293 THROMBIN GENERATION IN WOMEN WITH PREECLAMPSIA DIETMAR SCHLEMBACH[1], ANDREA ROSENKRANZ[2], MICHAEL HIDEN[2], EVA-CHRISTINE WEISS[1], UWE LANG[1] WOLFGANG MUNTEAN[2], [1]Medical University of Graz, Obstetrics and Gynecology, Graz, Austria, [2]Medical University of Graz, Dept. of Pediatrics, Graz, Austria

OBJECTIVE: Levels of thrombin generation markers such as prothrombin fragments 1+2 (F 1+2) and thrombinantithrombin complex (TAT) are elevated in women with preeclampsia. We investigated the thrombin generation (TG) by means of calibrated automated thrombography (CAT). The endogenous thrombin potential (ETP) has been shown to correlate with plasma-based hypercoagulable states and the individual's risk of possibly being affected by thrombosis.

STUDY DESIGN: TG was measured in platelet poor plasma by means of CAT in 17 late onset preeclamptic women (PE) and 80 healthy pregnant women (HP) during III. trimester. In addition, TAT and F1+2 were measured in plasma using commercially available ELISA assays (Enzygnost TAT® and Enzygnost F1+2®, Dade Behring, Marburg, Germany). For comparison of parameters the Mann-Whitney U test was performed. All statistical analyses were performed with SPSS©, software (SPSS Inc., Chicago, Illinois, USA).

RESULTS: ETP was significantly higher in preeclamptic women when compared to controls. The time until the thrombin peak was reached (TTP) was significantly prolonged in preeclamptic women, while no significant difference was found for the thrombin peak ($p=0.157$) or lag time ($p=0.446$). F1+2 and TAT were significantly higher in preeclamptic women when compared to controls ($p=0.0001$).

CONCLUSION: These data show that preeclamptic women generate significantly higher amounts of thrombin and that the time until the thrombin concentration approaches zero is significantly elongated in comparison to women with uncomplicated pregnancies. In addition, F1+2 and TAT were significantly higher in preeclamptic women, which is in accordance with previous studies on coagulation in preeclamptic pregnancies and representing an increased activity of the coagulation system. Our study demonstrates that ETP values are higher in preeclamptic women when compared to normal controls. These results suggest that it might be worthwhile to investigate, whether an increased ETP in early pregnancy is predictive for the development of future preeclampsia.

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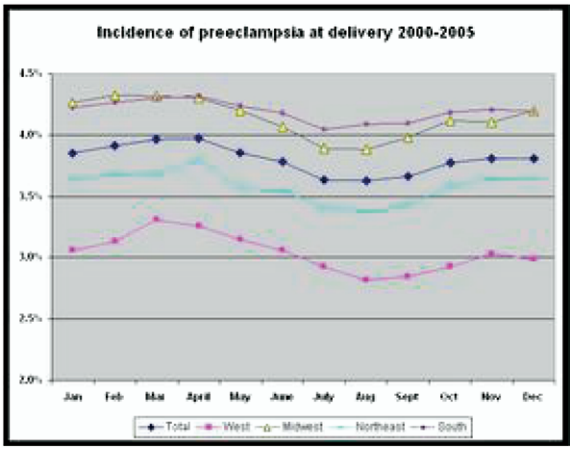
294 REGIONAL AND MONTHLY VARIATION IN RATES OF PREECLAMPSIA AT DELIVERY AMONG US BIRTHS JEFFREY SPENCER[1], SATYA POLAVARAPU[1], DIANE TIMMS[1], KATHLEEN SMITH[1], [1]University of Connecticut Health Center, Farmington, Connecticut

OBJECTIVE: To determine if there is a correlation between calendar month and rates of preeclampsia at delivery among US births divided by geographic region.

STUDY DESIGN: We queried the National Center for Health Statistics birth data from 2000-2005. We collected data on total deliveries and deliveries complicated by pregnancy induced hypertension. Data were stratified by month and by the four regions of the US as defined by the US Census, excluding AK and HI.

RESULTS: There were 24,446,824, deliveries in the US from 2000-2005 with 928,854 cases of preeclampsia (incidence=3.8%). Rates of preeclampsia in all four geographic regions showed a similar monthly variation. The Midwest and South had higher rates of preeclampsia at delivery during all months of the year ($p<0.001$).



CONCLUSION: All four geographic regions of the continental US show similar monthly variation in rates of preeclampsia at delivery, and the Midwest and South have higher overall rates of preeclampsia at delivery when compared with the West and East.

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295 CAUSES OF MASSIVE OBSTETRIC HAEMORRHAGE AND OUTCOMES OF MEDICAL AND SURGICAL MANAGEMENT STRATEGIES. DONAL O'BRIEN[1], E BABIKER[2], ORLA O'SULLIVAN[3], FIONNUALA MCAULIFFE[1], MICHAEL GEARY[3], B BRYNE[2], [1]UCD School of Medicine and Medical Science, Obstetrics & Gynaecology, National Maternity Hospital, Dublin, Ireland, [2]Royal College of Surgeons in Ireland, Coombe Womens and Infants University Hospital,, Dublin, Ireland, [3]Rotunda Hospital,, Dublin, Ireland

OBJECTIVE: Maternal mortality from haemorrhage in the UK increased in last triennium, but 80% of deaths received substandard care. The aetiology of massive obstetric haemorrhage (MOH) is changing and the efficacy of recent developments in medical and surgical management requires evaluation. This prospective audit was established to determine the incidence and aetiology of MOH and the success rates of medical and surgical interventions in our population

STUDY DESIGN: This prospective study from Jan 1st 2004 to Dec 31st 2007 recorded cases requiring acute transfusion of five or more units of blood (RCC) from the three Dublin maternity hospitals, with circa 24,000 deliveries annually

RESULTS: Of 93,291 deliveries over four years, there were 129 cases: the incidence of massive obstetric haemorrhage was 1.38/1000. There was one maternal death. The mean number of RCC transfused was 8 units +/− 5(SD). 60% of the women were multiparous and 26% had had a previous caesarean section (CS). Leading causes of MOH were uterine atony (49%), retained placental tissue (24%), placenta praevia (18%), cervical or vaginal trauma (16%), placenta accreta (12%) and broad ligament or uterine tears (10%). Medical therapies employed were oxytocin infusion (81%), misoprostol (64%), carboprost (33%), ergometrine (31%) and recombinant factor VIIa (4%). Medical management alone was successful in 28%. The success rate of surgical interventions was as follows: the hydrostatic balloon (80%), B-Lynch suture (40%), uterine artery ligation (50%) and internal iliac artery ligation (36%). 28 hysterectomies were performed. Interventional radiology was employed in one case

CONCLUSION: The incidence of MOH in our population is comparable to other reports using similar definitions. Uterine atony is the main cause of MOH but many women had multiple causes (38%). Although placenta praevia and accreta remain important causes of MOH, preventative methods for MOH should focus on early identification and aggressive management of uterine atony, retained placental tissue, and cervical and vaginal tears

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296 FETAL MACROSOMIA INCREASES RISK OF STILLBIRTH RADEK BUKOWSKI[1], FERGAL D. MALONE[2], T. FLINT PORTER[3], DAVID A. NYBERG[4], CHRISTINE H. COMSTOCK[5], GARY HANKINS[1], KEITH EDDLEMAN[6] SUSAN J. GROSS[7], LORRAINE DUGOFF[8], SABRINA CRAIGO[9], ILAN E. TIMOR-TRITSCH[10], STEPHEN R. CARR[11], HONOR M. WOLFE[12], MARY E. D'ALTON[13], [1]University of Texas Medical Branch, Obstetrics and Gynecology, Galveston, Texas, [2]Royal College of Surgeons in Ireland, Department of Obstetrics and Gynaecology, Dublin, Ireland, [3]The University of Utah / Intermountain Med. Center, Maternal-Fetal Medicine, Murray, Utah, [4]The Fetal & Women's Center of Arizona, OB/GYN Ultrasound, Scottsdale, Arizona, [5]William Beaumont Medical Center, Division of Fetal Imaging, Royal Oak, Michigan, [6]Mount Sinai School of Medicine, Obstetrics and Gynecology, New York, New York, [7]Jacobi Medical Center, Obstetrics and Gynecology, Bronx, New York, [8]University of Colorado Health Sciences Center, Obstetrics and Gynecology, Denver, Colorado, [9]Tufts University, Obstetrics and Gynecology, Boston, Massachusetts, [10]NYU Medical Center, School of Medicine and Obstetrics & Gynecology, New York, New York, [11]Women and Infants Hospital, Department of Obstetrics and Gynecology, Providence, RI, [12]University of North Carolina at Chapel Hill, Obstetrics and Gynecology, Chapel Hill, NC, [13]Columbia University, Maternal Fetal Medicine, New York, NY

OBJECTIVE: To determine the association between extremes of fetal growth and the risk of stillbirth.

STUDY DESIGN: In a cohort of 23,807 singleton pregnancies we calculated achieved percentile of individual fetal growth potential (GP). GP was calculated based on maternal and early pregnancy characteristics and categorized into growth restriction (GP<10%), normal growth (GP 10%-90%) and macrosomia (GP>90%). Gestational age was based on ultrasound in the first trimester. Stillbirth was defined as fetal death after 20 weeks of pregnancy in the absnce of chromosomal or congenital abnormalities.The association between GP and risk of stillbirth was adjusted for maternal characteristics (age, height, weight, race/ethncity, parity, smoking status) and pregnancy complications (chronic hypertension, preeclampsia, pre- and gestational diabetes).

RESULTS: There were 58 (0.24%) stillbirths without chromosomal or congenital abnormalities. Among them 59% had growth restriction and 14% were macrosomic. Fetal macrosomia was associated with a 3-fold and growth restriction with a 13-fold increased risk of stillbirth (RR, 95%CI = 3.2, 1.4-7.4, $p=0.008$ and 13.4, 7.4-24.3, $p<0.001$, respectively). Adjustment for maternal characteristics and complications of pregnancy did not have a material effect. Stillbirths of macrosomic fetuses occurred earlier than growth restricted fetuses (median, IQR = 29.0, 24.2-35.5 and 38.1, 33.8-39.6 weeks, $p=0.018$). There was no significant association between traditionally defined large for gestational age fetus and risk of stillbirth.

CONCLUSION: Extremes of fetal growth are associated with increased risk of stillbirth. Increased risk of stillbirth in macrosomia is independent of diabetes and other complications of pregnancy. Association between macrosomia and stillbirth is plausible because fetuses significantly larger than their optimal growth potential are vulnerable to external insults due to large nutritional demands relative to maternal supply. Monitoring of macrosomic in addition to growth restricted fetuses could potentially decrease rate of stillbirth.

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