



Vascular Access Hemostasis

External Vascular Access Hemostasis

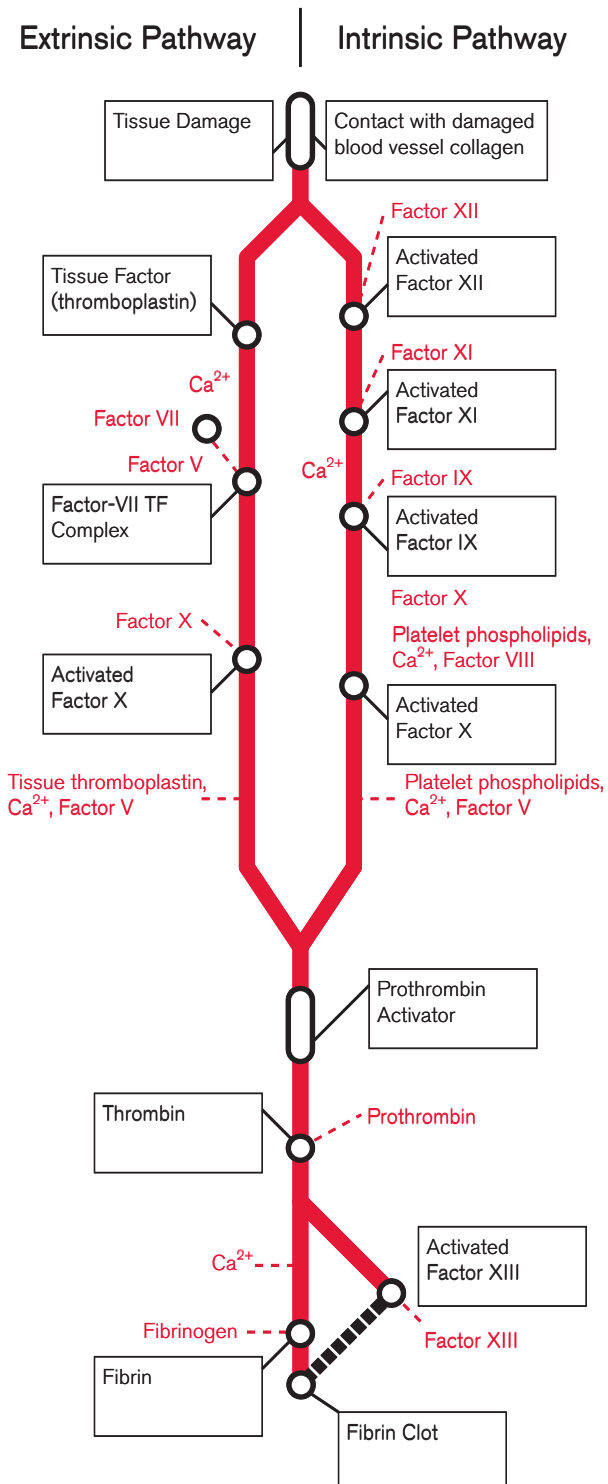
Hemostasis is a natural process — given enough time, the body heals itself. But when anticoagulation therapy or other conditions impair the blood's ability to clot, achieving hemostasis is a challenge that adds unnecessary risk. Syvek® topical dressings employ highly concentrated strands of the poly-N-acetylglucosamine (p-GlcNAc) polymeric fiber we call Syvek® S3D. This fiber has a unique three-dimensional structure that presents a vast array of bonding sites for the natural clotting agents in blood, accelerating hemostasis.



Syvek®
Hemostasis Innovation™

The Coagulation Cascade

Two biochemical pathways lead to the formation of a fibrin clot; intrinsic and extrinsic. The **intrinsic pathway** is a response to blood vessel abnormalities, while the **extrinsic pathway** is triggered by the tissue injury. Though initiated by distinct mechanisms, they converge along a common pathway that leads to clot formation.



Adapted from *Medical Biochemistry, 2nd Edition*, Baynes & Dominiczak, p. 70.
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Syvek® dressing is applied by external pressure to the surface of the wound.



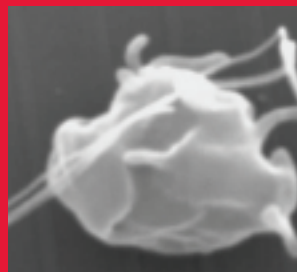
Blood comes in contact with Syvek® S3D fiber on the surface of the Syvek® dressing.

Vournakis et al. Journal of Trauma.
Vol 57 No.1 Pg. S2-S8. 2004



Protein molecules in the blood plasma rearrange to bond with S3D fibers, exposing surfaces that attract platelets.

Thatté et al. Journal of Trauma.
Vol 57 No.1 Pg. S13-S21. 2004



Bound platelets trigger vasoconstrictors, agents that limit the amount of blood flow in capillaries, slowing blood loss.

Thatté et al. Journal of Trauma.
Vol 57 No.1 Pg. S13-S21. 2004



Successive fibrin networks attract more platelets expediting clot formation.

Scanning Electron Micrograph courtesy of
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Syvek[®]



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