ACQUIRED INHIBITORS OF FACTOR V. Donald I. Feinstein, USC School of Medicine, Los Angeles, California, U.S.A.

Twelve patients with an acquired inhibitor of Factor V have been reported thus far in the literature. Of these, only one occurred in a patient with hereditary Factor V deficiency. Six patients received streptomycin in close temporal relationship to the appearance of the inhibitor. Six patients had been previously transfused, including four of those who received streptomycin. In nine of the eleven spontaneously occurring inhibitors, major surgery preceded the appearance of the inhibitor. The degree of clinical bleeding in these patients varied. One patient had no bleeding, six patients had mild to moderate bleeding, and four patients had severe bleeding. The inhibitor disappeared in less than eight weeks in seven patients, whereas in one patient it persisted for more than two years. Most of these inhibitors have the physicochemical properties of antibodies. Six of the spontaneous inhibitors appeared to be IgG, whereas in two patients inhibitor activity was found in both IgM and IgG fractions. Three inhibitors have been typed with light chain antisera and all contained both kappa and lambda chains. Plasmas from seven patients with hereditary Factor V deficiency have been tested with three of these inhibitors for inactive factor V antigenic material and none has been detected. In addition, plasma from a patient with hereditary factor V deficiency has been tested with heterologous factor V antibody and no antigenic material has been found. Thus hereditary factor V deficiency probably represents a deficiency of factor V molecules, rather than the synthesis of a defective molecule.

THE "LUPUS" ANTICOAGULANT: MANIFESTATIONS AND POSSIBLE MECHANISMS OF ACTION. C. Hougie, Department of Pathology, University of California, San Diego, La Jolla, California, U.S.A.

With the more general use of the partial thromboplastin time for pre-operative screening, an increasing number of patients with an inhibitor of the type associated with lupus erythematosus are being detected. This is now the most frequent type of inhibitor seen by us. Although the inhibitor is referred to as the "lupus" anticoagulant, in at least one-third of cases this disease is not present. The patients have a prolonged partial thromboplastin time (PTT), while their plasma prolongs the PTT of normal plasma, and this lengthening is not accentuated by prior incubation of the mixture. Thromboplastin generation is normal except when the patient's plasma is used as substrate. It has been suggested that the inhibitor interferes with the reaction of prothrombinase with prothrombin, more specifically by an action on the phospholipid moiety. In a small proportion of cases, the prothrombin time is prolonged due to a true deficiency of prothrombin. Those factors involved in the intrinsic system are usually decreased when measured by conventional one-stage techniques. As the level of the factor is often higher when tested at a 1:10 or 1:20 dilution rather than the conventional 1:5 dilution, the decrease is generally attributed to non-specific inhibition rather than a true deficiency. New evidence will be presented indicating that in some cases a true deficiency of one or more of these factors may exist, probably due to specific inhibition. The lupus anticoagulants studied by us differed in certain respects from each other in patient to patient. Their mode of action and clinical significance will be discussed.