## **European Society for Paediatric Research Abstracts for Oral Presentations**

 $\label{eq:linear} \begin{array}{c} 1 \\ \text{VIRUSES IN ACUTE OTITIS MEDIA} \\ \text{Hannu Sarkkinen*, MD^1, Olli Ruuskanen, MD^2, } \\ \text{Olli Meurman*, MD^1, and Jussi Eskola*^3, MD} \\ \text{Departments of Virology}^1 \text{ and Microbiology}^3, \text{University} \\ \text{of Turku, and Department of Pediatrics}^2, \text{Turku University Hospital, SF-20520 Turku, FINLAND} \end{array}$ VIRUSES IN ACUTE OTITIS MEDIA

There is epidemiologic evidence that otitis media is associated with viral respiratory infections but mini-mal information exists to prove such an etiology. We have tested antigens of respiratory viruses by immuno-assay in the middle ear fluid and nasopharyngeal se-cretions of 137 children with acute otitis media (Sarkkinen et al, submitted). We found that 1) an epi-demic of respiratory executial virus (PSV) infection demic of respiratory syncytial virus (RSV) infection caused a significant increase in the occurrence of acute otitis media, 2) 15% of the children had RSV in the middle ear fluid and in 7% RSV was the sole pathogen found. In addition, an adenovirus was found in middle ear fluid of 3% of the children, 3) bacteriological findings in otitis media related and unrelated to virus (RSV) infection were similar. These findings confirm and extend the results obtained earlier in this laboratory and indicate that otitis media is associated with virus infection and that in the early stages of infection the disease may be due solely to virus infection. Thus, at least during respiratory virus epidemic, treatment failures e.g. fever and earache unresponsive to antimicrobial therapy, may be due to a viral etiology of acute otitis media.

 $2~{\rm effect}$  of intravenous indomethacin on plasma  $6{\rm kpgf}_{1{\rm sc}}$  and  ${\rm txB}_2$  concentrations in neonates with patent ductus arteriosus and pulmonary disease. A WILKINSON, I ALEXANDER\*, M MITCHELL\*, John Radcliffe Hospital, Oxford, UK and Green Center, Dallas, USA.

The immediate effect of 0.2mg/kg indomethacin on the plasma concentration of 6-keto-prostaglandin  $F_{l^{\alpha}}$  $(6-KF_{1\alpha})$  and thromboxane  $(TxB_2)$  was studied in 10 preterm babies with sympatomatic patent ductus arteriosus (PDA) in the first week of life.

Gestational age ranged from 25-31 weeks and birthweight 660-1295g. The concentration of  $6-KF_{1\alpha}$  before indomethacin, range 255-3487pg/ml was much higher than previously reported in well preterm babies. This decreased after indomethacin in 6 babies. In 3 there was a small initial increase but 6 hours later the concentration was lower than pre-indomethacin. TxB,, also higher than in well preterm babies, range 24724991pg/ml, increased in all who continued to need assisted ventilation (5) but decreased in all those who could be extubated within 72 hours of treatment (5). These results suggest that co-existing lung disease in babies with PDA is associated with a paradoxical effect of indomethacin. This may be responsible for the phenomenon of "transient closure" which, in a clinical study of 30 babies was significantly related to the presence or absence of pulmonary disease (p<0.005). Indomethacin is unlikely to lead to permament closure of PDA when there is co-existing pulmonary disease which may alter prostaglandin metabolism.

RESPIRATORY FAILURE AND THE MATURATION OF THE 3 AMBIENT THERMAL STIMULUS TO BREATHING DURING SLEEP IN LAMBS D C Andrews, J C Wollner,

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Breathing frequency (f) in lambs falls with increasing They rely on powerful airway reflexes postnatal age. to maintain lung volume and an effective respiratory rhythm during sleep between 2-6 weeks of age. Small changes in ambient temperature (Ta) were observed to modulate f by effecting expiratory time (Te) and thus the dependancy on intra-airway vagal reflexes. Irreversible respiratory failure could thus occur.

The age relationship of Ta on f during sleep was therefore studied in detail in 9 chronically instrumented self-reared lambs at three Tas  $10^\circ$ ,  $15^\circ$ ,  $20^\circ$ C (in some  $5^\circ$ ,  $25^\circ$  and  $30^\circ$ ) at 2, 6, 12 and 28 days of age.

f decreased significantly with postnatal age (39.7 (3D) to 27.5 (28D) p > .001) regardless of Ta (10-20°C). No change in f occurred between 10 and 20°C at 2 or 6 days of age but a significant fall in f occurred between 20 and  $10^{\circ}$ C at 12 days (p < .001) and 28 days (p < .01). Shivering was observed in lambs < 6 days at  $10^{\circ}$ C and f increased markedly between 25 and  $30^{\circ}$ C.

Thus, f is sustained for the first 2 weeks by metabolic demands and then, as thermal efficiency improves, by vagal airway reflexes modulated by Ta before the chemical drive to breathing fully matures. A postneonatal vulnerability to sleep related respiratory failure perhaps analogues to SIDS is suggested by these results.

4 Selective increase of CSF lactate and pyruvate, and persistent normalisation after temporary

J.JAEKEN, P.CASAER\*, L.CORBEEL, M.RAES\*, G.MASSA\*, P.DEGRAEUWE\*, E.EGGERMONT\*. Dept. Paediatrics, Leuven. A boy was admitted at the age of 5 months with convulsions, irritability, dullness and hypotonia. Develop-mental age was about 2 months. He also showed hyperventilation from birth on. His parents were unrelated. Ges-tational age was 37 wks and birthweight 2.5kg. There was no peripartal asphyxia. Antiepileptic therapy could not completely control the convulsions. Plasma alanine and blood pyruvate and lactate levels were normal, also after glucose loading, but CSF alanine (48  $\mu$ mol/L;nl $\langle$  30), pyruvate (0.9mmol/L;nl $\langle$  0,15) and lactate (10mmol/L; nl $\langle$  1.5) were markedly increased. Amino acid and organic acid analysis was otherwise normal. Protidorhachia, funduscopy, EMG, nerve conduction velocity, EEG, brain evoked responses and CAT-scan of the brain were normal. Peroral thiamine (3x100mg/day) caused a striking clini-cal improvement and complete biochemical normalisation within a few days. Antiepileptic therapy could be dis-continued. Thiamine therapy was stopped after two months. Symptomatology has not returned now six months after discontinuation and psychomotor development is satisfac-tory. Conclusion : this patient suffered from a defect tory. <u>Lonclusion</u>: this patient suffered from a detect of pyruvate oxidation (probably pyruvate dehydrogena-se) limited to brain. Temporary thiamine therapy appa-rently cured this defect that is tentatively attribu-ted to "immaturity". The finding of normal CSF thiamine levels before therapy excludes thiamine deficiency.