

**Abstract O-079 Table 1** Demographics of Study Population (n = 30)

GA at time of study	31.7 (30.7, 33.5)
Postnatal age in days	43 (23.5, 53.5)
Weight in kg at time of study	1.4 (1.1, 1.6)
Insignificant PDA/PFO	6/8
Surfactant (given at birth / 2nd dose)	25/7
Ventilated before study	27
Bubble CPAP /ventilator CPAP	24/6
CPAP requirement (5/6/7 cm H <sub>2</sub> O)	13/7/10
O <sub>2</sub> requirement (%)	30 (25,30)
Capillary gas: pH	7.35 (7.34, 7.39)
Capillary gas: pCO <sub>2</sub> (mmHg)	53 (47, 55)
Capillary gas: HCO <sub>3</sub> <sup>-</sup> (mmol/l)	29 (25, 34)
Capillary gas: base excess	2.6 (-0.4, 4.8)
Respiratory support at 36 weeks GA	12
O <sub>2</sub> requirement at 36 weeks GA	25

Values given as median (IQR) or absolute numbers.

**Abstract O-079 Table 2** Haemodynamic parameters on three nCPAP levels

	4 cm H <sub>2</sub> O	6 cm H <sub>2</sub> O	8 cm H <sub>2</sub> O	p
RVO (ml/kg/min)	411 ± 110	418 ± 94	414 ± 93	0.98
LVO (ml/kg/min)	407 ± 121	405 ± 140	403 ± 123	0.96
SVC flow (ml/kg/min)	157 ± 53	149 ± 4	155 ± 42	0.58
LPA flow (ml/kg/min)	136 ± 57	142 ± 61	136 ± 53	0.45
TAPSE (mm)	7.9 ± 1.8	8.1 ± 1.5	8.0 ± 1.2	0.58
BP systolic (mmHg)	73 ± 9	71 ± 9	74 ± 13	0.34
BP mean (mmHg)	52 ± 9	53 ± 8	55 ± 11	0.28
BP diastolic (mmHg)	40 ± 1	43 ± 1	46 ± 12	0.11
HR (beats/min)	163 ± 13	164 ± 13	162 ± 13	0.62

**Results** Thirty infants with a median (IQR) gestational age of 25.9 (25.6–26.8) weeks and a birth weight of 0.78 (0.66–0.94) kg were studied at a median age of 43 (24–53) days. There were no significant differences in any cardiovascular parameters at different levels of nCPAP.

**Discussion** We conclude that nCPAP levels between 4 and 8 cm H<sub>2</sub>O did not have an effect on CO in our study population of stable preterm infants with evolving chronic lung disease.

## Nosocomial Infections in the ICU

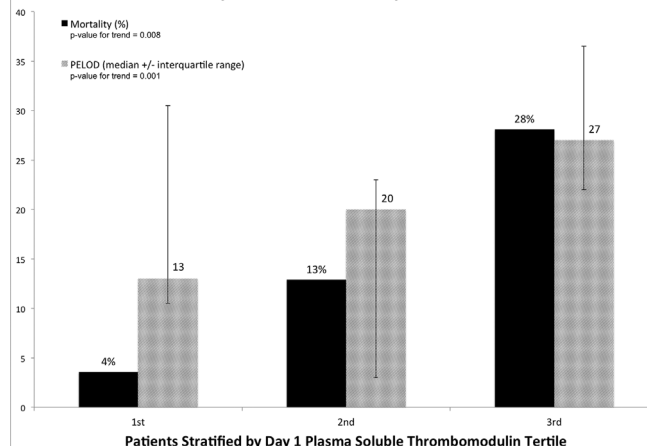
### O-080 ELEVATED SOLUBLE THROMBOMODULIN IS ASSOCIATED WITH INCREASED MORTALITY AMONG CHILDREN WITH INDIRECT ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

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10.1136/archdischild-2014-307384.147

**Abstract O-080 Table 1** Cohort characteristics comparing Direct and Indirect Injury-related ARDS

	Direct (n=133)	Indirect (n=91)
Age (y)	6.2	7.5
Male (%)	58	51
White (%)	62	66
Pneumonia	124	0
Aspiration	9	0
Sepsis	0	46
Trauma	0	13
Multiple Transfusions and others	0	32
Vasopressor Use (p=0.001)	33	55
PRISM II (SD)	16 (10)	13 (9)
PELOD (SD)	19 (12)	21 (14)
Mortality (%)	12	15

**ARDS Mortality and PELOD Score by Tertile of sTM Levels****Abstract O-080 Figure 1** ARDS Mortality and PELOD Score by Tertile of sTM Levels

**Background** Inflammation and endothelial damage accelerate cleavage of endothelium bound Thrombomodulin (TM). Elevated soluble TM (sTM) in plasma is associated with adverse outcomes in sepsis and DIC in adults, but this has not been studied among children with ARDS.

**Objective** Test the relationship of plasma sTM with clinical outcomes in paediatric ARDS.

**Design/methods** We measured sTM in plasma collected within 24 h of onset of ARDS (diagnosed by Berlin criteria) in an ongoing multi-centre observational cohort. We used non-parametric Mann-Whitney and trend tests, and regression models.

**Results** Baseline characteristics of study population are shown in Table. Among children with indirect lung injury, mean sTM levels were higher in non-survivors [241 ng/mL (102–134)] compared to survivors [118 ng/mL (107–374)] (p = 0.004). Mortality and Paediatric Logistic Organ Dysfunction (PELOD) score increased stepwise by tertile of plasma sTM (figure). On logistic regression, the odds of death increased by 4.5 for every log increase in plasma sTM and the association was independent of age, race, gender and severity of illness. No such relationship existed for direct ARDS.

**Conclusions** Higher plasma sTM is associated with increased mortality and organ failure in children with indirect ARDS. This supports the importance of endothelial injury and TM in pathobiology of indirect ARDS and suggests that early elevation in plasma sTM is an independent risk factor for mortality.