Time

09.30 **0849** STYPVEN TIME MEASUREMENT OF PLASMA AFTER FILTRATION: EFFECTS OF CIGARETTE SMOKING

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Normal plasma contains nonsedimentable platelet factor-3 (NS-PF3) activity, thought to be caused by circulating platelet membrane fragments. Stypven time (ST), an assay for PF-3 activity, of plasma prepared by differential centrifugation and by filtration through 0.22 μ Millipore filters were investigated. The average ST for platelet-rich plasma (PRP), low-spin platelet-poor plasma (LSPPP), medium-spin PPP (MSPPP), high-spin PPP (HSPPP) and filtered PPP (FPPP) was 28.0, 40.4, 43.4, 61.7 and 65.5 sec, respectively (27 determinations). Filtration of plasma did not affect factor V and X activities. Material eluted from filters after filtration of plasma consisted of membrane vesicles with high PF-3 activity. ST were then measured in plasma preparations obtained from smoking (S) (>15 cigarettes/day) and nonsmoking (NS) healthy male individuals, ages (A) between 45-64. Data obtained were grouped according to age and smoking habits (Gr. I, 9 S, A 45-64; Gr. II, 14 NS, A 45-54; Gr. III, 7 S, A 55-64; Gr. IV, 14 S, A 55-64) and subjected to two-way analysis variance employing the BMDP2V program. Significant shortening of ST was noted in LSPPP ($p<0.02$) and MSPPP ($p<0.02$) in smoking groups which, however, showed no significant differences in PRP ($p>0.8$), HSPPP ($p>0.06$) and FPPP ($p>0.4$). Results suggest smoking individuals exhibit significantly higher NS-PF3 activity in plasma.

09.45 **0850** BETA THROMBOGLOBULIN AND MYOCARDIAL INFARCTION

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Beta thromboglobulin, a platelet specific protein liberated during the release reaction, has been measured in normal individuals (n=285), and in patients presenting with acute chest pain. The latter group consisted of those with acute myocardial infarction (n=19), those with acute myocardial ischaemia (n=21), and those with chest pain of non cardiac origin (n=7). In the patient groups beta thromboglobulin was measured on admission to hospital, and thereafter daily until the patient was discharged.
There was no significant difference between the normal population (mean 22.5 ng/ml), and the patients with with non cardiac chest pain (mean 24 ng/ml). There was a significant difference between the normal population and the patients with acute myocardial infarction (mean 34 ng/ml), and acute myocardial ischaemia (mean 33 ng/ml), $p<0.001$. There was also a significant difference between these two groups and the patients with non cardiac chest pain, $p<0.01$.
We would conclude that platelet activation occurs in acute myocardial infarction and ischaemia, but it is not clear if this is a primary or a secondary phenomenon.

10.00 **0851** PLATELET AGGREGATION AND SEROTONIN RELEASE, COAGULATION, FIBRINOLYSIS, ANTIPLASMIN, FACTOR XIII, AND ANTITHROMBIN III IN ACUTE TRANSMURAL NON-COMPLICATED MYO-CARDIAL INFARCTION.

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Platelet aggregation in vitro (ADP and adrenalin), Serotonin release, APTT, Factor VIII, Factor XIII, FR-a, plasma ethanol gelation test, euglobulinclotlysistime, antiplasmin and antithrombin III were estimated in 12 patients with acute transmural non-complicated myocardial infarctions receiving no drugs with any known influence on the actual parameters. Distinct, uniform, changes were found. Initially distinct decrease in aggregation and release was present, changing within a week into increased aggregability, as estimated by threshold concentrations, and increased release function. The platelets remained "hyperactive" in above 50% at the discharge. Positive gelation test appeared after 1-2 days with peak level day 5, became afterwards negative in all. The fibrinolytic activity was within normal range day 1-2. It decreased rapidly with lowest activity on day 5-6 contemporarily with a peak activity of antiplasmin. Afterwards it increased slowly, but was still low in around 50% at the discharge. Factor VIII activity increased significantly with peak day 5-6. Factor XIII decreased biologically with lowest activity on day 5-6, increased to normal levels. Antithrombin III remained unchanged, in upper part of normal range. Thus, changes in platelet patterns are demonstrable at the onset of the infarction, while the other parameters develop later.