

**Table 2.** Doppler abnormalities for SGA twins and SGA singletons stratified by gestational age at delivery

Doppler indices	Gestational age at birth <34 weeks			Gestational age at birth 34-37 weeks			Gestational age at birth 34-37 weeks		
	Twins (n=36)	Singletons (n=115)	p-value	Twins (n=242)	Singletons (n=264)	p-value	Twins (n=75)	Singletons (n=764)	p-value
Any Doppler abnormality	21 (58.3%)	88 (76.5%)	0.034	59 (22.3%)	80 (33.1%)	0.007	6 (8.0%)	62 (8.1%)	0.972
UA/PD>95 <sup>th</sup> percentile	10 (27.8%)	20 (17.4%)	0.173	7 (2.7%)	11 (4.5%)	0.251	2 (2.7%)	4 (0.5%)	0.036
MCA-PI<5 <sup>th</sup> percentile	14 (38.9%)	56 (48.7%)	0.303	39 (14.8%)	57 (23.0%)	0.012	4 (5.3%)	50 (6.5%)	0.683
CPR<5 <sup>th</sup> percentile	12 (33.3%)	46 (40.0%)	0.473	45 (17.0%)	63 (26.0%)	0.014	2 (2.7%)	29 (3.8%)	0.621
REFD or AEDF	3 (8.3%)	43 (37.4%)	0.001	1 (0.4%)	5 (2.1%)	0.080	0 (0.0%)	0 (0.0%)	N/A

#### 417 Cerebro-placental ratio at third trimester screening as a predictor of adverse perinatal outcome

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**OBJECTIVE:** The aim of this study is to determine the value of cerebroplacental ratio (CPR) at routine third trimester scan in an unselected obstetric population to predict adverse perinatal outcome.

**STUDY DESIGN:** A quasi-experimental study was carried out including 5071 consecutive routine third trimester ultrasound scans (32+0-34+6 weeks) performed in a University Hospital. The CPR was calculated in unselected patients according to the explorer's criteria, as a ratio of the middle cerebral artery to the umbilical artery pulsatility indices. CPR below the 5 centile by gestational age was defined as abnormal. Exclusion criteria were major congenital anomalies, aneuploidy and multiple pregnancy. Ultrasound, clinical and perinatal data were collected from computerized clinical databases. Statistical analysis was performed with SPSS 20

**RESULTS:** CPR was assessed in 3325 pregnancies (65.6%). Those patients with abnormal CPR presented a statistically significant increase in preeclampsia (7.4% vs. 1.4% vs 1.3%,  $p = 0.024$ ), prematurity (37% vs. 6.4% vs 8.4%,  $p = 0.001$ ), born small for gestational age (SGA) (48.1% vs. 11.8% vs 7.2%,  $p = 0.001$ ), Apgar 5 minutes <7 (3.7% vs. 0.5% vs 1.1%,  $p = 0.009$ ), neonatal admission (25.9% vs. 4.5% vs 4.5%,  $p = 0.001$ ) and cesarean section rate (48.1% vs. 20.2% vs 21.9%,  $p = 0.001$ ), compared with normal or not performed. (Table 1) No differences were found among socio-demographic variables between groups.

The cohort in which the CPR was evaluated, whether normal or pathological, had a decrease in the prematurity rate (6.7% vs. 8.4%,  $p = 0.028$ ); Apgar test 5 minutes <7 (0.5% vs. 1.1%,  $p = 0.020$ ) and perinatal mortality (0.1% vs. 0.5%,  $p = 0.011$ ) compared with patients in whom CPR was not evaluated. (Table 2) Abnormal CPR was a best predictor of prematurity, Apgar 5 minutes <7, cesarean sections and neonatal admission than the isolated evaluation of the middle cerebral artery (MCAP) or the umbilical artery (UAPI).

**CONCLUSION:** Routine third trimester cerebroplacental ratio screening is associated with a decrease in the rate of prematurity, SGA, Apgar 5 minutes <7 and perinatal mortality. These results should be confirmed by randomized clinical trials.

**Table 1.** Perinatal outcome in pathological CPR.

N= 5071	Normal CPR (n=3298; 65,1%)	Pathological CPR (n=27; 0,5%)	Non evaluated CPR (n=1746; 34,4%)	p
Maternal age (SD)	32,05 (5,44)	33,09 (5,14)	32,27 (5,61)	0,284
Multipara (%)	1646 (49,9%)	9 (33,3%)	896 (51,3%)	0,323
BMI > 25 (%)	1190 (36,1%)	6 (22,2%)	686 (39,3%)	0,022
Smoking (%)	476 (14,4%)	4 (14,8%)	241 (13,8%)	0,827
Preeclampsia (%)	45 (1,4%)	2 (7,4%)	22 (1,3%)	0,024
Male newborn (%)	1657 (50,2%)	16 (59,3%)	902 (51,7%)	0,027
Prematurity (%)	212 (6,4%)	10 (37%)	146 (8,4%)	0,001
Gestational age at delivery (SD)	39,48 (1,59)	37,14 (3,52)	39,19 (2,31)	0,001
SGA (%)	389 (11,8%)	13 (48,1%)	126 (7,2%)	0,001
Neonatal weight (SD)	3228,53 (489,31)	2401,30 (875,59)	3272,55 (581,95)	0,001
Gestational age in ultrasound (SD)	33,20 (1,40)	32,98 (3,03)	33,81 (1,76)	0,001
pH < 7,10 (%)	72 (2,4%)	0 (0%)	42 (2,7%)	0,626
Apgar 5' < 7 (%)	16 (0,5%)	1 (3,7%)	19 (1,1%)	0,009
Cesarean section(%)	665 (20,2%)	13 (48,1%)	383 (21,9%)	0,003
Neonatal admission (%)	147 (4,5%)	7 (25,9%)	79 (4,5%)	0,001
Perinatal death (%)	3 (0,1%)	0 (0%)	8 (0,5%)	0,059

**Table 2.** Perinatal outcome in pregnancies with evaluated CPR vs. Not evaluated.

N= 5071	Not evaluated CPR (n=1746)	Evaluated CPR (n=3325)	p
Maternal age (SD)	32,27 (5,61)	32,05 (5,44)	0,180
Multipara (%)	896 (51,3%)	1655 (49,8%)	0,466
BMI > 25 (%)	686 (39,3%)	1196 (36%)	0,020
Smoking (%)	241 (13,8%)	480 (14,4%)	0,540
High blood pressure (%)	22 (1,3%)	47 (1,4%)	0,654
Male newborn (%)	902 (51,7%)	1673 (50,3%)	0,363
Prematurity (%)	146 (8,4%)	222 (6,7%)	0,028
Gestational age at delivery (SD)	39,20 (2,31)	39,47 (1,62)	0,320
Neonatal weight <P10 (%)	126 (7,2%)	402 (12,1%)	0,001
Neonatal weight (SD)	3272,55 (581,95)	3228,53 (498,99)	0,001
Gestational age in ultrasound (SD)	32,99 (1,76)	33,21 (1,42)	0,375
pH < 7,10 (%)	42 (2,7%)	72 (2,4%)	0,496
Apgar 5' < 7 (%)	19 (1,1%)	17 (0,5%)	0,020
Cesarean section(%)	383 (21,9%)	678 (20,4%)	0,001
NICU admission (%)	79 (4,5%)	154 (4,6%)	0,542
Perinatal death (%)	8 (0,5%)	3 (0,1%)	0,011

#### 418 Changes in fetal pulmonary artery doppler indices in response to maternal hyperoxygenation

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**OBJECTIVE:** The reactivity of the pulmonary vascular bed to the administration of oxygen is well established in the post-natal

circulation. We sought to evaluate changes in the pulmonary artery doppler following maternal hyperoxygenation in utero, during the third trimester of pregnancy.

**STUDY DESIGN:** Forty six women with a singleton gestation greater than or equal to 31 weeks gestational age were prospectively recruited to the study. A fetal echocardiogram was performed on all subjects to exclude congenital heart disease. Pulsatility index (PI), Resistance index (RI), Peak systolic (PSV) and end diastolic velocity (EDV), acceleration time (AT), and ejection time (ET) were taken within the proximal portion of the fetal main pulmonary artery (PA). AT:ET was used to assess pulmonary vascular resistance (PVR). Doppler measurements were taken at baseline and repeated immediately following maternal hyperoxygenation for 10 minutes (O2 100% v/v inhalational gas) at a rate of 12L/min via a partial non-rebreather mask. Doppler waveform measurements were also taken of the umbilical artery (UAD), middle cerebral artery (MCA) and the ductus arteriosus (DA).

**RESULTS:** The median gestational age was 35 [33 – 37] weeks. There was a decrease in fetal PA PI following maternal hyperoxygenation (from 2.37 [2.04 – 2.70] to 2.05 [1.69 – 2.41], p=0.001). The resistance index of the PA decreased from (0.86 [0.81- 0.91] to 0.78 [0.69-0.87]. There was an increase in PA AT (57 [42-71] to 66 [49 – 82] ms, leading to an increase in AT:ET following maternal hyperoxygenation (0.32 to 0.34), p=0.001) (Table 1). There were no significant changes in the resistance indices of the UAD and DA. There was a significant increase in MCA blood flow, but not in MCA resistance indices.

**CONCLUSION:** Maternal hyperoxygenation offers the opportunity to assess the reactivity of the pulmonary vasculature before birth. Our findings would indicate a reduction in fetal pulmonary vascular resistance with secondary increased fetal pulmonary blood flow. This was not achieved at the expense of ductal constriction. There was evidence of improved MCA peak systolic velocity parameters; this was likely due to the positive impact of improved pulmonary venous return on left ventricular preload. The hyperoxygenation test can inform us of functional rather than anatomical information in relation to the pulmonary arteries and this warrants further exploration in a larger cohort.

Table 1: Changes in Doppler indices at baseline and following Maternal hyperoxygenation

	Pre MH	Post MH	p-value
PA PI	2.37 [2.04–2.70]	2.05 [1.69–2.41]	<0.001
PA RI	0.86 [0.81- 0.91]	0.78 [0.69-0.87]	<0.001
PA PSV	66.4 [50.9-81.9]	62.3 [50.6-74.0]	0.142
PA EDV	9.5 [4.7-14.3]	14 [7.5-20.5]	<0.001
PA AT	56.6 [42-71]	65.5 [49–82]	<0.001
PA ET	181.2[145.2-217.2]	193.3 [158.3-228.3]	0.001
AT:ET	0.32 [0.23-0.40]	0.34 [0.27-0.41]	0.029
UAD PI	0.96 [0.71-1.21]	0.96 [0.76-1.16]	0.95
UAD RI	0.61 [0.52-0.70]	0.62 [0.54-0.69]	0.67
UAD PSV	39.8 [29.8-49.8]	41.6 [30.8-52.4]	0.28
UAD EDV	16.1 [10.5-21.7]	16.2 [11.2-21.2]	0.85
MCA PI	1.7 [1.1-2.3]	1.7 [1.2-2.2]	0.98
MCA RI	0.78 [0.69-0.87]	0.80 [0.67-0.93]	0.27
MCA PSV	33.3 [21.8-44.8]	40.5 [23.5-57.5]	0.005
MCA EDV	7.0 [3.7-10.3]	8.8 [3.2-14.4]	0.043
DA RI	0.87 [0.77-0.97]	0.88 [0.76-1.0]	0.74
DA PSV	50.3 [39.9-60.7]	50.4 [38.4-62.4]	0.62
DA EDV	7.3 [4.2-10.4]	7.6 [5.7-9.5]	0.81

MH, maternal hyperoxygenation; PA, pulmonary artery; PI, pulsatility index; RI, resistance index; PSV, peak systolic velocity; EDV, end-diastolic velocity; AT, acceleration time; ET, ejection time; UAD, umbilical artery Doppler; MCA, middle cerebral artery; DA, ductus arteriosus

**419 Prediction of fetal hemoglobin after multiple intrauterine transfusions**

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**OBJECTIVE:** A mathematical formula has been developed to estimate fetal hemoglobin based on the middle cerebral artery peak systolic

