122 TRENDS IN NEAR-TERM PRETERM BIRTH ADAM BORGIDA[1], SARAH HOPKINS[1], CAITLIN SAINT-AUBIN[1], DEBORAH FELDMAN[1], VICTOR FANG[1], CHARLES INGARDIA[1], [1]Hartford Hospital, Hartford, Connecticut

OBJECTIVE: We reviewed the trends in near-term (34 to 36 weeks) preterm birth (PTB) at our institution.

STUDY DESIGN: We performed a retrospective cohort analysis of our hospital's perinatal and neonatal databases to identify all singleton near-term PTBs from 1998 to 2006. We reviewed the trends in near-term PTBs by year and reviewed the modes of delivery. We also determined trends in NICU admission rates for near-term PTBs.

RESULTS: There were 35,689 singleton deliveries during the study period. Overall, 2,222 (6.2%) of births were between 34 to 36 weeks gestation. There was no significant increase in the rate of near-term PTB over the course of the study, overall or for any specific gestational age. However, the rate of cesarean birth increased significantly from 27% to 48%, $p<.0001$. The rate of NICU admission also increased significantly from 25% to 37%, $p<.05$.

CONCLUSION: Recently, studies have reported an increasing rate of near-term PTB, a rising cesarean rate and an increase in neonatal respiratory complications attributable to both. We did not find an increased risk of near-term PTB over a 9 year period at our facility. We did confirm the changing mode of delivery and the increasing rate of NICU admission. These findings provide further evidence that mode of delivery significantly affects neonatal complications.

0002-9378/$ - see front matter
doi:10.1016/j.ajog.2008.09.149

123 LOCAL INTERLEUKIN-1BETA IN PREGNANT WOMEN WITH BACTERIAL VAGINOSIS: IMPLICATIONS FOR PRETERM BIRTH JENNIFER CULHANE[1], SABINA CAUCI[2], [1]Children's Hospital of Philadelphia, Philadelphia, Pennsylvania, [2]University of Udine, Biomedical Sciences and Technologies, Udine, Italy

OBJECTIVE: Bacterial vaginosis (BV) is a microbial/mucosal immunity disorder associated with preterm birth, low birth weight, and miscarriage. Vaginal proinflammatory cytokines, particularly interleukin (IL)-1beta, have been suggested as determinant of both BV and preterm birth. We aimed to assess whether specific levels of vaginal IL-1beta in pregnant women with BV are associated with adverse pregnancy outcome.

STUDY DESIGN: Concentrations of IL-1beta were measured in vaginal fluid of 400 singleton pregnant women with BV (Nugent score 7-10) enrolled in Philadelphia in early gestation (12 weeks), comprising 105 adverse pregnancy outcomes including 66 preterm births (20 to <37 weeks' gestation), and 295 normal term controls (>37 weeks' gestation, >2500g at birth). Presence of concurrent STDs was recorded. The upper (>66th percentile) and lower (<33rd percentile) tertiles of IL-1beta concentrations were compared with the middle tertile (33rd to 66th percentile).

RESULTS: Vaginal IL-1beta concentrations were not significantly associated with any adverse pregnancy outcome. None of the tertiles of vaginal IL-1beta was associated with increased risk for preterm birth and miscarriage. To avoid confounding ([1]), in a secondary analysis, women with concurrent STDs were excluded, however, levels of IL-1beta were not associated with any adverse outcome.

CONCLUSION: High levels of IL-1beta in vaginal fluid has been suggested as risk factor for preterm birth. In our study, neither the highest nor the lowest levels of vaginal IL-1beta concentrations in BV positive pregnant women were associated with preterm birth. Our study is the largest one determining the relationship of IL-1beta with adverse pregnancy outcome among BV positive women. We observed that IL-1beta is not a risk marker for preterm birth among BV positive women in early gestation.

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0002-9378/$ - see front matter
doi:10.1016/j.ajog.2008.09.150

124 SEQUELAE OF PRIMARY CESAREAN DELIVERY IN SUCCESSIVE PREGNANCIES DARIOS GETAHUN[1], MICHAEL J FASSETT[2], CORINNA KOEBNICK[1], STEVEN J JACOBSEN[1], [1]Kaiser Permanente Southern California, Research & Evaluation, Pasadena, California, [2]Kaiser Permanente West Los Angeles Medical Center, Department of Maternal-Fetal Medicine, Los Angeles, California

OBJECTIVE: To examine whether a primary cesarean delivery (CS) in a first pregnancy increase risk of adverse outcomes in subsequent pregnancy.

STUDY DESIGN: We conducted a retrospective cohort analysis using the Matched Perinatal Services System, Hospital Inpatient and Physician encounter datasets, including birth certificate data and ICD-9 codes from hospitalization and outpatient physician encounter in all Kaiser Permanente Southern California hospitals (1991-2006). The study comprised of primiparous women with singleton births delivered at ≥20 weeks and fetuses weighing ≥500g linked to their consecutive pregnancies (n=62,067). Patients were excluded from the study if they had a prior history of the outcome under study. Odds ratio (OR) and 95% confidence interval (CI) were used to quantify the association after adjustment for confounders.

RESULTS: The rate of primary CS during the first birth was 19%. Risks of CS in the second pregnancy among women with and without previous primary CS were 6.9% and 67.5%, respectively (OR 26.1, 95% CI 24.7-27.5). Preterm (<37 wks) birth (OR 1.2, 95% CI 1.1-1.3), fetal distress (OR 1.6, 95% CI 1.4-1.8), chorioamnionitis (OR 3.3, 95% CI 2.9-3.9), endometritis (OR 3.2, 95% CI 2.5-4.2), preterm PROM (OR 1.2, 95% CI 1.0-1.5), placental abruption (OR 1.4, 95% CI 1.1-1.7), placenta previa (OR 1.4, 95% CI 1.1-1.7), uterine rupture (OR 46.7, 95% CI 18.7-116.8), stillbirth (OR 1.4, 95% CI 1.0-1.9), and neonatal mortality (OR 1.6, 95% CI 1.0-2.6) were significantly associated with prior primary CS. Risks of SGA birth, IUGR and neonatal mortality in the second pregnancy were unaffected by a prior primary CS.

CONCLUSION: Findings of this study suggest that a prior cesarean delivery may increase risk of adverse outcomes in subsequent pregnancy. This may be due to suboptimal implantation of the placenta in the vicinity of cesarean delivery scars and the cesarean scar may also induce a subclinical inflammation extending to subsequent pregnancy.

0002-9378/$ - see front matter
doi:10.1016/j.ajog.2008.09.151

125 MECHANISMS OF ADVERSE NEUROLOGICAL OUTCOMES FROM CHORIOAMNIONITIS AT TERM IRINA BURD[1], JINGHUA CHAI[1], ELLA OFORI[1], MICHAL ELOVITZ[1], [1]University of Pennsylvania, OBGYN; Maternal and Child Health Research Program, Philadelphia, Pennsylvania

OBJECTIVE: Infection during pregnancy conveys a significant increased risk for motor, cognitive and neurobehavioral abnormalities in the offspring. To date, research has focused on the effect of inflammation on preterm, not term, brain injury. While adverse neurological outcome is significantly increased in preterm infants, there is a growing body of data suggesting that inflammation at term is associated with increases in neurological injury, specifically cerebral palsy. The objective of this study was to elucidate whether prenatal inflammation at term results in brain injury.

STUDY DESIGN: A mouse model of intrauterine inflammation has been utilized for these studies. At E18.5, lipolysacharide (LPS) was injected in to uterine horn (n=6); controls received no intervention (n=6). QPCR was performed to assess cytokine expression in fetal brains. Morphological changes in neurons were investigated using an established cortical culture technique. Primary neuronal cultures were established 6 hours after exposure to LPS. Confocal microscopy (NF200 and MAP2) of neuronal cultures were performed for comparison of neuronal morphology. The number of dendritic processes at division day 3 (DD3) were recorded.

RESULTS: Fetal brains exposed to LPS had significanlty increased IL-1beta mRNA (12.4-fold, P=0.017), IL-6 (3.2-fold, P=0.04 and TNF-a (2.8 fold,P=0.08) compared to controls. IL-10 mRNA was not different. E18.5 LPS exposed neuronal cultures demonstrated significant morphological differences, including decreased aggregation of cells and abnormal growth of processes. On DD3, the number of dendritic processes were significantly decreased in LPS compared to control neurons ($p<0.001$).

CONCLUSION: Inflammation at term results in cytokine elevation in the fetal brain and altered neuronal morphology. These studies may provide a critical mechanism for the observed long term adverse neurological sequelae after exposure to inflammation at term. Further studies are needed in order to begin to develop therapeutic strategies to prevent adverse neurological outcome from inflammation at term.

0002-9378/$ - see front matter
doi:10.1016/j.ajog.2008.09.152