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# New Understandings of Platelet Function

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Platelets are disk-shaped, non-nucleated blood elements that tend to adhere to uneven or damaged surfaces, such as the site of vascular injury. Platelet activity is controlled by various regulatory mediators which affect the platelet count and its function. The platelet function is related to its adhesion and aggregation, which is critical for hemostatic plug formation. Platelet membrane receptors interact with exposed collagen in damaged vessels and can start the coagulation cascade with surface-bound adhesive ligands. Sites of vascular injury and atherosclerotic lesions, promote cellular interactions that result in localization, adhesion, and high concentration of mediators. Shearing stress in blood circulation is also an important factor in vascular cell adhesive capacity and other critical parameters are the diameter of the vessel, flow rate and the viscosity of blood. Adhesion receptors located on the surface of blood platelets have a key role in blood flow rate. Absence of platelet receptors glycoprotein (GP) Ib-IXV, GPVI, and the integrin aIIbβ3 (GPIIb-IIIa), results in prolonged bleeding times and also clinical bleeding symptoms. These same platelet receptors regulate the thrombosis episodes such as heart attack or stroke.

Recent technical advances have demonstrated platelet adhesion and aggregation mechanisms which are much more complex and dynamic than previously described. The identification of mechanisms of functional platelet performance and vascular-bed-specific inhibitors of platelet aggregation is important in better understanding the hemostasis and thrombosis process. In future; producing safer and more effective antiplatelet agents to reduce thrombosis complications and also prevent complications from platelet dysfunction, might be possible based on this new understanding.