Prostaglandin use in neonatal transport

The West of Scotland transport team introduced a protocol for management of neonates on intravenous prostaglandin that necessitated intubating infants on over 5 ng/kg/min prostaglandin for <48 hours. The respiratory management of infants on prostaglandin during transfer is controversial and often subjective. We reviewed the safety profile of our prostaglandin transports over a two year period and compared our policy with other transport services in the United Kingdom to try to provide additional information on guidelines for safe transfer.

Transfers were identified by manually searching the transport records and case notes. A postal questionnaire was sent to the lead consultant of all transport services in the United Kingdom about factors that influence transport of infants on prostaglandin including dose and length of time on prostin, mode and length of transfer, and clinical stability.

Thirty seven transfers were identified with a dose range of 5–100 ng/kg/min (median 10 ng/kg/min); 15% of babies were receiving a dose of 5 ng/kg/min and 85% ≥ 10 ng/kg/min.

Length of time on prostaglandin was: 68% <24 hours, 16% 24–48 hours, and 16% >48 hours.

Thirty infants (81%) were ventilated before transfer, 12 by the transport team and 18 by the referring hospital. Of the infants ventilated by the referring hospital, most were for clinical condition (most commonly cyanosis before establishment of a diagnosis) although 5/18 were at the request of the transfer team. Most infants (90%) were ventilated because they were on a higher dose of prostin (≥ 10 ng/kg/min) and the duration of treatment was short (<48 hours). Two infants were ventilated despite receiving low dose prostin (5 ng/kg/min) as they had been on this for under 24 hours. One infant was on low dose prostin for several days and was intubated for clinical instability. Seven infants were transferred self ventilating (all those on 5 ng/kg/min prostin for >48 hours).

Distance transferred ranged from 6 to 490 km (road transfer <10 km, eight infants; road transfer 20–150 km, 12 infants; air transfer >200 km, 17 infants). The most common diagnoses were hypoplastic left heart syndrome (35%) and pulmonary atresia variants (27%). No prostaglandin related incidents or complications of intubation or ventilation were recorded during the transfer.

The rate of response to the questionnaire was 91%. In 70% of transport services, there was no protocol guiding management of prostaglandin transfers. Factors cited that influence respiratory management included dose and length of time on prostaglandin, clinical condition of baby, and distance and mode of transfer. Existing protocols specifying dosage advocated ventilation of infants on >10 ng/kg/min for long distance transfers or clinical instability. Most transfers were by specialist registrars (80%). Only one unit had recently audited their transfers.

We conclude that a protocol based approach to airway management is safe during transfer for our population of neonates. The potential complications of electively ventilating infants, especially those with parallel circulation such as hypoplastic left heart syndrome, do, however, need to be recognised. We recommend audit of prostaglandin transfers and consideration of a protocol appropriate to each region.

L McGlone, L Jackson

Royal Hospital for Sick children, Glasgow, Scotland, UK

Dr McGlone, Royal Hospital for Sick children, Dalnair Street, Glasgow G3 8SJ, Scotland, UK; mcglonelaura@hotmail.com

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Plasma leptin, insulin, and neuropeptide Y response to feeding in newborn infants

Although the functions and regulation of leptin, insulin, and neuropeptide Y (NPY) are well studied, their role and regulatory mechanisms in neonates are poorly understood.

We have reported that, compared with umbilical concentrations, on the 4th day of life fasting plasma leptin and insulin concentrations are significantly decreased, and plasma NPY concentrations increased.¹ The effects of feeding on plasma concentrations of these hormones are controversial^{2–3} or have not been studied.

We have investigated the effects of breast feeding on plasma concentrations of leptin, insulin, and NPY in 30 healthy, full term neonates, exclusively breast fed, on the 4th day of life.

At 30 minutes after breast feeding, the mean (SD) plasma concentrations of leptin and insulin increased (leptin from 4.91 (6.05) to 9.85 (8.09) ng/ml ($p < 0.001$), and insulin from 3.12 (1.24) to 9.41 (6.06) μ U/ml ($p < 0.001$)), whereas NPY did not show any significant change (73.87 (29.22) v 69.37 (26.52) pmol/l).

In adult humans, plasma leptin concentrations do not change in response to meals, suggesting that, in the regulation of energy balance, leptin works as a long term adiposity signal rather than a short term meal related factor.⁴

The discovery that the stomach can also produce and store leptin and release it in response to food intake has suggested that leptin may also have a role in the short term control of energy balance.^{5–6} Recently, it has been shown that the onset of leptin mRNA expression and leptin synthesis in the human stomach takes place around the 11th week and increases sharply in older fetuses.⁷ Moreover, in neonatal rats, it has been shown that orally supplied leptin is directly taken up by the immature stomach and may regulate short term feeding.⁸ The early post-feeding increase in plasma concentrations of leptin, observed by us, in agreement with Cinaz *et al.*,² may suggest that the neonate adsorbs leptin produced by the gastric mucosa. This leptin may play a potential role in acute regulation of feeding behaviour of neonates, and, downregulating endogenous leptin produced by adipocytes, it could be the cause of the fasting plasma leptin decrease observed on the 4th day of life.

The lack of effect of feeding on plasma concentrations of NPY reinforces our hypothesis that the high circulating concentrations of NPY in the neonate are only due to neuronal release of NPY, in response to the general activation of the sympathetic system present at birth.

C Mami, R Manganaro, L Marseglia, G Saitta, M Gemelli

Department of Pediatric and Surgery Sciences, Division of Neonatology, University of Messina, Messina, Italy

F Martino

Department of Radiologic Sciences, University of Messina

Correspondence to: Professor Gemelli, Divisione di Neonatologia, Policlinico Università di Messina, 98100 Messina, Italy; Marina.Gemelli@unime.it

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Circulatory effects of antenatal betamethasone therapy in low birthweight infants

We read with great interest the paper of Vural *et al.*¹ on the cardiac effects of single course antenatal betamethasone in preterm infants. In line with the observations of Dimitriou *et al.*² on the effect of betamethasone on neonatal renal function, there is still need to further document the impact of antenatally administered steroids on various extrapulmonary outcome variables such as cardiovascular stability.

We therefore would like to report on the use of dopamine in the first week of life in a cohort of 282 consecutively admitted low birthweight (LBW) infants—that is, <1500 g—of whom 243 survived until discharge. Our algorithm to initiate dopamine has recently been described.³

Data on antenatal betamethasone were not retrieved in six cases, resulting in observations on 237 LBW survivors. The clinical characteristics of the LBW infants either treated ($n = 178$) or not ($n = 59$) before birth with betamethasone can be summarised as follows (betamethasone/no betamethasone): number = 178/59; mean (range) gestational age (weeks) = 29 (24–34)/30 (24–35) (NS); mean (SD) birth weight (g) = 1142 (272)/1062 (303) (NS); mean (range) CRIB score = 2 (0–13)/2 (0–13) (NS); inotropic agents before day 7 = 38%/46% (NS); mean (range) duration (hours) of inotropic agents in first week when all survivors are included = 0 (0–168)/0 (0–144) (NS), and when only LBW infants treated with inotropic agents are included = 50 (4–168)/56 (8–144) (NS).

In brief, we were not able to find any difference in the administration or duration