0031-3998/84/0795-0811$02.00/0
PEDIATRIC RESEARCH

Vol. 18, No. 8, 1984
Printed in U.S.A.

European Society for Paediatric Research
Abstracts for Oral Presentations

1 VIRUSES IN ACUTE OTITIS MEDIA

Hannu Sarkkinen*, MD[1], Olli Ruuskanen, MD[2], Olli Meurman*, MD[1], and Jussi Eskola*[3], MD
Departments of Virology[1] and Microbiology[3], University of Turku, and Department of Pediatrics[2], Turku University Hospital, SF-20520 Turku, FINLAND

There is epidemiologic evidence that otitis media is associated with viral respiratory infections but minimal information exists to prove such an etiology. We have tested antigens of respiratory viruses by immunoassay in the middle ear fluid and nasopharyngeal secretions of 137 children with acute otitis media (Sarkkinen et al, submitted). We found that 1) an epidemic 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 EFFECT OF INTRAVENOUS INDOMETHACIN ON PLASMA 6KPGF$_{1\alpha}$ and 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$_{1\alpha}$ (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$_2$, also higher than in well preterm babies, range 247-4991pg/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.

3 RESPIRATORY FAILURE AND THE MATURATION OF THE AMBIENT THERMAL STIMULUS TO BREATHING DURING SLEEP IN LAMBS

D C Andrews, J C Wollner, L Fedorko, P Johnson, Nuffield Department of Obstetrics & Gynaecology, University of Oxford, U K

Breathing frequency (f) in lambs falls with increasing postnatal age. They rely on powerful airway reflexes 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°, 15°, 20°C (in some 5°, 25° and 30°) 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°C at 12 days ($p < .001$) and 28 days ($p < .01$). Shivering was observed in lambs < 6 days at 10°C and f increased markedly between 25 and 30°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 thiamine therapy.

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. Developmental age was about 2 months. He also showed hyperventilation from birth on. His parents were unrelated. Gestational 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 µmol/L;nl< 30), pyruvate (0.9mmol/L;nl< 0,15) and lactate (10mmol/L; nl< 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 clinical improvement and complete biochemical normalisation within a few days. Antiepileptic therapy could be discontinued. Thiamine therapy was stopped after two months. Symptomatology has not returned now six months after discontinuation and psychomotor development is satisfactory. Conclusion : this patient suffered from a defect of pyruvate oxidation (probably pyruvate dehydrogenase) limited to brain. Temporary thiamine therapy apparently cured this defect that is tentatively attributed to "immaturity". The finding of normal CSF thiamine levels before therapy excludes thiamine deficiency.