

In addition to contributing to platelet function, inflammation plays a role in both coagulation-dependent thrombus formation and thrombus resolution. Both polymorphonuclear neutrophils and monocytes/macrophages contribute to multiple overlapping thrombotic functions, including fibrinolysis, chemokine and cytokine production, and phagocytosis.

THE DISTINCTION BETWEEN ARTERIAL AND VENOUS THROMBOSIS

Although there is overlap, venous thrombosis and arterial thrombosis are initiated differently, and clot formation progresses by somewhat distinct pathways. In the setting of stasis or states of hypercoagulability, venous thrombosis is activated with the initiation of the coagulation cascade primarily due to exposure of tissue factor; this leads to the formation of thrombin and the subsequent conversion of fibrinogen to fibrin. In the artery, thrombin formation also occurs, but thrombosis is primarily promoted by the adhesion of platelets to an injured vessel and stimulated by exposed extracellular matrix (Figs. 117-1 and 117-2). There is wide variation in individual responses to vascular injury, an important determinant of which is the predisposition an individual has to arterial or venous thrombosis. This concept has been supported indirectly in prothrombotic animal models in which there is poor correlation between the propensity to develop venous versus arterial thrombosis.

Despite considerable progress in understanding the role of hypercoagulable states in VTE, the contribution of hypercoagulability to arterial vascular disease is much less well understood. Although specific thrombophilic conditions, such as factor V Leiden and the prothrombin G20210A mutation, are risk factors for DVT, pulmonary embolism, and other venous thromboembolic events, their contribution to arterial thrombosis is less well defined. In fact, to the contrary, many of these thrombophilic factors have not been found to be clinically important risk factors for arterial thrombotic events, such as acute coronary syndromes.

Clinically, although the pathophysiology is distinct, arterial and venous thrombosis do share common risk factors, including age,