MIXED POSTERS VI

Thrombosis

THROMBIN TIME DILUTION TEST: A SIMPLE METHOD TO CONTROL HEPARIN THERAPY. J. Pizzuto, M.P. Reyna, S. García-Méndez and M.R. Morales, Servicio Hematología, Hospital General, Centro Médico Nacional, Mexico City, Mexico.

A modification of thrombin time (TT) is described, allowing quantitative measurement of plasma heparin activity during therapy. When heparin-containing plasma shows prolonged TT, TT is corrected by diluting with platelets rich plasma (PRP) thus finding the adequate heparin activity; the latter is estimated when prolonged TT of test plasma is corrected by PRP in the proportion between 1:4 and 1:8 ($\pm 20\%$). Results of 30 PRP samples studied *in vitro* by adding different amounts of heparin showed that samples diluted 1:4 and 1:8 did not correct prolonged TT ($P < 0.05$) when they contained respectively, more than 0.2 or 0.3 U of heparin/ml. Therefore within these levels of heparin one should place the adequate dosage of heparin when using this method. Patients treated with 100 U of heparin/Kg every 6 h were thus studied: 52 without previous coagulation defects and 22 with disseminated intravascular coagulation (DIC). The results in the first group showed good heparin effect in 29 cases, overdosage in 12 and underdosage in 11. Hemorrhage occurred in 5 of the overdosage group. In the cases of DIC, 4 with underdosage of heparin did not improve; in 11 out of 18 with adequate levels of heparin, both hemorrhage and coagulopathy disappeared and the other 7 who had more severe complications did not improve. To sum up, the diluted TT is a very useful test to assess heparin therapy even in patients with previous coagulation abnormalities.

FIBRINOLYTIC SYSTEM-HEPARIN INTERACTION. G. A.-Villaverde, L. Badimón, E. Bastida and F. G.-Valdecasas, Dept. Farmacología, Consejo Superior Investigaciones Científicas, Barcelona, Spain.

It was studied *in vitro* the effect of Heparin(Hp) directly on Urokinase(UK) induced fibrinolysis, Hp on rat euglobulins(Eu)-UK activated, and Hp on whole rat blood UK-activated (through the fractions: whole and diluted blood, whole and diluted plasma, Eu and inhibitors). For testing the influence of Hp with UK we used the polyvalent method on fibrin plate that we presented in the X International Congress of Angiology, Tokyo, Sept. 1976. We have assayed UK concentrations from 0.25 to 100 Pl.U./ml. and Hp concentrations from 0.001 to 1000 I.U./ml. It was not found significant modifications in the Hp effects on UK. In the system Eu-UK-Hp we have obtained decrease in fibrinolytic response of Eu-UK activated with every Hp concentration. Studying the above mentioned hematic fractions, the results were different related to the Hp concentration, thus the low dose produced increase of fibrinolytic activity, while higher dose produced clear inhibition in each fraction. Assaying isolated inhibitors UK-mixed, in order to quantify the fibrinolytic response in Hp presence, we found a progressive increase of the inhibitory potential accordingly with the increase of Hp concentration. In rat, *in vivo*, we obtained significant inhibition in every tested blood fraction with Hp higher dose.