In women post CS, anti-Xa levels do not reflect the full anticoagulant profile of tinzaparin and thrombin production is effectively reduced even when anti-Xa levels are negligible.

### P.37

**The use of low-molecular-weight heparin nadropran calcium by pregnant women with thrombophilia**

M. Huklova*, J. Hulikova. Center of Haemostasis and Thrombosis, HEMO MEDICA Kosice, Slovakia

Most women with a thrombophilia have healthy pregnancies. However, the thrombophilia can contribute to many pregnancy complications (preeclampsia, intrauterine growth retardation, abortion, placenta, stillbirth) and recurrent miscarriages. We evaluated the pregnancy outcome by women with inherited / acquired thrombophilia, treated with Fraxiparine.

**Methods:** 292 women with multiple genetic thrombophilic mutations / acquired thrombophilia with a history of last two recurrent miscarriages, or one of the pregnancy complications, were treated during their consecutive pregnancies with Fraxiparine, from the time of verification of the pregnancy throughout gestation, until 4–6 weeks in puerperium. In most cases, a fixed dose of Fraxiparine (0.3 ml s.c.) was administered. The women were tested for the mutation of FVLeiden, FII 20210, MTHFR, FXIIIC46T, FXIII val34leu, PAI-1, fibrogen G455A and antiphospholipid antibody.

**Results:** Before any untreated pregnancies (n=802), the rate of fetal loss (early and late) was 65.1% (average number 1.6), 33.6% live infants survived. By treated women we had a good pregnancy outcome (delivery 87%, fetal loss only 13%), Abortion risk ratio: 0.21 and number necessary to treat: 1:67. We didn't find out any statistically significant connection between the type of thrombophilia and the way of terminating the pregnancy.

**Conclusion:** Our results indicate that a therapy with nadropran calcium by pregnant women with thrombophilia significantly reduces recurrent miscarriage, pregnancy complications and improves maternal and fetal outcome.

### P.38

**Genetic variability of LXRbeta gene might contribute to preeclampsia**

K. Mouzat1 *, E. Mercier2, A. Polge1, A. Evrard1, J-M. Lobaccaro3, J-P. Brouillet1, S. Lumbroso1, J-C. Gris3, 1Biochemistry Laboratory and 2Hematology Laboratory, University Hospital, Nîmes, 3GReD Laboratory, UMR CNRS 6247 – Clermont University and UMR INSERM U931, Aubière, France

Preeclampsia (PE) is a frequent complication of pregnancy and is one of the leading causes of perinatal mortality. Both genetic and environmental factors may affect its risk. Lipid metabolism, especially cholesterol metabolism is associated to the pathology. Among the actors implicated in this metabolism, Liver X Receptors alpha (LXRalpha, NRH3) and beta (LXRbeta, NRH12) play a central role. They belong to the nuclear receptors superfamily and are activated by cholesterol derivatives. Their implication in preeclampsia is also suggested as they can modulate trophoblast invasion and they regulate Endoglin (CD105) gene expression, a marker of angiogenesis. A previous study has suggested that defects in these genes may affect its risk. Lipid metabolism, especially cholesterol metabolism is associated to the pathology.

**Aim:** To study ETP in a healthy female population in order to identify higher risk of thrombosis.

**Materials and Methods:** We studied 80 healthy females aged 34.02 ± 6.8. The characteristics of the studied population are: Medical history: 6 (7.5%), Arterial hypertension: none, Oral contraceptive: no use, Family medical history: 6 (7.5%), Arterial hypertension: none, Oral contraceptive: no use, Alcohol use: none. Thrombin generation was performed on platelet poor plasma with the ETP reagent of Siemens in the automated analyzer BCS-XP. We also determined INR with Thromboel S (Siemens). From ETP we studied AUC (ma) – area under curve, INR cal., Tlag (s), T_max (s). We also determined INR with Thromboel S (Siemens). From ETP we studied AUC (ma) – area under curve, INR cal., Tlag (s), T_max (s).

**Results:** See the tables.

<table>
<thead>
<tr>
<th>INR</th>
<th>AUC (ma)</th>
<th>AUC cal</th>
<th>Tlag (s)</th>
<th>T_max (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.99 ± 0.11</td>
<td>411.7 ± 53.8</td>
<td>107.2 ± 13.2</td>
<td>20.8 ± 4.6</td>
<td>55.1 ± 12.4</td>
</tr>
</tbody>
</table>

**Group**

<table>
<thead>
<tr>
<th>AUC (ma)</th>
<th>AUC cal</th>
<th>Tlag (s)</th>
<th>T_max (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking: (n=38)</td>
<td>409 ± 60</td>
<td>106 ± 15.8</td>
<td>213.5 ± 5.1</td>
</tr>
<tr>
<td>Non smoking: (n=42)</td>
<td>413 ± 47</td>
<td>108 ± 10.4</td>
<td>203.1 ± 4.05</td>
</tr>
<tr>
<td>Abodem.Obesity: (n=25)</td>
<td>433.5 ± 59</td>
<td>113 ± 15</td>
<td>217.5 ± 5.4</td>
</tr>
<tr>
<td>Non Abodem.Obesity: (n=35)</td>
<td>402.4 ± 48</td>
<td>104 ± 11</td>
<td>20.8 ± 4.2</td>
</tr>
</tbody>
</table>

The data analysis was made with SPSS 16.0. There was found a significant difference of AUC (ma) in the groups with abdominal obesity against the group without abdominal obesity (p=0.001) and of AUC cal in the same groups (p=0.02). All the other groups have no significant differences (NS).

### P.39

**Decreased fibrin clot porosity in patients with antiphospholipid syndrome**

A. Vikerforst1 *, E. Svennungsson1, K. Bremme1, M. Holmström4, A. Antovic5. 1Dept of Medicine, 2Unit of Rheumatology and 3Coagulation Unit, Haemotology Centre, Karolinska University Hospital, 4Dept of Clinical Sciences, Danderyds Hospital, 5Dept of Women’s and Children’s Health, Division of Obstetrics and Gynaecology, Karolinska Institutet, Stockholm Sweden

**Background:** It has been reported that patients with type 1 diabetes and young males with myocardial infarction form a fibrin clot, which is tighter and more resistant to fibrinolysis in comparison to the fibrin clot formed by healthy controls.

**Materials and Methods:** We evaluated fibrin clot porosity in plasma-samples from 46 patients with APS, strictly fulfilling the Sydney criteria. Previously established flow measurement technique was used to determine the fibrin clot porosity, as expressed as the Darcy constant (Ks). A low Ks level indicates a tighter fibrin clot. Ks-levels were compared to reference Ks values from healthy individuals.

**Results:** The mean Ks-levels were significantly lower in the samples from patients with APS (6.7 ± 2.9) compared to reference Ks values (10.7 ± 1.6), p < 0.0001. Within the APS-group Ks-levels did not vary substantially depending on age, clinical APS – manifestations or aPL – pattern supporting the diagnosis at inclusion. However IgM antibodies to Cardiolipin and dalteparin-treatment seemed inversely related to fibrin gel porosity. There was also a trend towards lower Ks-levels for the subgroup of patients with previous obstetric morbidity.

**Conclusion:** Patients with an antiphospholipid syndrome form a tighter and more stable fibrin clot compared to the clot formed by healthy controls. To our knowledge this is a new finding. Future studies including larger patient materials and controls may shed further light on the aetiology of APS, which could contribute to better risk assessment and management for APS-patients.