THE EFFECT OF THE FIBRINOGEN CONCENTRATION AND WHITE CELL COUNT ON INTRAVASCULAR FIBRIN DEPOSITION.

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The relationship between blood fibrinogen concentration and intravascular fibrin deposition was examined in EACA-treated rabbits infused with a standard amount of thrombin or simiparasin sufficient to produce fibrin monomer (FM) but not defibrination. Fibrin in organs was assayed by a previously described quantitative technique using 125I-fibrinogen. A significant (p<0.01) positive correlation was found between the baseline fibrinogen concentration and fibrin deposition 3 hours after infusion. A parallel relationship between fibrinogen concentration and enzymatic clotting as well as non-enzymatic fibrin formation from soluble FM. Release of fibrinogen from circulating FM is facilitated by a raised fibrinogen concentration by a mechanism that cannot be explained by the well known in vitro interaction between FM and fibrinogen. Second, white cells appear to participate in the intravascular precipitation of circulating soluble FM complexes.

FIBRINONEPTIDE A (FPA) LEVELS IN VENOUS THROMBOSIS AND PULMONARY EMBOLISM BEFORE AND DURING AN-TICOAGULANT THERAPY. I. Yadav, M.L. Nosse, E.L. Kaplan and J. Hirsh, Department of Medicine, Columbia University College of Physicians and Surgeons, New York, New York, U.S.A., and Department of Pathology, McMaster University Medical Centre, Hamilton, Ontario, Canada.

FPA levels were measured in 60 patients subjected to venography and to lung scan for symptoms suggestive of venous thromboembolism. In 43 patients with negative venography and/or lung scan, FPA levels were in the normal range (<1.5 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 5.2 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. 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 recurred. 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 one patient FPA elevations occurred in the absence of symptoms while the repeat lung scan showed new lesions. Subcutaneous anticoagulation and/or the transition from heparin to Coumadin preceded the recurrence of symptoms in 6 out of 10 episodes. FPA levels vary frequently in asymptomatic patients with evidence of venous thrombosis as shown by 125I-fibrinogen uptake scan. These results suggest that FPA measurements may be useful in the diagnosis and in monitoring therapy of symptomatic thromboembolism.