CIRCULATING PLATELET AGGREGATES DURING EMOTIONAL STRESS AND CIGARETTE SMOKING. G.F. Gensini, R. Abbate, D. Prisco and G.G. Neri Serneri. University of Florence Medical School, Florence, Italy.

Increased platelet aggregation has been observed in various hypercoagulable states, but its predictive value for thrombosis is so far uncertain. We studied the effect of emotional stress and of cigarette smoking on circulating platelets by platelet aggregates ratio (PAR) according to Wu and Hoak (1974) in medical students aged 20-22 years. The emotional stress was undergoing a University examination. PAR was measured immediately before the examination, at the end and 15 and 30 min after the examination. PAR was significantly lowered in all the subjects at the end ($P < 0.01$) and after 15 min ($P < 0.01$) but returned toward normal values after 30 min. The decrease of PAR suggests the production of reversible circulating platelet aggregates. The effect of smoking a cigarette has been investigated in 8 students. PAR has been determined before smoking, at the end and after 2, 5 and 10 min. Smoking lasted 4 min. In 5 subjects we observed a decrease of PAR at 2 min ($P < 0.01$), whereas at 5 and 10 min PAR value became normal. Lettuce cigarette smoke did not affect PAR value. Our results indicate that: 1) - Platelet aggregates are very easily produced in circulating blood; 2) - A low value of PAR does not necessarily indicate a platelet hyperaggregability clinically significant.

INCREASED PLATELET AGGREGATION DUE TO A PLASMATIC AGGREGATING ACTIVITY. G.G. Neri Serneri, R. Abbate, G.F. Gensini, S. Favilla and C. Mugnaini. University of Florence Medical School, Florence, Italy.

In various clinical states an increased platelet aggregation has been observed, but its mechanism(s) is not yet completely understood. Plasma of some patients with history of myocardial infarction (MIP) or with cerebrovascular disorders (CVP) delays platelet disaggregation after aggregation by ADP (Neri Serneri et al 1974) and induces morphological changes in control platelets (Schatz and Gross 1975). In a group of 27 MIP and 20 CVP we identified 16 MIP and 12 CVP with plasmatic aggregating activity (AA) by cross-matches in a modification of the method of Wu and Hoak (1974) for platelet aggregates. As the AA disappeared after heparin treatment (12,500 U x 2 for 7 days) we investigated whether the AA was related to an activation of clotting processes. At this purpose we measured in these patients and in those without AA the concentration of high molecular weight fibrinogen complexes (HMWFC) by agarose 4% gel-filtration. The patients with AA showed a significantly ($P < 0.01$) higher HMWFC concentration ($9.68 \pm 2.15\%$) than those without AA ($5.41 \pm 1.7\%$) but the AA was not related to the HMWFC themselves, which on the contrary showed a mild antiaggregating activity. On gel-chromatography of the whole plasma the AA was eluted at an elution volume at which factor X is usually collected.

EFFECTS OF LIPID-LOWERING DRUGS UPON FASTING-INDUCED PLATELET AGGREGATION. K. Gjesdal. Aker Hospital, Oslo, Norway.

During fasting, plasma free fatty acid (FFA) concentration increases, correlating with an increase in the percentage of venous reversible platelet aggregates (Gjesdal et al, Thromb. Haemostas. 36, 325, 1976). In the present study 20 healthy males, who had fasted for 72 hours, received either 1 g of sodium salicylate (SS) or 0.2 g of a nicotinic acid analogue (NAA) orally. Blood was collected before, and 1/2, 1 and 2 hours after drug intake. FFA was assayed by titration and platelet aggregates according to Wu and Hoak (Lancet 2, 924, 1974).

After SS, mean FFA concentration was reduced ($p < 0.01$) from 1.87 to 1.34 (1/2h), 1.49 (1h) and 1.66 (2h) mmol/l, whereas NAA resulted in a reduction ($p < 0.01$) from 1.43 to 0.94 (1/2h), 0.65 (1h) and 0.67 (2h) mmol/l. Concomitantly, platelet aggregate percentage decreased transiently (not significantly) from mean values of 38% (SS group) and 32% (NAA group) to minimum levels of 21 and 23% respectively. During lipolysis inhibition, no significant correlation was found between FFA concentration and aggregate percentage, suggesting that other factors than FFA then had the greater influence upon reversible platelet aggregation.