Animal physiology-lecture (4)

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Hemostasis and Blood Coagulation

The term hemostasis means prevention of blood; hemostasis is achieved by several mechanisms (figure 1):

(1) Vascular constriction,

(2) Formation of a platelet plug,

(3) Formation of a blood clot as a result of blood coagulation, and

(4) Eventual growth of fibrous tissue into the blood clot to close the hole in the vessel permanently.

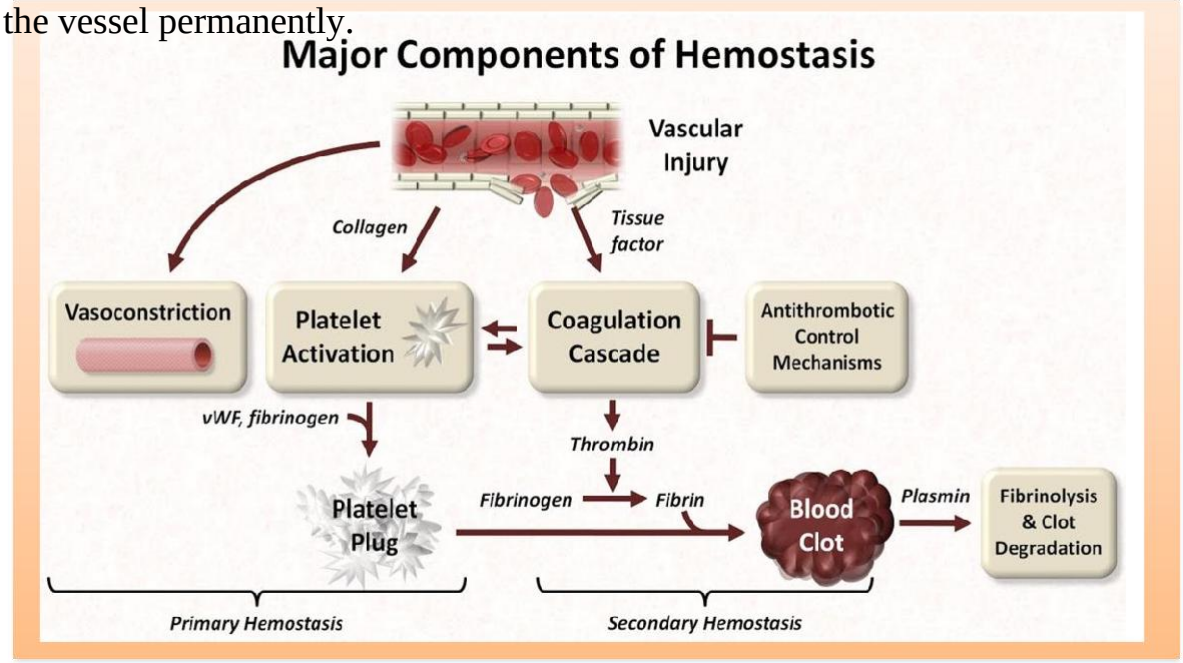

Figure 1: Major components of hemostasis.

Vascular Constriction

Immediately after a blood vessel has been cut or ruptured, the trauma to the vessel wall itself causes the smooth muscle in the wall to contract; this instantaneously reduces the flow of blood from the ruptured vessel.

The contraction results from:

(1) Local myogenic spasm,

(2) Local autacoid factors from the traumatized tissues and blood platelets, and

(3) Nervous reflexes.

Formation of the Platelet Plug

If the cut in the blood vessel is very small—indeed, many very small vascular holes do develop throughout the body each day—the cut is often sealed by a platelet plug, rather than by a blood clot.

Platelets have many functional characteristics of whole cells, even though they do not have nuclei and cannot reproduce. **In their cytoplasm are such active factors as:**

(1) Actin and myosin molecules, which are contractile proteins similar to those found in muscle cells, and still another contractile protein, thrombosthenin, that can cause the platelets to contract;

(2) Residuals of both the endoplasmic reticulum and the Golgi apparatus that synthesize various enzymes and especially store large quantities of calcium ions;

(3) Mitochondria and enzyme systems those are capable of forming adenosine triphosphate (ATP) and adenosine diphosphate (ADP);

(4) Enzyme systems that synthesize prostaglandins, which are local hormones that cause many vascular and other local tissue reactions;

(5) An important protein called fibrin-stabilizing factor; and

(6) 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.

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, especially with collagen fibers in the vascular wall, the platelets themselves immediately change their own characteristics drastically. They begin to swell; they assume irregular forms with numerous irradiating pseudopods protruding from their surfaces; their contractile proteins contract forcefully and cause the release of granules that contain multiple active factors; they become sticky so that they adhere to collagen in the tissues and to a protein called von Willebrand factor that leaks into the traumatized tissue from the plasma; they secrete large quantities of ADP; and their enzymes form thromboxane A2. The ADP and thromboxane in turn act on nearby platelets to activate them as well, and the stickiness of these additional platelets causes them to adhere to the original activated platelets. Therefore, at the site of any opening in a blood vessel wall, the damaged vascular wall activates successively increasing numbers of platelets that themselves attract more and more additional platelets, thus forming a platelet plug. This is at first a loose plug, but it 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 attach tightly to the platelets, thus constructing an unyielding plug.

Blood Coagulation in the Ruptured Vessel

The third mechanism for hemostasis is formation of the blood clot. The clot begins to develop in 15 to 20 seconds if the trauma to the vascular wall has been severe and in 1 to 2 minutes if the trauma has been minor. Activator substances from the traumatized vascular wall, from platelets, and from

blood proteins adhering to the traumatized vascular wall initiate the clotting process. The physical events of this process are shown in figure 2. Within 3 to 6 minutes after rupture of a vessel, if the vessel opening is not too large, the entire opening or broken end of the vessel is filled with clot. After 20 minutes to an hour, the clot retracts; this closes the vessel still further.

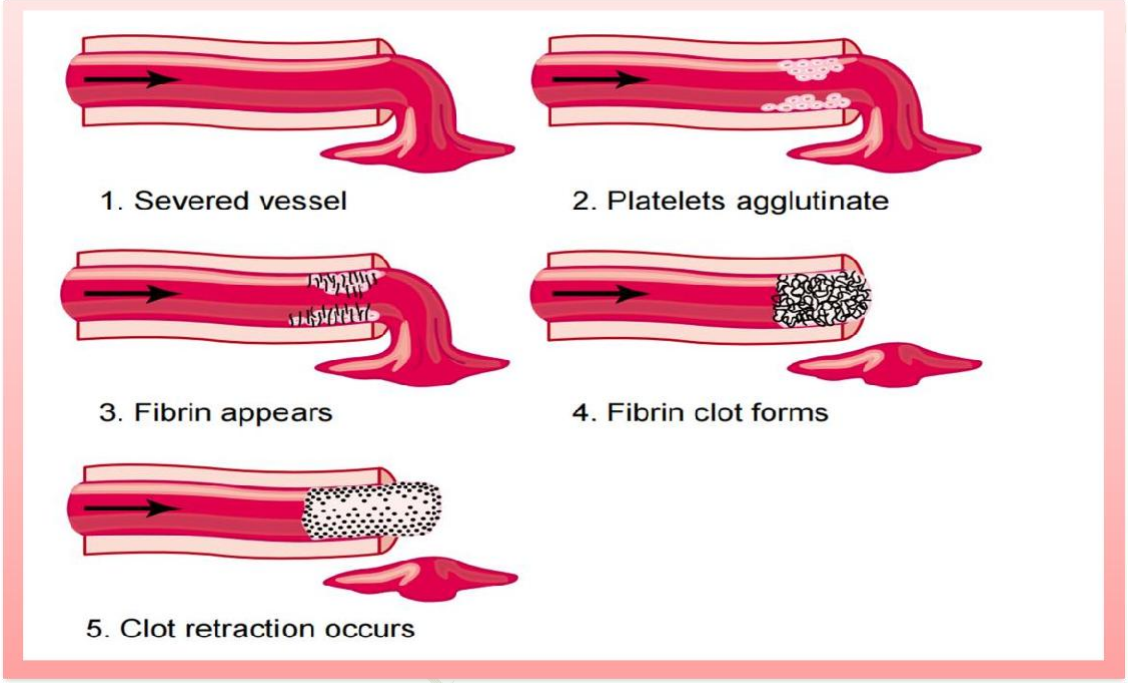

Figure 2: Clotting process in a traumatized blood vessel.

Fibrous Organization or Dissolution of the Blood Clot

Once a blood clot has formed, it can follow one of two courses:

(1) It can become invaded by fibroblasts, which subsequently form connective tissue all through the clot, or

(2) It can dissolve.

The usual course for a clot that forms in a small hole of a vessel wall is invasion by fibroblasts, beginning within a few hours after the clot is formed (which is promoted at least partially by growth factor secreted by platelets).

This continues to complete organization of the clot into fibrous tissue within about 1 to 2 weeks. Conversely, when excess blood has leaked into the tissues and tissue clots have occurred where they are not needed, special substances within the clot itself usually become activated.