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PROTEIN C AND AT III IN ESSENTIAL HYPERTENSION.
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Since hypertension is generally accepted as a risk factor for atherosclerosis and,on the other hand,alterations in haemostasis were reported in the disease,we focused our interest on two plasma inhibitors-protein C and antithrombin III, which play critical roles in the regulation of blood clotting process. We studied 19 patients with newly diagnosed essential hypertension at 1st and 2nd degree of the disease (acc.to WHO criteria). Protein C antigen level was measured by means of ELISA Protein C test (Boehringer Mannheim GmbH). AT III and Factor VIII R:Ag concentrations were measured by rocket immunoelectrophoresis acc.to Laurell using monospecific antisera (Behringwerke AG,Marburg). In comparison with healthy subjects the patients with essential hypertension revealed statistically significant decrease in protein C antigen level (79 ± 21% vs 101 ± 16; $p < 0.01$) and substantially increased F.VIII R:Ag values (174 ± 72% vs 103 ± 33%; $p < 0.01$). No significant correlation was found between F.VIII R:Ag and protein C level. The patients had also elevation of AT III concentration of no statistical significance. Since F.VIII R:Ag is produced and released by endothelial cells its elevated level in patients with hypertension may reflect injury to the vascular wall. Decreased levels of protein C probably due to its increased consumption observed in the patients may contribute to imbalance in the haemostatic system and promote vascular complications.

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PLATELET FUNCTION AND KALLIKREIN SYSTEM IN PATIENTS WITH ESSENTIAL HYPERTENSION.
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Since the pathogenesis of hypertension is still discussed the aim of this study was to investigate behaviour of platelets and kallikrein system in patients suffering from this disease. In 30 patients with essential hypertension, aged 23-51 years and 20 normotensive healthy subjects, aged 21-55 years the following parameters of platelet function were studied: platelet aggregation induced with ADP,platelet activating factor (PAF) and arachidonic acid (AA) according Born's method, plasma beta-thromboglobulin (Beta-TG) and platelet factor 4 (PF4), plasma thromboxane B_2 (TXB_2) and cyclic AMP using radioimmunoassay kits. The activity of kallikrein and factor XII was also determined using Chromozym PK (Boehringer Mannheim GmbH). The hypertensive patients demonstrated a greater platelet aggregability by ADP and PAF, elevated concentrations of Beta-TG and TXB_2 as well as decreased level of cyclic AMP in comparison to normotensive subjects. No significantly changes in platelet aggregability by AA and activity of PF4 were found in the group of hypertensive patients. There were also significantly decreased plasma concentrations of kallikrein and factor XII in these patients. Obtained results have shown hyperfunction of blood platelets and depletion of kallikrein system in the patients with essential hypertension. These results suggest that among different pathogenic factors function of blood platelets and kallikrein system should be taken into consideration.

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FACTOR VIII AND FACTOR XII LEVELS IN BORDERLINE HYPERTENSION.
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Borderline hypertension causes mortality and morbidity rates similar to those associated with estabilished hypertension. However, there is no univocal guideline for its therapeutic management. Hypercoagulability in hypertension has been demonstrated. The aim of our study was to evaluate some coagulation factors in a group of patients affected by borderline hypertension. The following tests were carried out: PT and PTT, Factor VIII coagulant activity, FVIII antigen and FVIII ristocetin cofactor, Factor XII and Factor XI activities. These tests were selected for their relationship to the contact coagulative activation near the vascular wall. In our patients statistically significant higher FVIII and FXII coagulant activities than normal control subjects were found. Moreover, an evident even though not statistically significant PTT shortening was seen. Other tests taken into consideration were all within normal limits. Our results suggest that an increased FVIII and FXII synthesis and/or release is present, and an activated coagulation system exists in borderline hypertension. Furthermore, it is not clear why an excess of FVIII:C over FVIIIR:Ag and FVIIIR:RCof was found in our patients. In conclusion, an activation of haemostatic mechanism was found in borderline hypertension. The young age of patients and the absence of evident hypertensive angiopathy are in agreement with an overactivity of blood vessel tone. Haemostatic activation could be an useful marker in favour of the precious management of patients with borderline hypertension.

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TREATMENT OF RAISED BLOOD PRESSURE WITH NISOLDIPINE REDUCES "SPONTANEOUS" PLATELET AGGREGATION IN WHOLE BLOOD.
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Hypertensive patients are at risk of premature vascular disease, and abnormal platelet function in hypertensive subjects may contribute to vascular damage. In a placebo controlled, double blind, cross over study, we have investigated the effect of treatment of moderate hypertension with nisoldipine (a 1,4, dihydropyridine calcium antagonist) on several aspects of platelet function. In 12 hypertensive subjects, venous blood samples were taken for platelet count, PCV, βTG, PF_4, and aggregation to standard doses of ADP and adrenaline in whole blood. Platelet aggregation in whole blood which occurred during processing (spontaneous aggregation) was also recorded. Samples were collected on four occasions: after 4 weeks treatment with placebo (A), after 6 weeks and 12 weeks of the crossover phase with either nisoldipine (B) or placebo (C), and finally after re-establishment of blood pressure control on routine therapy (D). Nisoldipine was effective in lowering blood pressure, with mean values during the 4 treatment phases of: A 119 mmHg, B 104 mmHg ($p < 0.01$), C 114 mmHg, D 103 mmHg.

Neither nisoldipine nor routine treatment significantly affected platelet count, PCV, βTG, PF_4 or aggregation in whole blood to adrenalin or ADP. "Spontaneous aggregation" in whole blood however, was significantly inhibited by reduction in blood pressure during treatment both with nisoldipine and routine drugs. Treatment of hypertension appears to raise the threshold of blood platelets for aggregation regardless of the pharmacological agent used.

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