PLATELET FUNCTION IN PRE-ECLAMPSIA. K.A.E. Whigham, P.W. Howie, A.H. Drummond and C.R.M. Prentice. University Department of Medicine, Royal Infirmary, Glasgow, Scotland.

As pre-eclampsia is associated with thrombocytopenia, it is possible that platelets contribute to the intravascular coagulation in this disease. Platelet functions were studied in 10 pre-eclamptic and normal pregnant women, and in women with mild or severe pre-eclampsia. Platelet aggregation was measured in response to six aggregating agents; maximum aggregation rates and the concentration of each agent required to induce half-maximal aggregation (E.C. 50 value) were determined. Platelets from women with severe pre-eclampsia were less responsive than normal, with significantly lower maximum aggregation rates in response to collagen, vasopressin and arachidonic acid, and a higher than normal E.C. 50 value for collagen. Women with severe pre-eclampsia also had a significantly raised plasma adenine nucleotide concentration (2.99 ± 2.35 vs 0.74 ± 0.85 nmol/ 10^8 cells) and a reduced platelet 5-hydroxytryptamine concentration (0.190 ± 0.293 vs 0.508 ± 0.340 μ g/ 10^8 cells). In mild pre-eclampsia, only the maximum rate of collagen-induced aggregation differed from normal. Platelets in patients with severe pre-eclampsia may have undergone the release reaction in vivo, in keeping with the close association between pre-eclampsia and intravascular coagulation.

COMPARATIVE STUDY ON THE EFFECT OF CARBENICILLIN, INHIBITORS OF PLATELET FUNCTION AND HEPARIN ON EXPERIMENTAL THROMBUS FORMATION. R. Zimmermann, K. Andrassy, C. Zeltzsch, D. Lange and F. Hof. Med. Universitätsklinik, Heidelberg, G.F.R.

Previous studies documented an impairment of haemostasis by synthetic penicillins (Thromb. Haem. 34:115, 1976) and penicillin (Lancet II:1039, 1976). Therefore the antithrombotic activity of synthetic penicillin (carbenicillin) (C) was compared with that of aspirin (ASA), dipyridamole (DIPY) and heparin in 150 rabbits. Thrombus formation was induced by standardized endothelial lesions. The dose of C was adjusted to a 4.2 fold prolongation of bleeding time, similar to that seen in clinical patients. Analysis and composition of thrombi was done by measurement of incorporation of labeled blood elements (^{51}Cr labeled platelets, ^{125}I -fibrinogen and ^{59}Fe labeled red cells). The 'specific thrombus/blood ratio' with values of 19.1 and 50.9 (^{51}Cr) in venous and arterial thrombi evidenced the significance of platelets in this model. In the venous system C reduced formation of thrombi by 43%, ASA by 34%, ASA and DIPY by 55% and heparin by 90%. In the arterial system C inhibited thrombus formation by 89%, ASA by 15%, ASA and DIPY by 46% and heparin by 60%. It is concluded, that C effectively prevents thrombus formation in the arterial system and to lower extent in the venous system. The results prove the importance of platelets in arterial thrombogenesis and the efficacy of platelet aggregation inhibitors in preventing thrombi in the arterial system. In comparison with other known antiplatelet drugs it seems, that C is the most effective platelet aggregation inhibitor to date.

KINETIC STUDIES OF FIBRINOGEN IN PREGNANT RABBITS. R.-M. Moeller, I. Mahn, and G. Müller-Berghaus. Justus-Liebig-Universität, Giessen, West Germany.

During gestation increased concentrations of fibrin(ogen) derivatives are observed indicative of intravascular action of thrombin. The aim of this study was to elucidate the kinetics of fibrinogen during gestation. The elimination of homologous I-125-fibrinogen was studied in 14 pregnant rabbits during the first as well as during the third trimesters of gestation. Control studies were performed with 10 non-pregnant rabbits.

The mean distribution volume of labeled fibrinogen did not significantly differ between pregnant and non-pregnant rabbits. During the third trimester pregnant rabbits demonstrated a pronounced shortening of T 1/2 of labeled fibrinogen from a mean of 55.3 hr during the first to a mean of 29.7 hr during the third trimester. The experiments showed a significant increase in the fractional catabolic rate from 45.0 to 69.9% per day in the course of gestation. The shortening of T 1/2 of labeled fibrinogen correlated to the number of fetuses per litter.

This study indicates an average acceleration of the fibrinogen turnover during gestation of about 50%. These direct quantitative measurements demonstrate that fibrinogen catabolism is pronouncedly accelerated during pregnancy.