

Blood Groups and Transfusions

Losses of about 15 to 30 percent of blood lead to pallor and weakness. Losses of over 30 percent cause 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) 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. The plasma membranes of RBCs, like those of all body cells, bear genetically determined proteins (antigens), which identify each person as unique. An **antigen** 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. The person's RBC proteins will be recognized as foreign if transfused into another person with differ RBCantigen. The recognizer 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 RBCs to clump, a phenomenon called **agglutination**, 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 and cause kidney failure.

There are over 30 common RBC antigens in humans, allowing each person's blood cells to be classified into different blood groups. However, it is the antigens of the ABO and Rh blood groups that cause the most vigorous transfusion reactions.

As shown in Table (1), 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 possession of either A or B antigen yields type A or B blood, respectively. In the ABO blood group, antibodies are formed 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, while those with type A antigens (group A) form anti-B antibodies, and so on.

Table (1): ABO Blood Group

Blood group	Frequency	RBC antigen (agglutinogens)	Plasma antibodies (agglutinins)	Blood that can be received
AB	5	A, B	None	A, B, AB, O Universal recipient
B	27	B	Anti-A	B,O
A	28	A	Anti-B	A,0
O	40	None	Anti-A Anti-B	O Universal donor

The **Rh blood groups** are so named because one of the eight Rh antigens (agglutinin D), was originally identified in Rhesus monkeys. Later the same antigen was discovered in human beings. Most people 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 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 donor and recipient before blood is transfused is obvious. The general procedure for determining ABO blood type is briefly outlined in Figure (12). 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.

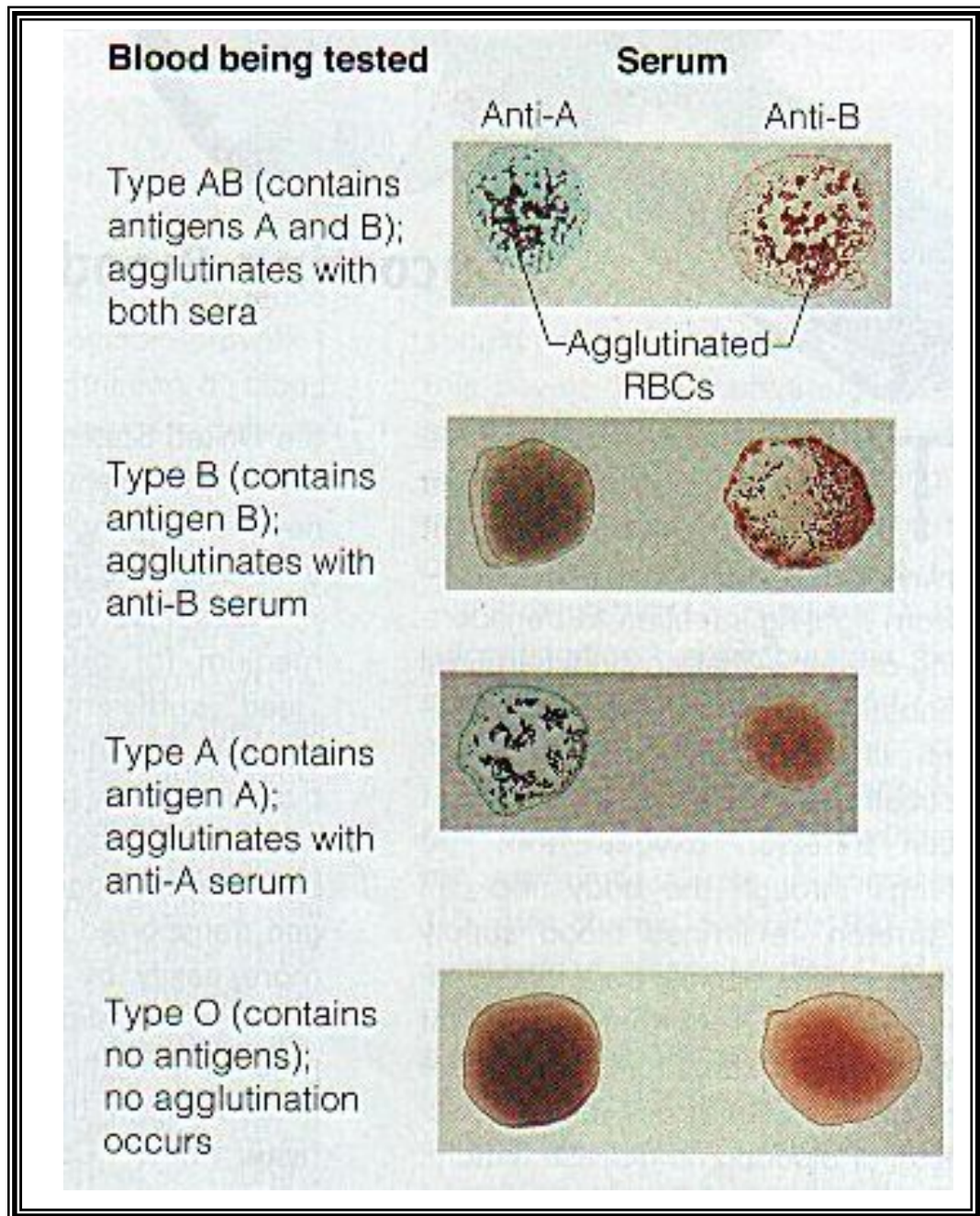


Figure (12) : Blood typing of ABO blood group