I. Rákóczi, B. Garadnay, L. Arnold and I. Gáti (Clinic for Gynecology, 7643 Pécs, Hungary):
Effect of Heparin on Antithrombin III Activity During Delivery and Early Puerperium. (152)

It is known that antithrombin III (AT III) is the main inhibitor of blood coagulation. It inactivates thrombin and activated X factor. Heparin increases the AT III activity in vitro as well as in vivo. The authors have studied the AT III activity in sera of 30 pregnant women, at term, before labour started (2–24 hrs) and 30 as well as 60 min after the birth of placenta and daily for five days after delivery. The AT III activity was determined using the modified method of Gerendas. Out of the 30 women 15 were given 5000 IU heparin subcutaneously $1^1/_2$–2 hours before delivery. In the 15 women with no heparin treatment AT III activity of serum significantly decreased after birth of placenta compared with the predelivery value and became normal on the fifth postpartum day. Heparin given subcutaneously prevented development of this decrease.

O. Egeberg (The Institute for Thrombosis Research, Rikshospitalet, Oslo, Norway):
Inherited Antithrombin III Deficiency and Thrombo-Embolism. (153)

Thrombophilia due to inherited deficiency of blood antithrombin III (AT III, heparin cofactor, anticonvertin) in a Norwegian family was published 1965, Thromb. D. h. *13*, 516 & *14*, 473. Only a few families with this defect have since then been described in different countries. In another Norwegian family, two sisters, age 42 and 30, and a brother, 35, have had episodes of venous thrombosis and pulmonary embolism from the age of 24–29. Their father suffered from thrombosis and died at 67. The two sisters have blood AT III level about half of normal average, measured with a two-stage coagulation assay. Data from both families are compatible with an autosomal dominant inheritance of the plasma protein deficiency. Venous thrombosis in the families is remarkably often complicated with embolizations; this might also relate to an inadequate platelet function. Platelet aggregation time of PRP with added thrombin or ADP was found prolonged. In coumarin treatment of the patients, AT III assaying gave increased levels.

I. Nagy and H. Losonczy (I. st Dept. of Int. Med. University Pécs, 7643 Hungary):
The Significance of the Chronic Anticoagulant Treatment in Recurrent Thromboembolie Caused by Hereditary Antithrombin III Deficiency. (154)

It has been known since the publication of Egeberg (1965, 1970) and Marciniak (1974) that herditary antithrombin III deficiency could be the cause of recurrent venous thromboembolism.

The authors observed in 5 cases of severe repeated venous thrombosis in young patients an antithrombin III decrease, which proved to be a hereditary abnormality. In the case of a 15 years old girl the late introduced anticoagulant treatment could not save the life of the patient, she died after repeated deep vein thrombosis. In the other cases the long-lasting anticoagulant treatment resulted in a perfect clinical improve, while the behaviour of antithrombin III was different; in some cases its quantity (determined by radial-immunodiffusion) and functional activity (examined by modified method of Gerendás and Rák) remained decreased, while in the other cases its functional activity increased during the anticoagulant treatment as it was found by Marciniak, too.

It is most likely, that there are two types of hereditary antithrombin III decrease; in one of them the quantitative and functional decrease goes parallel, in the other there is mainly a functional decrease, which improves during the chronic anticoagulant treatment. The authors demonstrated the significance of the prolonged anticoagulant treatment in the patients with hereditary antithrombin III decrease.