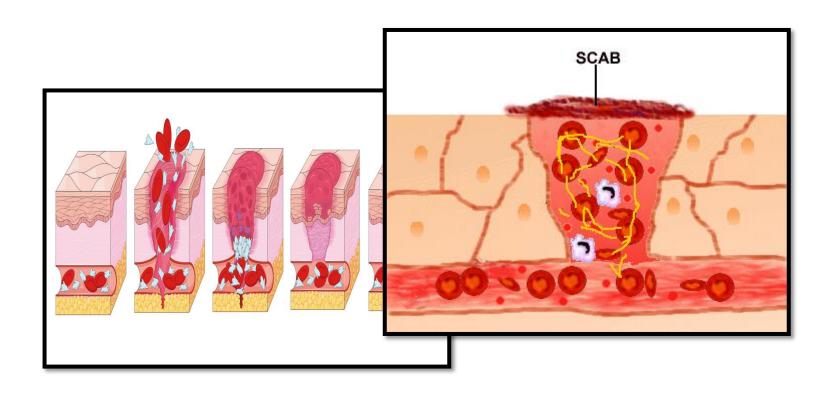
Blood Clotting



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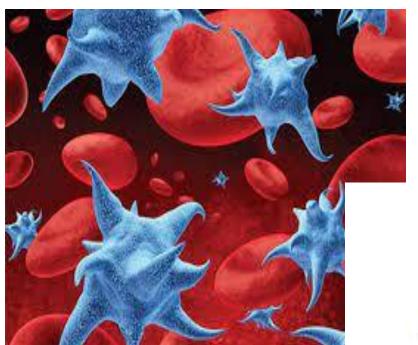
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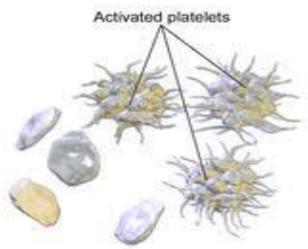
THROMBOCYTES (Platelets)

- Small, irregularly-shaped, oval non-neocleated cells, 2-4 µm in diameter.
- Derived from fragmentation of precursor megakaryocytes (bone marrow).
- Reference range (SI units) is 150,000 450,000 μL.
- -The average lifespan of a platelet is between 8 and 12 days.
- Old platelets are destroyed by phagocytosis in the spleen and by Kupffer cells in the liver.

Platelet functions:

- Maintenance of Vascular Integrity.
- Formation of the Primary Hemostatic Plug.
- Accelerate thrombin formation.





Platelets

HEMOSTASIS (blood clots)

Blood coagulation or clotting is an important phenomenon to prevent excess loss of blood in case of injury or trauma. The blood stops flowing from a wound in case of injury. The blood clot or 'coagulum' is formed by a network of fibrin threads.

HEMOSTASIS

- 1) Constriction of the blood vessel.
- 2) Formation of a temporary "platelet plug."
- 3) Activation of the coagulation cascade.
- 4) Formation of "fibrin plug" or the final clot.

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Vessel wall (vasoconstriction) Platelets (Production and activation, platelets plug formation) Blood coagulation (Formation of fibrin to form a clot) Fibrinolysis
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Prothrombin is the inactive form of thrombin that is present in the plasma. Thrombokinase converts prothrombin to active thrombin which in turn activates fibrinogen to fibrin. All these clotting factors help in blood coagulation.

An injury stimulates platelets or thrombocytes to release various factors that initiate the blood clotting cascade. Calcium ions play an important role in blood coagulation.

Human Blood Clotting Clotting Factors Prothrombin Thrombin Fibrinogen Fibrin (soluble) (insoluble) Broken vessel Activated platelet Fibrin strands Blood clot Damaged Blood Vessel Formation of Platelet Plug **Development of Clot** Injury to vessel lining triggers Vasoconstriction limits blood flow Fibrin strands adhere to the the release of clotting factors and platelets form a sticky plug plug to form an insoluble clot

Primary hemostasis (platelet clotting)

Primary hemostasis is when your body forms a temporary plug to seal an injury. To accomplish that, platelets that circulate in your blood stick to the damaged tissue and activate. That activation means they can "recruit" more platelets to form a platelet "plug" to stop blood loss from the damaged area. That clot works much like a cork or bottle stopper, keeping blood in and debris or germs out.

Primary hemostasis may also involve constriction (narrowing) of the damaged blood vessel, which can happen because of substances that activated platelets release.

Secondary hemostasis (coagulation cascade)

The platelet plug is the first step to stop bleeding, but it isn't stable enough to stay in place without help. The next step, which stabilizes the plug, is secondary hemostasis. This step, sometimes called coagulation, involves molecules in your blood called "coagulation factors." Those factors activate in sequence, the "coagulation cascade," which amplifies clotting effects as the sequence continues. Ultimately, the coagulation cascade forms a substance called fibrin. During this step, the platelet plug acts like bricks and the fibrin acts like mortar. Together, they form a solid, stable clot.

Coagulation of blood occurs through a series of reactions due to the activation of a group of substances called clotting factors.

There are 13 clotting factors identified and named after the scientists who discovered them or as per the activity. Only factor IX or Christmas factor is named after the patient in whom it was discovered.

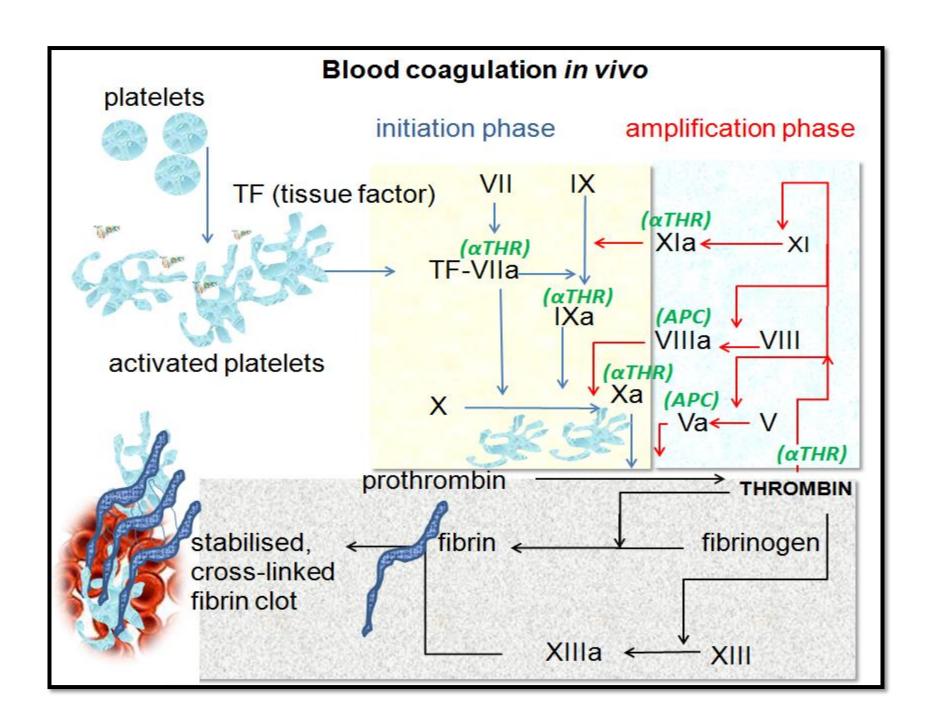
Factor I Fibrinogen	Factor VI Unassigned	Factor XI Plasma thromboplastin antecedent
Factor II Prothrombin	Factor VII Stable factor	Factor XII Hageman factor
Factor III Thromboplastin	Factor VIII Antihemophilic factor	Factor XIII Fibrin-stabilising factor
Factor IV Calcium Factor	Factor IX Christmas factor	
Factor V Labile factor (Proaccelerin)	Factor X Stuart-Prower factor	

Prothrombin activators are a group of substances which convert prothrombin to thrombin in two ways:

- 1. Extrinsic pathway: (the main pathway to initiate coagulation) The process is started when injured endothelial cells produce tissue factor (factor III), which activates factor VII.
- 2. Intrinsic pathway: (which amplifies coagulation) involves the activation of factors XII, XI, IX, and factor VIII.

The prothrombin activator converts prothrombin to thrombin in the presence of enough ionic Ca++ from platelets.

The most significant ions in the process of coagulation of blood are calcium ions. Because calcium ions are required for the conversion of thromboplastin into prothrombinase and prothrombin into thrombin (active).



Fibrin clot remodeling

The last stage of hemostasis is when your body remodels the existing clot into a fibrin clot. Your body does that because blood clots are a temporary patch, not a permanent solution. That removal involves a process called fibrinolysis. During fibrinolysis, your body remodels the clot into the same kind of tissue that was there before the injury.

The two final phases in the hemostatic process, plasma coagulation with the formation of a fibrin clot, and fibrinolysis leading to the dissolution of fibrin clots