FREE COMMUNICATIONS X

Plasma Inhibitors.

CHANGES IN LEVELS OF PLASMA PRE-KALLIKREIN, KALLIKREIN AND KALLIKREIN INHIBITORS DURING ENDOTOXIN SHOCK IN DOGS. M.J. Gallimore, A.O. Aasen and E. Amundsen. Institute for Surgical Research, Rikshospitalet, Oslo, Norway.

Plasma protease activity is known to be increased during endotoxin shock and recent studies have indicated that the plasma kallikrein-kinin system becomes activated by circulating endotoxin. Plasma levels of pre-kallikrein kallikrein and kallikrein inhibitors were therefore determined in samples from dogs infused with E.coli endotoxin, using assays with a chromogenic substrate for plasma kallikrein (Chromozyme -PK, Pentapharm, Basle, Switzerland). "Fast-reacting" and "time-dependent" inhibitors of kallikrein were studied using purified human plasma kallikrein. Considerably reduced levels of plasma pre-kallikrein and increased levels of kallikrein were detected in the late phase of shock and significant reductions in "fast-reacting" and "time-dependent" inhibition of kallikrein was observed. These results show that during endotoxin shock plasma pre-kallikrein becomes activated to kallikrein and indicate that kallikrein inhibitors play an important mediatory role in the pathophysiology of endotoxin shock.

A NEW PHYSIOLOGICAL INHIBITOR OF BLOOD COAGULATION IN THE NEWBORN. H.C.Hemker, A.D.Muller and J.H.v.Door, Department of Biochemistry, State University Limburg, Maastricht, The Netherlands.

In more than half of a series of samples of cord blood a hitherto unrecognized inhibitor of blood coagulation could be demonstrated. At three days of age this inhibitor is found in all babies. In 27 out of 43 samples the disappearance rate of thrombin in the two-stage prothrombin assay was significant (> 5 times) greater than it uses to be in normal human adult plasma. In the 16 others it was no more than 1.8 times greater than normal. On basis of this observation we divided the plasma in an I (inactivation) and non-I group. We determined thrombin inactivating proteins in I and in non-I plasmas respectively and found no significant differences in antithrombin 3, α_1 -antitrypsin, α_2 -macroglobulin, antipain, C₁ esterase inhibitor or inter- α -antitrypsin inhibitor. A non-protein fraction can be prepared from blood containing this inhibitor that has no appreciable inhibitory action of its own but that markedly enhances the activity of antithrombin 3. We found as yet no reasons to assume that the inhibitor is something else than heparin.