APPARENT VISCOSITY OF ARTIFICIAL RED-WHITE AND WHITE THROMBI DURING ANAESTHESIA AND SURGERY. L. Dintenfass and B. Aronson. Medical Research, Kanematsu Memorial Institute, Sydney Hospital, Sydney 2000, and Department of Medicine, University of Sydney, Sydney 2006, Australia; and Department of Anaesthesiology, Hadassah University Medical School and Hospital, Mount Scopus, Jerusalem.

This is a pilot study intended to explore some aspects of thrombus formation which might be of value in understanding the dynamics of anaesthesia, on the one hand, and the effects of anaesthesia and surgery on tissue perfusion, on the other. Patients studied included six subjects undergoing operations for retinal detachement (5), upper abdominal surgery (1), and a major orthopaedic procedure (1). Artificial thrombi, of morphology of red/ /white and white arterial thrombi, were formed in vitro by means of VFTV, variable frequency thrombo viscometer, at temperature 37C, on freshly shed blood, at mean shear rates of 26.8 and 80 sec^{-1}. Blood samples were drawn immediately prior to commencement of anaesthesia, and then at half-hourly intervals. Anaesthesia was induced with thiopentone sodium, and halothane or meperidine and droperidol. In general, the apparent viscosity of artificial thrombi increased during surgery.

CLOTTING STUDIES PERFORMED ON BLOOD STORED IN HALF-STRENGTH ACD-A. S. Cederholm-Williams, J.M. Mishler and J.H. Darley. Department of Haematology, Radcliffe Infirmary, Oxford, England.

Six hundred ml of whole blood from each of five healthy male donors was equally divided and stored at 4-6°C in either standard ACD-A (2.2g trisodium citrate, 0.8g citric acid, 2.45g dextrose/dl anticoagulant solution, pH 4.9) or half-strength ACD-A (1.1g trisodium citrate, 0.8g citric acid, 2.45g dextrose, pH 4.3) to determine if low citrate concentrations adversely effected the following: prothrombin time (PT), thrombin time (TT), Kaolin-cephalin clotting time (KCCT), ethanol gel (EG) and fibrinogen levels. Low citrate concentrations had no significant effect (Student's t test for paired scores) on any clotting indice tested (see table below).

ACD-A Strength	PT(sec)	TT(sec)	KCCT(sec)	EG	Fibrinogen(mg/dl)
Standard					
control	19.0	15.2	42.8	negative	270.4
7d	23.3	9.6	53.8	negative	224.6
14d	19.2	10.8	52.4	negative	214.4
21d	21.4	11.8	50.6	negative	204.8
Half					
control	19.0	15.2	42.8	negative	270.4
7d	24.6	8.8	52.6	negative	245.0
14d	18.0	9.6	49.2	negative	217.4
21d	20.2	13.0	52.4	negative	202.4

ABNORMAL PLATELET ULTRASTRUCTURE IN FULMINANT HEPATIC FAILURE. Bullock, G.*, Weston, M.J.+, Rubin, M.H.+, Roberts, J.*, Langley, P.G.+, White, Y.+ and Williams, R.+ *CIBA Laboratories, Horsham, England and +Liver Unit, King's College Hospital, London, England.

Platelets obtained from eight patients with varying degrees of liver damage have been studied with respect to their ultrastructure. These platelets were isolated from platelet-rich plasma which had been utilised in the pharmacological studies described by Dr. Weston (previous abstract) and were compared with control platelets isolated from five normal subjects. The latter were chosen for normal bleeding times and response of their platelets to aggregation with ADP and collagen.

Marked differences were seen between control platelets and those from the test group in that there was an alteration in the microtubular content and disposition. In addition, these changes were partially reversed during the recovery period suggesting that production of new normal platelets was taking place. This is one of the few conditions where platelet structure has been correlated with a clinical disorder.