1050 RISTOCETIN INDUCED PLATELET FACTOR 3 ACTIVITY

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In human platelet rich plasma a pronounced Pf 3 activity was induced by aggregating doses of ristocetin. Following centrifugation of aggregated platelets no Pf 3 activity could be detected in the supernatant, i.e., it remained associated with the altered platelet surface membrane. At a ristocetin dose that induced two wave aggregation aspirin pretreatment prevented irreversible aggregation but not Pf 3 availability. In contrast, both second wave aggregation and the development of Pf 3 activity were inhibited by the potent antiaggregating agent PGI_2. In platelets isolated on albumin density gradient and free of plasmatic VIIIR:WF no Pf 3 activity was induced by ristocetin, however, aggregability as well as Pf 3 availability to ristocetin could be restored by platelet poor plasma replacement. Estimation of ristocetin induced Pf 3 activity appears to be a procedure of diagnostic value in von Willebrand disease.

1051 COAGULATION ACTIVITY OF SURFACE ACTIVATED PLASMA.

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Vessel stasis itself does not cause a thrombus in experimental animals. Some additional stimulus to coagulation has to be superimposed for a thrombus to occur. Previously injections of serum have been used for this purpose. In the present study small amounts of human plasma, activated against a surface in vitro, injected prior to ligation of the jugular vein caused thrombosis in rabbits. Plasma not activated failed to do so. The coagulation enhancing principle was dependent upon factors XII, XI, IX and VIII and was stable in vitro but rapidly disappeared in vivo. Heparin inhibited this activity and did so more easily when added prior to surface activation as compared to after. Recalcification time of surface activated plasma was studied in vitro using a thrombometer[R]. This instrument measures the light scattering caused by the clot. Recalcification time of surface activated plasma was shorter than non activated plasma. This shortening was dependent upon factors XII and XI and was also inhibited by heparin. Also in this case heparin was more effective if added prior to surface activation. Surface activated plasma added in minute amounts to normal plasma shortened the recalcification time of the latter. Patients on low dose heparin theraphy had a prolonged recalcification time postoperatively whereas patients without such theraphy showed a shortened time postoperatively. Plasma from patients without low dose heparin was more readily surface activated.

1052 THE EVALUATION OF THE ROLE OF THE KALLIKREIN-KININ SYSTEMS WITH COAGULATION FACTORS DURING PREGNANCY DELIVERY AND PUERIPERIUM

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(Purpose) While it is a well-known fact that the majority of blood coagulation factors are increasing in the latter half of Pregnancy and Delivery, the correlation between Kallikrein-Kinin Systems and Coagulation factors has not yet been brought to a full explanation. From this point of view, Kininogens and individual coagulation factors were hereby determined in order to clarify the state of hypercoagulability and DIC during Pregnancy and Delivery.

(Method) 1) In 20 normal cases from Pregnancy through to Delivery and Pueriperium and 10 cases of DIC, Kininogen were measured with the method of Bioassay using Rat-Uterus. (DINIZ Method) 2) The state of hypercoagulability in the background of DIC was evaluated by determining many coagulation-factors including Contact Factors.

(Results) 1) In the latter half of the period of gestation, individual factors (excluding Factor XIII) were increasing, especially before Delivery. 2) The high levels of the kininogen in plasma were proved during Pregnancy and before Delivery(8.4 ± 3.4 μg/ml) than that of nonpregnancy (4.3 ± 2.1 μg/ml) as well as many other coagulation factors. 3) In the cases of DIC, the extremely low level of Kininogen (2.2 ± 1.4 μg/ml) were proved.

(Conclusion) The levels of Kininogen seems to play an important role in the mechanismus of DIC which start before and immediately after Delivery.