

EVALUATION OF THE ANTITHROMBOTIC PROPERTIES OF SULOCTIDIL IN PATIENTS WITH SUBSTITUTE HEART VALVES. M. Moriau, A. Ferrant, C. Col-De Beys, A. Hurlet, C. Chaland, R. Ponlot, P. Jaumain, M. Goenen and R. Masure. Haemostasis and Thrombosis Research Unit, University of Louvain, Belgium.

Isotopic platelet survival time (PST), platelet count, adhesiveness, aggregation, PF₄ release and coagulation parameters were examined in 23 patients with prosthetic heart valves (6 mitral, 10 aortic and 7 mitroaortic Starr Edwards or Bjork valves) treated with or without suloctidil.

The patients were distributed in 2 comparative groups: the treated group with 11 patients with VKA (nicoumalone) and suloctidil (600 mg/day) and the control group with 12 patients with VKA alone. The biological parameters were performed before (the 10th postop. day) and 6 weeks after treatment with or without suloctidil.

Before treatment were: PST shortened, platelet adhesiveness and aggregation normal and PF₄ release, factors I, VIII-C, VIII-R-Ag increased in both groups.

After treatment, PST returned to normal in the treated group, but remained unchanged or was more decreased in the control group. Platelet adhesiveness and aggregation were unchanged in both groups. PF₄ release was reduced in the treated group and unchanged in the control group. Platelet count, factors I, VIII-C, VIII-R-Ag returned to the preoperative values in both groups.

Two severe thromboembolic complications appeared in the control group, none in the treated group.

INHIBITION OF THE PLATELET FUNCTION BY THE EFFECT OF PLASMIN ON HUMAN FACTOR VIII. R. Castillo, S. Maragall, E. Martín, E. Argelagués and A. Ordinas. Facultad de Medicina. Hospital Clínico y Provincial. Barcelona. Spain.

Plasmin-treated human purified factor VIII prepared from normal and afibrinogenemia plasmas inhibits the ADP-induced aggregation and the collagen-induced 14C serotonin release of normal and von Willebrand human platelet rich plasmas (PRP). These results reconfirm the fact that the inhibition of the human platelet function by the effect of plasmin on the plasmatic protein is mainly linked to the digested factor VIII but not to the fibrinogen, fact that we had previously proved when ADP-induced aggregation of normal washed platelets was inhibited by plasmin-treated normal human serum, but not by plasmin-treated von Willebrand plasma. The factor VIII breakdown products were revealed by crossed immunoelectrophoresis, sepharose 4B chromatography and SDS polyacrylamide electrophoresis.

The defective ADP-induced aggregation that we have observed in PRP from patients between the 8th and the 24th hour of thrombolytic treatment, is only due to the proteolytic effect of plasmin on the plasmatic factor VIII and not to a direct action of the plasmin on the platelet membrane, since washed platelets from these patients aggregate normally by ADP with normal platelet poor plasma (PPP), and the collagen-induced 14C serotonin release is also correct.

THE HEPARIN THROMBIN CLOTTING TIME - A MEASURE OF PLATELET ACTIVATION? J.R. O'Brien, M.D. Etherington, Sandra Jamieson and R. Shuttleworth. Portsmouth & South East Hampshire District Pathology Service, Portsmouth, Hampshire, England.

The heparin thrombin clotting time (HTCT) of platelet poor plasma (PPP) measures non-specifically the total heparin neutralizing activity (HNA) of PPP. This HNA must include any platelet factor 4 (PF₄) liberated in vivo from "activated" platelets. It is suggested that released PF₄ is the main determinant of clinical abnormalities of the HTCT for the following reasons. (1) There is an inverse relationship between the intra-platelet PF₄ determined by a clotting method and the HTCT (Thromb. Haemostas., 36, 649, 1976). (2) There is minimal activity in plasma from aplasia and I.T.P. (3) There is a mild excess of activity in groups of patients at risk of thrombosis and (4) most activity during thrombosis, operations, acute myocardial infarction and infections, in all of which conditions platelets presumably are involved and "activated". There is a significant inverse correlation between the HTCT and (5) the plasma "low activity PF₄" of Niewiarowski and (6) the β thromboglobulin (radioimmunoassay).

In most healthy people this test is remarkably constant, suggesting a steady state. But there are marked inter-person differences which could indicate different degrees of platelet activation and different thrombotic risks. This test can be routinely carried out in 2 minutes in a hospital laboratory with consistent results over the years. It is already of great clinical use and importance. Since this test can now be normalized therapeutically the question of patient benefit can be raised.