*N. van Zandwijk, Th. F. J. Lenssen, Elisabeth M. Prakke, J. van der Meer, A. S. Groen and C. A. Wagenvoort* (Central Laboratory of the Netherlands Red Cross Blood Transfusion Service, Amsterdam, The Netherlands): **Leucocytes and Platelets in Isolated Lung Perfusion.** **(246)**

Removal of leucocytes and platelets from homologous blood prevents early functional and morphologic deterioration of an isolated rabbit lung preparation. Lungs perfused with whole blood have a marked ability for granulocyte sequestration. Control experiments in which the perfusion system was not connected with the lung revealed an increase of plasma serotonin and histamine levels due to platelet injury and/or aggregation generated by the perfusion circuit. When an isolated lung was connected with the circuit, plasma serotonin was largely metabolized. However, the metabolic uptake of serotonin by lungs perfused with whole blood decreased after 30 min while pulmonary vascular resistence (PVR) increased. In contrast, perfusion experiments with blood from which almost all leucocytes had been removed, were characterized by a less pronounced rise of plasma serotonin level and no or little change in PVR.

It is concluded that leucocytes particularly granulocytes most likely traumatized by the extracorporeal procedure, have a detrimental effect on the integrity of perfused isolated lungs. Damage of endothelium, which has been recognized as a site of serotonin metabolism, could then be an early symptom. The results of our experiments in which the perfused isolated lung preparation has been used as a model for pulmonary dysfunction after extracorporeal circulation and massive transfusion warrants more attention to be paid to the role granulocytes and platelets play in the genesis of respiratory distress after such procedures.

*H. J. Krzywanek and K. Breddin* (Center of Internal Medicine, Department of Angiology, J. W. Goethe University, D-6 Frankfurt a.M.): **The Photometric Aggregation Test (PAT III). A Screening Test for Enhanced Platelet Aggregation.** **(247)**

PAT III has been developed for the photometric registration of "spontaneous" platelet aggregation. Citrated PRP is rotated at 20 rpm and 37° C using a disc shaped polystyrol cuvette. Aggregation inducing substances are not used. Changes in optical density which occur when platelets aggregate, are registered on a chart recorder.

The following parameters are of special interest: $T_r$ = time from start of rotation to onset of aggregation; angle alpha$_2$ as a measure of max. aggregation speed; and $\alpha_2/T_r$. These parameters allow a more precise differentiation of the spontaneous platelet aggregation.

Our previous findings with PAT I were confirmed by our recent studies: In 124 normal subjects we found a gradual decrease of missing spontaneous aggregation ($\alpha$ 0–40°) with rising age, whereas the number of persons with enhanced aggregation ($\alpha$ 41–90°) reached 70–80% beyond the age of 50. The percentage of 183 patients with diabetes mellitus and enhanced platelet aggregation was greater than that of age matched controls.

*Ulla Sivertsen* (Institute for Experimental Research in Surgery, University of Copenhagen, Denmark): **Platelet Aggregation in whole Blood.** **(248)**

A semi-micromethod for testing the aggregability of platelets in whole blood is described. Aggregation is induced with ADP at 37° C in 0,1 ml samples of citrated blood. At 30 seconds intervals aggregation is stopped and the number of nonaggregated platelets are determined.

The main results as found in "normal" human donors (having received no drugs) are:

1) Platelet aggregation in whole blood is less dependent on the pH, the platelet number, the time elapse from blood sampling to aggregation test as compared with platelet aggregation in plasma (a.m. Born).

2) Disaggregation is only observed to a minor degree (after 180 seconds the maximal disaggregation amounts to 30% of the aggregated platelets).