

**HEMOLYSIS PROCESS AND CLASSIFICATION OF ANEMIA DISEASE,
MECHANISMS OF ORIGIN**

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Annotation: Hemolysis, breakdown or destruction of red blood cells so that the contained oxygen-carrying pigment hemoglobin is freed into the surrounding medium. Hemolysis occurs normally in a small percentage of red blood cells as a means of removing aged cells from the bloodstream and freeing heme for iron recycling. It also can be induced by exercise.

Key words: Hemolysis, anemia, blood cells, red blood cells, white blood cells, thrombocyte, hemolytic anemia.

In disease, hemolysis typically is associated with hemolytic anemia, whereby increased or accelerated hemolysis shortens the life span of red blood cells, causing them to die more quickly than they can be replenished by the bone marrow. Hemolytic anemia may involve either intravascular hemolysis, in which red blood cells are destroyed within the circulation, or extravascular hemolysis, in which the cells are destroyed in the liver or spleen. The cause may be intrinsic or extrinsic in nature. Causes of intrinsic hemolytic anemia include inherited defects in red blood cells, such as hereditary spherocytosis, sickle cell anemia, and thalassemia. Extrinsic disease may be caused by antibodies that attack and destroy red blood cells, such as in paroxysmal cold hemoglobinuria (a type of autoimmune hemolytic anemia); by diseases or infections that cause the spleen to become overactive (hypersplenism); or by other factors that result in the destruction of red blood cells, including chemicals, infections, trauma (such as repeated impact of the feet in running), venoms, or the toxic products of microorganisms. In erythroblastosis fetalis (hemolytic disease of the newborn), a mismatch in antibody compatibility between fetal and maternal blood results in the destruction of fetal red blood cells by maternal antibodies that cross the placenta.

Hemolysis may be produced in the laboratory by various physical agents: heat, freezing, flooding with water, sound. In certain situations it is used as a specific laboratory test for antigen-antibody reactions.

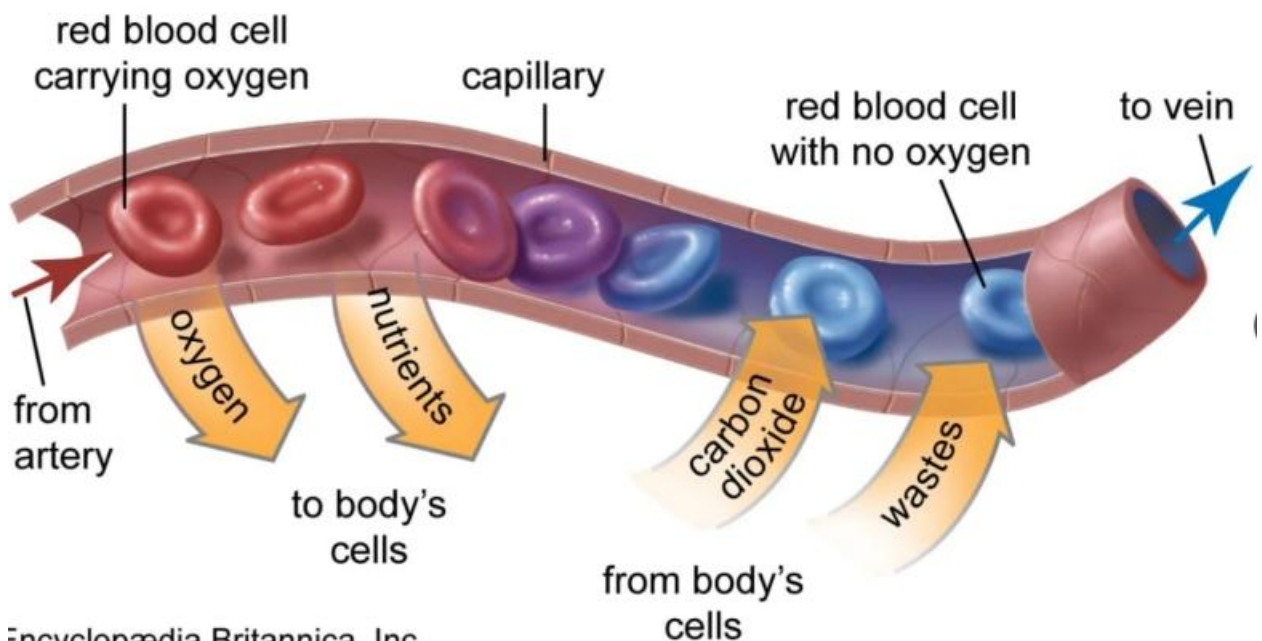


Picture 1. Hemolysis.

Red blood cell, cellular component of blood, millions of which in the circulation of vertebrates give the blood its characteristic colour and carry oxygen from the lungs to the tissues. The mature human red blood cell is small, round, and biconcave; it appears dumbbell-shaped in profile. The cell is flexible and assumes a bell shape as it passes through extremely small blood vessels. It is covered with a membrane composed of lipids and proteins, lacks a nucleus, and contains hemoglobin—a red iron-rich protein that binds oxygen.

The function of the red cell and its hemoglobin is to carry oxygen from the lungs or gills to all the body tissues and to carry carbon dioxide, a waste product of metabolism, to the lungs, where it is excreted. In invertebrates, oxygen-carrying pigment is carried free in the plasma; its concentration in red cells in vertebrates, so that oxygen and carbon dioxide are exchanged as gases, is more efficient and represents an important evolutionary development. The mammalian red cell is further adapted by lacking a nucleus—the amount of oxygen required by the cell for its own metabolism is thus very low, and most oxygen carried can be freed into the tissues. The biconcave shape of the cell allows oxygen exchange at a constant rate over the largest possible area.

The red cell develops in bone marrow in several stages: from a hemocytoblast, a multipotential cell in the mesenchyme, it becomes an erythroblast (normoblast); during two to five days of development, the erythroblast gradually fills with hemoglobin, and its nucleus and mitochondria (particles in the cytoplasm that provide energy for the cell) disappear. In a late stage the cell is called a reticulocyte, which ultimately becomes a fully mature red cell. The average red cell in humans lives 100–120 days; there are some 5.2 million red cells per cubic millimetre of blood in the adult human.



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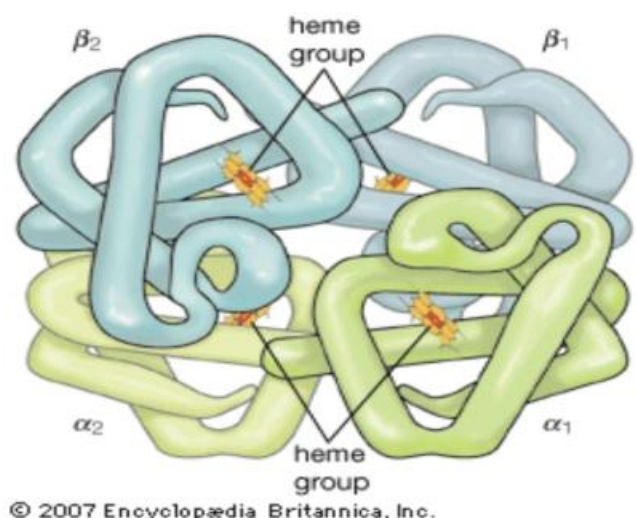
Picture 2, capillary

Though red cells are usually round, a small proportion are oval in the normal person, and in certain hereditary states a higher proportion may be oval. Some diseases also display red cells of abnormal shape—e.g., oval in pernicious anemia, crescent-shaped in sickle cell anemia, and with

projections giving a thorny appearance in the hereditary disorder acanthocytosis. The number of red cells and the amount of hemoglobin vary among different individuals and under different conditions; the number is higher, for example, in persons who live at high altitudes and in the disease polycythemia. At birth the red cell count is high; it falls shortly after birth and gradually rises to the adult level at puberty.

Hemoglobin, iron-containing protein in the blood of many animals—in the red blood cells (erythrocytes) of vertebrates—that transports oxygen to the tissues. Hemoglobin forms an unstable reversible bond with oxygen. In the oxygenated state, it is called oxyhemoglobin and is bright red; in the reduced state, it is purplish blue.

Hemoglobin develops in cells in the bone marrow that become red blood cells. When red cells die, hemoglobin is broken up: iron is salvaged, transported to the bone marrow by proteins called transferrins, and used again in the production of new red blood cells; the remainder of the hemoglobin forms the basis of bilirubin, a chemical that is excreted into the bile and gives the feces their characteristic yellow-brown colour.



Picture 3 , hemoglobin

Each hemoglobin molecule is made up of four heme groups surrounding a globin group, forming a tetrahedral structure. Heme, which accounts for only 4 percent of the weight of the molecule, is composed of a ringlike organic compound known as a porphyrin to which an iron atom is attached. It is the iron atom that binds oxygen as the blood travels between the lungs and the tissues. There are four iron atoms in each molecule of hemoglobin, which accordingly can bind four molecules of oxygen. Globin consists of two linked pairs of polypeptide chains.

Hemoglobin S is a variant form of hemoglobin that is present in persons who have sickle cell anemia, a severe hereditary form of anemia in which the cells become crescent-shaped when oxygen is lacking. The abnormal sickle-shaped cells die prematurely and may become lodged in small blood vessels, potentially obstructing the microcirculation and leading to tissue damage. The sickling trait is found mainly in people of African descent, though the disease also occurs in persons of Middle Eastern, Mediterranean, or Indian descent.

Anemia, condition in which the red blood cells (erythrocytes) are reduced in number or volume or are deficient in hemoglobin, their oxygen-carrying pigment. The most noticeable outward symptom of anemia is usually pallor of the skin, mucous membranes, and nail beds. Symptoms of tissue oxygen deficiency include pulsating noises in the ear, dizziness, fainting, and shortness of breath. Compensatory action of the heart may lead to its enlargement and to a rapid pulse rate. There are close to 100 different varieties of anemia, distinguished by the cause and by the size and hemoglobin content of the abnormal cells.

Anemia results when the destruction of red blood cells exceeds production, production of red blood cells is reduced, or acute or chronic blood loss occurs. Increased destruction of red blood cells (hemolysis) may be caused by hereditary cell defects, as in sickle cell anemia, hereditary spherocytosis, or glucose-6-phosphate dehydrogenase deficiency. Destruction also may be caused by exposure to hemolytic chemicals (substances causing the release of hemoglobin from the red cells) such as the antibiotic drug sulfanilamide, the antimalarial drug primaquine, or naphthalene (mothballs), or it may be caused by development of antibodies against the red blood cells, as in erythroblastosis fetalis. Reduced production of red cells may be caused by disorders of the bone marrow, as in leukemia and aplastic anemia, or by deficiency of one or more of the nutrients, notably vitamin B₁₂, folic acid (folate), and iron, that are necessary for the synthesis of red cells. Lower production may also be caused by deficiency of certain hormones or inhibition of the red-cell-forming processes by certain drugs or by toxins produced by disease, particularly chronic infection, cancer, and kidney failure.

Structurally, the anemias generally fall into the following types: (1) macrocytic anemia, characterized by larger-than-normal red cells (e.g., pernicious anemia), (2) normocytic anemia, characterized by a decrease in the number of red cells, which are otherwise relatively normal (e.g., anemia caused by sudden blood loss, as in a bleeding peptic ulcer, most cases of hemophilia, and purpura), (3) simple microcytic anemia, characterized by smaller-than-normal red cells (encountered in cases of chronic inflammatory conditions and in renal disease), and (4) microcytic hypochromic anemia, characterized by a reduction in red-cell size and hemoglobin concentration (frequently associated with iron-deficiency anemia but also seen in thalassemia).

The treatment of anemia varies greatly, depending on the diagnosis. It includes supplying the missing nutrients in the deficiency anemias, detecting and removing toxic factors, improving the underlying disorder with drugs and other forms of therapy, decreasing the extent of blood destruction by methods that include surgery (e.g., splenectomy), or restoring blood volume with transfusion.

Iron-deficiency anemia, anemia that develops due to a lack of the mineral iron, the main function of which is in the formation of hemoglobin, the blood pigment that carries oxygen from the blood to the tissues. Iron-deficiency anemia, the most common anemia, occurs when the body's loss of iron is high and its iron stores are depleted—as during periods of rapid growth, pregnancy, or menstruation or other sources of chronic blood loss. The condition may also develop when dietary iron intake is low or metabolism of iron is inefficient (e.g., starvation or hookworm infestation). It is estimated that iron-deficiency anemia affects approximately 15 percent of the population worldwide. Symptoms include weakness, fatigue, and sometimes pallor, shortness of breath, coldness of extremities, changeable appetite, sore tongue, loss of hair, brittle fingernails, or dry skin. Treatment consists of the administration of iron; quick improvement is common.

Megaloblastic anemia, the production in the bone marrow of abnormal nucleated red cells known as megaloblasts, develops as the result of dietary deficiency of, faulty absorption of, or increased demands for vitamin B₁₂ or folic acid. When such a vitamin deficiency occurs, bone marrow activity is seriously impaired; marrow cells proliferate but do not mature properly, and erythropoiesis becomes largely ineffective. Anemia develops, the number of young red cells (reticulocytes) is reduced, and even the numbers of granulocytes (white cells that contain granules in the cellular substance outside the nucleus) and platelets are decreased. The mature red cells that are formed from megaloblasts are larger than normal, resulting in a macrocytic anemia. The impaired and ineffective erythropoiesis is associated with accelerated destruction of the red cells, thereby providing the features of a hemolytic anemia (caused by the destruction of red cells at a rate substantially greater than normal). Vitamin B₁₂ is a red, cobalt-containing vitamin that is found in animal foods and is important in the synthesis of deoxyribonucleic acid (DNA). A deficiency of vitamin B₁₂ leads to disordered production of DNA and hence to the impaired production of red cells. Unlike other vitamins, it is formed not by higher plants but only by certain bacteria and molds and in the rumen (first stomach chamber) of sheep and cattle, provided that traces of cobalt are present in their fodder. In humans, vitamin B₁₂ must be obtained passively, by eating food of an animal source. Furthermore, this vitamin is not absorbed efficiently from the human intestinal tract unless a certain secretion of the stomach, intrinsic factor, is available to bind with vitamin B₁₂.

The most common cause of vitamin B₁₂ deficiency is pernicious anemia, a condition mostly affecting elderly persons. In this disorder the stomach does not secrete intrinsic factor, perhaps as the result of an immune process consisting of the production of antibodies directed against the stomach lining. The tendency to form such antibodies may be hereditary. Patients with pernicious anemia are given monthly injections of vitamin B₁₂. Oral treatment with the vitamin is possible but inefficient because absorption is poor.

Other forms of vitamin B₁₂ deficiency are rare. They are seen in complete vegetarians (vegans) whose diets lack vitamin B₁₂, in persons whose stomachs have been completely removed and so lack a source of intrinsic factor, in those who are infected with the fish tapeworm **Diphyllobothrium latum** or have intestinal cul-de-sacs or partial obstructions where competition by the tapeworms or by bacteria for vitamin B₁₂ deprives the host, and in persons with primary intestinal diseases that affect the absorptive capacity of the small intestine (ileum). In these conditions, additional nutritional deficiencies, such as of folic acid and iron, are also likely to develop.

Blood changes similar to those occurring in vitamin B₁₂ deficiency result from deficiency of folic acid. Folic acid (folate) is a vitamin found in leafy vegetables, but it is also synthesized by certain intestinal bacteria. Deficiency usually is the result of a highly defective diet or of chronic intestinal malabsorption as mentioned above. Pregnancy greatly increases the need for this vitamin. There is also an increased demand in cases of chronic accelerated production of red cells. This type of deficiency also has been observed in some patients receiving anticonvulsant drugs, and there is some evidence that absorption of the vitamin may be impaired in these cases. Often several factors affecting supply and demand of the vitamin play a role in producing folic acid deficiency. Unless folic acid deficiency is complicated by the presence of intestinal or liver disease, its treatment rarely requires more than the institution of a normal diet. In any event the oral administration of folic acid relieves the megaloblastic anemia. Some effect can be demonstrated even in pernicious anemia, but this treatment is not safe because the nervous

system is not protected against the effects of vitamin B₁₂ deficiency, and serious damage to the nervous system may occur unless vitamin B₁₂ is given.

In addition to the above conditions, megaloblastic anemia may arise in still other situations. Selective vitamin B₁₂ malabsorption may be the consequence of a hereditary defect. Deranged metabolism may play a role in some instances of megaloblastic anemia that accompany pregnancy. Metabolic antagonism is thought to be the mechanism underlying the megaloblastic anemia associated with the use of certain anticonvulsant drugs and some drugs employed in the treatment of leukemia and other forms of cancer. In fact, one of the earliest drugs used to treat leukemia was a folic acid antagonist.

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