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## 0678 COMPARISON BETWEEN PLASMA LEVELS OF β-THROMBOGLOBULIN AND PLATELET FACTOR 4 IN VARIOUS DISEASES

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Concentrations of β-thromboglobulin (β-TG) and platelet factor 4 (PF-4) in platelet poor plasma were determined using radioimmunoassay in 97 healthy subjects and 307 hospitalized patients. In normal subjects, mean values (and SEM) of β-TG and PF-4 were 37±2 ng/ml and 8.3±1.5 ng/ml, respectively. Levels of β-TG increased with age. A statistically significant correlation between concentrations of β-TG and PF-4 was observed in 30 normal subjects ($r=0.77$, $p<0.001$). Levels of these protein increased in cases of DIC, acute deep vein thrombosis, acute myocardial infarction, acute cerebral infarction, transient ischemic attacks, diabetes mellitus with retinopathy or nephrotic syndrome, whereas these decreased in patients with aplastic anemia or acute leukemia. In a case of DIC with suppressed production of platelets in bone marrow, concentrations of β-TG and PF-4 remained within normal limits. There were few cases, in whom discrepancies between levels of β-TG and PF-4 were observed. Following oral administration of dipyridamole (300 mg daily), concentrations of β-TG and PF-4 decreased. From these results, it is concluded that elevation of β-TG or PF-4 indicates hypercoagulable states.

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## 0679 ASSESSMENT OF CLINICAL HYPERCOAGULABILITY TENDENCY

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Optimal assessment of hypercoagulability which may result in various types of intravascular clotting requires the following procedures. No storage of blood specimens, analyzation right after withdrawal of blood (with few persons involved in work), blood obtained with siliconized, not plastic syringes. Use of citrate Na as anticoagulant by only 20 % dilution, further dilution increases but does not decrease coagulability. Hypercoagulability can be correctly assessed with the thrombin generation test resulting in a curve leaving clotting factors in their natural relation and platelets in suspension. Synthetic substrates can be misleading. Some patients with a prothrombin time in therapeutic range have a normal thrombin generation showing that they are not protected. Short antithrombin III times reflect reduction of another protection against intravascular clotting. They must become prolonged by prothrombin-depressing agents, if not these agents are ineffective. Platelet aggregation induced by stirring only reveals another type of hypercoagulability tendency which cannot be treated with anticoagulants. Prolonged euglobulin lysis time reflects loss of yet another type of protection against an intravascular clotting potential. Fast test for fibrin monomeres should be added. The tests mentioned revealed various types of hypercoagulability in most of the many patients who were referred to us for assessment of this tendency.

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## 0680 PLASMINOGEN ACTIVITY IN PLASMA AND IN SERUM BEFORE AND AFTER VENOSTASIS

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When venostasis is applied in men for 10 minutes at a pressure half-way between diastolic and systolic, various results may be obtained as far as fibrinolysis is concerned. Normally the euglobulin lysis time reduced from above 3 hours to less than one hour. In these conditions, after venostasis the estimation of plasminogen after activation by streptokinase (S.K.) on synthetic substrate S2251, shows 20-80 % greater activity in serum than in plasma. In some patients at high risks of thrombosis, the euglobulin lysis time is unchanged after venostasis. In these cases, the biological activity of plasminogen is slightly greater in plasma than in serum, as is observed before venostasis in all subjects.

In order to explain these observations, experiments were performed using placental or plasmatic (glu or lys) plasminogen ; these show that for the same antigen activity, the biological activity of placental plasminogen previously activated by S.K. is greater than that of plasmatic plasminogen. We suggest a modification of the structure of plasminogen in the presence of activators and of fibrin clots. Further experiments will be necessary to explain the modification of plasminogen structure.