Figura 1. The endothelium regulates initiation of coagulation. The resting endothelium (1a) deactivates platelets by secretion of nitric oxide and prostacycline; activates antithrombin (AT), and enhances fibrinolysis through thrombomodulin and endothelial protein C receptors (EPCR) that facilitate protein C activation by thrombin. The activated endothelium (1b) sheds its antithrombotic mediators into circulation, and secretes the platelet anchoring von Willebrand Factor (vWF) instead of platelet deactivating mediators.

**Fig.1a**

![Diagram of non-activated and anti-thrombotic endothelium](image)

**Fig.1b**

![Diagram of activated and procoagulant endothelium](image)
Figura 2. Primary haemostasis is the adhesion and activation of platelets leading to formation of a continuous platelet plug covering the vascular injury. Glycoprotein (GP), von Willebrand Factor (vWF), adenosine dihosphate (ADP), Thromboxane A2 (TXA2).
Figura 3. Initiation of coagulation occurs on the sub-endothelial cells following vascular injury. They present large amounts of tissue factor (TF) that activates coagulation factor (F) VII. Coagulation is rapidly shut down by tissue factor pathway inhibitor (TFPI) and the small amount of thrombin produced must traverse to adherent platelets. Suffix "a" indicate activated coagulation factors.
Figura 4. Amplification of coagulation occurs on the adherent platelets. The thrombin activates the platelet and the coagulation factors (F) bound in high concentrations to the anionic platelet membrane. The activation process is reinforced by autocrine signalling including degranulation of dense granules and thromboxane A2 (TXA2) formation. Suffix "a" indicate activated coagulation factors. Protease activated receptor (PAR), adenosine diphosphate (ADP) cyclooxygenase 2 (COX-2), arachidonic acid (AA)
Figura 5. The propagation of coagulation occurs on the highly activated platelet that amplifies thrombin production. Besides the formation of the fibrin mesh, thrombin also activates coagulation factors (F) XIII that cross-links the fibrin polymers increasing strength and resistance to fibrinolysis. Suffix “a” indicate activated coagulation factors.