outcomes of interest: PPROM < 21 days, PTB < 28 weeks, and PTB < 32 weeks. For each outcome, multiple logistic models were fitted to examine the effect of trocar location, controlling for other potential risk factors. Odds ratios (OR) are reported with 95% confidence intervals.

RESULTS: 751 patients were studied. In bivariate analysis, LUS location was associated with PPROM < 21 days (18.3% vs. 8.8%, p = 0.0355), but not with PTB < 28 weeks (10.7% vs. 9.4%, p=0.72) or PTB < 32 weeks (12.2% vs. 8.2%, p = 0.09). Lateral location was not associated with any of the 3 outcomes. Results were confirmed with logistic regression models. Patients with LUS location were twice as likely as those with a more superior location to have PPROM < 21 days (OR = 2.17, 1.06-4.46), after controlling for placental location, Quintero stage, gestational age at procedure, preoperative vaginal bleeding, and study center.

CONCLUSION: After controlling for potential confounders, we found that trocar insertion in the LUS appeared to be associated with an increased risk of PPROM but not preterm birth. Inherent differences in the local milieu of the fetal membranes may explain these findings. Although the site of trocar insertion is often restricted by case-specific factors, it is reasonable to avoid the lower uterine segment if technically feasible.

219 Neonatal morbidity in preterm growth-restricted fetuses: does mode of delivery matter?

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OBJECTIVE: Fetal growth restriction (FGR) is associated with increased morbidity and mortality especially in preterm neonates. Cesarean rates among growth-restricted, premature fetuses are reported as high as 50%. The objective of our study was to examine neonatal outcomes in premature growth-restricted fetuses based on mode of delivery.

STUDY DESIGN: We performed a retrospective cohort study of patients with antenatally diagnosed FGR (estimated fetal weight less than 10th percentile) from 2006-2016. We included singleton, live born pregnancies delivering between 30-36 weeks' gestation. Neonates with suspected chromosomal abnormalities or anomalies were excluded. Maternal factors such as smoking, hypertension, parity, and abnormal Dopplers were examined. Neonatal outcomes included respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), necrotizing enterocolitis (NEC), 5-minute Apgar <7, neonatal death, and length of NICU stay. Mode of delivery and neonatal outcome were analyzed using Chi square. Logistic regression was used for composite neonatal morbidity. Length of NICU stay was tested using Kruskal-Wallis H test.

RESULTS: Complete data for 200 patients were available for our study. Of these, 50.3% underwent induction of labor and 49.5% underwent planned Cesarean. Ultimately 70.3% of the study population delivered by Cesarean. Composite morbidity was significantly higher in patients undergoing planned Cesarean delivery when compared to those who were induced and delivered vaginally (OR 2.73 [95% CI 1.10-6.75]). This finding remained significant after controlling for hypertension, smoking, parity, and abnormal Doppler studies. There was not an increase in composite morbidity in those patients who underwent induction but ultimately delivered by Cesarean compared with those delivering vaginally. No neonatal deaths occurred in our study population. Neonates delivered by planned Cesarean and Cesarean after induction were found to have a longer stay in the NICU (p <0.001).

CONCLUSION: Cesarean delivery is common among pregnancies complicated by FGR. Our data demonstrated a lower risk for neonatal morbidity in vaginal deliveries compared with planned Cesareans. These results are helpful for counseling patients regarding mode of delivery in growth restricted fetuses requiring preterm delivery.

Mode of Delivery	Induction/Vaginal	Induction/Cesarean	Planned Cesarean	P value
RDS (n, %)	6 (3%)	7 (3.5%)	22 (11%)	0.163
IVH (n, %)	0	0	2 (1%)	0.357
NEC (n, %)	0	0	1 (0.5%)	0.599
5-minute Apgar <7 (n, %)	0	0	3 (1.5%)	0.211
NICU stay (mean, days)	12.8	15.5	24.2	<0.001

220 Neonatal morbidity is increased with the inaccurate diagnosis of fetal growth restriction

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OBJECTIVE: With the limited accuracy of fetal growth ultrasound, sonographic screening for fetal growth restriction (FGR) introduces the potential for misdiagnosis of an appropriate-for-gestational age (AGA) fetus as FGR, which may increase the likelihood of iatrogenic prematurity and associated perinatal complications. We sought to determine the frequency of misdiagnosis of FGR in newborns with AGA birth weights, and to test the hypothesis that misdiagnosis of FGR increases the likelihood of prematurity and adverse perinatal outcomes.

STUDY DESIGN: In this retrospective cohort study, the exposed cohort consisted of all singleton, liveborn, non-anomalous infants delivered at Magee-Womens Hospital from 2003-2009 that were AGA at birth, but characterized as FGR on antenatal ultrasound. For each exposed infant, we selected 9 unexposed controls whose birth weight percentile fell within 3 points of the exposed infant and were considered AGA on growth ultrasound during the same gestational week that the exposed infant was considered FGR. Consistent with institutional protocol, fetal and neonatal weights were evaluated using the growth standard published by Fenton. We compared outcomes in exposed and unexposed infants using chi-square test and logistic regression, adjusting for education, tobacco use, marital status, race, and nulliparity.

RESULTS: Out of 40,577 AGA newborns that met inclusion criteria, 10,549 (26.0%) underwent fetal growth ultrasound beyond 24 weeks. Of these, only 78 (0.7%) were misdiagnosed as FGR. AGA neonates identified as FGR on ultrasound were more likely to deliver preterm, and more likely to require NICU admission (Table). There was only 1 neonatal death in the entire study cohort, and no cases of 5 min. Apgar<4. Misdiagnosis of FGR was not associated with an increased risk of Cesarean delivery.

CONCLUSION: With use of the Fenton nomogram, the antenatal diagnosis of FGR in AGA neonates was rare, but associated with an increased likelihood of preterm delivery and NICU admission. These risks must be considered when evaluating the utility of fetal growth ultrasound or adoption of less stringent fetal growth standards.

Rates of adverse outcomes in AGA neonates misdiagnosed as FGR and unexposed controls

Outcome	AGA birth- FGR US	CTL	Unadjusted p-value (Chi²)	Adjusted Odds Ratio (95% C.I.)
Preterm delivery	19/78 (24.4%)	28/702 (4.0%)	<0.001	7.13 (3.55-14.3)
NICU admission	20/78 (25.6%)	87/702 (12.4%)	0.001	2.60 (1.42-4.74)
Cesarean delivery	23/78 (29.5%)	186/702 (26.5%)	NS	1.18 (0.69-2.04)
Neonatal death	1/78 (1.3%)	0/702	-	-
5 min. Apgar<4	0/78	0/702	-	-

221 Impact of inclusion of fetal growth restriction in severe preeclampsia diagnosis

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OBJECTIVE: Based on the newest ACOG guidelines (2013), fetal growth restriction (FGR) is no longer a criterion for diagnosis of preeclampsia (PE) with severe features. This study examines the impact of including FGR as criteria for the diagnosis of PE with severe features on neonatal and maternal outcomes.

STUDY DESIGN: Retrospective cohort of women with PE at a tertiary care center from 2011-2015. Inclusion criteria was diagnosis of PE <37 weeks. Women with preeclampsia with neither severe features (blood pressure $\geq$ 160/110 or a serum laboratory abnormality) nor FGR were excluded. Exposure groups include PE with severe features and no FGR (SPEnoFGR), PE with FGR and no severe features (SPEbyFGR), and PE with severe features plus FGR (SPE+FGR). The primary outcome was perinatal death (stillbirth or neonatal death). Secondary outcomes were abruption, delivery for non-reassuring fetal heart tones (NRFHTs), intrapartum or postpartum eclampsia, and a composite of adverse maternal outcomes (cardiomyopathy, ICU admission, intubation, and readmission for hypertension). Groups were compared with ANOVA and χ^2, as appropriate.

RESULTS: Of the 618 women included, 527 (85.3%) were SPEnoFGR, 20(3.4%) were SPEbyFGR, and 70(11.3%) were SPE+FGR. The gestational age at delivery was earliest with SPE+FGR (31.1 wks) and latest in SPEbyFGR (35.5 wks). In spite of this, the presence of FGR in PE with or without other severe features was associated with an increased incidence of perinatal death (p<0.01). Presence of FGR in PE with or without other severe features was also associated with an increased incidence in delivery for NRFHTs (p<0.01). Maternal outcomes did not differ significantly between groups.

CONCLUSION: Although utilizing FGR as a severe feature would reclassify relatively few cases of PE, the presence of FGR has a significant impact on neonatal mortality.

Neonatal and maternal outcomes with FGR included in SPE diagnosis (presented as n (%))

	Severe Preeclampsia no FGR	Severe Preeclampsia FGR only	Severe Preeclampsia plus FGR	P value
	n=527	n=21	n=70	
Perinatal death	24 (4.6%)	2 (9.5%)	11 (16.4%)	<0.01
Stillbirth	16 (3.0%)	1 (4.8%)	4 (5.8%)	0.47
Neonatal death	11 (2.1%)	1 (4.8%)	7 (10.5%)	<0.01
Abruption	5 (1.0%)	0	2 (2.9%)	0.32
Non-reassuring fetal heart tones	40 (7.6%)	5 (23.8%)	23 (32.9%)	<0.01
Adverse maternal outcomes	35 (6.6%)	1 (4.8%)	3 (4.3%)	0.72
Eclampsia (intrapartum/postpartum	3 (0.6%)	0	0	0.77

222 Global transcriptomic analysis of human placenta in the setting of intrauterine growth restriction (IUGR) using RNA sequencing (RNA-Seq)

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OBJECTIVE: Appropriate fetal growth is linked to normal placental development and function. Identification of key pathogenic pathways that may be responsible for the increased morbidity and mortality of fetuses with IUGR is critical. We performed a genome-wide transcriptome RNA-seq analysis to discover placental villous trophoblast transcripts differentially expressed in the setting of preeclampsia (PE) as opposed to idiopathic IUGR.

STUDY DESIGN: Total RNA was extracted from 25 placental villous tissue samples of women with: 1) idiopathic PTB (iPTB, n=5, GA: 32±1wks; 2) idiopathic IUGR (n=5, GA: 29±1 wks); 3) severe PE & normal fetal growth (sPE, n=8, GA: 30±1 wks); 4) sPE & IUGR (n=7, GA: 30±1 wks). All IUGR fetuses had birthweights <10% (85% were at <3%) and abnormal Dopplers. Cord blood Insulin-like growth factor 1 (IGF-1) was measured as a marker of IUGR. RNA-seq was performed using the Illumina platform. Following data curation and quality control, statistical analyses were performed using 'DESeq2' and 'edgeR' (FDR<0.1).

RESULTS: Relative to iPTB, idiopathic IUGR resulted in altered expression of 3,908 placental transcripts. Relative to iPTB, placentas of sPE & IUGR exhibited altered expression of 1,639 genes. This group profile was consistent with significantly lower cord blood IGF-1 levels in idiopathic IUGR vs. iPTB newborns (P<0.001). Conversely, 576 genes were differentially expressed in the setting of sPE alone. Overall, the global transcriptional profiles of sPE and sPE & IUGR placentas were highly similar, with differential expression of <10 genes. In contrast, idiopathic IUGR differed from sPE & IUGR by 91 transcripts and from sPE & normal fetal growth by 543 transcripts. Idiopathic IUGR was associated with dysregulation of gene sets related to cell cycle regulation, oxidative phosphorylation, and insulin growth factor regulation. sPE was linked to pathways associated with altered adipokine signaling, lipid and sugar metabolism, and amyloid precursor processing.

CONCLUSION: The transcriptome of villous trophoblasts of pregnancies complicated by sPE and sPE & IUGR is remarkably similar, implying that in sPE, IUGR cannot be linked to a markedly different gene expression profile. The large variability of placental transcriptome in idiopathic IUGR emphasizes again the heterogeneity of the growth restricted phenotype.

223 Fetal growth restriction is associated with different patterns of placental abnormalities in twin and singleton pregnancies

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OBJECTIVE: The rate of fetal growth restriction (FGR) is higher in twin compared with singleton pregnancies. However, the mechanisms underlying FGR may differ between twins and singletons. Our aim was to compare the placental findings between twin and singleton pregnancies complicated by FGR.

STUDY DESIGN: This was a retrospective cohort study of all singleton and twin SGA (birth weight <10th%) newborns delivered in a single tertiary referral center between 2001-2015. SGA was diagnosed in twins and singletons using the recently published NICHD twins- and