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PM.19 WITHDRAWN BY AUTHOR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 malplacentalation including miscarriages and pre-eclampsia. We hypothesise that thyroid hormones (TH) play an important role within human decidua in regulating placentalation.

Methods Deciduas from human pregnancy were obtained from 1st (8–11 weeks) and 2nd trimester (12–20 weeks) surgical terminations of pregnancy. Primary cultures of total decidual cells (TDC), and immunomagnetic bead isolated populations of stromal-enriched (CD10⁺^{ve}) and stromal-depleted (CD10^{-ve}) cells, uterine natural killer cells (uNKs; CD56⁺^{ve}) and macrophages (CD14⁺^{ve}) 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 α 1, TR β 1) required for TH-responsiveness in uNKs and macrophages from early gestation. The viability of TDC and cell isolates were unaffected by T3. In 1st trimester, T3 reduced IL-10 secretion by TDC and CD10^{-ve} cells ($p < 0.01$), and reduced GM-CSF, IL-10, IL-1 β , IL-6, MCP-1 by macrophages ($p < 0.01$). In 2nd trimester, T3 increased IL-10 by TDC ($p < 0.01$) and reduced IL-10 by uNKs ($p < 0.001$). T3 increased VEGF secretion by 1st trimester uNKs ($p < 0.05$), and angiopoietin-2 by 2nd 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 3rd leading cause of maternal death in the UK¹. In order to minimise VTE risk in the postnatal period, we introduced a new scoring system in June 2011, based on RCOG guidelines². 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 VEGF₁₆₅ 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% O₂) environment up to 10–12 weeks and normoxia (>20% O₂) thereafter. First trimester VEGF₁₆₅ b levels are low in women destined to later develop pre-eclampsia, so we investigated whether VEGF₁₆₅ 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 VEGF₁₆₅ b and a VEGF₁₆₅ b blocking antibody clone 56–1. Cell survival was studied via cytotoxicity experiments. Production of VEGF₁₆₅ b by trophoblasts was determined via enzyme linked immunoassay (ELISA).

VEGF₁₆₅ b production by trophoblasts was increased in response to hypoxia (hypoxia: 1812 ± 33 pg/ml vs. normoxia: 1407 ± 95 pg/ml, unpaired t test, $p = 0.016$), and inhibition of VEGF₁₆₅ b increased