umbilical arteries of healthy term SGA, LGA, and AGA newborns (n = 12 in each group). Optical densities of proteins of interest were normalized in relation to that of glyceraldehyde-3-phosphate dehydrogenase (GAPDH) as a standard.

Results: Protein expression of NO synthases, of prooxidative enzymes, and of antioxidative enzymes was similar in the three groups.

Conclusions: Protein expression patterns of enzymes involved in endothelial function and redox status did not differ in umbilical arteries of term SGA, LGA, and AGA newborns. Thus, indicators of arterial endothelial dysfunction in terms of aberrant enzyme expression in SGA or LGA newborns vs. AGA newborns were not present at birth.

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RANDOMIZED CLINICAL TRIAL ON ECHOCARDIOGRAPHICALLY GUIDED (ECHO) VERSUS STANDARD IBUPROFEN TREATMENT (SIBT) FOR PATENT DUCTUS ARTERIOSUS (PDA): PILOT STUDY

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Background: Pharmacological closure of PDA with SIBT protocol consists of three doses of ibuprofen (10-5-5 mg/kg) given 24h apart. Associated side effects during treatment have been reported. Therefore, treatment strategies deserve further evaluation.

Aims: To explore whether successful PDA closure with ibuprofen could be accomplished with a reduce number of doses.

Methods: Preterm babies older than 12 h of postnatal life with PDA ≥ 1.5 mm were randomized to EchoG (additional ibuprofen doses only if PDA ≥ 1.5 mm) or SIBT. Infants were serially scanned on days 1, 2, 3, 4, 7, 10 and 17 after being included in the study. The primary outcome was PDA reopening rate needing further treatment. Non-parametric tests were used for comparisons.

Results: 17 (EchoG) and 28 (SIBT) infants were enrolled [gestational age 28 (2.2) vs 29.6 (2.7) wk, $p < 0.05$; birth weight 1032 (280) vs 1237 (382) g, $p = 0.08$, respectively]. Patients received 2.8 (2.2) (EchoG) and 3.5 (1.3) (SIBT) ($p = 0.06$)

ibuprofen doses, at a mean age of 56.1 (17-168) h, without differences between groups. PDA was closed after the first ibuprofen dose in 44% of the study population. Rates for PDA reopening were 10.7% and 0% ($p = 0.28$), and rates for surgical closure 28.6% and 5.9% ($p = 0.12$), in EchoG and SIBT, respectively. Trends pointed towards lower incidence of necrotizing enterocolitis (7.1% and 17.6%, $p = 0.35$), urine output change (+8% and -30% $p = 0.05$), and serum creatinine rise (+5.9% and +34.2%, $p = 0.08$) in EchoG vs SIBT.

Conclusions: EchoG PDA treatment is a feasible strategy and avoids unnecessary ibuprofen doses.

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MECHANISM OF CARDIOVASCULAR DYSFUNCTION IN INFANTS OF ACTIVE SMOKERS

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Recently we showed that blood pressure and heart rate control is impaired in infants of mothers who smoke. This dysfunction resembles classic orthostatic intolerance and could indicate susceptibility to raised blood pressure later on. We analyzed whether this dysfunction reflects abnormal autonomic neural modulation of the heart, or the vasculature, or both.

Methods: Subjects: term-born infants of mothers who smoked during pregnancy (15 cigarettes/day; $n = 15$) and infants of non-smokers ($n = 27$). All were examined at least twice between 1 week and 18 months age. Supine blood pressure (BP; Finometer) and heart period (HP) were recorded for 4 min during quiet sleep. We used autoregressive cross-spectral analysis to compare age-related changes in low (LF; 0.04-0.15 Hz) and high frequency (HF, 0.15-0.4 Hz) power of HP and systolic BP (SBP). The gain of the heart-pressure relationship (α) was estimated by frequency domain analysis. We considered (i) LF_{HP} and LF_{SAP} power as markers of the sympathetic efferent modulation of the heart and blood vessels, respectively, and (ii) α as an index of baroreflex sensitivity.

Results: Absolute values of, and variance in HP / BP of infants of smokers and non-smokers were comparable. In tobacco-exposed infants LF_{SAP} (but not LF_{HP}) decreased and α increased with age.