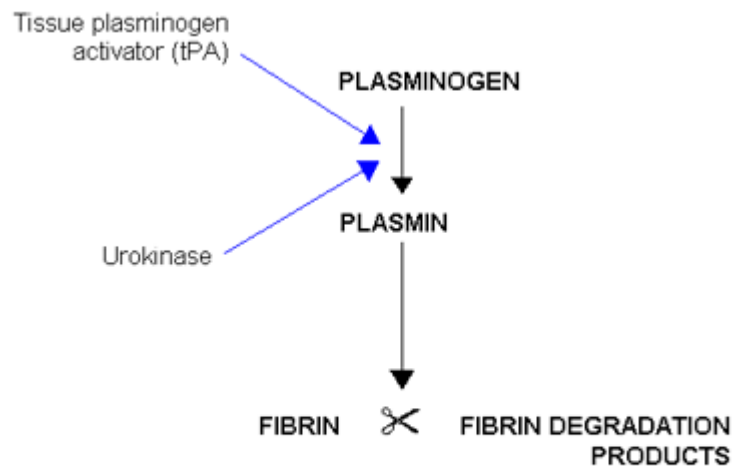


2.2.4

Clot Retraction and Fibrinolysis

Keep in mind that blood clots form upon and in conjunction with a preexisting platelet plug. Upon clot formation, a process known as **clot retraction** occurs. During clot retraction, actin and myosin contained within platelets of the platelet plug begin to contract. As these proteins contract, the web of platelets connected by molecules of fibrinogen begins to retract and condense. This, in turn, causes the connected fibrin blood clot to retract and condense as well. Consequently, clot retraction decreases the size of the cut or gash by drawing the damaged ends of the blood vessel toward one another. Fibroblasts and epithelial cells proliferate in and around the clot. This serves to help repair the damaged vessel.

Within several days of clot formation, an enzyme known as **plasmin** completely degrades fibrin, thus dissolving the clot through a process known as **fibrinolysis**. Plasmin is the active form of an inactive plasma protein known as plasminogen. Plasminogen is synthesized and released by the liver and is converted to plasmin by a number of different molecules, one of which is called tissue plasminogen activator (tPA). Endothelial cells produce tPA. In the presence of fibrin, tPA greatly accelerates its enzymatic function to convert plasminogen to plasmin, thus initiating the process of clot dissolution. It is interesting to note that by activating tPA, fibrin initiates its own degradation. tPA can be given to patients who are experiencing heart attacks or strokes caused by an embolism (floating clot). However, the treatment seems to be most effective if it is administered within 90 minutes of onset of symptoms.



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