Neonatal Society

Meeting held on 8 November 1979 at the Institute of Child Health, London

Blood pressure variations in the healthy neonate. 
F M Cowan (introduced by K W Cross). Neonatal Research Group, Department of Physiology, The London Hospital Medical College.

To date, noninvasive estimations of neonatal blood pressure (BP) have often necessitated disturbance of the child and have generally been limited to systolic pressure. The Dinamap neonatal BP monitor 847 gives long-term noninvasive monitoring without disturbing the child, who remains clothed and comfortable in his cot. It measures the mean arterial pressure (MAP), and the pulse rate (PR), and deduces values for the systolic and diastolic pressures.

10 normal term babies were studied to see the effect of different sleep states on their BPs. All were 3 days old and had been fed at least 2 hours before the measurements. Sleep state was monitored using a bi-parietal electroencephalogram. The results confirmed previous findings that the MAP, PR, systolic and diastolic pressures were higher in active than in quiet sleep (P<0.01, P<0.01, P<0.05, P<0.05 respectively), and also that the Dinamap is sufficiently sensitive to detect small physiological variations. A study was undertaken to record the effects of breast feeding on BP. In each baby the BP, MAP, and PR rose considerably during the feed but within minutes of its end had returned to resting levels, and any variations during the next hour were explicable by changes in sleep state or body movement.

Positive pressure ventilation and cranial volume in newborn infants. D W A Milligan (introduced by K W Cross). Research Institute, Hospital for Sick Children, Toronto, Canada.

It has been suggested that treatment of sick preterm infants with positive airways pressure might predispose to intracranial haemorrhage.1 In order to establish whether a rise in cranial volume takes place when positive pressure is applied to the airway, cranial volume, pleural pressure, and airway pressure were measured simultaneously in 15 infants requiring ventilatory assistance. Cranial volume changes were calculated from changes in occipitofrontal circumference measured with a mercury-in-Silastic strain gauge. The study had ethical approval.

The increase in cranial volume which occurred with any application of positive pressure to the airways was found to depend on the proportion of that pressure which was transmitted to the pleural space, the relationship between these two being approximately linear. Increases of cranial volume of 0.1% or greater occurred when pleural pressure was +4 cmH2O or more or reached 20% of the pressure applied at the airway. The mean maximum increase in cranial volume was 0.025% for infants with hyaline membrane disease and 0.23% for those with 'normal' lungs (P<0.001 on 2-tailed t test).

The findings emphasise the need for more critical monitoring during periods of rapidly changing lung compliance.

References

Prognosis for very low birthweight infants treated by mask ventilation. A P Lipscomb, A Stewart, and E O R Reynolds. Department of Paediatrics, University College Hospital and Medical School, London.

In 1974 we described a method for mechanically ventilating very low birthweight infants through a face mask, lightly held in place with tubular elastic netting.1 Pressure preset ventilators were used and the technique was used only when peak airways pressure no higher than about 12 cmH2O was needed. Subsequently Pape et al.2 reported high incidences of intracranial haemorrhage (particularly into the cerebellum) in infants who died after mask ventilation, and of neurological sequelae in survivors. These they attributed to compression of the head.

We therefore examined the outcome for infants weighing <1500 g treated with mask ventilation at this hospital. 274 infants weighing <1500 g were admitted between 1975 and 1977. Necropsies were done on 77 of the 99 who died. No excess of cerebral haemorrhages was found in mask-ventilated infants. 82 of the 175 survivors were mechanically ventilated. At follow-up, aged 15 to 42 months, 8 of the 41...
Infants ventilated only through endotracheal tubes and 8 of the 41 infants whose treatment included mask ventilation were found to have major handicaps. We therefore found no evidence that mask ventilation per se caused cerebral damage in our population of infants. This might have been because compression of the head was kept to a minimum.

References


Transcutaneous oxygen tension measurements during sleep in the newborn baby and infant. E A Carse, D J Henderson-Smart, P Johnson, P Whyte, and A R Wilkinson. Department of Paediatrics, and Nuffield Institute for Medical Research, University of Oxford.

We made sequential 6-hour polygraphic studies of 14 healthy babies at 3–7 days and at 1, 3, and 6 months of age. Five babies were the siblings of infants who had died from sudden infant death syndrome (SIDS). The heart and respiratory rates and transcutaneous oxygen levels for each minute during sleep in the two groups were compared.

Heart rate was higher in active than in quiet sleep in both groups at all ages. This difference was significant (P<0.05) in the normal babies at 1 week, 1 month, and 3 months, and in the siblings of SIDS at 1 and 3 months. There was no significant difference in respiratory rate and transcutaneous oxygen level in active or quiet sleep in either group.

Between 1 week and 1 month heart rates rose in both groups of babies (P<0.05) but fell again at 3 and 6 months. In the normal babies respiratory rate did not change with age but in the siblings of SIDS it was higher at 1 week, and at 1 month it was significantly higher than in the normal group (P<0.05). It then fell progressively at 3 and 6 months.

Transcutaneous oxygen level rose in the normal babies between 1 week and 1 month and in siblings of SIDS between 1 week and 3 months (P<0.05).

There was no significant difference in transcutaneous oxygen level between groups at 1 month, when the siblings of SIDS had raised respiratory rates. This does not support the hypothesis that a higher respiratory rate in siblings of SIDS in the early months of life is due to chronic mild hypoxia.

Effect of severe physical illness on the behaviour of very low birthweight infants. A Whitelaw, K Minde, and J Brown. Division of Perinatal Medicine, and Department of Psychiatry, Hospital for Sick Children, Toronto, Canada.

10 very low birthweight infants (<1500 g) with serious medical complications, many requiring ventilation, were observed during their severe illnesses, 10 different behavioural patterns being continuously recorded on a multichannel event recorder. These 10 sick infants were paired with 10 infants of approximately the same birthweights, gestational ages, and postnatal ages, who were well and had no medical problems, except for the need for oxygen in two of them. The well infants spent significantly longer with their eyes open, moving their arms, opening their mouths, and moving their heads than did the sick infants. The 10 sick infants were observed again, 2 weeks later, by which time they had recovered from their life-threatening complications. When recovered, they spent significantly longer with eyes open, mouths open, and moving their heads. Grimacing, smiling, hand to mouth, crying, yawning, arm or leg movements did not vary according to whether the infant was sick or well. Parents visiting their sick infants touched their babies and smiled at them much less than did parents of babies who were well. Thus attachment by parents to such small sick infants may be inhibited not only by Perspex walls and fear of death or damage, but also by reduced activity by the infant.

Bacteriological findings in necrotising enterocolitis—a controlled study. M F Smith, M W Caswell, G S Clayden, and S P Borriello (introduced by J W Scopes). Department of Paediatrics and Microbiology, St Thomas's Hospital, and Bacterial Metabolism Research Laboratory, London.

The role of bacterial infection in the aetiology of neonatal necrotising enterocolitis (NEC) remains unclear. Many organisms have been postulated, the most recent contenders are bacteria of the genus Clostridium, particularly Clostridium butyricum and Clostridium welchii. The interpretation of clostridial isolates from affected infants is made difficult by the lack of accurate quantitative and qualitative data on clostridial species in these infants.

In a recent outbreak of NEC at St Thomas’s Hospital, stools from 6 NEC and 8 healthy infants were examined quantitatively and qualitatively for all Clostridium sp. Environmental specimens were also taken from 52 sites and cultured for Clostridium sp.
19 stools from 6 NEC and 8 control infants all showed Clostridium sp. (except one from Case 10). Total clostridial counts ranged from $10^4$ to $10^8.5$ clostridia/g wet weight of stool. No infant harboured more than four species at any time. C. butyricum was the most common isolate and was found in stools from 5 NEC patients, but also in all but one of the 8 control infants. Clostridium difficile was isolated from 2 NEC infants during the active stage of their disease, but also from 7 of 8 controls. A toxin, neutralised by Clostridium sordellii antitoxin, was found in the stool of one NEC and 4 control infants. Clostridium perfringens was isolated from 2 infants and, although Clostridium tertium and Clostridium paraputrificum are usually present in the faeces of term infants, these species were not isolated from any of the 19 stools.

These results do not implicate a single aetiological agent of the Clostridium sp. C. difficile, the aetiological agent in adult pseudomembranous colitis, must also be considered an unlikely organism in NEC. The continued occurrence of NEC in `epidemic' form must surely indicate a transmissible agent, possibly of a viral nature. (Results reported in full—Journal of Infection 1980, in press.)

References


Amikacin was studied in 35 newborn infants with suspected septicaemia. Median and (range) of gestation was 30 (24–40) weeks, of birthweight 1500 (700–2900) g, and of age 5 (1–94) days. The initial dosage of 15 mg/kg per day was increased to 20 mg/kg per day because of low plasma levels. The higher dose gave satisfactory peak (derived by inspection of curves) and 12-hour trough levels in infants <1400 g and >4 weeks.

Multivariate analysis showed that plasma creatinine accounted for 70% of the total variance of trough levels. Effects of postnatal age and weight were partly due to differences in plasma creatinine concentrations. Arterial pH and $P_{CO_2}$ had significant effects in the whole group but not when those with renal impairment were excluded.

13 infants had impaired renal function but no evidence of renal toxicity. Plasma urea was a very poor guide to renal function in this group of infants.

Two infants died out of 7 with positive blood cultures—one with Staphylococcus aureus infection and one with Alcaligenes. One treatment failure had a positive blood culture with the original organism (Escherichia coli) after a week of treatment but responded well to ampicillin. Eight other infants had bacteriological and clinical evidence of infection and responded satisfactorily.

Unwanted effects were confined to colonisation with a 'new' organism during treatment in 5 infants, one of the organisms being an amikacin-resistant Pseudomonas sp.

Insulin injection of rabbit fetuses in utero increases somatomedin and cartilage metabolic activity. D J Hill and R D G Milner. Department of Paediatrics, University of Sheffield, Children's Hospital, Sheffield.

The effect of exogenous insulin on plasma somatomedin activity and cartilage metabolism in rabbit fetuses is reported.

One fetus in each of 12 litters of Dutch rabbits was injected with one unit of IZS insulin subcutaneously on day 27 of gestation and a control fetus was injected with the same volume of saline. The litter was delivered by caesarean section on day 29 and plasma and costal cartilage were collected from all fetuses. Plasma somatomedin activity was determined by the fetal rabbit cartilage bioassay.1 Costal cartilage from each fetus was incubated in medium containing $^3$H-thymidine or $^{35}$S-sulphate as indicators of cell replication and matrix synthesis respectively. Individual values for somatomedin activity or cartilage isotope uptake were ranked within a litter.

In each case the rank in the litter of the insulin-injected fetus, but not the saline-injected fetus, was significantly higher than the mean rank of the litter. Insulin (10 $\mu$U/ml—1 U/ml) added to the cartilage incubation medium with or without fetal rabbit plasma had no direct action on cartilage metabolism in vitro.

The results suggest that insulin may act as a fetal 'growth hormone' by stimulating skeletal growth via the somatomedin pathway.

Reference

Although there are guidelines for vitamin D supplementation in term infants, the requirements of preterm infants are uncertain and doses of 400 to 1000 IU/day have been recommended. Hillman and Haddad\(^1\) found no increase in plasma 25-hydroxyvitamin D (25-OHD) levels with oral vitamin D supplements until after 36 weeks' gestational age. Wolf \textit{et al.}\(^2\) reported an increased plasma 25-OHD in response to oral vitamin D at 32 to 34 weeks. 24-OHD levels were studied in two groups of preterm infants, A and B, given 400 and 1000 IU of oral vitamin D daily. Both groups of babies showed increases in plasma 25-OHD at 36 weeks compared with presupplementation levels (group A, \(P<0.025\); group B, \(P<0.0025\)). Plasma 25-OHD levels at 36 weeks were not higher in group B than in group A. The rise in 25-OHD after supplementation correlated with the initial plasma 25-OHD value (\(R = -0.68, P<0.0025\)). There were no differences in plasma calcium and alkaline phosphatase.

Our results demonstrate the ability of preterm infants to absorb 25-hydroxylate vitamin D, but do not exclude the possibility of impaired 1\(\alpha\) hydroxylation or end organ unresponsiveness to 1:25-OHD, which could cause rickets in the presence of normal plasma 25-OHD.

We conclude that a daily oral supplement of 400 IU of vitamin D is sufficient to produce normal plasma 25-OHD levels by 36 weeks' gestational age.

References
