Results

Abstract 169 Table 1

Hospital mortality of infants by SpO2 target overall

	SpO2 85–89%	SpO2 91–95%	Risk Ratio (95% CI)	p value
Hospital mortality	235/1220 (19.3%)	202/1217 (16.6%)	1.16 (0.98–1.38)	0.09

Abstract 169 Table 2

Hospital mortality of infants by SpO2 target, by old vs new software

	Old software				New software			
	SpO2 85–89%	SpO2 91–95%	Risk Ratio (95% CI)	p value	SpO2 85–89%	SpO2 91–95%	Risk Ratio (95% CI)	p value
Hospital mortality*	98/629 (15.6%)	109/630 (17.3%)	0.90 (0.70–1.16)	0.41	137/591 (23.2%)	93/587 (15.8%)	1.46 (1.15–1.85)	0.0015**

There was no significant mortality difference between SpO_2 targets overall. There was significant heterogeneity between old and new software on mortality (test for interaction p=0.006).* Using new software, targeting 91–95% increased hospital survival by 7.4% (from 76.8% to 84.2%) versus targeting 85–89% (p=0.0015).**

Conclusions Pending the primary outcome of disability free survival at 2 years it appears wise not to target 85–89%.

References

1. Askie. BMC Pediatrics 2011; 11:6.
2. Stenson. NEJM 2011; 364:1680–2.
3. Johnston. Arch Dis Child Fetal Neonatal Ed. 2011; 96:429–33.

170 MATERNAL ALLOPURINOL ADMINISTRATION DURING TERM LABOR IS NEUROPROTECTIVE IN CASE OF FETAL HYPOXIA; A MULTICENTER RANDOMIZED PLACEBO CONTROLLED TRIAL

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Background Hypoxic-ischemic encephalopathy due to perinatal hypoxia-induced free radical formation is an important cause of long-term neurodevelopmental disabilities. Allopurinol reduces the formation of free radicals, which potentially limits hypoxia-induced reperfusion damage. With this trial we aimed to assess whether maternal allopurinol treatment during fetal hypoxia would reduce the release of brain-tissue-specific biomarkers associated with neonatal brain damage.

Methods We performed a randomized double blind placebo controlled multicenter trial (NCT00189007) studying laboring women at term with imminent fetal hypoxia. Fetal distress was suspected in case of an abnormal fetal heart rate trace, ST-wave abnormalities on fetal ECG or fetal scalp pH<7.20. Women were allocated to receive allopurinol 500 mg IV or placebo immediately prior to delivery. Endpoints were S100B and neuroketal in cord blood, which are brain-tissue-specific biomarkers for brain damage. Because S100B followed a non-normal distribution, we used a poisson regression model with associated RR (95%CI). For neuroketal we report geometric mean differences.

Results We randomized 222 women to allopurinol (n=111) or placebo (n=111). S100B was significantly lower in the allopurinol-group (median 43.4; IQR 20.2–71.5) compared to the placebo-group (median 54.9; IQR 26.8–94.7), RR 0.91 (95%CI 0.88–0.94). Neuroketal did not significantly differ between groups, geometric mean difference –7.57 (95%CI –15.6; 3.57).

Post-hoc analysis showed a marked gender difference in treatment effect in favor of girls for S100B (RR 0.63 (95%CI 0.59–0.68)) and neuroketal (geometric mean difference –16.5 (95%CI –24.6; -1.83)).

Conclusion Maternal treatment with allopurinol during fetal hypoxia reduces damage to neuronal cells as indicated by brain-tissue-specific chemical biomarkers.

171 NIPPV DOES NOT REDUCE BRONCHOPULMONARY DYSPLASIA (BPD) OR DEATH IN EXTREMELY LOW BIRTH WEIGHT INFANTS - A RANDOMISED TRIAL

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Background Standard care of preterm infants includes nCPAP & NIPPV. We compared rates of BPD or death in a randomised trial of NIPPV or nCPAP.

Methods Eligible infants were preterm, birth weight < 1000g; requiring either (i) non-invasive respiratory support within first seven days, "no intubation/early extubation group", or (ii) were < 28 days at first extubation - "prior intubation". Central block randomisation to NIPPV or nCPAP was conducted via the web. Primary outcome was a composite of death (prior to 36 weeks' gestational age [GA]) or BPD at 36 weeks' GA: defined as requiring ventilation; $FiO_2 > 30\%$; or positive oxygen reduction test (ORT). Sample size 1000 (β 80%; 2-tailed α 5%) to demonstrate 20% reduction in primary outcome.

Results 36 international sites enrolled 1007 infants. Observed rates of BPD or death were similar in the two groups. BPD outcome,