Abstract

All materials as yet evaluated induce thrombosis when placed in the vascular circulation. How thrombogenesis occurs on artificial surfaces is incompletely understood. In this study the platelet is examined for its potential role in this process. Since the platelet is known to respond differentially to different surfaces ex vivo, the nature of the control of this differential response is evaluated: Is the surface activation of the platelet determined primarily by previously adsorbed proteins and other plasma factors or can the platelet also modulate its own response to surfaces?

The shape-change activation of individual platelets ex vivo (in canines and macaques) to four polymer surfaces was compared to the response of purified platelets in vitro from the same subjects thus controlling for haematological variation between individuals. The differential in vitro responses to the four polymers were observed to closely mimic the ex vivo responses. Thus platelets are capable of responding differentially to surfaces in the same manner as they do ex vivo without modulation or control by other plasma factors.

The response of human platelets in vitro was then compared to those of the canine and the macaque. The order

of extent of shape-change activation to the four polymers was the same for all three species, canines, humans, and macaques. This suggests that the surface properties which influence thrombogenicity are conserved across specie lines. However, differences were noted between these species. The macaque platelet responses were considerably attenuated in comparison to those of the canine and human, which were in general quite similar. Thus, the macaque may be a less desirable model for human thrombgenesis.