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THE EFFECTS OF CIGARETTE SMOKING ON THE HEMOSTATIC SYSTEM. T.N. Meade, R. Chakrabarti, A.P. Raines, W.R. North and Yvonne Stirling. MRC-DRSS Epidemiology and Medical Care Unit, Northwick Park Hospital, Harrow, U.K.

The main purposes of the Northwick Park Heart Study (NPHE) are to improve the prediction of ischemic heart disease (IHD) and to elucidate its pathogenesis. Measures of hemostatic function are made along with those of variables already known to be associated with IHD; one characteristic of NPHE, particularly age and social class, are correlated with both cigarette smoking and with levels of some hemostatic variables, multiple regression analysis has been used to identify smoking effects independent of associations between smoking and these other characteristics. The hemostatic variables included in this analysis are factors V, VII and VIII, fibrinogen, antithrombin III, fibrinolytic activity, platelet count and platelet adhesiveness. In men, factor VIII levels and fibrinolytic activity are significantly lower in smokers than non-smokers; fibrinogen levels are significantly higher. In women, factor VIII levels are also significantly lower in smokers, but there is no difference in fibrinogen; for fibrinolytic activity there is an interaction between smoking and the use of oral contraceptives (OC). Activity in non-smokers on OC is substantially higher than in those not on OC, but activity in smokers on OC is the same as in women not on OC. It therefore seems that smoking abolishes the protective rise in fibrinolytic activity caused by OC. In men, cigarette smoking may promote thrombosis through effects on fibrinogen and fibrinolytic activity.

RELATION BETWEEN PLATELET VOLUMES AND PLATELET KINETICS IN IDIOPATHIC THROMBOCYTOPHENSIS PURPURA (ITP). T. Tsukada, Tsurumori General Hospital, Tokyo, Japan.

Platelet volume distribution and appearance rate of megathrombocytes were measured with Coulter counter Model-B in 24 patients with ITP during platelet kinetic studies. Normal range of platelet volumes in EDTA-blood is as follows: mode: 6.8 ± 0.8 μm³(mean ± 2 SD), median: 7.6 ± 0.8 μm³ and mean volume: 9.4 ± 1.1 μm³. In 15 patients platelets were increased in mean volumes, 8 patients showed normal size and one patient decreased in mean volumes. Increase of megathrombocytes (larger than 14 μm³) was noted in 14 cases. Remaining 10 cases showed normal appearance rate of megathrombocytes (15 ± 4.7 %). In 5 patients, which was contributed to the smaller elements although the mean volumes were remained in normal range or increased. These findings suggest that at least two population of platelets are present simultaneously in these cases. No close correlation was noted between platelet volumes and platelet counts, mean platelet survival time or platelet turnover. The appearance rate of megathrombocytes also had no correlation with the results of platelet kinetics. Three out of 11 cases whose platelet counts increased to the normal after splenectomy showed still larger platelet volumes than the normal.

SPECIFICITY OF FACTOR VIII INHIBITOR FOR PROCOAGULANT ACTIVITY. H. Yang and H. Kubur.
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Nine hemophilia A patients with an inhibitor to factor VIII procoagulant and eight without an inhibitor were studied for the presence of an inhibitor to von Willebrand factor (vWF). After a 3 hour incubation at 37°C, test plasma, normal pooled plasma and test plasma mixed with normal pooled plasma were assayed for vWF in the quantitative ristocetin-induced platelet aggregation system. The mean vWF was 1.315±0.59 unit/ml in the non-inhibitor group and 1.66±0.52 in the inhibitor group. In the non-inhibitor group the predicted vWF (sumation of the test sample and normal plasma) was 1.308±0.77 unit/ml, whereas the actual vWF of the mixture was 1.22±1.5. In the inhibitor group the predicted vWF was 1.65±0.33 unit/ml whereas the actual vWF of the mixture was 1.44±0.60. The results in the two groups were not significantly different. Neither group had inhibitory activity for vWF. The factor VIII inhibitor is highly specific for the procoagulant part of the factor VIII complex.