Activated platelets play an integral role in thrombin formation by presenting a procoagulant surface. This surface results from loss of plasma membrane phospholipid bilayer asymmetry; upon platelet stimulation, the anionic aminophospholipid phosphatidylserine (PS) is translocated from the inner membrane leaflet, where it is normally sequestered, to the outer leaflet. Negatively-charged PS facilitates assembly of the intrinsic tenase and prothrombinase coagulation complexes on the activated platelet surface, thereby accelerating thrombin generation.

There is heterogeneity in platelets in their ability to form a procoagulant surface, and determinants of PS exposure will be described. Persistence of the procoagulant surface will be discussed; it does not reverse readily as had previously been expected. The remarkable morphological transformation into spherical ‘balloons’, almost devoid of their normal internal architecture, that PS-exposing platelets undergo will be illustrated. Even in this zombie-like state, procoagulant platelets participate in thrombosis; the reduction of platelet accumulation in a thrombosis model by blocking PS will be described.

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