ReACT TEST: RECALCIFICATION OF ACTIVATED WHOLE BLOOD USING THE SONOCLOT. G.E. Ens*, R.D. Hamstra**. Colorado Coagulation Consultants*, University of Colorado Medical Center**, Denver, Colorado, U.S.A.

A rapid, simple, reliable laboratory method for detecting thrombotic tendency, estimating heparin dosage, and monitoring heparin effect is available using a new thrombokinet instrument (Sonoclot) and recalcified activated clotting (ReACT). The same method can be used to follow bleeding disorders such as hemophilia. 0.2 ml of carefully mixed, citrated whole blood (1 part citrate plus 9 parts blood) was measured into a disposable unsiliconized glass cuvette containing a disposable magnetic stir bar. 0.1 ml of ReACT mixture (siliceous earth plus 0.025 M CaCl_2) was added to the cuvette, mixed for 5 seconds, and allowed to clot while monitoring with the Sonoclot. The ReACT procedure was modified for hemophilic studies, platelet poor plasma was clotted with siliceous earth plus 0.05 M CaCl_2 . A continuous line chart was analyzed for Clot Onset (C_0), an arbitrary time end point, ReACT Time (RT), and Clot Formation Rate (CFR). Studies from normal adults (50) show a C_0 of 100-150 sec., and RT of 140-190 sec., and CFR of 10-20 chart units/minute. Patients with clinically evident clotting problems exhibit a slightly shorter C_0 , shortened RT and increased CFR. Heparin added in vitro at doses of 0.05 u/ml to 0.5 u/ml lengthens the C_0 and RT while the CFR changes little. ReACT results from heparinized patients are similar. Estimation of needs based on in vitro data have been made with smooth heparization. Platelet poor plasma from untreated classical hemophiliacs shows a markedly prolonged C_0 and RT with a low CFR. Serial dilution with normal plasma causes normalization of the Sonoclot curve between 0% and 10% factor VIII levels.

THE SONOCLOT: A NEW THROMBOKINETIC COAGULATION INSTRUMENT. R.D. Hamstra*, G.E. Ens**, S. Simons***. University of Colorado Medical Center*, Colorado Coagulation Consultants**, Sienco, Inc.***, Denver, Colorado, U.S.A.

A new coagulation instrument (Sonoclot) has been evaluated in the laboratory, at the bedside, and in surgery. It was compared to the prothrombin time, activated partial thromboplastin time, activated clotting time, Lee-White clotting time, fibrinogen and thrombelastograph. Most coagulation tests are reported as time to a clot end point while the Sonoclot records a continuous thrombokinet pattern by measuring the energy required to maintain axial vibration of a hollow plastic probe in 0.4 ml of whole blood while it clots. Disposable plastic probes and non-siliconized glass cuvettes are rapidly changed after testing. Results are a line pattern which has been analysed for Clot Onset (C_0) and Clot Formation Rate (CFR). Native whole blood from normal adults (54 studied) has a C_0 of 2-4.5 minutes and CFR of 4-8 chart units/minute. The only similar clotting instrument is the thrombelastograph (TEG). Sonoclot and TEG patterns correlate, the Sonoclot sensing change before the TEG. Whole blood clotting measured with the Sonoclot is a new method which distinguishes hypercoagulation, various levels of heparin, and severe bleeding problems from normal. The device is durable, stable, easy to operate, and portable, providing a permanent record in a few minutes.

EFFECT OF MINI-DOSE OF CHLORPROMAZINE ON THE BLEEDING TIME AND PLATELET AGGREGATION. J. Zahavi and G. Schwartz. University of Tel-Aviv School of Medicine, Tel-Aviv, Israel.

The impairment of hemostasis by chlorpromazine (C) at concentrations below those which inhibit platelet release reaction, has been evaluated in 24 normal subjects. In each individual, modified Ivy bleeding time and platelet aggregation (PAG) induced by adenosine diphosphate (ADP) 0.6 μM and 4 μM , collagen 0.003% and 1-epinephrine 3 μM as well as platelet serotonin concentration, were performed prior to and 20 min. after intramuscular administration of (C) or saline. Normal saline was injected to 6 subjects and 5, 12.5 and 20 mg. of (C) to another 8, 5 and 5 subjects respectively. There was a slight but significant prolongation of the bleeding time and a decrease in the initial wave of aggregation to ADP 0.6 μM in the subjects on (C) compared to those on saline. Platelet serotonin, however, and (PAG) to collagen, 1-epinephrine and ADP 4 μM , were the same in both groups. The impaired hemostasis by mini-dose of (C) might be attributed to the prevention of leakage of ADP from intact erythrocytes in-vivo and, if so, may signify an important new application of this drug in the treatment of thrombotic disorders.