261 **CIRCULATING ADRENOMEDULLIN IS INCREASED IN POST-HYPOXIC-ISCHEMIC BRAIN INJURY IN THE NEWBORN** EMANUELA MARINONI[1], ROMOLO DI IORIO[1], GABRIELE URBAN[2], DIEGO GAZZOLO[3], ERMELANDO COSMI V[4]; [1]University "La Sapienza," 2nd Dept. of OB/GYN, Rome; [2]New York University, Dept. of OB/GYN, Division of Maternal Fetal Medicine, New York, NY; [3]Giannina Gaslini Children's University Hospital, Dept. of Pediatrics, Genoa; [4]University "La Sapienza," 2nd Dept. of OB/GYN, Rome

OBJECTIVE: A substantial part of post-hypoxic-ischemic brain injury occurs upon reperfusion and reoxygenation by the excess release of vasoactive factors. Adrenomedullin (AM) is a newly discovered vasodilator peptide that participates in the regulation of cerebral blood flow. The aim of this study was to investigate whether AM is involved in the occurrence of IVH in asphyxiated infants.

STUDY DESIGN: A case-control study was performed on 20 full-term asphyxiated newborns: 10 developed IVH (group A) and 10 did not develop IVH (group B). Twenty term healthy newborns represented the control group. Routine laboratory parameters, neurological patterns, cerebral US scanning and Doppler velocimetry were assessed at 12 and 72 hours from birth. Plasma AM were determined at 12 hours.

RESULTS: No differences were found with respect to laboratory monitoring parameters and neurological examination at 12 hours from birth between asphyxiated infants developing or not developing IVH. At this time-point, in all but two infants, cerebral ultrasound examination was negative for pathological findings. MCA PI values at 12 hours were significantly higher in group A than in group B or controls ($P < .05$, for all). AM levels were significantly higher ($P < .001$) in group A (23.4 ± 6.7 fmol/mL) than in group B (8.8 ± 6.9 fmol/mL) or controls (9.6 ± 6.5 fmol/mL). In asphyxiated newborns AM concentration correlated with MCA PI values recorded at 12 hours ($P < .05$; $r = 0.44$).

CONCLUSION: Increased concentration of AM at 12 hours from birth in asphyxiated newborns who will develop IVH suggests that this peptide may play a role in the loss of cerebral vascular autoregulation which leads to brain injury due to reactive excessive vasodilatation. This study indicates that measurement of AM in post-asphyxial period may be useful in early detection of IVH when clinical examination and cerebral ultrasound are still silent.

262 **OBSTETRIC ANTECEDENTS OF CONGENITAL HEARING LOSS** KAREN CHU[1], ANDREW ELIMIAN[1], JAMIE BARBERA[1], J. GERALD QUIRK[1], [1]State University of New York at Stony Brook, Dept of Obstetrics, Gynecology and Reproductive Medicine, Stony Brook, NY

OBJECTIVE: Significant hearing loss is one of the most common major abnormalities present at birth. When undetected, hearing loss hampers speech, language, and cognitive development. Our objective is to determine what obstetric factors are associated with congenital hearing loss (CHL).

STUDY DESIGN: We reviewed maternal, neonatal hospital charts of all neonates found on neonatal hearing screening to have partial or complete CHL as well as a cohort matched by date of birth with normal audiometric findings from 1/1/98 through 12/31/2000. The two groups defined by the presence or absence of CHL were compared for various maternal and perinatal variables. Student t-test, χ test and logistic regression were used for statistical analysis.

RESULTS: 64 neonates had complete or partial CHL while the matched cohort consisted of 271 neonates. There were no differences between groups when compared for maternal age, parity, race, exposure to prescribed or illicit drugs, clinical or histiologic chorioamnionitis, and various perinatal outcome variables. The mean gestational age and birth weight (35.9 ± 5.9 vs 38 ± 3.1 wks, $P = .01$), (2698 ± 1173 vs. 3195 ± 816 g, $P = .002$) were significantly lower in the CHL group. Although univariate analysis revealed no significant differences between groups with regards to most various maternal, obstetric and perinatal variables, there was a significant association between CHL and maternal HTN, antenatal steroids, lower GA and BW at delivery, lower Apgar score, RDS, and structural/chromosomal anomalies. However on logistic regression, the OR, 95% CI and P values for BW ≤1500 g, 5 min. Apgar scores <7, FHx of CHL, and structural/chromosomal anomalies were (7, 3, 18, .001), (5, 2, 18, .009), (17, 2, 197, .02) and (64, 14, 292, .0001), respectively.

CONCLUSION: Congenital structural/chromosomal anomaly is a significant risk factor for the development of CHL. VLBW, low Apgar scores and a positive FHx were also independently associated with CHL.

263 **MATERNAL AND NEONATAL CARDIAC TROPONIN T LEVELS AFTER MATERNAL EXPOSURE TO MAGNESIUM SULFATE** MATTHEW ALVAREZ MD[1], ANDRA JAMES MD[2], SIHE WANG PHD[3], TAMMY SMITH BS[2], FRANK SEDOR PHD[1]; [1]Duke University, Department of Obstetrics and Gynecology, Durham, NC; [2]Duke University, Obstetrics and Gynecology, Durham, NC; [3]Duke University, Durham, NC

OBJECTIVE: Elevated serum troponin T, a protein unique to cardiac muscle, indicates myocardial injury. A study performed at our institution demonstrated that fetal troponin T is elevated when mothers are exposed to magnesium sulfate intrapartum. There have been no similar studies to evaluate troponin T levels in magnesium-exposed mothers. The purpose of this study was to compare troponin T levels in magnesium exposed and non-exposed mothers, as well as neonates.

STUDY DESIGN: Patients were identified intrapartum as having magnesium exposure (subjects) or not (controls). Umbilical cord and maternal venous blood was obtained after delivery. Samples were analyzed using the AVL 988-4 Electrolyte Analyzer to determine ionized magnesium levels and the Immunoassay Analyzer (Boeringer) to determine troponin T levels. Ionized magnesium levels were used to quantify magnesium exposure. Troponin T levels of exposed mothers and neonates were compared with controls. The Mann Whitney U Test (StatView.SAS) was used for statistical analysis.

RESULTS: Samples were analyzed from 14 subjects and 23 controls. The mean gestational age was 37 weeks (33-41) for subjects and 39 weeks (36-42) for controls. The reason for magnesium in all cases was preeclampsia. Ionized magnesium levels were significantly higher in mothers and neonates exposed to magnesium (see Table 1). Exposure to magnesium had no significant effect on maternal troponin T levels. In neonates exposed to magnesium, however, troponin T levels were significantly elevated (see Table 2).

CONCLUSION: After maternal exposure to magnesium, neonates, but not mothers, demonstrate elevated troponin T levels. This may reflect fetal cardiac effect or injury.

Table 1

Median ionized magnesium levels in mg/dL for subjects and controls

	SUBJECTS (N = 14)	CONTROLS (N = 23)	*P* VALUE
Mothers	1.381	.422	<.0001
Neonates	1.330	.474	<.0001

Table 2

Median troponin T levels in ng/dL for subjects and controls

	SUBJECTS (N = 13)	CONTROLS (N = 23)	*P* VALUE
Mothers	.013	0	.66
Neonates	.070	.034	<.01

264 **SITE-SPECIFIC DIFFERENCES IN FETAL WHOLE BLOOD COAGULATION** NIGEL SIMPSON[1], KEVIN WINKLE[1], LORNA ROBINSON[1], HEATHER GORTON[2], GORDON LYONS[2], JAMES WALKER[3]; [1]University of Leeds, Obstetrics & Gynaecology, Leeds; [2]University of Leeds, Department of Obstetric Anaesthetia, Leeds; [3]University of Leeds, Leeds

OBJECTIVE: Effective and rapid clotting is a feature of fetal blood, yet there are few data on the dynamics of clot generation within the umbilical circulation. Thromboelastography (TEG) is a technique which enables dynamic assessment of whole blood coagulation. This study set out to characterise clotting profiles in blood obtained simultaneously from umbilical artery and vein (UA and UV), and maternal vein (MV).

STUDY DESIGN: Maternal blood samples were obtained via a 16G cannula sited in a forearm vein, placed prior to elective cesarean section. Analysis by the TEG analyser (Haemoscope, Skokie, IL) was commenced at 4 minutes after venepuncture using native whole blood. At delivery a segment of umbilical cord was clamped, and samples were obtained from the umbilical artery and vein. Two samples from each were taken, one sample for TEG analysis and one for blood gas analysis. Intergroup differences were ascertained using non-parametric analysis.

RESULTS: The rate of clot formation was highest in UA (vs UV, $P = .002$; vs MV, $P = .002$), and similar in UV and MV (P = NS). The final clot strength was greatest in MV (vs UA, $P < .001$, vs UV, $P < .001$) with no difference between UA and UV. There was no correlation between any TEG parameter, blood gas measurement, birth weight, or haemoglobin concentration.

CONCLUSION: Coagulation is most rapidly achieved within the umbilical arterial circulation, providing an effective mechanism to avoid fetal-maternal haemorrhage at the placental interface. The mechanism by which subsequent passage through the placental circulation affects fetal coagulation is uncertain, and requires further investigation. (Supported by grants from The Burton Charitable Trust and Pfizer.)

Figure
alpha angle.