HEMOSTATIC DEFECTS IN ACUTE LEUKEMIAS: SPECIAL INVESTIGATIONS INCLUDING AGAROSE-GEL-FILTRATION OF PLASMA SAMPLES. P. Ashbeck, U. Schmitz-Huebner, B. Kirchhof, E. Zimmermann, J. van de Loo. Department of Medicine and Institute of Physiology, University of Muenster, Muenster, W.-Germany.

22 patients with acute leukemias were investigated before and during cytostatic polychemotherapy. The mean decrease of peripheral leukocytes was 38 000/mm³ within the first 3 days of the treatment. The bleeding tendency could be attributed to thrombocytopenia and one or more of the following mechanisms:

1) Only in febrile patients the development of a DIC could be shown: increase of fibrinogen and soluble fibrin; decrease of prothrombin (S.C. - activation, chrom. substrate), F. V, F. VIII, and antithrombin III (chrom. substrate, immunological). In these patients, no consumption of antithrombin was demonstrable.

2) Non-febrile patients did not develop a DIC during the cytostatic treatment.

3) In some patients an isolated fibrinolysis could be shown already before treatment: increase of soluble fibrin and FDP; marked decrease of fibrinogen, antithrombin, and F. XIII, which were correlated with the amount of cytoreduction. In these patients, there was no consumption of AT III.

4) Asparaginase produced the well-known defect in protein synthesis. No further influence of either cytostatic therapy or different cytological classifications of the blasts could be demonstrated.

ALTERNATIONS OF HEMOSTASIS DURING CARDIOPULMONARY BYPASS (CPB): A COMPARISON BETWEEN MEMBRANE AND BUBBLE OXYGENATORS. R.L. Rick, N.R. Arbogast and W.R. Schmalhorst. San Joaquin Hemostasis Oncology Medical Group, California Coagulation Laboratories, San Joaquin Community Hospital, Bakersfield, California, and UCLA Center for the Health Sciences, Los Angeles, California.

Alterations of hemostasis during cardipulmonary bypass (CPB) using bubble oxygenators have been previously defined and found to consist of a severe platelet function defect, a primary hyperfibrinogeno(gel)ytic syndrome, and minimal thrombocytopenia. This study compares defects in hemostasis with membrane oxygenators and bubble oxygenators. 30 consecutive patients were studied and all patients studied were undergoing elective coronary artery bypass surgery. Tests of hemostasis included thrombin and reptilase times, protamine corrected thrombin times, soluble fibrin monomer, fibrinogen degradation products, fibrinolytic assays, platelet counts, and tests of platelet function. Studies were drawn pre-bypass, mid-bypass, and 1 hour post bypass. It was found that thrombocytopenia was much less in membrane patients.

All patients developed a primary hyperfibrinogeno(gel)ytic syndrome and the degree of this was equal in bubble or membrane oxygenators. Platelet dysfunction also was seen in all patients but was significantly different between the two oxygenation systems. At one hour postop, membrane patients showed no correction of platelet function as assessed by adhesion (14%), while those perfused with bubble oxygenators showed significant correction (67%) at one hour postop.

In conclusion, the primary hyperfibrinogeno(gel)ytic syndrome occurring during cardipulmonary bypass appears to be of equal significance regardless of oxygenation mechanism. Less thrombocytopenia, but more platelet dysfunction is seen with the membrane system.

IDENTIFICATION OF MEMBRANE-LINKED PROTEINS ACTING ON BLOOD RED CELL FILTRATION. STUDY IN CEREBROVASCULAR ACCIDENT AND THROMBOSIS. M.R. Baiseau, M.F. Lorenzo-Roudaut, J.P. Manau, P. Blanchard, E. Crockett, M. Briceaud - Hôpital Cardioangiologique de Bordeaux et INSERM, U 6 de Cardiologie, PESSAC (France).

The red blood cell filtration (RCF) through nucleopore filters (5 μm) decrease in cerebrovascular accidents (CVA) and thrombosis. This disorder is related to circulating factors, especially fibrinogen and factor VIII. But after washing it also appears to be related to membrane-linked proteins. In this study, isolation and identification of these proteins were realized in patients with increased RCF.

20 patients with CVA and/or other thrombosis, 8 days after the onset and compared to 30 controls.

After the RC have been washed twice the proteins were eluted from the membrane, concentrated and submitted to usual electrophoresis and immunoelectrophoresis. The identification was made with specific antisera against factor VIII, fibrinogen, fragments D and E, transferrin, coaguloplasmin and using SDS polyacrylamid gel electrophoresis with, as controls, purified fibrinogen, fragments D and E, transferrin, coaguloplasmin.

The results were: in controls the eluted proteins are similar with an albumin/globulin ratio (A/G)>1. In patients with a high disorder of RCF the proteins were increased, with an inversion of the A/G. Two kinds of proteins were found: (1) a β population composed of inflammatory proteins, mainly transferrin, (2) a α-β population constituted by fibrin-related products. Concerning the whole population of patients with increases in RCF, 80 % exhibited proteins induced by inflammation. In 20 % it was found proteins related to the coagulation, i.e.: fibrin related products. Finally, the disorder of RCF in thrombosis is much more due to these membrane disturbances than to circulating factors. During the clinical care of the patients it also has been observed that pentoxyphiline improved the RCF, certainly acting at the membrane level.