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HORMONAL REPLACEMENT THERAPY IN POSTMENOPAUSAL WOMEN (TRANSDERMAL ESTRADIOL AND MEDROXYPROGESTERONE ACETATE ORALLY) AND STATUS OF COAGULATION AND FIBRINOLYTIC SYSTEM

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Prospective studies that included 116 postmenopausal women on hormonal replacement therapy (transdermal estradiol and medroxyprogesterone acetate orally) were performed. Plasma levels of prothrombin fragment F1+2 (F1+2), thrombin-antithrombin III complex (TAT), activities of antithrombin III (AT III), protein C (PC), protein S (PS), plasminogen activator inhibitor -1 (PAI-1) and fibrinogen level were measured before, after 6 and 12 months of continuous treatment. A comparison between the levels of the coagulation markers (F1+2, TAT complex) and endogenous anticoagulants (AT III, PC, PS) revealed no statistically significant differences. Plasma activity of PAI-1 showed significant decrease after 6 month of therapy. Plasma fibrinogen concentration significantly decreased ($p < 0.05$) after 12 months of treatment (271.27 mg/dl/71.08 vs. 238.33 mg/dl/34.48).

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CHANGES IN SOME PARAMETERS OF THE COAGULATION AND FIBRINOLYSIS SYSTEMS FOLLOWING FACIO-MANDIBULAR SURGERY.

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Thrombotic complications are still a major risk factor following surgery, despite modern operating techniques and anticoagulation therapy. Numerous studies have been performed examining coagulation and fibrinolytic parameters following surgery but to our knowledge, no in depth studies have been performed in blood samples from patients undergoing facio-mandibular surgery (FMS).

In this study we compared 2 groups of 20 patients with cancer undergoing FMS and anticoagulated with either Fragmin (FG) or Fraxiparin (FP) (5000 anti-FXa units as a bolus injection). Blood samples were taken immediately after and 1-4 days post surgery and levels of various parameters of the coagulation and fibrinolytic systems were measured. Significant differences between the two groups were found for some parameters. FXII levels were lower one day post surgery and kallikrein like activities significantly higher at all sampling times in the FP group. D-dimers were significantly elevated from day 1 to day 4 following surgery in the FP group and TAT complexes were significantly higher in this group. t-PA levels were also significantly higher and PAI levels significantly lower in the FP group.

Our results indicate that reduced contact system activation, coagulation and fibrinolysis occurred in the patients treated with Fragmin which might be of significance for future anticoagulant selection for FMS.

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ACTIVATION OF THE HEMOSTATIC SYSTEM DOES NOT CAUSE NEUTROPHIL DEGRANULATION IN UNSTABLE ANGINA.

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To assess whether activation of the thrombotic system may induce neutrophil degranulation in unstable angina (UA), we studied the time course of the intracellular index of myeloperoxidase (MPXI), a marker of neutrophil activation, in relation to levels of Thrombin-antithrombin III complexes (TAT) and of Fragment 1+2 (F1+2) as markers of activation of the coagulation system in 20 patients (pts) with UA and in 20 healthy volunteers (N). Samples were taken on admission, and at 6, 24, 48 and 72 hours.

Results (median and range): On admission no differences were found between UA and N in TAT e F1+2 values: TAT: 2.05 ng/ml (range 0.5/-14.4) vs 2.1 ng/ml (range 1.05/4.2); F1+2: 0.83 nmol/l (range 0.14/-1.65) vs 0.59 nmol/l (range 0.41/0.98), conversely MPXI levels were significantly different in UA vs N: respectively -4 (range +5/-15) vs -1.3 (range +5.5/-7, $p < 0.01$). During the study 11 episodes of elevation of TAT and/or F1+2 (6 symptomatic and 5 asymptomatic) followed by at least one sample for MPXI were observed. No changes in MPXI values after the episodes of activation of the coagulation as compared to baseline values were observed (-3.5, range -11.4/1.2 vs -4.7, range -13.1/4.5 respectively). There was no correlation between MPXI and TAT, and MPXI and F1+2.

Conclusions: Our data suggest that activation of the coagulation system doesn't elicit systemic neutrophil activation in unstable angina; this may suggests that a primary, yet unknown inflammatory cause, may be responsible for the neutrophil activation observed in unstable angina.

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FIBRINOLYSIS ACTIVATION IN SYNOVIAL FLUID FROM KNEE JOINT.

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Nothing is known about fibrinolytic process within synovial fluid which complicated joint trauma.

The aim of study was to evaluate fibrinolytic activity of this kind synovial fluid (s.f.) from knee joint. The study group consisted of 30 patients aged 22- 66 in whom synovial fluid appeared in knee joint 1- 8 months after trauma. The s. f. was removed from joint to prevent the destruction of joint cartilage. In the s.f. and in the blood of patients tissue plasminogen activator antigen (t- PA Ag), urokinase plasminogen activator (u-PA) activity and plasmin- alfa-2- antiplasmin complexes (PAP) were measured.

The results are given in the table.

parameter	synovial fluid		plasma	
	ng/ml	ng/ mg of protein	ng/ml	ng/mg of protein
PAP complexes	2363,5 ± 1128,2	63,3	181 ± 68,0	2,80
t-PA Ag	2,0 ± 1,14	0,54	9,86 ± 1,85	0,152
u-PA Act	0,27 ± 0,23	0,0072	0,28 ± 0,13	0,0043

In the synovial fluid we detected very high concentration of PAP complexes and the presence of t-PA antigen and u-PA activity. Calculation per 1 mg of protein showed that in s.f. concentration of PAP complexes was about 23 times and u-PA activity 1,6 times higher than in plasma, but t-PA Ag 3 times lower.

We concluded that fibrinolysis activation observed in s.f. from knee joint depended on u-PA and t-PA.