

ACCELERATED HEMOSTASIS IN CORONARY ARTERY DISEASE. T. Wajima and L.L. Burkett, Wilford Hall USAF Medical Center, San Antonio, TX and VA Hospital, Memphis, Tenn., USA.

Reduced antithrombin III levels and positive paracoagulation tests occur in some cases of coronary artery disease. This could be related to the cause of atherosclerosis or it could be the result of the disease itself. Thirty-one patients who had arteriosclerotic heart disease, well documented coronary artery occlusions (1-2 vessels), and were subjected to coronary artery bypass surgery were studied for active hemostatic mechanisms of coagulation. Plasma fibrinogen (FPA) levels, fibrinogen, paracoagulation tests, and antithrombin III assays were performed. In addition, PT, PTT, TT, ECLT, and EDP were examined. The blood samples were taken 2-3 days before surgery. Ten of 31 had elevated levels of FPA, and 21 had normal FPA. Eleven patients had positive paracoagulation tests. Six of 31 showed decreased antithrombin III. Seven had an increased fibrinogen level (over 500 mg%). Four of ten patients with elevated FPA had positive tests for paracoagulation, decreased antithrombin III and increased fibrinogen. PT, PTT, TT, Platelet counts, FDP, and ECLT were normal in all patients, except three who had shortened euglobulin clot lysis time. Evidence for activated fibrinolysis was not observed except in 3 cases with shortened euglobulin clot lysis time. There was no difference between elevated FPA groups and normal groups in the postoperative period. The degree or extent of coronary artery occlusion was not correlated with the level of FPA or positive paracoagulation tests. Since there were no clinical and laboratory data suggesting disseminated intravascular coagulation, the increased FPA, positive paracoagulation and the reduced level of antithrombin III strongly favor an accelerated hemostasis, probably of localized nature.

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THROMBOLYTIC THERAPY WITH STREPTOKINASE IN ACUTE DEEP VEIN THROMBOSIS. R. Schmützler et al. Klinik Bergisch-Land, Wuppertal, West Germany.

The difficulty is known to determine the real date of the beginning of venous thrombosis. Acute deep thrombosis of the femoral and pelvic veins can be followed by two severe complications: pulmonary embolism and chronic venous insufficiency. Primarily causal treatment has to be aspired: thrombectomy or thrombolysis. Confrontation of two groups: 34 successfully thrombolysed occlusions with streptokinase (SK) contrary to 34 heparin treated occlusions without success. Control 3 years later: clear-cut advantage of thrombolytic therapy. Evaluation of 93 patients in a study over ten years with acute, deep venous thrombosis of the extremities after SK treatment: success rate 68%. Comment on the situation of acute postoperative thrombosis with the possibility of thrombolytic treatment based on our experimental examinations. In summary, it can be stated that thrombolytic treatment cannot be started before the seventh postoperative day without risk of hemorrhage. Side effects respectively complications of SK treatment have to be mentioned like febrile reactions and hemorrhages. Fever appeared in 57% of the patients in a large study, hemorrhaging in 29% of the cases, 22% of which could be considered mild. Pulmonary embolism during thrombolysis occurred in 4-8%, 1-2% lethal. An over-all lethality rate for SK therapy in the mentioned study is between 1,2 to 1,4%.

INCREASE IN PLATELET VOLUME AND AGGREGABILITY IN ADENOSINE DIPHOSPHATE INDUCED PULMONARY MICRO THROMBOEMBOLISM AND EFFECT OF ACETYSALICYLIC ACID TREATMENT. T. Motomiya and H. Yamazaki, Division of Cardiology Research, Tokyo Metropolitan Institute of Medical Science, Tokyo, Japan.

Several reports suggest that functional and morphological changes of platelet may occur in atherosclerotic and thromboembolic diseases and acetylsalicylic acid (ASA) has been considered as one of the possibly beneficial treatments of such conditions. Thirty male rabbits including 7 pretreated with ASA 200 mg/Kg p.o. for 4 days were catheterized. ECG, respiration, arterial and central venous pressures were recorded throughout the experiment. ADP 2 mg/Kg was injected into the central vein and citrated blood was obtained before, 30 seconds, 3, 10 and 45 minutes after the injection for platelet studies. Platelet count and volume were measured using with a Coulter Counter Zbi coupled with a Channelyzer C-1000. Platelet aggregability was determined by a Sienco aggregometer. Within several seconds after completion of ADP injection the animal developed bradycardia, premature beats, ischemic ST-T wave changes, hypotension, apnea and convulsion. Histology of the lung revealed small vessels being obstructed with platelet aggregates. In rabbits received ASA developed less bradycardia and hypotension and no ischemic ST-T wave changes, apnea or convulsion. Platelet count decreased at 30-second and increased at 10-minute after injection. Significant increases in platelet volume and aggregability were noted with no overt change in platelet morphology at 3 and 10 minutes after ADP injection. Correlation between platelet volume and aggregability was significant ($r=0.60$, $P<0.05$). This experiment demonstrated that platelet size or volume is not only dependent on its age but changes transiently under certain circumstances such as thrombosis. Treatment with ASA prevented cardiorespiratory disorders and changes in platelet volume and aggregability induced by administration of ADP.