EFFECTS OF A SPLENIC EXTRACT ON COAGULATION. C. R. SPILLERT, L. P. PARMER, O. M. HASTINGS AND E. J. LAZARO, CMDNJ, NEW JERSEY MEDICAL SCHOOL, NEWARK, N. J., U. S. A.

We have previously described the ability of a lipoidal splenic extract (LSE) to decrease the coagulability of blood in laboratory animals. (Spillert, C. R. et al.: Hematological effects of a splenic extract. In Micro-circulation. Vol 1, Grayson, J and Zingg, W. (eds) New York and London, Plenum Press 1976, p207). We report here the effects of LSE on platelet aggregation (PA) in blood of myocardial infarction prone and advanced malignant disease patients, (2) thromboelastographic values on the blood of patients with a variety of clinical states, and (3) survival of elderly mice with E. Coli endotoxemia (ECE). LSE was added to platelet rich plasma prior to determination of PA, and to citrated whole blood before recalcification in the thromboelastographic studies. One-year-old Swiss mice received LSE (10 mg/kg), or saline (controls), 2 hours prior to ECE (5 mg/kg) challenge. The decrease in PA (LSE versus controls) is indicated by the 16.0% ($p < 0.001$) and 20.2% ($p < 0.05$) inhibition of ADP induced PA in the blood of myocardial infarction prone ($n = 27$) and advanced malignant disease ($n = 6$) patients respectively. Thromboelastographic parameters R_t , $R_t + K$, and T_{50mm} increased 17.5%, 17.7% and 21.2% respectively. ($p < 0.005$). Forty-eight hour survivals in mice with ECE were 44.1% (saline, $n = 34$) and 79.5% (LSE, $n = 39$) ($p < 0.01$). These results demonstrate the inhibitory effects of LSE on certain parameters of the coagulation process. It appears likely that the protective property of LSE in ECE is related to these anti-coagulation effects.

PLATELET-LIKE BODIES IN URINE IN SOME BLOOD DISORDERS. N. Kōno, T. P. Chen, N. Suzawa and T. Mizokami, Tokyo Electric Power Hospital, Tokyo, Japan, Keio University, School of Medicine, Tokyo, Japan, Meguroekimae Clinic, Tokyo, Japan.

We had reported that it is always observed a small body resembling a platelet in urine of normal and various sick persons and the small body is a platelet itself or a ruin of platelet (Lancet, 1, 1339, 1975., N. Engl. J. Med., 293, 44, 1975., Nihon Univ. J. Med., 17, 117, 1975., Japan. J. Clin. Hemat., 17, 632, 1976., Jap. J. Urol., 67, 903, 1976.). We had studied the Platelet-like bodies in urine of 3 cases suffered from ITP, 3 cases suffered from AML, 2 cases suffered from GML, and one case suffered from MCL, SLE and reticulosarcoma, respectively. The small bodies were also observed in all urine of these patients. By transmission electron microscopy, these small bodies were a platelet itself or a ruin of platelet. And the TEM findings showed difference in each blood disorder and treatment.

EFFECT OF THROMBIN ON FACTOR VIII/VON WILLEBRAND FACTOR (FVIII/vWF). Mary Ellen Switzer and P. A. McKee, Duke University Medical Center, Durham NC, U.S.A.

The sodium dodecyl sulfate (SDS) gels and immunological properties of FVIII/vWF and thrombin-activated (thr-act) FVIII/vWF are identical. Hence thrombin must either modify FVIII/vWF by very minor proteolysis or cleave only a few FVIII/vWF molecules. The procoagulant (PC) activity of FVIII/vWF and thr-act FVIII/vWF eluted sharply in the void volume (V_0) from 4% agarose in 0.15 M NaCl, with >65% loss of the PC activity of the thr-act FVIII/vWF by 3 hrs. The PC activity of FVIII/vWF and thr-act FVIII/vWF was stabilized by 0.25 M CaCl₂. When FVIII/vWF and thr-act FVIII/vWF were filtered on 4% agarose in 0.25 M CaCl₂, the protein eluted in the V_0 , but most of the PC activity eluted later in a region of little detectable protein; however, the delayed PC peak from thr-act FVIII/vWF was greatly enhanced above control levels. Nonreduced, neither activity peak protein entered a 7.5% SDS gel. After reduction, the gel pattern for the PC activity peak protein from FVIII/vWF showed major bands of 195x, 79x, 61x, 51x and 18x(10³) molecular weight (MW) and several minor bands >100,000 MW. The reduced PC protein from thr-act FVIII/vWF lacked all bands >100,000 MW, but the four lower MW bands were present and well-resolved. Thrombin activation did not affect the vWF activity which was proportional to protein concentration throughout any chromatogram; the 195,000-dalton subunit was not necessary for vWF activity. FVIII/vWF, which had the peak PC activity in the V_0 and no other PC activity peak on 4% agarose-0.25 M CaCl₂, was thr-act before filtration under the same conditions. Then, the greatly enhanced PC activity eluted well after the V_0 , with <10% PC activity in the V_0 . We conclude that modification of a few FVIII/vWF molecules by thrombin and their stabilization by 0.25 M CaCl₂ causes the PC peak that elutes aberrantly from 4% agarose-0.25 M CaCl₂.