

Abstract O-021 Table 1

	S+B(131)	S(134)	OR(95% CI)	P
B. W.(g)	882 ± 249	935 ± 283		0.81
G. A (wks)	26.5 ± 2.2	26.8 ± 2.2		0.91
Age study (hrs)	2.0 ± 1.5	1.8 ± 1.6		0.85
Death	17/131(13%)	22/134(16%)	0.76(0.38, 1.51)	0.54
BPD	38/131(29%)	67/134(50%)	0.49(0.29, 0.81)	0.008
BPD or death	55/131(42%)	89/134(66%)	0.45(0.27, 0.73)	0.001
BPD*	57/131(44%)	88/134(66%)	0.4(0.25, 0.66)	<0.001
BPD* or death	74/131(56%)	110/134(82%)	0.28(0.06, 0.49)	<0.001
mild*	19/131(15%)	21/134(16%)	0.91(0.47, 1.79)	0.79
mod*	26/131(20%)	41/134(31%)	0.56(0.32, 0.99)	0.048
severe*	12/131(9%)	26/134(19%)	0.42(0.20, 0.87)	0.017

**Background/aims** Intra-tracheal instillation of surfactant/budesonide significantly improves pulmonary status in animals. The aim is to investigate if this therapy would decrease the incidence of BPD or death.

**Methods and materials** This randomised controlled trial comprised 265 VLBW infants who had: 1) severe radiographic RDS, 2) requirement of IMV ( $\text{FIO}_2 \geq 0.5$ ) shortly after birth: 131 received surfactant (S) (100 mg/kg) and budesonide (B) (0.25 mg/kg) (S+B gr.), 134 received S only (100 mg/kg) (S gr.). The sample size was determined based on the hypothesis that 60% of infants in the S group and 40% in the S+B group would die or develop BPD defined at 34 weeks postm. age.

**Results** The S+B infant had lower tracheal aspirate interleukins 1, 6 and 8, lower OI, lower MAP in the early course of therapy, higher chance to wean to room air ( $p = 0.03$ ). No significant immediate and long term adverse effects were observed. \* NIH criteria

**Conclusions** In VLBW infants with severe RDS, administration of surfactant/budesonide significantly decreases the incidence of BPD and BPD or death with no apparent adverse side effects.

### O-022 TREATMENT OF VENTILATED PRETERM (PT) INFANTS WITH LATE SURFACTANT DOES NOT INCREASE SURVIVAL WITHOUT BRONCHOPULMONARY DYPLASIA (BPD) AT 36 WK PMA

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**Background/aims** The pathogenesis of BPD is multifactorial. In preterm infants  $\leq 28$  wk GA requiring ventilation at 7–14 days, >60% have surfactant dysfunction. Survival without BPD in these infants is <25%. Inhaled nitric oxide (iNO) may improve outcome in some infants (Schreiber, NEJM 2003, Ballard NEJM, 2006).

**Methods** Preterm infants  $\leq 28$  wk GA requiring mechanical ventilation at 7–14 days were enrolled in a RCT at 25 US centres. All infants received iNO and were randomised to receive surfactant (Infasurf) or sham instillation behind a screen every 1–2

days; maximum of 5 doses. Infants were evaluated by physiologic oxygen/flow reduction at 36 and 40 wk. Pulmonary outcome to 18 months is being collected.

**Results** Between January 2010 and September 2013, 511 of the planned 524 infants were enrolled. There was no difference between groups in mean BW ( $701 \pm 164$  grams), GA ( $25.2 \pm 1.2$  wk), percentage under 26 wk (70.6%), race, gender, severity of disease at enrollment or co-morbidities of prematurity. Survival without BPD was not different between treated vs. controls at 36 wk (31.3% vs.31.7%; relative benefit 0.98 (0.75, 1.28  $p = 0.89$ ) or 40 wk (58.7% vs. 54.1%; relative benefit 1.08 (0.92, 1.27  $p = 0.33$ ). Overall survival without BPD at 36wk in African Americans was better than whites (37.2% vs. 25.4% $p = 0.008$ ).

**Conclusions** Late treatment with surfactant in ventilated preterm infants did not improve survival without BPD at 36 or 40 weeks PMA. Overall better outcome in African-American infants may be due to a racial response to iNO. Pulmonary and neurodevelopmental assessment are on-going.

## Cardiac Failure in Congenital Diaphragmatic Hernia: Cause or Consequence?

### O-023 CANDIDATE BIOMARKERS OF PULMONARY HYPERTENSION AND CARDIAC DYSFUNCTION IN CONGENITAL DIAPHRAGMATIC HERNIA

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**Background and aims** In infants with congenital diaphragmatic hernia (CDH) plasma peptides which mediate, or are produced in response to pulmonary hypertension (PH) and cardiac dysfunction may be useful clinical biomarkers of disease severity. This study investigated correlation between candidate biomarkers and existing measures of oxygenation, PH, and cardiac function in CDH.

**Methods** Prospective observational study. Plasma samples were obtained for measurement of BNP, NTpro-BNP, VEGF-A, PLGF, and Tropinin1. Concomitant echocardiographic measures of pulmonary artery pressure (derived from TR jet velocity [PAP<sub>est</sub>]; and PDA flow ratio [PDA<sub>R:L</sub>]) and cardiac function (Tissue Doppler Imaging of systolic [S'] and diastolic [E'] velocities and tricuspid valve diastolic flow ratio [TV<sub>E:A</sub>]) were obtained. Oxygenation index was calculated OI.

Abstract O-023 Table 1 Correlations (r value) between candidate biomarkers and measures of oxygenation, PH and cardiovascular function

Candidate biomarker	PH measures				Septal TDI velocities	
	OI	PAP <sub>est</sub>	PDA <sub>R:L</sub>	TV <sub>E:A</sub>	IVS S' (systolic)	IVS E' (early diastolic)
BNP	<b>0.65</b>	0.15	<b>0.43</b>	0.22	0.09	-0.02
NTproBNP	<b>0.62</b>	<b>0.43</b>	<b>0.39</b>	0.04	0.08	-0.12
Troponin 1	<0.01	<0.01	<b>0.26</b>	<b>-0.30</b>	<b>0.26</b>	0.04
VEGF-A	<b>0.64</b>	0.18	0.10	<b>-0.30</b>	-0.11	-0.18
PLGF	<b>-0.34</b>	<b>-0.47</b>	<b>-0.48</b>	<b>0.27</b>	-0.01	<b>0.25</b>

Numbers represent r values, significant correlations in bold ( $p < 0.05$ )