Hemostasis

The term hemostasis means prevention of blood loss. This achieved by several mechanisms, including:

1. **Vascular spasm.**
2. **Formation of a platelets plug.**
3. **Formation of blood clot.**
4. **Growth of fibrous tissue into the blood clot to close the whole vessel permanently.**

Vascular Spasm

The most immediate protection against blood loss is vascular spasm, a prompt constriction of the broken vessel. Several things trigger this reaction. An injury stimulates with pain receptors, some of which directly innervate nearby blood vessels and cause them to constrict. Immediately after a blood vessel is cut or ruptured, the stimulus of the trauma to the vessel causes the wall of the vessel to contract due to nervous reflexes, local myogenic spasm and local humoral factors from blood platelets.

Formation of a Platelet Plug.

Platelets are not cells, but small fragments of megakaryote cytoplasm. Although they were once called thrombocytes. Platelets are small round or oval discs 2-4 µm in diameter.

They are formed in the bone marrow from megakaryocytes which are extremely large cells of the hemopoietic series in the bone marrow that fragment into platelets. The normal concentration of platelets in the blood is between 150,000 and 400,000 per µL.

Platelets have many functional, even through these do not have nuclei and can not reproduce. In their cytoplasm are such active factor as:

1. **They secrete growth factors that stimulate mitosis in fibroblasts and smooth muscle and help to maintain the linings of blood vessels.**

2. **They secrete vasoconstrictors that cause vascular spasm in broken vessels.**

3. **They form temporary platelet plugs to stop bleeding.**

4. **They phagocytize and destroy bacteria.**

5. **They secrete chemicals that attract neutrophils and monocytes to sites of inflammation.**

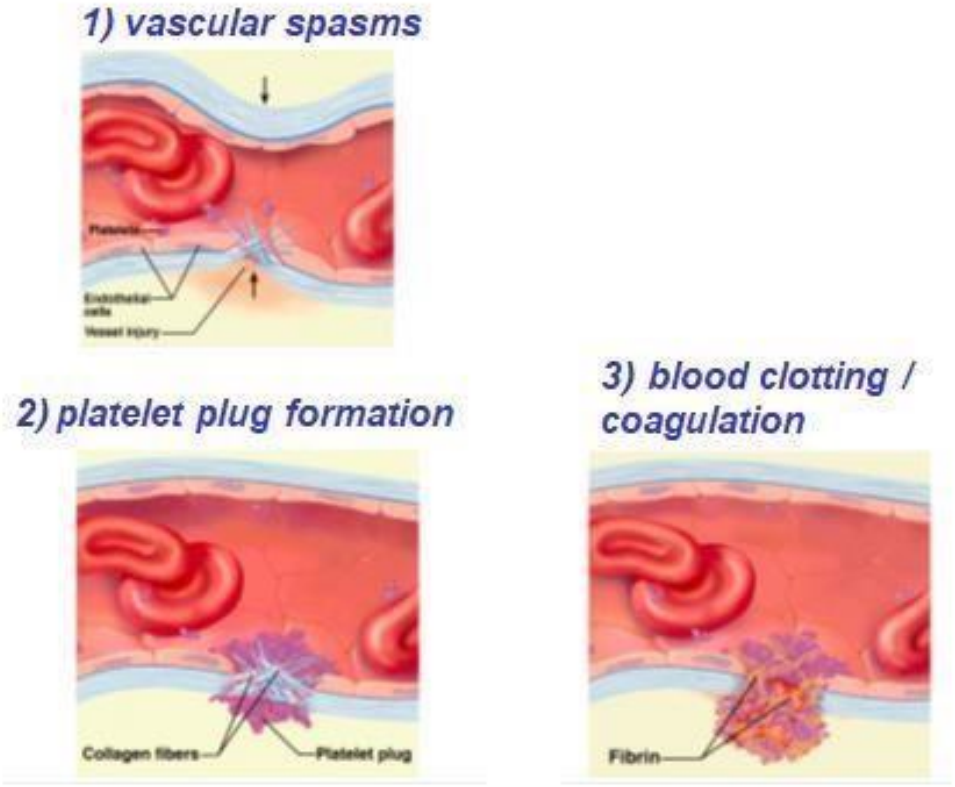
6. **They dissolve blood clots that have outlasted their usefulness.**

The cell membrane of the platelets is also important, on its surface is a coat of glycoproteins that causes it to avoid adherence to normal endothelium and yet to adhere to injured areas of the vessel wall.

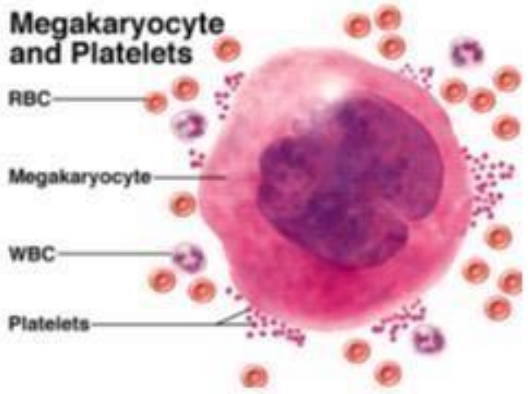
Mechanism of the Platelet Plug

Platelet repair of vascular openings is based on several important functions of the platelet itself, when platelets come in contact with a damaged vascular surface, such as the collagen fibers in

the vascular wall or damaged endothelial cells, they immediately change their characteristics. They begin to swell, they assume irregular forms and become sticky so that they stick to the collagen fibers, they secrete large quantities of ADP and their enzymes form thromboxane A2 in turn act on nearby platelets to activate them as well forming a platelet plug.



Platelets



Platelets are not cells but cytoplasmic fragments of extraordinarily large (up to 60 μm in diameter) cells called ***megakaryocytes***.

Normal Platelet Count: 150,000 – 400,000/μl