SIMULTANEOUS MEASUREMENT OF PLATELET FACTOR 4 (PF4) AND β-THROMBOGLOBULIN (βTG) RELEASE AND FIBRINOPEPTIDE A (FFP) CLEAVAGE. K.L. Kaplan and H.L. Nossel. Department of Medicine, Columbia University College of Physicians and Surgeons, New York, New York, U.S.A.

Platelet activation and fibrin formation occur in thromboembolism, arterial disease, and intravascular coagulation. Selective involvement in certain disease entities and combined involvement in others has been suggested on the basis of turnover studies. The development in this laboratory of sensitive and specific radioimmunoassays for βTG, PF4, and stromabolin as indices of fibrin formation, and the availability of the radioimmunoassay for FFP as an index of fibrin formation, have allowed studies of the physiologic basis for differential involvement of platelets and fibrin formation. Simultaneous measurement of platelet activation, monitored by radioimmunoas-

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THE EFFECT OF THE FIBRINOGEN CONCENTRATION AND WHITE CELL COUNT ON INTRAVASCULAR FIBRIN DEPOSITION. Victor Ouerwich and Boguslaw Lipinski. St. Elizabeth’s Hospital, Boston, Mass., U.S.A.

The relationship between blood fibrinogen concentration and intravascular fibrin deposition was examined in BACI-treated rabbits infused with a standard amount of thrombin or plasmin sufficient to produce fibrin monomer (PM) but not defibrination. Fibrin in organs was measured by a microtiter plate assay and by a quantitative enzyme-linked technique. A significant (p<0.01) positive correlation was found between the baseline fibrinogen concentration and fibrin deposition 3 hours after infusion. By contrast, in vitro there was an inverse relationship between fibrinogen concentration and enzymatic clotting as well as non-enzymatic fibrin formation from soluble PM. When PM-treated leukocytic rabbits were infused, fibrin deposition was inhibited despite the fact that the animals’ plasma fibrinogen concentration was substantially increased by the PM infusion. When leukocytosis was induced by treatment with endotoxin, fibrin deposition was potentiated. It is concluded that fibrin deposition from circulating PM is facilitated by a raised fibrinogen concentration by a mechanism that cannot be explained by the well known in vitro interaction between PM and fibrinogen. Second, white cells appear to participate in the intravascular precipitation of circulating soluble PM complexes.

FIBRINOPEPTIDE A (FPA) LEVELS IN VENOUS THROMBOSIS AND PULMONARY EMBOLISM BEFORE AND DURING ANTICOAGULANT THERAPY. I. Yudelson, H.L. Nossel, K.L. Kaplan and J. Hirsh. Department of Medi-

FPA levels were measured in 60 patients subjected to venography and to lung scan for symptoms suggestive of venous thromboembolism. In all 23 patients with negative venography and/or lung scan, FPA levels were in the normal range (<1.3 pmol/ml, mean 0.6 pmol/ml). The FPA levels were elevated in 34 of the 37 patients with a positive lung scan and/or venogram. The range was 0.4-112 pmol/ml, median 6.2 pmol/ml. The FPA levels were measured serially in patients with confirmed thromboembolism who were treated with heparin. In 14 of the 15 patients there was a marked drop in FPA levels in the first 15 minutes after the initial dose of heparin. In 1 patient the FPA levels only reached the normal range after 48 hours of heparin therapy. In 4 patients FPA levels were measured daily in 10 patients while on anticoagulant therapy. In 4 patients FPA levels became normal and remained so and no symptoms occurred. In the other 6 patients there were 13 episodes of FPA elevations. 10 of these were preceded by a recurrence of the initial symptoms. In 1 patient FPA elevations occurred in the absence of symptoms while the repeat lung scan showed new lesions. Subsequent anticoagulation and/or the transition from heparin to Coumadin preceded the recurrence of symptoms in 6 out of 10 episodes. FPA levels were frequently normal in asymptomatic patients with evidence of venous thrombosis as shown by 14C fibrinogen uptake scan. These results suggest that FPA measurements may be useful in the diagnosis and in monitoring therapy of symptomatic thromboembolism.