, wastes, proteins, salts, and so on. Plasma composition changes as body cells remove or add substances to it, but homeostatic mechanisms act to keep it relatively constant. Plasma makes up 55 percent of whole blood.
3. Formed elements, the living blood cells that make up about 45 percent of whole blood, include the following:
   a. Erythrocytes, or RBCs—disc-shaped, anucleate cells that transport oxygen bound to their hemoglobin molecules. Their life span is 100 to 120 days.
   b. Leukocytes, or WBCs—amoeboid cells involved in protecting the body.
   c. Platelets—cell fragments that act in blood clotting.
4. A decrease in oxygen-carrying ability of blood is anemia. Possible causes are decrease in number of functional RBCs or decrease in amount of hemoglobin they contain. Polycythemia is an excessive number of RBCs that may result from bone marrow cancer or a move to a location where less oxygen is available in the air (at high altitude, for example).
5. Leukocytes are nucleated cells, classed into two groups:
   a. Granulocytes include neutrophils, eosinophils, and basophils.
   b. Agranulocytes include monocytes and lymphocytes.
6. When bacteria, viruses, or other foreign substances invade the body, WBCs increase in number (leukocytosis) and fight them in various ways.
7. An abnormal decrease in number of WBCs is leukopenia. An abnormal increase in WBCs is seen in infectious mononucleosis and leukemia (cancer of blood-forming bone marrow).
8. All formed elements arise in red bone marrow from a common stem cell, the hemocytoblast. However, their developmental pathways differ. The stimulus for hematopoiesis is hormonal (erythropoietin in the case of RBCs).

Hemostasis (pp. 346–348)

1. Stoppage of blood loss from an injured blood vessel, or hemostasis, involves three steps: vascular spasms, platelet plug formation, and blood clot formation.
2. Hemostasis is started by a tear or interruption in the blood vessel lining. Vascular spasms and accumulation of platelets at the site temporarily stop or slow blood loss. Platelet PF_3 and tissue factor initiate the clotting cascade, leading to formation of fibrin threads. Fibrin traps RBCs as they flow past, forming the clot.
3. Normally, clots are digested when a vessel has been permanently repaired. An attached clot that forms or persists in an unbroken blood vessel is a thrombus; a clot traveling in the bloodstream is an embolus.
4. Abnormal bleeding may reflect a deficit of platelets (thrombocytopenia), genetic factors (hemophilia), or inability of the liver to make clotting factors.

Blood Groups and Transfusions (pp. 348–352)

1. Blood groups are classified on the basis of proteins (antigens) on RBC membranes. Complementary antibodies may (or may not) be present in blood. Antibodies act to agglutinate (clump) and lyse foreign RBCs.
2. The blood group most commonly typed for is ABO. Type O is most common; least common is AB. ABO antigens are accompanied by preformed antibodies in plasma, which act against RBCs that have "foreign" antigens.
3. Rh factor is found in most Americans. Rh^- people do not have preformed antibodies to Rh^+ RBCs but form them once exposed to Rh^+ blood.

Developmental Aspects of Blood (p. 352)

1. Congenital blood defects include various types of hemolytic anemias and hemophilia. Incompatibility

between maternal and fetal blood can result in fetal cyanosis, resulting from destruction of fetal blood cells.

2. Fetal hemoglobin (HbF) binds more readily with oxygen than does HbA.
3. Physiologic jaundice in a newborn reflects immaturity of the infant's liver.
4. Leukemias are most common in the very young and very old. Older adults are also at risk for anemia and clotting disorders.

Review Questions

Multiple Choice

More than one choice may apply.

1. Which would lead to increased erythropoiesis?
   a. Chronic bleeding ulcer
   b. Reduction in respiratory ventilation
   c. Decreased level of physical activity
   d. Reduced blood flow to the kidneys
2. Sickling of RBCs can be induced in a person with sickle cell anemia by
   a. blood loss.
   b. vigorous exercise.
   c. stress.
   d. fever.
3. A child is diagnosed with sickle cell anemia. This means that
   a. one parent had sickle cell anemia.
   b. one parent carried the sickle cell gene.
   c. both parents had sickle cell anemia.
   d. both parents carried the sickle cell gene.
4. Polycythemia vera will result in
   a. overproduction of WBCs.
   b. exceptionally high blood volume.
   c. abnormally high blood viscosity.
   d. abnormally low hematocrit.
5. Which of the following is not typical of leukocytes?
   a. Amoeboid movement
   b. Phagocytic (some)
   c. Nucleated
   d. The most numerous cells in the bloodstream
6. The leukocyte that releases histamine and other inflammatory chemicals is the
   a. basophil.
   b. monocyte.
   c. eosinophil.
   d. neutrophil.
7. Which of the following formed elements are phagocytic?
   a. Erythrocytes
   b. Neutrophils
   c. Monocytes
   d. Lymphocytes
8. A condition resulting from thrombocytopenia is
   a. thrombus formation.
   b. embolus formation.
   c. petechiae.
   d. hemophilia.
9. Which of the following can cause problems in a transfusion reaction?
   a. Donor antibodies attacking recipient RBCs
   b. Clogging of small vessels by agglutinated clumps of RBCs
   c. Lysis of donated RBCs
   d. Blockage of kidney tubules
10. If an Rh^- mother becomes pregnant, when can hemolytic disease of the newborn *not possibly* occur in the child?
    a. If the child is Rh^-
    b. If the child is Rh^+
    c. If the father is Rh^+
    d. If the father is Rh^-
11. Plasma without the clotting proteins is called
    a. serum.
    b. whole blood.
    c. fibrin.
    d. tissue factor.
12. Albumin
    a. is a blood buffer.
    b. helps maintain blood's osmotic pressure.
    c. distributes body heat.
    d. transports certain molecules.

Short Answer Essay

13. What is the blood volume of an average-sized adult?
14. Name as many different categories of substances carried in plasma as you can.
15. Define *formed elements.* Which category is most numerous? Which makes up the buffy coat?
16. Define *anemia,* and give three possible causes.
17. Name the granular and agranular WBCs. Give the major function of each type in the body.
18. Name the formed elements that arise from myeloid stem cells. Name those arising from lymphoid stem cells.

19. What WBC type resides primarily in the tissues of the body?
20. Describe the process of hemostasis. Indicate what starts the process.
21. How can liver dysfunction cause bleeding disorders?
22. What are agglutinins?
23. Name the four ABO blood groups.
24. What is a transfusion reaction? Why does it happen?
25. Explain why an Rh^- person does not have a transfusion reaction on the first exposure to Rh^+ blood. Why is there a transfusion reaction the second time he or she receives the Rh^+ blood?
26. If you had a high hematocrit, would you expect your hemoglobin determination to be high or low? Why?

Critical Thinking and Clinical Application Questions

27. A patient on renal dialysis has a low RBC count. What hormone, secreted by the kidney, can be assumed to be deficient?
28. A bone marrow biopsy of Mr. Bongalonga, a man on a long-term drug therapy, shows an abnormally high percentage of nonhematopoietic connective tissue. What condition does this indicate? If the symptoms are critical, what short-term and long-term treatments are indicated? Will infusion of whole blood or packed red cells be more likely?
29. A woman comes to the clinic complaining of fatigue, shortness of breath, and chills. Blood tests show anemia, and a bleeding ulcer is diagnosed. What type of anemia is this?
30. A patient is diagnosed with bone marrow cancer and has a hematocrit of 70 percent. What is this condition called?
31. A middle-aged college professor from Boston is in the Swiss Alps studying astronomy. He arrived two days ago and plans to stay the entire year. However, he notices that he is short of breath when he walks up steps and that he tires easily with any physical activity. His symptoms gradually disappear; after two months, he feels fine. Upon returning to the United States, he has a complete physical exam and is told that his erythrocyte count is higher than normal. (a) Attempt to explain this finding. (b) Will his RBC count remain at this higher-than-normal level? Why or why not?
32. Why is someone more likely to bleed to death when an artery is cleanly severed than when it is crushed and torn?
33. Explain how fetal hemoglobin, HbF, enhances oxygen transfer across the placenta from the mother to the fetus.
34. Jenny, a healthy young woman, had a battery of tests during a physical for a new job. Her RBC count was at the higher end of the normal range at that time, but four weeks later it was substantially elevated beyond that. When asked if any circumstances had changed in her life, she admitted to taking up smoking. How might her new habit explain her higher RBC count?