Poster Board P6-108

0358 MANAGEMENT OF HEMOSTASE IN ACUTE LIVER FAILURE

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In coma hepaticum perfusion with baboon liver in by-pass is a last remaining possibility.In order to maintain hemostase in the extra corporal circuit heparin was found to be necessary. This is of interest since clotting tests are prolonged and clotting factors like X,XII and AT III are often based to zero values. AT III* was substituted with 250-500 AT III units every 3 hours resulting in 40-80 % of normal range and heparin was given in a dosage of 500-750 USPE hep./h resulting in 0.1-0.2 USPE hep./ml plasma. The therapy was monitored 4-8 times per day by chromogenic substrates.Further analysis of factor X, AT III heparin and hematocrit was performed in the arterial and venous part of the liver in by-pass. After blood passage there was a significant reduction of heparin; factor X,AT III and hematocrit showed constant values. With the controlled AT III substitution and minimal heparinization thrombus formation in the liver in by-pass was never observed, hemostase was maintained, but the fate of the patients depend on liver regeneration.

*kindly provided by Behringwerke and Kabi.

P6-109 0359 LIVER DEVASCULARISATION IN PIG:ALTERATIONS OF COAGULATION&FIBRINOLYSIS

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All arteries leading to the liver were ligated and an end-to-side portaval shunt was performed in twelve pigs.Before and 1,3,6 and 9 hrs after laparatomy a blood sample was drawn by femoral artherial catheter. APTT,Prothrombin,Factor VIII AHF(one-stage method),platelet and red cells counts, fast-acting Antiplasmin by S-2251,AT III by S-2238 were performed on each sample.Prothrombin slowly but progressively decreased while APTT reduced. VIII AHF showed a little increase at first but the higher level,about the 160% of initial value,was recorded at the 9th hr.AT III increased during the 1st hr but,as Antiplasmin,slowly decreased to the 70% in the following hours.The AT III and the first VIII AHF increase occurred during portal stasina clumping,simultaneously to a very deep fall of blood pressure.The stasis of portal system and an adrenergic stress may explain this finding.The survival of the pigs was quite costant,more than 24 hrs: this model of hepatic failure has been chosen for application of articial devices to support liver function.

P6-110 0360 PROCOAGULANT PROPERTIES OF ASCITIC FLUID IN HEPATIC CIRRHOSIS

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Re-infusion of ascitic fluid into the vasculature of patients with liver cirrhosis corrects many of the protein, fluid, and electrolyte abnormalities. Such infusion can lead to D.I.C. Three such cases are reported here. One had intermittent drainage and re-infusion, the other two had Le Veen shunts installed. All three patients showed laboratory evidence of D.I.C. Ascitic fluid on one patient showed procoagulant material by the thromboplastin activation test. In the presence of ascites from another patient, plasmas deficient in Factors XI, IX, and VIII gave recalcification and non-activated partial thromboplastin times similar to those of normal plasma. RCT's and PTT's of normal heparinized plasma and of plasmas deficient in Factors X and VII-X were shortened to a lesser extent. Clotting times of Factor V deficient plasma were prolonged by ascites. These results indicate that an activator of Factor X is present in the ascitic fluid. Additional tests such as RVV and prothrombin times suggest that some activated Factor X and/or tissue activator (endotoxin and leukocytes) may have been formed in vivo.

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