

share the same primary pathology. The clinical features of pre-eclampsia are consistent with hypoxia and the changes in oxygen delivery and consumption indices in women with severe disease are similar to that observed in distributive shock. However, so far there are no studies done on maternal tissue oxygenation levels in pregnancies complicated by pre-eclampsia and IUGR.

**Methodology** Women in their third trimester with pre-eclampsia, IUGR and normal pregnancy ( $n = 16, 6, 16$  respectively) were recruited for the study. Filtrass strain gauge plethysmography was used to compare calf blood flow and Mediad iPOX pulse oximeter was used to compare the oxygenation in the three groups.

**Results** The resting peripheral blood flow was significantly reduced in pre-eclampsia group compared to normal pregnancy group (mean  $\pm$  SEM  $[2.1 \pm 0.22$  vs.  $1.01 \pm 0.1]$ ,  $p = 0.003$ ), however no change was demonstrated in IUGR group compared to normal pregnancy group (mean  $\pm$  SEM  $[2.1 \pm 0.22$  vs.  $1.9 \pm 0.5]$ ,  $p = 0.92$ ). No significant difference was noted in maternal tissue oxygenation between the normal pregnancy, pre-eclampsia and IUGR groups (mean  $\pm$  SEM  $[97.13 \pm 0.4, 96.69 \pm 0.33, 97.83 \pm 0.47]$  respectively)  $p = 0.26$ ). No correlation was found between blood flow and tissue oxygenation.

**Conclusion** This study demonstrated that there is reduced resting peripheral blood flow in women with pre-eclampsia but not in IUGR and the reduction in blood flow in pre-eclampsia is not associated with changes in tissue oxygenation.

#### PM.19 WITHDRAWN BY AUTHOR

#### PM.20 THYROID HORMONE ACTION IN THE DECIDUA DURING HUMAN PREGNANCY

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**Introduction** Maternal thyroid dysfunction is associated with complications of malplacentaion including miscarriages and pre-eclampsia. We hypothesise that thyroid hormones (TH) play an important role within human decidua in regulating placentaion.

**Methods** Deciduas from human pregnancy were obtained from 1<sup>st</sup> (8–11 weeks) and 2<sup>nd</sup> trimester (12–20 weeks) surgical terminations of pregnancy. Primary cultures of total decidual cells (TDC), and immunomagnetic bead isolated populations of stromal-enriched (CD10<sup>+</sup><sup>ve</sup>) and stromal-depleted (CD10<sup>-ve</sup>) cells, uterine natural killer cells (uNKs; CD56<sup>+</sup><sup>ve</sup>) and macrophages (CD14<sup>+</sup><sup>ve</sup>) were treated with T3 (0.10, 100 nM). Assessments were made of cell viability (MTT assay), cytokine and angiogenic growth factor secretion (immuno-mediated assay) and the effects of decidual cell-conditioned media on extravillous trophoblast (EVT) invasion through Matrigel®.

**Results** Immunohistochemistry showed the expression of TH transporters (MCT8, MCT10) and receptors (TR $\alpha$ 1, TR $\beta$ 1) required for TH-responsiveness in uNKs and macrophages from early gestation. The viability of TDC and cell isolates were unaffected by T3. In 1<sup>st</sup> trimester, T3 reduced IL-10 secretion by TDC and CD10<sup>-ve</sup> cells ( $p < 0.01$ ), and reduced GM-CSF, IL-10, IL-1 $\beta$ , IL-6, MCP-1 by macrophages ( $p < 0.01$ ). In 2<sup>nd</sup> trimester, T3 increased IL-10 by TDC ( $p < 0.01$ ) and reduced IL-10 by uNKs ( $p < 0.001$ ). T3 increased VEGF secretion by 1<sup>st</sup> trimester uNKs ( $p < 0.05$ ), and angiopoietin-2 by 2<sup>nd</sup> trimester TDC and uNKs ( $p < 0.05$ ). Conditioned media from T3-treated TDC and macrophages did not alter EVT invasion compared to untreated controls.

**Conclusion** TH regulate decidual cytokine and angiogenic growth factor secretion in a cell-specific and gestation-dependent manner. The summation of TH effects upon the secretome do not affect EVT invasion.

#### PM.21 COMPLIANCE WITH POSTNATAL THROMBOPROPHYLAXIS

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**Background** Venous thromboembolism (VTE) is the 3<sup>rd</sup> leading cause of maternal death in the UK<sup>1</sup>. In order to minimise VTE risk in the postnatal period, we introduced a new scoring system in June 2011, based on RCOG guidelines<sup>2</sup>. Every woman's VTE risk is scored, and those who meet predetermined criteria are discharged on a seven days (7/7) or six weeks (6/52) course of low molecular weight heparin (LMWH). There were concerns regarding patient compliance and so a survey was conducted to explore this.

**Method** Pharmacy data identified 113 postnatal women who were discharged in November 2011 on LMWH. A telephone survey in February 2012 assessed understanding of the need for LMWH, and compliance.

**Results** 52 women were successfully contacted: 29 had been prescribed a 7/7 course, and 23 a 6/52 course.

- 100% of women understood the need for LMWH.
- 96% of those on a 7/7 course completed all injections.
- Only 32% completed the 6/52 course.

We identified reasons for non-compliance and the destination of unused LMWH.

**Conclusion** Non-compliance has implications for both patient safety and cost. The survey highlighted the importance of effective patient education and identified a need for improved communication between primary and secondary care. A multidisciplinary approach, with all healthcare professionals emphasising the importance of LMWH in the postnatal period may improve long-term compliance. A patient information leaflet has since been introduced.

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#### PM.22 THE ROLE OF VEGF<sub>165</sub> B IN TROPHOBLAST SURVIVAL – IMPLICATIONS FOR PRE-ECLAMPSIA PATHOPHYSIOLOGY

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It is widely accepted that the pathophysiological foundations of pre-eclampsia are laid down in the first trimester, with inadequate invasion of placental trophoblasts into maternal spiral arteries of the uterus, resulting in defective arterial remodelling. The angiogenic VEGF family of glycoproteins are expressed in first trimester trophoblasts and are important factors in placental development, which occurs in a hypoxic (<2% O<sub>2</sub>) environment up to 10–12 weeks and normoxia (>20% O<sub>2</sub>) thereafter. First trimester VEGF<sub>165</sub> b levels are low in women destined to later develop pre-eclampsia, so we investigated whether VEGF<sub>165</sub> b plays a role in early trophoblast survival and therefore pre-eclampsia pathophysiology.

Trophoblast cells were cultured in hypoxic and normoxic environments, in the absence and presence of VEGF<sub>165</sub> b and a VEGF<sub>165</sub> b blocking antibody clone 56–1. Cell survival was studied via cytotoxicity experiments. Production of VEGF<sub>165</sub> b by trophoblasts was determined via enzyme linked immunoassay (ELISA).

VEGF<sub>165</sub> b production by trophoblasts was increased in response to hypoxia (hypoxia:  $1812 \pm 33$  pg/ml vs. normoxia:  $1407 \pm 95$  pg/ml, unpaired t test,  $p = 0.016$ ), and inhibition of VEGF<sub>165</sub> b increased