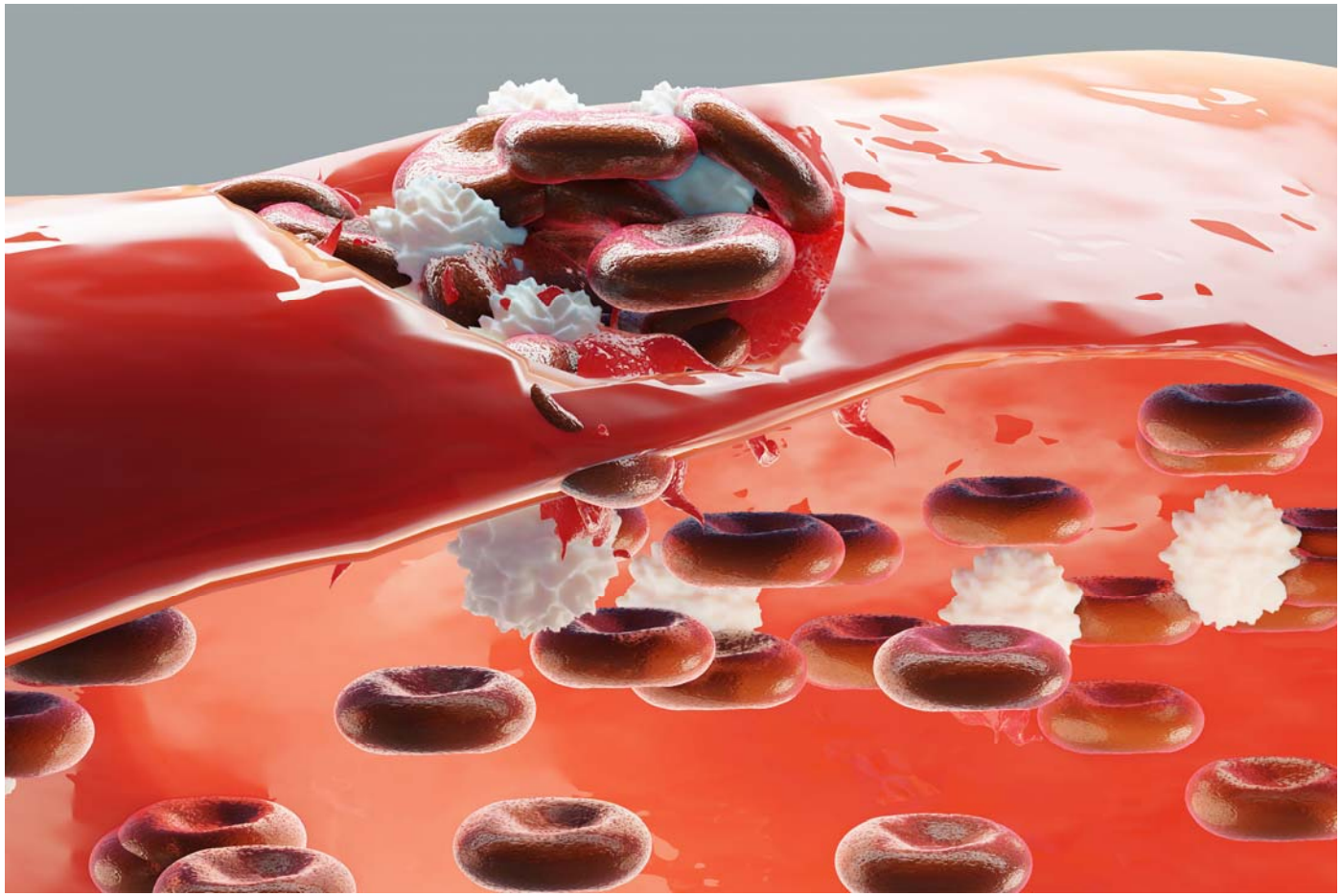


Assist. Lec.: Safa Jalil AL-Yassiri

blood clotting, groups and blood disorders

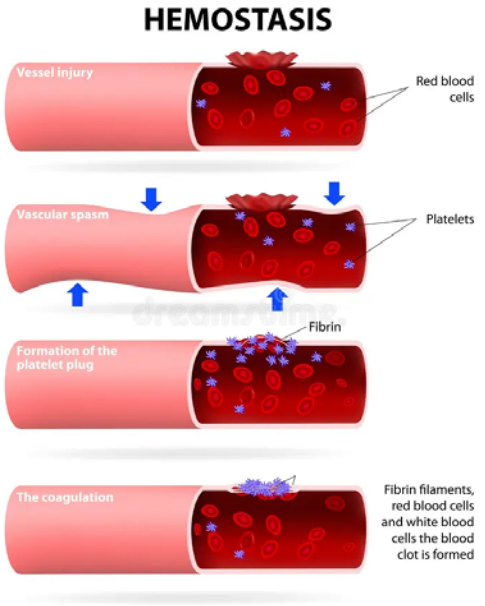
Physical and Chemical Characteristics of platelets:

Platelets have many functional characteristics of whole cells, even though they do not have nuclei and cannot reproduce. In their cytoplasm are the following:

- Actin and myosin molecules, which are contractile proteins similar to those found in muscle cells, and thrombosthenin.
- Residuals of both the endoplasmic reticulum and Golgi apparatus that synthesize various enzymes and especially store large quantities of calcium ions.
- Mitochondria and enzyme systems that are capable of forming adenosine triphosphate (ATP) and adenosine diphosphate (ADP).
- Enzyme systems that synthesize prostaglandins, which are local hormones that cause many vascular and other local tissue reactions.
- an important protein called fibrin-stabilizing factor.
- a growth factor that causes vascular endothelial cells, vascular smooth muscle cells, and fibroblasts to multiply and grow, thus causing cellular growth that eventually helps repair damaged vascular walls.
- On the platelet cell membrane surface is a coat of glycoproteins that repulses adherence to normal endothelium and yet causes adherence to injured areas of the vessel wall, especially to injured endothelial cells and even more so to any exposed collagen from deep within the vessel wall.
- the platelet membrane contains large amounts of phospholipids that activate multiple stages in the blood-clotting process.

Hemostasis and blood Coagulation:

The term hemostasis means prevention of blood loss. Platelets and proteins in the plasma work together to stop the bleeding by forming a clot over the injury. Within 3 to 6 minutes after rupture of a vessel, the entire opening or broken end of the vessel is filled with clot if the vessel opening is not too large.



Whenever a vessel is severed or ruptured, hemostasis is achieved by several mechanisms:

(1) vascular constriction.
(2) formation of a platelet plug.
(3) formation of a blood clot as a result of blood coagulation.
(4) growth of fibrous tissue into the blood clot to close the hole in the vessel permanently.

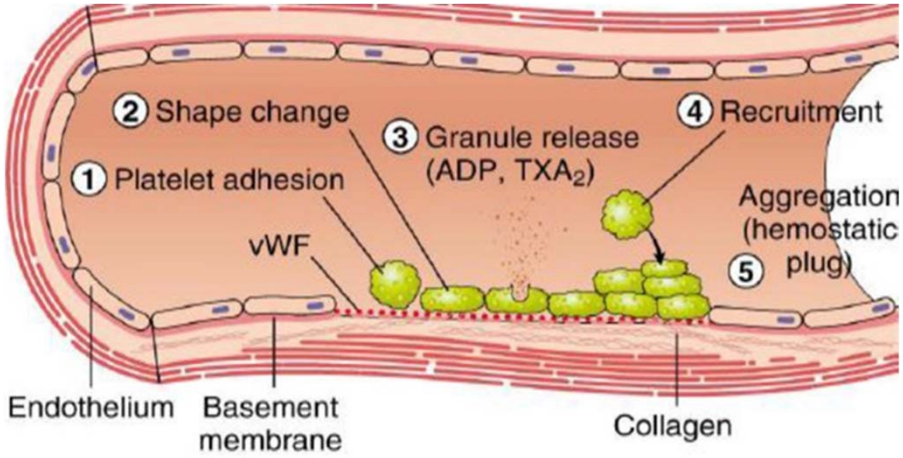
1. **Vascular spasm:** when a large vessel such as an artery or vein is severed, the smooth muscle in its wall contracts in response to the damage. Platelets in the area of the rupture release serotonin, which also brings about vasoconstriction. The diameter of the vessel is thereby made smaller, and the smaller opening may then be blocked by a blood clot. If the vessel did not constrict first, the clot that forms would quickly be washed out by the force of the blood pressure.

2. Platelet plug:

- The platelets come in contact with a damaged vascular surface, especially with collagen fibers in the vascular wall through von Willebrand factor(vWF), which leaks into the traumatized tissue from the plasma.

- the platelets rapidly change their own characteristics drastically. They begin to swell, they assume irregular forms with numerous irradiating pseudopods protruding from their surfaces (become spiky).

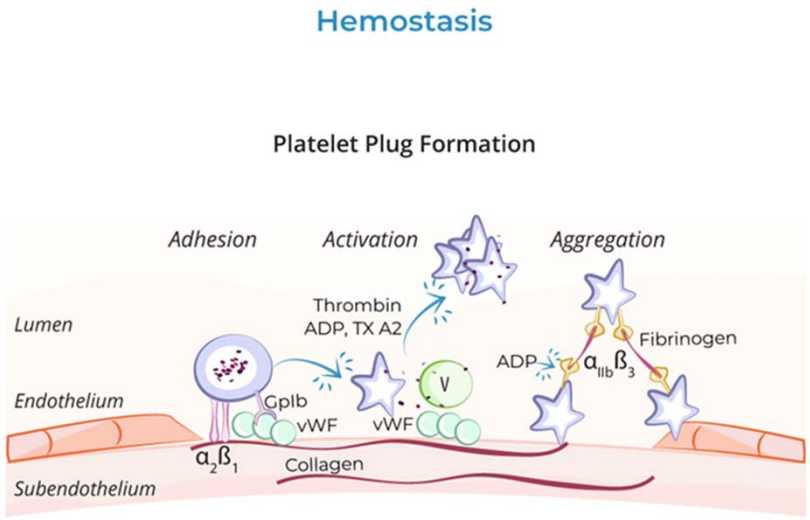
- The platelets contract and release of granules that contain multiple active factors.



- Therefore, at the site of a puncture in a blood vessel wall, the damaged vascular wall activates successively increasing numbers of platelets that attract more and more additional platelets, thus forming a platelet plug.

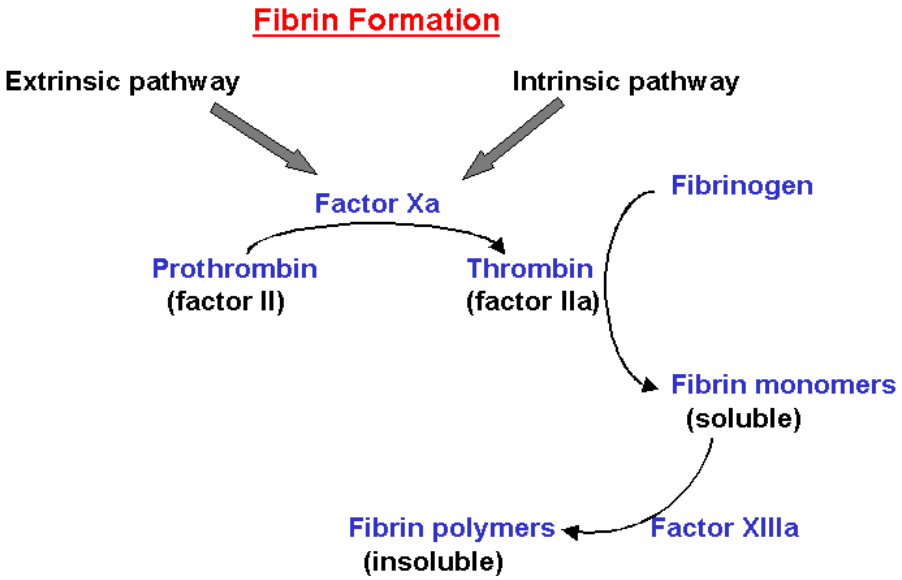
- This plug is loose at first but is usually successful in blocking blood loss if the vascular opening is small.

- Then, during the subsequent process of blood coagulation, fibrin threads form. These threads attach tightly to the platelets, thus constructing an unyielding plug



3. The blood clot:

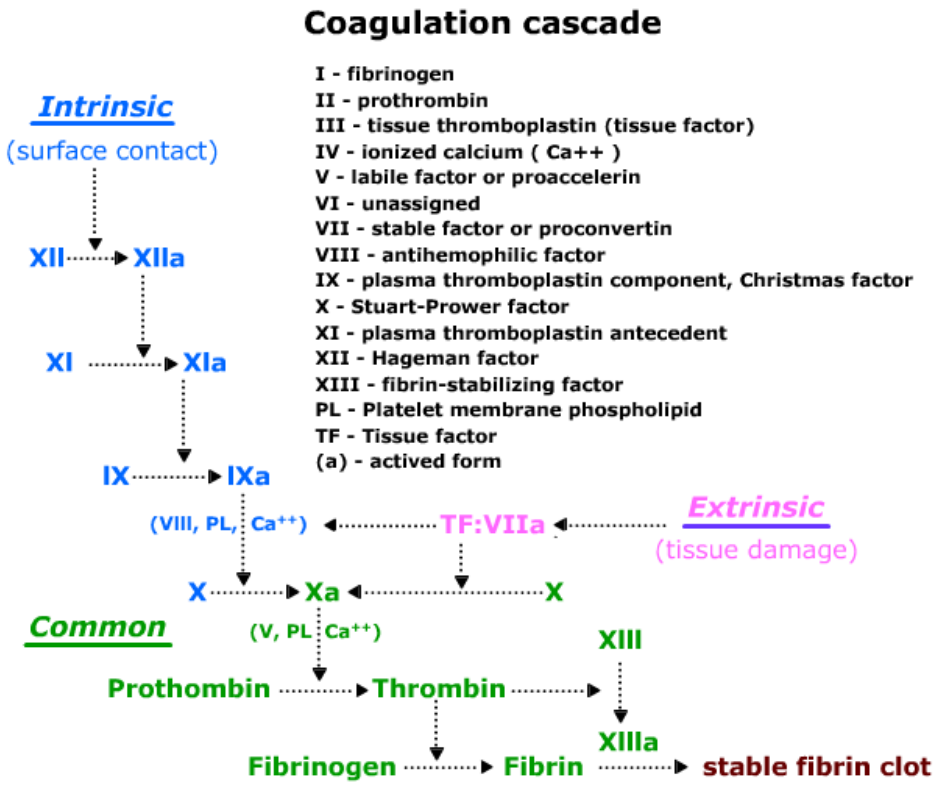
Is the third mechanism for hemostasis. At which **Clotting takes place in three essential steps:**



- ❑ Formation of prothrombin activator.
- ❑ Conversion of prothrombin into thrombin.
- ❑ The thrombin acts as an enzyme to convert fibrinogen into fibrin fibers that enmesh platelets.

Prothrombin activator is generally considered to be formed in two ways, although, in reality, the two ways interact constantly with each other by:

- **the extrinsic pathway** that begins with trauma to the vascular wall and surrounding tissues.
- **the intrinsic pathway** that begins in the blood.
- In both the extrinsic and the intrinsic pathways, a series of different plasma proteins called blood-clotting factors plays a major role. Most of these proteins are inactive forms of proteolytic enzymes. When converted to the active forms, their enzymatic actions cause the successive, cascading reactions of the clotting process.



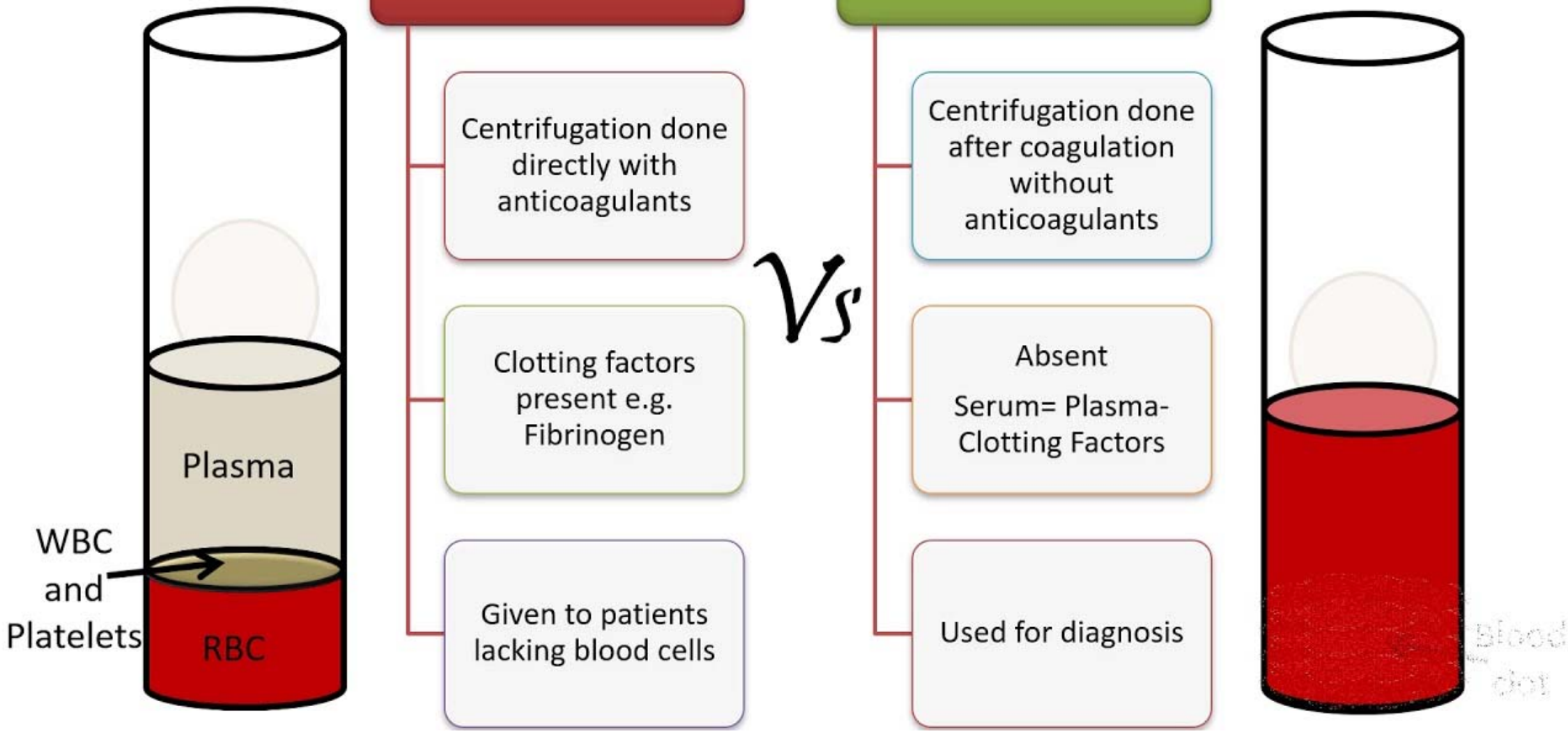
Blood Clotting Factors:

1.	Factor I	Fibrinogen
2.	Factor II	Prothrombin
3.	Factor III	Thromboplastin (Tissue factor)
4.	Factor IV	Calcium
5.	Factor V	Labile factor (Proaccelerin or accelerator globulin)
6.	Factor VI	Presence has not been proved
7.	Factor VII	Stable factor
8.	Factor VIII	Antihemophilic factor (Antihemophilic globulin)
9.	Factor IX	Christmas factor
10.	Factor X	Stuart-Prower factor
11.	Factor XI	Plasma thromboplastin antecedent
12.	Factor XII	Hageman factor (Contact factor)
13.	Factor XIII	Fibrin-stabilizing factor (Fibrinase).

Clotting factors were named after the scientists who discovered them or as per the activity, except factor IX. Factor IX or Christmas factor was named after the patient in whom it was discovered.

Role of Calcium Ions in the Intrinsic and Extrinsic Pathways:

- Except for the first two steps in the intrinsic pathway, calcium ions are required for promotion or acceleration of all the blood-clotting reactions.
- Therefore, in the absence of calcium ions, blood clotting by either pathway does not occur.
- In the living body, the calcium ion concentration seldom falls low enough to affect blood-clotting kinetics significantly.
- when blood is removed from someone, it can be prevented from clotting by reducing the calcium ion concentration below the threshold level for clotting by deionizing the calcium by causing it to react with substances such as citrate ion or by precipitating the calcium with substances such as oxalate ion.


Plasma
Serum
Centrifugation done directly with anticoagulants
Clotting factors present e.g. Fibrinogen
Given to patients lacking blood cells
Vs
Centrifugation done after coagulation without anticoagulants
Absent
Serum= Plasma- Clotting Factors
Used for diagnosis
Plasma
WBC and Platelets
RBC
blood clot

Blood disorders:

❑ **Anemia** is a deficiency of red blood cells, or insufficient hemoglobin within the red blood cells.

❖ **types of anemia:**

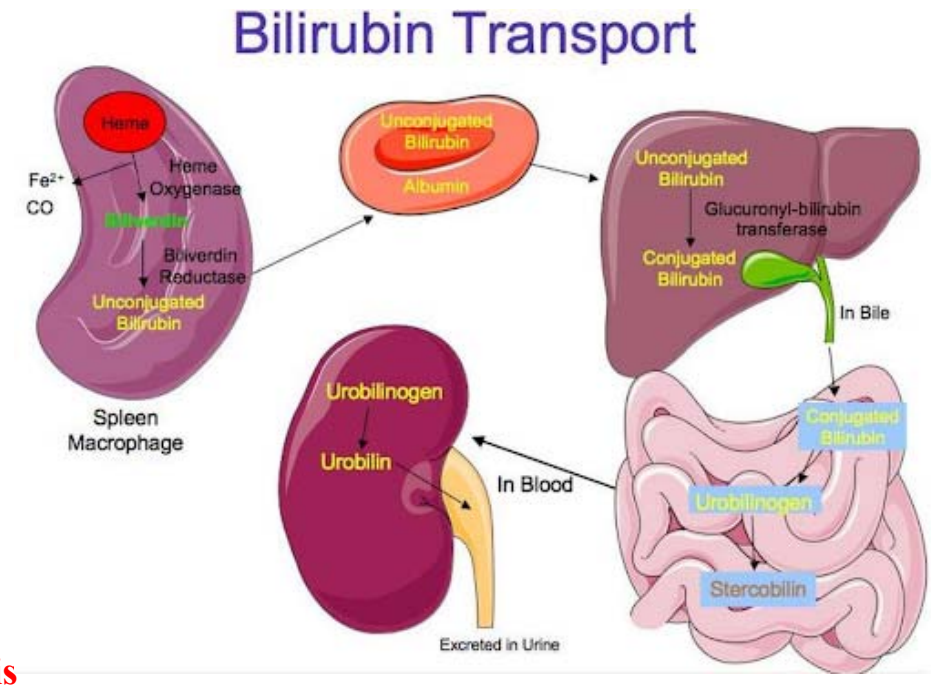
1. **Iron-deficiency anemia:** is caused by a lack of dietary iron, and there is not enough of this mineral to form sufficient hemoglobin. A person with this type of anemia may have a normal RBC count and a normal hematocrit, but the hemoglobin level will be below normal.
2. **Pernicious anemia**: caused by deficiency of vitamin B12, which is found only in animal foods, in which the RBCs are large, misshapen, and fragile.

3. **Sickle-cell anemia:** It is a genetic disorder of hemoglobin, which causes RBCs to sickle, and rupture. clog capillaries,
4. **Aplastic anemia:** is suppression of the red bone marrow, with decreased production of RBCs, WBCs, and platelets. This is a very serious disorder that may be caused by exposure to radiation, certain chemicals such as benzene, or some medications.

5. **Hemolytic anemia:** is any disorder that causes rupture of RBCs before the end of their normal life span. Sickle-cell anemia and Rh disease of the newborn are examples. Another example is malaria, in which a protozoan parasite reproduces in RBCs and destroys them. Hemolytic anemias are often characterized by jaundice because of the increased production of bilirubin

Jaundice:

- A part of the hemoglobin molecule is the heme portion, which cannot be recycled and is a waste product.
- The heme is converted to bilirubin. The liver removes bilirubin from circulation and excretes it into bile; bilirubin is a bile pigment. Bile is secreted by the liver into the duodenum and passes through the small intestine and colon, so bilirubin is eliminated in feces, and gives feces their characteristic brown color.
- In the colon some bilirubin is changed to urobilinogen by the colon bacteria. Some urobilinogen may be absorbed into the blood, but it is changed to urobilin and excreted by the kidneys in urine.
- If bilirubin is not excreted properly, it remains in the blood. **This may cause jaundice.**



❑ **Jaundice:** Jaundice is not a disease, but rather a sign caused by excessive accumulation of bilirubin in the blood. Because one of the liver's many functions is the excretion of bilirubin.

❖ **types of jaundice:**

1. **hepatic jaundice:** jaundice may be a sign of liver disease such as hepatitis or cirrhosis. because the problem is with the liver.

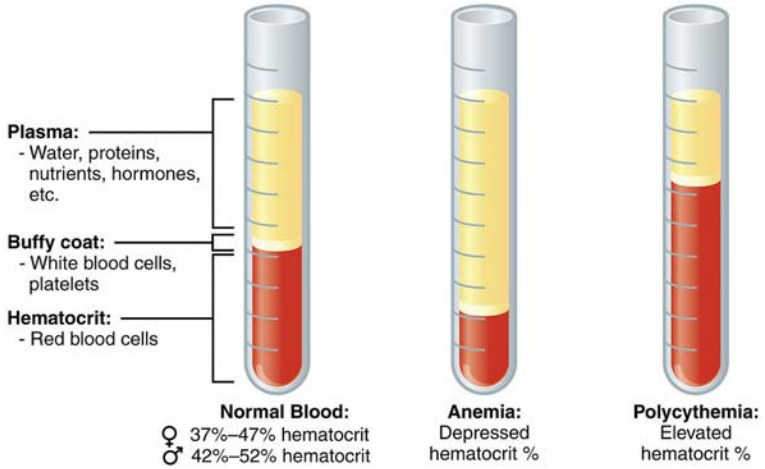
2. **Pre-hepatic jaundice:** means that the problem is "before" the liver; that is, hemolysis of RBCs is taking place at a more rapid rate. Rapid hemolysis is characteristic of sickle-cell anemia, malaria, and Rh disease of the new born; these are hemolytic anemias. As excessive numbers of RBCs are destroyed, bilirubin is formed at a faster rate than the liver can excrete it. The bilirubin that the liver cannot excrete remains in the blood and causes jaundice.

3. **Post-hepatic jaundice:** means that the problem is "after" the liver. The liver excretes bilirubin into bile, which is stored in the gallbladder and then moved to the small intestine. If the bile ducts are obstructed, perhaps by gallstones or inflammation of the gallbladder, bile cannot pass to the small intestine and backs up in the liver. Bilirubin may then be reabsorbed back into the blood and cause jaundice. Another name for this type is obstructive jaundice

- **Polycythemia:** is a blood disorder occurring when there are too many red blood cells. The excess red blood cells cause the blood to increase in volume and thicken, keeping it from flowing easily.

- **types of polycythemia:**

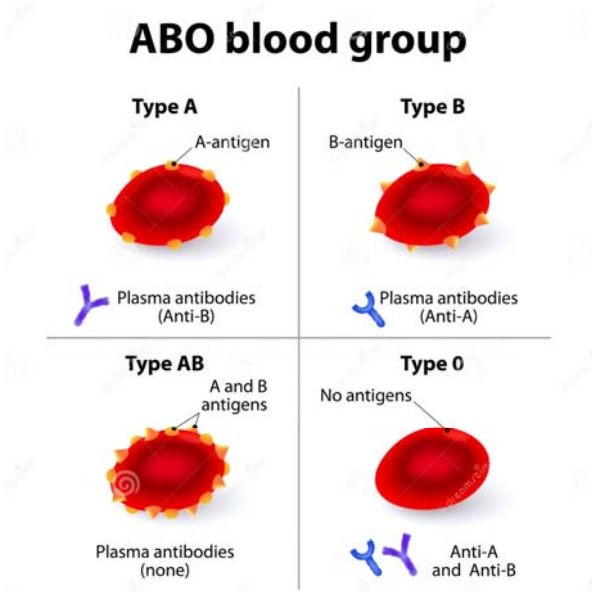
- **Primary polycythemia:** also called polycythemia vera (PV). which causes the bone marrow to create excess precursor blood cells that develop and function abnormally, leading to the production of too many red blood cells. A person with PV may also have increased numbers of other blood cells, such as white blood cells or platelets.

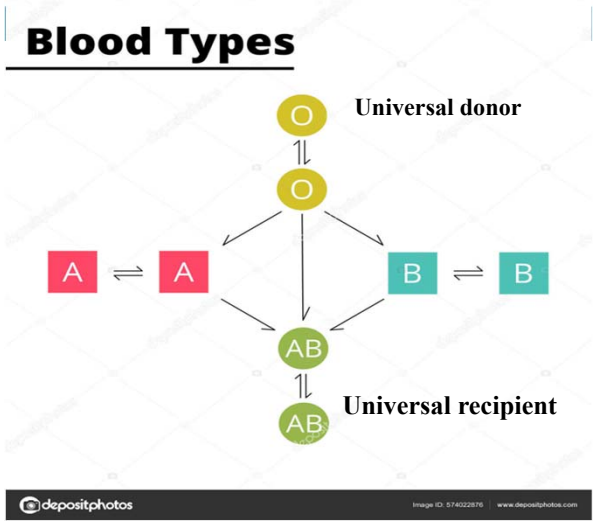


- **Secondary polycythemia:** Whenever the tissues become hypoxic because of too little oxygen in the breathed air, such as at high altitudes, or because of failure of oxygen delivery to the tissues, such as in cardiac failure, the blood-forming organs automatically produce large quantities of extra RBCs. This condition is called secondary polycythemia, and the RBC count commonly rises to 6 to 7 million/mm3, about 30% above normal.

Blood groups:

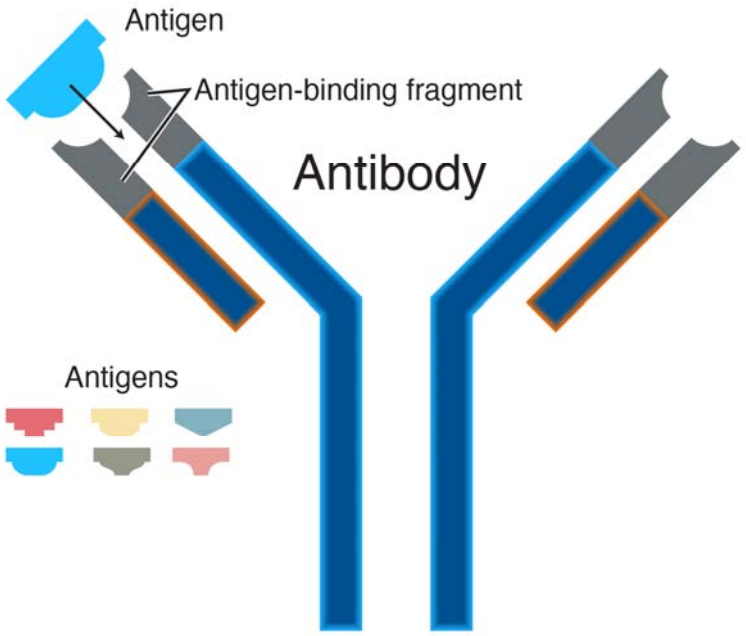
- Before giving a transfusion to a person, it is necessary to determine the blood type of the recipient and donor blood so that the bloods can be appropriately matched. This process is called blood typing and blood matching.
- Two particular types of antigens are much more likely than the others to cause blood transfusion reactions. They are the O-A-B system of antigens and the Rh system.
- The letters A and B represent antigens on the red blood cell membrane.
- A person with type A blood has the A antigen on the RBCs, and someone with type B blood has the B antigen. Type AB means that both A and B antigens are present, and type O means that neither the A nor the B antigen is present.
- Circulating in the plasma of each person are natural antibodies for those antigens not present on the RBCs. Therefore, a type A person has anti-B antibodies in the plasma; a type B person has anti-A antibodies; a type AB person has neither anti-A nor anti-B antibodies; and a type O person has both anti-A and anti-B antibodies.





An antigen: is any substance that prompts the body to trigger an immune response against it. include allergens, bacteria and viruses.

Antibodies: are Y-shaped proteins that the body produces when it detects antigens. Antibodies are produced by immune cells called B cells



The Rh factor:

- There are six common types of Rh antigens, each of which is called an Rh factor Rhesus (Rh) factor
- These types are designated C, D, E, c, d, and e.
- The type D antigen is widely prevalent in the population and is considerably more antigenic than the other Rh antigens.
- Anyone who has this type of antigen is said to be (Rh positive), whereas a person who does not have type D antigen is said to be (Rh negative).
- If an (Rh-negative) person receives (Rh-positive) blood by mistake, antibodies will be formed just as they would be to bacteria or viruses.
- A first mistaken transfusion often does not cause problems, because antibody production is slow upon the first exposure to Rh-positive RBCs. A second transfusion, however, when anti-Rh antibodies are already present, will bring about a transfusion reaction, with hemolysis and possible kidney damage.