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0342 PLATELET HYPERSENSITIVITY IN ACUTE MYOCARDIAL INFARCTION (M.I.) PATIENTS (PTS)

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A national multicentre controlled trial (ARIS) to determine the effect of sulfinpyrazone (S) (400 mg twice a day) on m.i. recurrence and cardiac death prevention is now in its 3rd year. Admission of new pts terminates on June 30, 1979. Various platelet (pl) and coagulatory parameters are measured in a randomised series of 175 pts at trial entry (10-20 days after m.i. episode) and at various times during treatment with S or placebo. This has provided the opportunity of comparing (factorial ANOVA analysis for unbalanced groups) basal results in m.i. pts and in 45 normal matching controls: no difference in pl count, pl retention and PTT; m.i. pts had significantly shorter bleeding time (146.99±80.47 vs 264.11±112.98 sec), heparin thrombin clotting time (HTCT) (29.97±16.47 vs 36.31±11.31), and significant increase of fibrinogenemia (339.85±179.94 vs 237.85±35.77 mg%) and PF_4 release (37.30±15.13 vs 28.00±12.38 U/ml). ADP (0.2, 0.4 and 0.8 μM) produced pl aggregation curves in m.i. pts showing significantly increased 1st wave slope with 0.2 μM and maximal extent at 0.2 and 0.4 μM; significantly higher frequency of 2nd wave at 0.4 and 0.8 μM. Collagen (1.25, 2.5 and 5 μg) produced pl aggregation curves with significantly reduced lag period at 2.5 μg, steeper slope and higher maximal aggregation at 1.25 and 5 μg. The only important correlation (inverse) was between HTCT and PF_4 release in the m.i. group.

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0343 RELATIONSHIP BETWEEN PLASMA CONCENTRATIONS OF FIBRONECTIN, α - LIPOPROTEIN AND PLASMINOGEN

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In plasma samples of 24 patients with angiographically documented coronary artery disease the concentration of 12 proteins was determined by radial diffusion of monospecific antisera against gels containing various dilutions of plasma. The concentration of fibronectin, recently discussed to promote phagocytosis by cells of the reticulo - endothelial system, showed a good correlation ($r = 0.541$, $2P \leq 0.01$) with that of α - lipoprotein, a potentially antiatherogenic factor. In addition a significant correlation ($r = 0.515$, $2P \leq 0.05$) was found between the amounts of α - lipoprotein and plasminogen suggesting a possible relationship between various protective factors, which in some cases may promote regression of atherosclerosis.

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0344 ABNORMAL PLATELET FUNCTION IN RAYNAUD'S PHENOMENON.

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Platelet aggregation (PA) induced by adenosine diphosphate (ADP) 0.6 and 4μM, l-ephinephrine 6μM, collagen 1u/ml and thrombin 0.2u/ml has been measured in citrated platelet rich plasma of 38 patients (mean age 42.2) suffering from Raynaud's Phenomenon. In 29 of them, plasma β-thromboglobulin (βTG), measured by radioimmunoassay was determined and the results were compared to age and sex matched healthy subjects and correlated to serum immunoglobulin (Ig) and plasma fibrinogen levels. Plasma βTG (mean 72.2ng/ml, ranged 20-209) was significantly elevated in the patients compared to the controls ($p \leq 0.005$). The rate and extent of platelet aggregation however, though higher in the patients than controls were significant only to ADP 0.6μM ($p \leq 0.045$) rate and $p \leq 0.002$ extent) and 4μM ($p \leq 0.005$ rate and $p \leq 0.004$ extent). Serum IgM levels were also abnormally increased in 12, IGA in one and IgG in 4 patients. There was no correlation between the platelet studies and plasma fibrinogen or serum immunoglobulin suggesting that these tests are measuring various aspects of the disease state. These results indicate that in-vivo platelet activation and "release reaction" are enhanced in patients suffering from Raynaud's Phenomenon presumably not only by abnormal plasma factors. Furthermore, they suggest that platelets may be involved in the pathogenesis of the disease and its complications.