

Formation of a Platelet Plug

Platelets do not normally attach to one another or to the smooth lining of the blood vessels. However, when the endothelial layer is damaged, blood platelets immediately attach to, and are activated by, the injured surface. This process is facilitated by a plasma glycoprotein, *von Willebrand factor* (vWF). Produced in both endothelial cells and platelets, vWF forms bridges between receptors on the surface of platelets and collagen fibers in the connective tissue. The platelets then begin to swell, and extend cell projections in an octopus-like manner. Simultaneously, the platelets release active substances that are stored in their vesicles, including adenosine diphosphate (ADP), which causes the surface of the platelets to become sticky. Thus, new platelets adhere to those already attached to the collagen fibers. The newly attached platelets also release ADP, and so the process is continued. In addition, platelets participating in the process form thromboxane A_2 from arachidonic acid, which is one of the fatty acids in the phospholipids of the cell membranes (p. 89). Thromboxane A_2 augments the aggregation of blood platelets directly, as well as indirectly by stimulating release of ADP.

The platelets continue to aggregate until a platelet plug sealing the blood vessel is formed (Fig. 9.10). Thromboxane A_2 , along with serotonin released from the platelets, contributes to the contraction of the injured vessel.

It is obviously important that formation of the platelet plug is restricted to the injured area, without extending into the uninjured part of the blood vessel. This is prevented mainly because ADP and other substances released from activated platelets stimulate uninjured endothelial cells to release nitric oxide (NO) as well as a prostaglandin called prostacyclin (Fig. 9.10). While thromboxane A_2 stimulates aggregation of platelets, NO and prostacyclin counteract it. Moreover, like most other cells in the body, platelets and intact endothelial cells have negatively charged surfaces, and thus repel each other. These mechanisms help to limit the formation of the platelet plug to the injured area.

Initially, the platelet plug is loosely constructed. However, shortly after vessel injury, the blood starts to coagulate, forming a lattice of fine fibrin threads (p. 329) in and around the platelet plug. This reinforces the platelet plug and ensures that it does not give way under the pressure of the

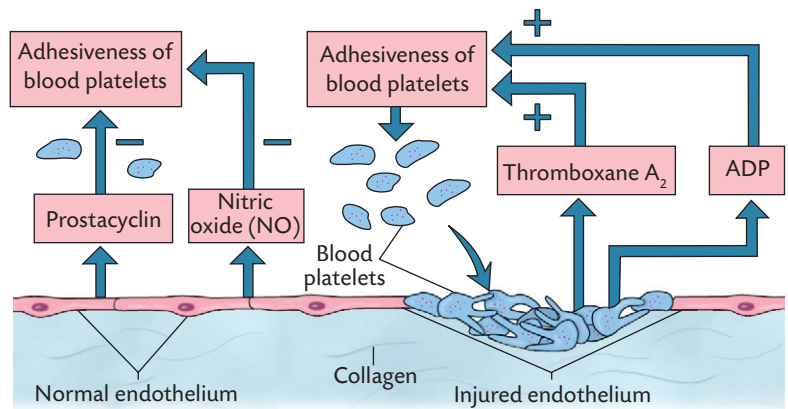


Figure 9.10 Formation of a platelet plug. When blood platelets adhere to collagen in the wall of a defective blood vessel, they release ADP and synthesize thromboxane A_2 . These substances increase the adhesiveness of the platelets, causing more platelets to aggregate at the site of injury. The platelet plug thus formed seals the injured vessel (right part of figure). While blood vessel injuries cause the release and increase in synthesis of substances that induce platelet aggregation (ADP and thromboxane A_2), an intact endothelium releases prostacyclin and nitric oxide, which inhibits platelet aggregation (left part of figure).

blood. After 30–60 min, the clot shrinks, drawing the edges of the wound together and further sealing off the vessel. The contractile apparatus of the platelets causes this shrinkage by retracting the platelet extensions. As the clot shrinks, a yellowish liquid, *serum*, is squeezed out. Serum has the same composition as plasma, except that serum does not contain the coagulation factors that have been used in the clotting process.

The importance of platelet plug formation in stopping bleeding depends on the extent of the



- 36** What is hemostasis?
- 37** What is a platelet plug?
- 38** Describe the mechanisms in the formation of a platelet plug.
- 39** What prevents the platelet plug from spreading to uninjured areas of the blood vessel?
- 40** What is the difference between serum and plasma?

Platelets form a plug that temporarily stops bleeding

Prostacyclin and nitric oxide prevent expansion of the platelet plug

Figure 9.11 Formation and stabilization of fibrin.

