

CORRELATION OF PLATELET WITH CLINICAL STUDIES IN PATIENTS WITH CORONARY ARTERY DISEASE PRE AND POST CORONARY ARTERY SURGERY. D.J. Doyle, C.N. Chesterman, J.F. Cade, F.J. Morgan. Melbourne University Department of Medicine St. Vincent's Hospital, Intensive Care Unit Royal Melbourne Hospital and St. Vincent's School of Medical Research.

80 patients with angina were studied preoperatively with measurements of ^{51}Cr platelet survival (PS), plasma PF4 and βTG (RIA), platelet aggregates and platelet count. Clinical data was recorded prospectively. 58 patients were restudied a mean of 34 weeks postoperatively, 44 of whom entered a double blind randomized trial of sulphinyprazone.

Mean preoperative PF4 and βTG were elevated above normal values (PF4 14 vs 8ng/ml, βTG 54 vs 32ng/ml) and the mean PS shortened (150 vs 174hrs). These tests returned towards normal postoperatively (PF4 10ng/ml, βTG 39ng/ml and PS 186hrs). This trend is partly due to a significant change in βTG , PF4 and PS in patients treated with sulphinyprazone.

A significant negative correlation was detected between platelet count and angina grade ($r = -0.30$, $p < 0.001$), number of previous transmural myocardial infarctions ($r = -0.36$, $p < 0.001$) and angiographic grading of severity of coronary artery disease ($r = -0.28$, $p < 0.05$). No other significant correlation of platelet studies with coronary artery disease (CAD) status was detected. There was however an association between elevated PF4 and βTG and shortened PS in the 4 postoperative patients with peripheral vascular disease (PVD) compared to those without PVD (mean PF4 21.8 vs 7.3ng/ml, βTG 98.1 vs 34.2ng/ml). In addition βTG was negatively correlated with postoperative (not preoperative) triglyceride concentrations ($r = -0.73$, $p < 0.05$) which were also correlated with PS ($r = 0.72$, $p < 0.05$).

Abnormalities in platelet parameters are present in patients with CAD but they do not appear to be sensitive indicators of CAD severity and may well be affected by other factors for example extent of atheroma elsewhere, serum lipids and preoperative stress.

0441

PLATELET SURVIVAL TIME IN MEN WITH POST MYOCARDIAL INFARCTION AND NORMAL CORONARY ARTERIOGRAM. F. Calsals, R. Castillo, L. Alfonso, J.L. Vidal, J.C. Paré and A. Betriu. Hospital Clínico y Provincial. Facultad de Medicina. Barcelona

Some patients fail to show any evidence of arteriographic coronary obstruction following an episode of myocardial infarction. This type of subjects would all present a shortened mean platelet survival.

Over a year's period, platelet survival time (Chromium 51 labelling) was measured in 8 subjects who, one month after suffering an acute transmural infarction, were found to have a normal coronary arteriogram (NC). Survival times were determined between 3 and 12 months after the infarction. At this moment, all the patients were asymptomatic.

As control, and simultaneously, platelet survival time was evaluated at random in 8 patients who had had a myocardial infarction with arteriographic evidence of coronary obstruction (AC), and in 11 normal subjects (NS).

Platelet life span was measured by fitting the gamma function at 11 days of survival experimental points and the goodness of fitting surveyed by a graphic terminal (Tektroniks 4051). Since a failure of fitting with this system was found in 30% of cases, the mean weight method of computing platelet life span had to be employed.

The results of the mean platelet survival time in the three groups of individuals show no significant differences: NS $7,76 \pm 0,89$; NC $7,48 \pm 1,68$; AC $7,64 \pm 0,87$ (days ± 1 SD). Only two patients with normal coronary arteriogram had values lower than the inferior range of normal subjects.

These results do not agree with others.

0440

IMPROVEMENT OF SHORTENED PLATELET SURVIVAL HALF-LIFE FROM THE EARLY TO THE LATE PHASE OF MYOCARDIAL INFARCTION. J.H. Chesebro, V. Fuster, J. Byrne. Mayo Clinic, Rochester, Minnesota, U.S.A.

The incidence of reinfarction, death and systemic thromboembolism is greatest in the first 2 months after acute myocardial infarction and decreases with time after infarction. Reasons for this are unknown, but increased platelet vascular wall interaction may be a contributing factor. Thirteen patients, mean age 53 years, had a platelet survival determination (platelet half-life calculated by least square analysis) within 1 month (mo) after acute, uncomplicated myocardial infarction (10 transmural, 3 subendocardial); a platelet survival study was repeated 3-8 mo (average 4 mo) later. All patients were on oral anticoagulants and during the time of observation none of the patients had any clinical thromboembolic events.

Ten of the 13 patients had a shortened (<92 hours) platelet survival half-life of 75 hours (avg) within 1 mo after myocardial infarction which increased significantly ($P < .01$) to 94 hours (avg) four mo (avg) later. Ten control patients with angiographically proven coronary artery disease, stable angina pectoris, and a shortened platelet survival of 85 hours (avg) had a repeat platelet survival 6 mo later that was not significantly changed (mean variability 5%).

The shortened platelet survival that improves with time after myocardial infarction is compatible with the increased incidence of reinfarction, death and thromboembolism early after myocardial infarction. Whether the increased platelet consumption reflects platelet deposition in the coronary vessels or in the left ventricle (as a mural thrombus) is at present under investigation with imaging of III-Indium-labeled platelets.

0442

CLOT IMPEDENCE AS AN INDICATOR OF PLATELET DYSFUNCTION FOLLOWING CARDIOPULMONARY BYPASS. A. Saleem, D.H. Yawn, S.A. Saleh and E.S. Crawford. Departments of Pathology, Anesthesiology and Surgery, Baylor College of Medicine, Houston, U.S.A.

Post-operative bleeding following cardiopulmonary bypass remains a serious problem. Recent studies have indicated platelet dysfunction may be responsible for altered hemostasis in a significant number of patients. Although evaluation of coagulation factors can usually be done with speed and precision, evaluation of platelet function is time-consuming. We have evaluated a clot impedance device (Sonoelot[®], Sienco Inc., Colorado) to measure platelet function. The device measures and records the clot impedance to a vibrating probe as the blood sample clots and retracts. In our evaluation of healthy subjects, we found the initial slope of the impedance curve and the entire retraction phase are influenced by the number of platelets. Extrapolating this information to the patients undergoing cardiovascular bypass, we found 7 out of 11 patients with post-operative bleeding had poor retraction phase in spite of an adequate platelet count. This suggested platelet dysfunction. All seven patients achieved satisfactory hemostasis after platelet transfusion. This was correlated with a normal clot impedance study. Four patients with normal clot impedance were found to have surgical bleeding. The test is easy to perform and the result is available within fifteen minutes of drawing the blood sample. In our hands, the measurement of clot impedance appears to be a reliable adjunct in the etiological diagnosis of post-operative bleeding.