ANTISTREPTOKINASE TITRES BEFORE, DURING AND AFTER STREPTOKINASE THERAPY. R. Naidu, H. Köstering, M.A. Guerrero and R. Hampe. Department of Internal Medicine, University of Göttingen, W. Germany.

We usually prefer streptokinase therapy for thrombolytic treatment in patients with thromboembolism. For this therapy it is necessary to know the antistreptokinase (ASTK) titre, in order to calculate the initial dose, due to streptokinase resistance in some patients. The ASTK titres in 108 normal subjects and in 442 patients were examined and showed that 4.66% needed one million units streptokinase, 7.86% 500,000, 21.7% 250,000, 23.3% 100,000 and 42.5% 50,000 units. The results varied greatly in the different groups of patients.

It is sometimes necessary to repeat streptokinase administration to patients. The ASTK titre was therefore determined daily for three weeks in 25 patients before, during and after streptokinase therapy. In another group of 70 patients, who had previously received this treatment for up to 72 months, the ASTK titres were elevated and 7 days following the beginning of the new treatment had increased to 15 million units and between the 11th and 16th day to 30 million units. This was followed by a slow but steady decrease to 6 million after one month, 2.5 million after 2 months and 900,000 after 3 months. After six months were were most often normal values and low titres were rarely seen. This means that thrombolytic therapy could be repeated at this time.

EVALUATION OF UROKINASE THERAPY FOR CEREBROVASCULAR OCCLUSIVE DISEASES. T. Abe, M. Kazama, I. Maruyama and R. Fukuda. Teikyo University School of Medicine, Tokyo, Japan.

Urokinase (UK) was administered to 55 cases of possibly occlusive cerebrovascular accidents and its clinical effects were analyzed. The indication was considered by the elapsing time from the onset, the gravity of clinical signs, the localization and size of brain damage, the angiographical feature and the supposed pathohistological changes in brain. UK administration was controlled mainly by PT, PTT, fibrinogen-, FDP-content, ELT, platelet count and its functions as the monitoring parameters. Average doses of UK for adults, 30,000 IU twice daily, were continued for the first week, and then decreased by half every two or three days. The thrombolytic treatment was maintained for two to three weeks. Administration of Warfarin or dextran sulfate was started several days before the discontinuation of UK therapy and continued for some weeks.

Depending on the response of the patients to this therapy, they were divided into five classes;

- a) clinical improvement with visible resolution of thrombi proved by angiography or computerized tomography (14 cases),
- b) clinical improvement without visible angiographic findings (35 cases),
- c) no improvement in clinical and laboratory findings (3 cases),
- d) clinical improvement with worsening of partial clinical signs (3 cases), and
- e) clinical exacerbation without any effect (no case).

SIMULATION OF COUPLED ENZYME ASSAYS FOR A NEW DETERMINATION OF PLASMINOGEN VIA THE CLOTTING TIME. O. Richter and E. Jacobi. Institute of Biomathematics and Second Departm. of Internal Medicine, University of D-4000 Düsseldorf, W.-Germany.

The enzymatic assay for the determination of plasminogen via the clotting time (JACOBI et al., Dtsch. med. Wochenschr. 101 (1976) 1220-1222

0.2 ml plasminogen reagent (test-kit Behringwerke/Hoechst AG)

0.1 ml plasma, diluted 1:100, incubation time 3 min. 37°C

0.05 ml thrombine (3 NIH U/ml), clotting time

is based on the following sequence of reactions: (plasminogen reagent = fibrinogen, bovine plasminogen and streptokinase, which does not react with bovine plasminogen) human plasminogen and streptokinase form an activator, which converts bovine plasminogen to bovine plasmin, which splits fibrinogen during the incubation time. The thrombine clotting time of the resulting mixture containing fibrinogen and fibrinogen degradation products depends on the amount of bovine plasmin in the assay, which is in turn a function of human plasminogen concentration. The assay is described by a kinetic model in terms of non-linear differential equations. On the basis of this model the clotting time can be computed as a function of the parameters of the assay: concentration of fibrinogen, human plasminogen, bovine plasminogen and incubation time. By computersimulation an optimal parameter set is obtained for which the resulting calibration curve is linear and not sensitive against the variances of the reagent. Furthermore, the model gives a detailed insight into the mechanism involved in the interaction of human plasminogen, fibrinogen, bovine plasminogen and thrombine.