Blood coagulation

Business

Coagulation of the blood is a process that prevents excess loss of blood from the body. The coagulation process occurs as a result of two steps: The first, Primary Homeostasis, is characterized by activation of the platelets for the formation of a plug at the punctured site. On being punctured, the von Willebrand Factor (vWF) of the body tissues become exposed resulting into the gathering of the collagen and other coagulating factors. The circulating platelets then adhere to the collagen; subsequently, a series of reactions producing other materials that cause more adhesion of collagen and platelets ensue.

More materials are then released by the bound platelets boosting their clumping thus helping the plug formation (Bose, 2011) Another step, Secondary Homeostasis, is characterized by a cascade of reactions that help in the formation of fibrin. This process is achieved by either the Intrinsic Pathway or the Extrinsic Pathway. Both this processes are necessary for producing prothrombin activator that furthers the coagulation process with the latter being more rapid. Moreover, the two steps above converge into a common pathway that aims to form thrombin. Plasma proteins fibrinogen and prothrombin are produced in the liver in an inactive form.

The prothrombin factor is instrumental in converting them into their active forms. First, it converts prothrombin to thrombin which in turn converts fibrinogen into fibrin strands. The fibrin strands creates a mesh that offers a structural support for the platelet plug made at the punctured site thus ending the coagulation process (Bose, 2011). At times, blood coagulation may occur uncontrollably due to some disorders. These results into a condition called thrombosis. This situation can be remedied by the

introduction of anti-coagulants like Antithrombin III which inhibits thrombin formation by binding to prothrombin, factor 9 and 10 thus inactivating them.

Heparin is another anticoagulant that enhances the functioning of Antithrombin III by binding to it to cause an allosteric change thus developing a higher affinity for thrombin (King, 2012).