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CHANGES IN FIBRINOLYTIC PARAMETERS AFTER DELIVERY. J.C. Kirchheimer (1), H. Kölbl (2), G. Christ (1) and G. Tatra (2), Lab. Clin. Exp. Physiol. (1) and 2nd Dept. Obstet. & Gynaecol. (2), Univ. Vienna, Austria

Recent studies by Astedt et al. have shown increasing levels of plasminogen activator inhibitor during pregnancy, but the origin of the inhibitor is unknown. Levels of fibrinolytic parameters were determined in plasma collected from 18 females (age 22.7 ± 3.2, mean ± SD) after a normal medically controlled pregnancy at the time of delivery and on the following 5 days. Tissue-type plasminogen activator (tPA) antigen was measured by enzyme immunoassay, urokinase type plasminogen activator (uPA) antigen by a radioimmunoassay and plasminogen activator inhibitor (PAI) by a functional assay. The results are summarized in the following table:

	tPA antigen (ng/ml)	uPA antigen (ng/ml)	PAI activity U/ml
delivery	7.63 ± 3.50	6.83 ± 0.55	11.32 ± 2.79
day 1 p.p.	7.60 ± 3.95	6.48 ± 0.61	8.10 ± 2.31
day 2 p.p.	4.85 ± 1.89	6.30 ± 0.73	7.86 ± 1.84
day 3 p.p.	—	5.65 ± 0.35	—
day 4 p.p.	4.65 ± 2.19	6.00 ± 0.98	7.85 ± 1.76
day 5 p.p.	—	6.50 ± 0.82	7.70 ± 1.26

Postpartal changes in tPA antigen and PAI have been found to be significant, both decreasing after delivery and reaching normal control values for tPA on day 2 and for PAI on day 1 while uPA antigen remained normal. Since tPA levels before delivery have been found to be normal, increased levels at delivery might be caused by a release or by hormonal changes, while the decrease in PAI might again be caused by hormonal changes or by removal of the placenta.

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A MONOCLONAL BASED ELISA FOR HUMAN u-PA QUANTIFICATION

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An ELISA has been developped for quantifiing the antigen levels of u-PA present in human plasma,tissue and cell extracts,conditioned medium and others biological fluids.

The assay was set up using PVC plates coated with rabbit anti u-PA IgG and a monoclonal antibody against human u-PA as second antibody (UKM_{23} obtained in our laboratory as previously described).Detection was performed with a rabbit anti-mouse IgG conjugated with horseradish peroxidase.

By immunoblotting technique the monoclonal antibody used UKM_{23} , recognizes all human molecular weight species and an additional band of 81 KD in human plasma. Also recognizes the u-PA present in conditioned medium from HT-1080 cell line.

The detection limit of ELISA assay is 0,1 ng of total u-PA.The first assay in human plasma from healty volunters, shows u-PA levels of 4,39 ± 0,94 ng/ml.

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ACQUIRED HAEMOPHILIA DUE TO FACTOR VIII INHIBITOR WITH SEVERE HAEMORRHAGES IN A 46-YEAR-OLD WOMAN SUCCESSFULLY TREATED WITH CYCLOSPORIN A. Z. Boda, J.Hársfalvi, K. Pecze and K.Rak. 2nd Department of Medicine, University Medical School, Debrecen, Hungary.

A formerly healthy 46-year-old woman suffering from acquired haemophilia caused by factor VIII antibodies was admitted in an unconscious state following subarachnoid haemorrhage. Treatment with prothrombin complex concentrates (taken as a whole 100 000 U of PCC, home made and 10 000U of FEIBA, Immuno), steroid (Prednison 50 mg/day) and cyclophosphamide (100 mg/day) was only partially successful: neurological state improved but the haemorrhagic tendency remained. Significant haematuria, and skin and mucosal bleeding characterized her clinical picture. In the meantime, signs of non-A non-B hepatitis were observed. After recovery treatment with Cyclosporin A (Sandimmun, Sandoz) was started (250 mg/day per os) together with small dose of Prednison (15 mg/day). No PCC was applied since that time and the partial thromboplastin times (PTT) became gradually shorter. Level of factor VIII inhibitor was 160 Bethesda unit prior and 9 unit after treatment, the duration of that was 60 days till now. Factor VIII coagulant activity (VIIIC) increased from value of less than 1 percent to 13.7 percent.

Treatment of acquired haemophilia caused by factor VIII antibodies, particularly in cases with central nervous system bleeding, may be very difficult. History of our patient may indicate that patients resistent to substitution therapy, steroid and cytostatics may response well to Cyclosporin A. Therefore, its use is recommended.

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MONOCLONAL PURIFIED FACTOR VIII:C (MONOCLATE) TREATMENT IN A PREVIOUSLY UNTREATED HAEMOPHILIA. L. Parapia, A. Minford, J.B. Hamilton, School of Biomedical Sciences, Bradford University and Bradford Haemophilia Centre, Bradford Royal Infirmary, England.

Monoclate is a new generation of Factor VIII concentrate produced by purification using mouse monoclonal anti-Factor VIII:R antibody. As the Factor VIII:C does not interact with the antibody it can be eluted by disrupting the Factor VIII:C - Factor VIII:R complex using a high concentration of calcium ions. The eluted Factor VIII:C is concentrated and purified. The method of manufacture has demonstrated efficacy in the elimination of infectious viral particles.

The first "virgin" haemophiliac to be treated by this has completed 20 weeks follow-up. The patient, a child of 18 months with a Factor VIII:C level of 2.8%, was treated with 190 x 4 units of the Factor VIII concentrate for a severe cut of the lower lip.

The HIV status has remained negative. The AST and ALT enzymes have remained within normal limits. Other parameters which have remained normal are Gamma GT, WBC and lymphocyte counts, T cell subsets and B cell ratios.

The patient has remained well and no side effects have been noted. Mouse antibody titres are being carried out and the results will be presented at the conference.