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#### MASIMO SIGNAL EXTRATION TECHNOLOGY (SET) DOES NOT OFFER AN ADVANTAGE DURING NEONATAL TRANSPORT

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Introduction: Neonatal transport presents challenges for physiological monitoring. Vibration and acceleration can cause motion artefact. Signal loss or inaccuracy could lead to unnecessary interventions or delay in diagnosis. Masimo signal extraction technology (SET) is a new type of oxygen saturation monitoring that has been shown to reduce data inaccuracies due to motion artefact in some environments.

Aim: To compare the reliability of monitoring using Masimo SET versus a standard saturation monitor (Nellcor) during neonatal transport.

Method: All infants transported by the South East Scotland Neonatal Transport Service who required monitoring were eligible for inclusion. A saturation probe from each monitor was attached to opposite feet. Data from both monitors and ECG heart rate were downloaded onto laptop during transport. ECG artefact was removed. Saturation monitor artefact was then defined to be present whenever the saturation derived HR differed from the ECG HR by more than 10 bpm. The proportion of time that each saturation monitor was affected by artefact was compared by paired T-test. HR from each saturation monitor was compared with ECG HR and precision and bias were calculated. Preliminary studies indicated that 20 transports would give 85% power to detect a 10% difference in artefact between the monitors.

Results: 23 studies were carried out on 20 infants. Mean gestation was 35.5 weeks. Mean weight was 2115g. 8 transports were urgent, 15 were non-urgent. Saturation artefact proportions for the Masimo SET and Nellcor monitors were 1.13% and 1.54% (p=0.714). Mean oxygen saturations were 91.0% and 91.1% (p=0.873). The mean HR bias for the Masimo and Nellcor monitors were -0.1177 and -0.4357 BPM (p=0.011) and the mean precisions were 3.099 and 2.334 BPM (p>0.001).

Conclusions: Data loss due to artefact was a relatively uncommon phenomenon during neonatal transport with either monitor. Masimo SET monitoring offered no clear advantage.

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## DOES THE METHOD USED FOR MONITORING AFFECT STABILITY OF OXYGENATION IN PRETERM INFANTS?

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Background: Oxygen saturation (SpO2) and transcutaneous oxygen tension (TcPO2) monitors are used widely to monitor oxygen levels in preterm neonates. Response to these monitors by changing fraction of inspired oxygen (FiO2), influences oxygenation levels. Oxygen excess and oxygen variability have been linked to increased risk

Aim: To determine whether the method of oxygenation monitoring influences oxygen variability and/or the

Aim: To determine whether the method of oxygenation monitoring influences oxygen variability and/or the absolute time spent with hyper- and hypoxia in a population of oxygen dependent preterm infants.

Methods: 19 infants requiring supplemental oxygen were studied. Consent was obtained. Infants were randomised to have their FiO2 guided by one monitoring method for 3 hours after which they switched to the other monitoring method for 3 hours. During the TePO2 monitoring period fiFO2 was adjusted to maintain the infants TePO2 between 6.0–9.0 kPa. During the SpO2 monitoring period the FiO2 was adjusted to maintain the infants SpO2 between 8.0–948. For each of the two periods alarms were enabled for the monitor under investigation, and readings from the other monitor although continuously recorded were not displayed. TePO2 and SpO2 readings were downloaded every second. For each infant and for each monitoring method, the SpO2 variability (SD of SpO2 readings), mean SpO2, percentage of time saturated above 94% and below 86% were calculated. Paired T-tests were used to compare monitoring methods.

Results: 13 infants were ventilated, 6 on CPAP. Mean (SD) Weight 1003 (416) gms, Mean (SD) CGA 27.3 (2.52) weeks, Mean (SD) FiO2 0.34 (0.123).

	TcPO <sub>2</sub>	SpO <sub>2</sub>	Significance
Mean SpO2 variability	2.99	4.00	P=0.023
Mean SpO2	92.1	91.0	P=0.095
Mean % of time saturated <86%	9.40	8.12	P=0.643
Mean % of time saturated >94%	30.8	16.6	P=0.044

Conclusion: Our results suggest that using the above target limits to control oxygenation, the use of SpO2 moritors lead to more oxygen variability. The method of oxygenation monitoring influences infant stability, which may impact on risk of adverse outcome.

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## NEONATAL HYDROCORTISONE TREATMENT RELATED TO 1H-MRS OF THE HIPPOCAMPUS AND SHORT-TERM MEMORY AT SCHOOL AGE IN PRETERM BORN CHILDREN.

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Introduction: Animal studies have shown that corticosteroids (dexamethasone) cause neuronal loss in the hippocampus and deficits in short term memory. Proton magnetic resonance spectroscopy measures

the impocampus and denotes in short term memory. Proton langitude resonance spectroscopy measures brain metabolites in vivo and gives an indication of neuronal integrity.

Aim of the study: To investigate whether prolonged administration of hydrocortisone during the neonatal period for bronchopulmonary dysplasia (BPD) in preterm born children changes the metabolism in the hippocampus measured at school age. Secondly, to investigate whether hippocampal metabolism and short-term memory and neurodevelopmental outcome are related.

Patients and methods: 37 Preterm born children (< 32 weeks (range 25.0–33.0) and/or a birth weight < 1500 grams) underwent proton spectroscopy of the hippocampus at school age. 18 Children were treated with hydrocortisone for BPD (starting dose 5 mg/kg/d tapered over a minimum period of 22 days, median duration 28 days) and 19 never received corticosteroids. N-acetyl aspartate/ Choline + (phospho)Creatine (NAA/(Cho+Cr)) ratios were determined. A 15 words recall memory test and IQ measurement were obtained on the same day.

Results: Hydrocortisone treated children were younger (27.9 vs 30.6 weeks, p<0.0001), lighter (1091)

vs 1379 grams, p=0.017) and sicker (artificial ventilation 100% vs 68%, p=0.02, need for inotropes 44% vs 16%, p=0.057) than their nonsteroid treated counterparts. Mean NAA/(Cho+Cr) ratios in the hippocampus were not significantly different in the hydrocortisone group compared with the nonsteroid group (0.67 vs 0.63, p=0.20). Performance on the 15 words memory test (6.61 vs 6.21, p=0.57) and IQ (97 vs 95, p=0.67) were similar in the two groups. There was no relation between NAA/(Cho+Cr) ratios and memory nor between NAA/(Cho+Cr) ratios and IQ.

Conclusion: Hydrocortisone administered in the neonatal period for BPD does not change metabolism

of the hippocampus as measured by proton MR spectroscopy nor outcome as tested by short-term memory and IQ. Hydrocortisone used in the mentioned dose appears safe for preterm neonates with BPD.

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#### INTRAVENTRICULAR HAEMORRHAGE ON THE LEFT SIDE OF THE BRAIN INDUCES LEFT-HANDEDNESS

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**Background:** Prematurely born children are more often left-handed than their term born peers. It is unknown if this excess left-handedness, a marker for the extent of cerebral lateralisation, is caused

Methods: In a cohort of 221 preterm born infants (gestational age <= 32 weeks and/or birth weight <= 1500 grams), brains were serially examined in the neonatal period using cranial ultrasound (US) to detect intraventricular haemorrhage (IVH), periventricular leukomalacia (PVL) and other abnormalities. At age 7 to 10 years, magnetic resonance imaging (MRI) was done, and hand preference and current IQ were measured. MRI and US were classified as normal, mildly abnormal, or severely abnormal, blinded to handedness data.

Results: Children with severely abnormal US (IVH grade III/IV, cystic PVL grade II/III, thalamic lesion, focal infarction or haemorrhage at the level of the convexity) had an increased chance to become left-handed: odds ratio (OR) severely abnormal US versus normal US 4.1, 95% CI 1.6 to 10.0, p=0.003. This was confirmed by MRI scans at school age: OR severely abnormal MRI versus normal MRI 4.0, 95% CI 1.5 to 10.7, p=0.005. Findings were attributable to IVH. Left-sided IVH showed a higher chance for left-handedness (OR left-sided IVH versus no IVH 4.4, 95% CI 1.7 to 11.3, p=0.002), whereas right-sided IVH did not. Neonates with left-sided mild IVH (grade I and II) still showed an increased chance for left-handedness: OR mild left-sided IVH versus no IVH 4.0, 95% CI 1.5 to 10.9, p=0.007. PVL was not related to left-handedness.

Conclusion: Our findings strongly indicate that even a small intraventricular haemorrhage affecting the left side of the brain induces left-handedness. This is likely related to the role of the subependymal germinal matrix in the developing brain.

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## HEARING ABNORMALITIES IN CHILDREN WITH FABRY DISEASE: DATA FROM FOS - THE FABRY OUTCOME SURVEY

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Background: Fabry disease is an X-linked glycosphingolipid storage disorder due to a deficiency of the enzyme alpha-galactosidase A. Accumulation of substrate results in a progressive and lifethreatening multisystemic disease. Early clinical manifestations include pain and gastrointestinal symptoms. Sensorineural hearing loss and vertigo are well-recognized features of the disorder, occurring in approximately 50% of adults with Fabry disease. We have investigated the audiological symptoms of Fabry disease in children using pure-tone and impedance audiometry.

Methods: Symptom history was obtained using a standardized questionnaire from FOS - the Fabry Outcome Survey. Hearing was measured using pure-tone audiometry of both air (250-8000 Hz) and bone (500-4000 Hz) conduction thresholds, with masking as appropriate. Impedance audiometry was performed to exclude effusions.

**Results:** Symptom questionnaires were completed by 46 boys and 52 girls, with a median age of 12 (range, 1–18) and 14 (range, 2–18) years, respectively. A total of 15% of both boys and girls complained of dizziness. Tinnitus was reported in 22% of boys and 35% of girls. Generally, the children with hearing loss were found to have different degrees of impairment in each ear. Air conduction thresholds in the ear with the most significant hearing loss were abnormal (> 20 dB) in 20-40% of boys and 10-25% of girls. Approximately 79% of children with ear-related symptoms also complained of neurological pain and gastrointestinal signs and symptoms

Conclusions: Audiological symptoms, especially tinnitus, and abnormal hearing thresholds are not uncommon in children with Fabry Disease. A large proportion of children with audiological symptoms also suffered from pain and gastrointestinal manifestations. A cluster of acroparaesthesiae, gastrointestinal manifestations and hearing abnormalities are therefore suggestive of Fabry disease.

## DO CYTOKINE OR CRP MEASURES ASSIST IN THE DIAGNOSIS OF NE-CROTISING ENTEROCOLITIS: A SYSTEMATIC REVIEW

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Objective: To conduct a systematic review of the use of cytokines or CRP in the diagnosis of necrotizing enterocolitis (NEC) in neonates.

Methods: MEDLINE was searched (keywords: NEC, pseudomembranous enterocolitis, diagnosis, newborn, and cytokine) from 1985 through 2005. Inclusion criteria were: (i) pre-term infants younger than 90 days (ii) proven NEC defined as intramural gas on Xray or surgical findings. The MEDLINE search was repeated (using keyword CRP instead of cytokine). Data were extracted to calculate specificity and sensitivity. Two independent reviewers selected and reviewed the articles.

Results: The search identified 22 potential studies, of which 5 allowed retrieval. The search identified 22 potential studies, of which 5 allowed retrieval.

Marker Ser	sitivity Spe	cificity	Likelih	nood ratio
IL-8	(Moore 1999)	.60	1.0	-
IL-6	(Ahmed 1998)	.85	.56	1.93
IL-11	(McLoy 2002)	.30	.79	1.43
TNF-alpha	(Caplan 1990)	0.55	1.0	-
CRP	(Isaacs 1987)	0.69	0.71	2.38

Conclusion: There are few studies which have examined the use of either cytokines or CRP as diagnostic tools for NEC. The possible association between NEC and cytokines is suggested in 22 studies. However, there is a lack of sufficient studies in which likelihood ratios could be calculated (n=4). Similarly, there is an insufficient number of studies which examine the use of CRP as a diagnostic tool for NEC. With the emergence of cytokines and CRP research in the last decade, studies that examine the link between cytokines and NEC should be further explored in larger cohorts. References Moore, A. 1999; Pediatr Res; 54: 114A. Ahmed, T. 1998; Pediatr Res 43:163A. McLoy, M.P. 2002. Pediatric Research. 51(6):756 Caplan, M.S. 1990 J Pediatrica 116:960 Isaacs, D. 1987 Acta