EVIDENCE FOR DISSEMINATED INTRAVASCULAR COAGULATION IN NEWBORNS WITH STREPTOCOCCAL SEPSIS. A.H. Sutor, F. Staudt, W. Pringsheim, and W. Künzer. Universitäts-Kinderklinik Freiburg, Germany FR.

Neonatal sepsis with Streptococci group B has a high mortality rate. 10 of our 13 patients died within a few days. In all patients we found low platelet counts. However, only the non-survivors showed additional coagulation findings compatible with DIC (low plasma coagulation factors, low plasminogen proactivator level, increased fibrin-fibrinogen degradation products, and low granulocyte numbers). Clinical symptoms were not present until shortly before death. The diagnosis of DIC was confirmed at autopsy. The clinical course was unchanged inspite of adequate antibiotic therapy. In 3 cases therapy with heparin or exchange transfusion was unsuccessful.

ALTERATION OF STREPTOKINASE-INDUCED FIBRINOLYSIS UNDER CLINICALLY RELEVANT CONDITIONS. A.H. Sutor, G. Wünsch-Macholz, and W. Künzer. Universitäts-Kinderklinik Freiburg, Germany FR.

Testing streptokinase-induced fibrinolysis in human plasma we obtained the following results: Optimal fibrinolysis takes part at pH-values between 6.8 and 7.25, at NaCl-concentrations between 0.04 and 0.08 g/l, at temperatures between 36° and 42°, and at streptokinase-concentrations between 0.05 and 2 U/ml plasma. Outside of these ranges fibrinolytic activity is reduced. No correlation was found between streptokinase-induced fibrinolysis and hemoglobin concentration, but a strongly positive correlation between fibrinolysis and erythrocyte stromata concentration.

PROBLEMS WITH ANTIPLATELET THERAPY IN CHILDREN WITH CYANOTIC HEART DISEASE. A.H. Sutor, B. Schmidt, H.P. Lorenz, and J. Vogt. Universitäts-Kinderklinik Freiburg, Germany FR.

Children with cyanotic heart disease are endangered by thromboembolic complications. However, in our 17 patients we could not find laboratory evidence for consumption coagulopathy but found evidence for isolated platelet consumption with increased platelet turnover. We therefore applied antiplatelet drugs (acetylsalicylic acid and dipyridamol) in order to prevent thromboembolic complications. With antiplatelet therapy the platelet count increased in average, however, 12 of 17 children with cyanotic heart disease did not show prolongation of the bleeding time as did controls. There is strong evidence that in these children an adequate blood-level of antiplatelet drugs was not obtained either by non-absorption (due to cardiac decompensation) or because of relative underdosage (due to increased platelet turnover) of the antiplatelet drugs. 2 of these patients who had a normal bleeding time, inspite of antiplatelet drugs, suffered from thromboembolic complications requiring fibrinolytic therapy. As a consequence we increase the dosage of antiplatelet drugs when the bleeding time does not prolong.