The ability of the body to control the flow of blood following vascular injury is paramount to continued survival. The process of blood clotting and then the subsequent dissolution of the clot, following repair of the injured tissue, is termed hemostasis. Hemostasis, composed of major events that occur in a set order following the loss of vascular integrity:

1. The initial phase of the process is vascular constriction. This limits the flow of blood to the area of injury.
2. Next, platelets become activated by thrombin and aggregate at the site of injury, forming a temporary, loose platelet plug. The protein fibrinogen is primarily responsible for stimulating platelet clumping. Platelets clump by binding to collagen that becomes exposed following rupture of the endothelial lining of vessels. Upon activation, platelets release numerous substances and proteins important for the coagulation cascade. In addition to induced secretion, activated platelets change their shape to accommodate the formation of the plug.
3. To insure stability of the initially loose platelet plug, a fibrin mesh (also called the clot) forms and entraps the plug. If the plug contains only platelets it is termed a white thrombus; if red blood cells are present it is called a red thrombus.
4. Finally, the clot must be dissolved in order for normal blood flow to resume following tissue repair. The dissolution of the clot occurs through the action of plasmin.

Two pathways lead to the formation of a fibrin clot: the intrinsic and extrinsic pathway. Although they are initiated by distinct mechanisms, the two converge on a common pathway that leads to clot formation. Both pathways are complex and involve numerous different proteins termed clotting factors. Nowadays the time based coagulation pathway takes the upper hand in describing the distinct mechanisms of the hemostasis process involving cells and coagulation factors into a homogenous pathway.

Blood coagulation is part of an important host defense mechanism termed hemostasis (the cessation of blood loss from a damaged vessel). Upon vessel injury, platelets adhere to macromolecules in the subendothelial tissues and then aggregate to form the primary hemostatic plug. The platelets stimulate local activation of plasma coagulation factors, leading to generation of a fibrin clot that reinforces the platelet aggregate. Later, as wound healing occurs, the platelet aggregate and fibrin clot are broken down. Mechanisms that restrict formation of platelet aggregates and fibrin clots to sites of injury are necessary to maintain the fluidity of the blood.

Hemorrhage can result from trauma, vascular defects, peptic ulcer disease), platelet abnormalities, or deficiencies of one or more of the plasma coagulation factors.
Thrombosis is a pathologic process in which a platelet aggregate and/or a fibrin clot forms in the lumen of an intact blood vessel or in a chamber of the heart. If thrombosis occurs in an artery, the tissue supplied by the artery may undergo ischemic necrosis (e.g., myocardial infarction due to thrombosis of a coronary artery).