

IMMUNE THROMBOSES OF THE PULMONARY VESSELS. S.V.ANDREEV, A.A.KUBATIEV. Academy of Medical Sciences, Moscow, USSR.

According to current concepts, pathogenesis of intravascular thrombus formation is underlain by three crucial factors: lesion of the vascular wall, impairment of hemodynamics and hemostatic properties of the blood. While admitting the important role of each of these factors, one should acknowledge that the true nature of thrombosis is much more complicated and does not always fit the framework of this triad. In our experiments on rabbits, it was demonstrated that the thrombotic process in the basin of the pulmonary vessels could be successfully reproduced even in intact animals under conditions of disturbed immune homeostasis. A distinctive feature of immune thrombosis of the pulmonary vessels was a generalized lesion of the microcirculatory bed, gradual increase in the thrombotic masses and involvement of the major branches of the pulmonary vessels (PV). Morphologically, a picture of lymphoid-cellular infiltration and localization of the antigenic complex in the affected PV was revealed. As a result of progressive decrease in the pulmonary arterial blood circulation and increasing resistance in the system of the lesser circulation there were noted, already during the first hours after the onset of the capillary thrombosis, a compensatory hyperfunction of the right ventricle of the heart which was manifested in its highly increased contractility, higher levels of cyclic AMP and phosphorylation potential. At a later period, however, the compensatory possibilities of the right ventricle of the heart failed to overcome this resistance with resulting incompetence of the organ starting to develop within 2 - 3 days.

CLINICAL USE OF PLATELET ADHESION. R.L. Bick, L.F. Fekete, and W.L. Wilson. Bay Area Hematology Santa Monica, California, U.S.A.

Platelet adhesion as a measurement of platelet function was performed in 50 normal and 100 abnormal patients. A Bowie-type glass bead column and a constant perfusion technique was used. Template bleeding times (TBT) and platelet aggregation was also obtained in abnormal patients. The abnormal population consisted of patients referred with a disorder of hemostasis thought to be due to platelet dysfunction. Results on the normal population revealed "normal" adhesion to be defined as 8% to 100% retention, with an average of  $94.8 \pm 2.7\%$  and a standard error of 0.41 (entire range for normals = 88% - 98%). In all 100 abnormal patients sent for evaluation, platelet adhesion correlated very well with aggregation, and more importantly, with clinical bleeding. In several cases abnormal adhesion and normal aggregation was seen; these 6 patients were classical von Willebrand's. In 4 patients normal adhesion and abnormal aggregation was noted; this was due to aspirin use. Four patients had borderline adhesion and aggregation, and in all others the pattern was either normal adhesion and aggregation or abnormal adhesion and aggregation. In the vast majority of abnormal patients there was absolute correlation between adhesion and the TBT; in those 6 patients where correlation was lacking, the TBT was normal and adhesion was abnormal. These 6 patients had a malignant paraprotein disorder with paraprotein-induced platelet dysfunction. The results of this study suggest platelet adhesion, when done in a standardized fashion, to be a highly reproducible modality to assess platelet function. Using this methodology the normal range is narrow and excellent correlation is noted between abnormal adhesion, abnormal aggregation and clinical bleedability.

THERAPY WITH FACTOR IX CONCENTRATE RESULTING IN D.I.C. AND THROMBOEMBOLIC PHENOMENA. E.W. Campbell, Jr., Sandra Neff, A.J. Bowdler. College of Human Medicine, Michigan State University, East Lansing, Michigan.

The development of subclinical D.I.C. in a factor IX hemophiliac receiving concentrate replacement therapy during surgery is discussed with respect to pertinent laboratory features. The case is unique in that factor IX levels were followed carefully and corresponded inversely to the intensity of the D.I.C. Of additional significance is the failure of heparin failure given in conjunction with factor IX concentrate to prevent D.I.C. The value of adequate laboratory monitoring during factor IX replacement therapy is apparent.