

Anemia

The term anemia comprises all conditions in which the capacity of the blood to transport O_2 is reduced. Anemia may be caused by a reduction in the number of circulating erythrocytes, less hemoglobin in each erythrocyte, or a combination of these factors. Anemia caused by a deficiency of vitamin B_{12} or folic acid is described in the main text (p. 319).

Iron-deficiency anemia. In humans, iron-deficiency anemia is the most common form of anemia. It may be caused by a deficient supply of dietary iron (most common in children and pregnant women), or reduced absorption of iron from the intestine (due to various gastric or intestinal diseases). It may also be due to increased loss of iron due to bleeding from gastric ulcers, tumors or polyps in the digestive tract, or heavy menstrual bleeding.

Iron deficiency is rare in adult domestic animals. Neonates of species that grow rapidly will, however, develop serious iron deficiency anemia unless extra iron is provided shortly after birth. This form of anemia is particularly serious in piglets raised in modern rearing systems. In such rearing systems the piglets do not have contact with soil or other natural iron sources.

Iron-deficiency in anemia in piglets is particularly dramatic because of the following factors:

- Piglets are born with an iron store that can maintain hemoglobin synthesis for only 1–2 days (about 5 mg).
- Sow milk contains very little iron
- Breeding practices have led to rapid growth of the piglets. Weight is usually increased 7–8 fold during the first four weeks of life.

In iron deficiency anemia in general, the erythrocytes exhibit a reduction in size and hemoglobin concentration. In piglets, the iron anemia deficiency also leads to a decrease in growth rate (Fig. 9.5).

Physiological anemia. In many domestic mammals, the hematocrit value, the hemoglobin concentration, and the number of erythrocytes per unit volume decrease during the first days, or weeks, after birth. These changes, called physiological anemia, can only be partially corrected by iron treatment. This form of anemia has been found in puppies, kittens, lambs, kids, baby rabbits, and piglets.

Hemolytic anemia. Hemolysis means destruction of erythrocytes. Hemolytic anemia develops if the erythrocytes are destroyed so rapidly that a normal bone marrow is unable to produce them at the same rate. In humans, hemolytic anemia is most often caused by inherited defects that result in fragile erythrocytes that are easily destroyed during passage through the capillaries. An example of such anemia is sickle-cell anemia, in which the erythrocytes contain a defective type of hemoglobin.

Inherited defects in erythrocytes are rare in domestic animals. However, erythrocytes can also have a shorter lifespan than normal if they are subjected to abnormal stresses. This is the case in parasitic diseases, such as piroplasmosis in horses, ruminants, dogs, and cats, malaria in humans, and plasmodiosis in birds (avian malaria). The parasites that cause these diseases, which are species-specific, penetrate the erythrocytes, where they multiply until the erythrocytes burst. Consequently, large numbers of parasites are emptied into the blood where they attack new erythrocytes, and the situation may become life-threatening. Binding of antibodies to antigens on erythrocyte membranes can also result in hemolytic anemia (p. 354). In contrast to iron-deficiency anemia, the number of reticulocytes in the blood rises during hemolytic anemia. This is caused by increased erythrocyte production, due to the higher blood concentration of erythropoietin caused by the anemia (p. 320).

Aplastic anemia. Some forms of anemia are due to the inability of the bone marrow to maintain a sufficiently high production rate of erythrocytes, despite all the necessary building blocks and growth factors being present. Such anemias are called aplastic, and may be due to bone marrow destruction by chemical substances (for example chemotherapeutic agents used in treatment of cancerous tumors), ionizing radiation, or to bone marrow invasion by cancer cells. Aplastic anemia can also be caused by bacterial or viral infections. In many cases of aplastic anemia, the production of leukocytes and platelets is also reduced.

Anemia during chronic renal disease. Patients with serious chronic renal disease usually suffer from anemia. This is primarily due to insufficient formation of erythropoietin by the kidneys. As a result, erythrocyte production in the bone marrow decreases.

Effects of anemia on cardiac output. The viscosity of the blood is primarily determined by the number of erythrocytes per unit volume. During serious anemias, the viscosity can be halved. This reduces the resistance to blood flow (p. 394), which, in turn, increases the venous return to the heart. The low O_2 content of blood during anemia causes dilation of the arterioles (metabolic autoregulation), which further increases the venous return to the heart. Consequently, cardiac output increases. Although each volume of blood transports less O_2 than normal, this is compensated for during rest by larger amounts of blood flowing through the systemic circulation. However, an anemic individual cannot increase cardiac output as much as a healthy individual during strenuous physical exercise, because the cardiac output of the anemic individual is already high while at rest. Consequently, O_2 deficiency may develop in the tissues and there may be a risk of heart failure.