PLATELET REACTIVITY AND FIBRINOGEN LEVELS IN UNSTABLE CORONARY ARTERY DISEASE (UCAD). E. Swahn (1), H. von Schenck (2) and L. Wallentin (1). Dept. of Cardiology (1) and Dept. of Clinical Chemistry (2), University Hospital S-581 85 Linköping, Sweden.

Unstable angina and non-Q-wave myocardial infarction represent the unstable phase of coronary artery disease (CAD). In UCAD a thrombosis in a coronary artery could be the triggering factor of the unstable phase. Increased platelet reactivity and hypercoagulability could be the predisposing factor for developing this condition. Therefore, in patients with UCAD the platelet reactivity was measured as the aggregation response to ADP and collagen, the platelet sensitivity to PGI2 and the release of platelet derived proteins. As an indicator of hypercoagulability the fibrinogen level in plasma was analysed. The control group consisted of patients with chest pain but without CAD. Blood samples for platelet tests and fibrinogen analysis were obtained in the acute phase and after one year.

Results: The only difference in platelet reactivity between cases and controls was noted in their sensitivity to PGI2. The difference remained in the females but not in the male UCAD group after one year.

Acute phase: UCAD Controls p
Pgi2 1.0 ng/ml Men 43.8±1.0 55.8±2.5 <0.01
Women 38.5±5.5 47.3±2.3

One year:
Pgi2 1.0 ng/ml Men 46.6±2.0 50.5±1.7 ns
Women 35.9±2.8 46.2±3.0

In the acute phase a diagnosis of UCAD, elevated weight index and smoking contributed independently to increased fibrinogen levels.

Conclusion: In patients with unstable CAD long term treatment with ASA 75 mg/day inhibits collagen induced platelet aggregation and hampers the ADP response. I.v. heparin tends to raise platelet reactivity and reduce the inhibitory effect of prostacyclin. Heparin induced platelet activation is reduced by simultaneous ASA therapy.

HEPARIN AND ACETYLSALICYLIC ACID (ASA) 75 MG/DAY IN UNSTABLE CORONARY ARTERY DISEASE - EFFECTS ON PLATELET REACTIVITY. Lars Wallentin, Ingrid Nyman Ulf Berglund, Eva Swahn. Dept. of Cardiology, Linköping, Sweden.

In unstable coronary artery disease (UCAD), i.e. unstable angina pectoris (UAP) or non-Q-myocardial infarction (NMI), treatment with heparin or ASA have given encouraging results. The present study attempts to visualize effects of i.v. heparin and of ASA and to evaluate the utility of ASA 75 mg/day (one year). Patients, admitted because of chest pain, who either develops NMI or signs of ischemia in resting or exercise ECGs are included. Within 72 hours patients are randomized to obtain Heparin+ASA, Heparin+Placebo, Placebo+ASA or Placebo+Placebo. Platelet reactivity is studied in vitro in platelet rich plasma (PRP) in a subgroup of patients. The aggregation response is studied after addition of severe adenine and ADP and after preincubation with prostacyclin before aggregation with ADP. The figures present results from 85 randomized patients tested before, 5 days, one month and one year after start of therapy.

Conclusion: In patients with unstable CAD long term treatment with ASA 75 mg/day inhibits collagen induced platelet aggregation and hampers the ADP response. I.v. heparin tends to raise platelet reactivity and reduce the inhibitory effect of prostacyclin. Heparin induced platelet activation is reduced by simultaneous ASA therapy.