A carefully taken family history and past history, physical examination, and examination of peripheral blood smear for platelets are suggested for more reliable and effective screening. (*Diamond, L. K., and Porter, F. S.: The Inadequacies of Routine Bleeding and Clotting Times, New England J. Med. 259: 1025 (Nov. 20) 1958.*)

BLOOD PLATELETS The survival period of blood platelets has been determined in thirty normal human recipients employing a standardized radioactive chromium tagging technique. "In vitro" measurements indicate that radioactive sodium chromate will bind to human blood platelets suspended in plasma. Determination of the radioactivity in the recipient's platelets indicates that the transfused platelets have a survival period of 9 to 11 days. There was no evidence of relabeling of the recipient's platelets or other blood elements. Platelet radioactivity in the recipient declines in a linear fashion suggesting that this method does measure the life span of the transfused platelets. (*Aas, K. A., and Gardner, F. H.: Survival of Blood Platelets Labeled with Chromium,*[51] *J. Clin. Invest. 37: 1257 (Sept.) 1958.*)

BLOOD PLATELETS It has become apparent that blood platelets, in addition to their role in blood coagulation, function as carriers of pharmacologically active amines. The distribution of adrenaline and noradrenaline between plasma and blood platelets has been studied by a fluorimetric method. In samples of human plasma the platelets contained a little over 50 per cent of the total catecholamines present in platelet-rich plasma. The platelet-bound proportion of adrenaline correlated with the platelet-bound proportion of noradrenaline. The concentration of catecholamines is about 125 times higher in platelets than in plasma. No catecholamines passed into serum from platelets during clotting. Lysis of platelets by freezing and thawing or by treatment with a surface-active agent resulted in a partial release. No uptake of adrenaline by platelets was observed in heparinized platelet-rich plasma at an adrenaline concentration of about 10 μg./l. An uptake resulting in a final concentration of platelet-bound adrenaline of about three times the initial concentration was found in citrated platelet-rich plasma at an adrenaline concentration of 80–200μg./l. Earlier results showing an increase of adrenaline in platelet-rich plasma after convulsion treatment was confirmed. (*Weil-Malherbe, H., and Bone, A. D.: The Association of Adrenaline and Noradrenaline with Blood Platelets, Biochem. J. 70: 14 (Sept.) 1958.*)

PLATELET SUBSTITUTE In the coagulation of blood, factors contained in platelets accelerate conversion of prothrombin to thrombin (Factor 1) and fibrinogen to fibrin (Factor 2); participate in the formation of thromboplastin (Factor 3); and neutralize the action of heparin (Factor 4). Not involved in coagulation but significant in hemostasis are the functions of platelets in promoting clot retraction and vasoconstriction. A soybean cephalin has been prepared that accelerates fibrin production from fibrinogen and has the same thromboplastic generation activity as platelets. However, it has no clot retractive or vasoconstrictor potency. Preliminary clinical trials indicate that this lipid may be useful in the therapy of thrombocytopenic states. (*Schulman, I., and others: Phosphatides as Platelet Substitutes in Blood Coagulation, Ann. New York Acad. Sc. 75: 195 (Oct. 13) 1958.*)

PLATELETS The function of platelets in hemostasis are considered from three aspects: (1) their clumping and fusing at the site of blood vessel injury; (2) their clotting function in which phospholipid seems the most important contribution; and (3) their role in maintaining resistance of capillaries to red cell extravasation. This may be chemical in nature and not depend on viable circulating platelets. Agglutination and fusion yielding hemostatic plugs *in vivo* are presumably produced by the same mechanisms causing viscous metamorphosis *in vitro*. pH of 7.4 seems essential. Fibrin needles adhere to the platelets and the clot is pulled together as the platelets agglutinate and fuse. Necessary for viscous metamorphosis and retraction are fibrinogen, thrombin, a nondialyzable serum factor, and calcium or magnesium. Since many types of enzyme inhibitors prevent retraction, many