NEONATOLOGY—THEN AND NOW

Plasma volume changes in the newborn (1958/59)

The fluid shift from the vascular compartment immediately after birth

DOUGLAS GAIRDNER, JOHN MARKS, JANET D ROSCOE, R O BRETTELL
Cambridge Maternity Hospital (Arch Dis Child 1958;33:489–98).

Postnatal plasma shift in premature infants

A C L CLARK, DOUGLAS GAIRDNER

Department of Medicine, University of Cambridge and Cambridge Maternity Hospital (Arch Dis Child 1960;35:352–4).

In 1952, Gairdner and his colleagues drew attention to the fact that there was a consistent rise in the haemoglobin of the newborn immediately after birth. Later they went on to challenge the theory current at the time that this rise was due to transfer of blood from the placenta and the summary of the first of these papers begins:

‘Immediately after birth there is a rise in concentration of haemoglobin and PCV [packed cell volume] in the majority of infants. The rise may be discernable within a few minutes of birth and is probably completed within an hour or two. It occurs in the absence of any transfer of placental blood’.

The conclusion reached was that these changes were due to a shift of fluid from the intravascular space:

‘... mainly whole plasma and its volume may amount to a large fraction of the circulating plasma at birth, a quarter or more. In addition there is a shift of fluid from the red cells, the volume of red cells contracting after birth by an average of 3%... These results imply that the foetus in utero is haemodilacemic, i.e. has a large plasma volume’.

The second paper confirms that a similar plasma shift occurs in preterm infants and while:

‘in this series the magnitude of the postnatal plasma shift bears no relation to the development of respiratory distress...’

‘It was suggested that the shift might take place particularly from the pulmonary circulation and so contribute to the development of pulmonary oedema and respiratory failure...’

Today. What to do, if anything, about the changes in haemoglobin, packed cell volume, plasma, and whole blood volume in the first day or two of life has exercised the minds of paediatricians for at least 30 years. These early observations have stood the test of time and it is now generally accepted that transfusion of extra blood from the placenta, once actively encouraged, can be harmful. It initially renders the baby hypervolaemic, encourages dilatation of the capillary bed with further transudate, which causes increased oedema and intravascular polycythaemia. It is also accepted that transudation from the pulmonary capillary is a precursor of hyaline membrane formation so retention of plasma within the vascular space would seem a sensible objective. The fact that an early diuresis signals a good prognosis in preterm infants with respiratory distress probably indicates return of this oedema fluid into the intravascular space with the associated improved haemodynamics similar to that seen after haemodilution, the therapeutic equivalent in infants with hyperviscosity.

I have always felt that the advantages of improved oncotic pressure (albeit transient) from infusions of albumin with the consequent reduction of tissue oedema and improved renal output outweigh any disadvantage there might be in increasing blood volume and so possibly prolonging patency of the ductus arteriosus. The fact that oedema does not correlate well with serum albumin is not the point. Whether the baby benefits from its administration surely is and recent work would encourage the practice of infusion of salt free albumin.

LETTERS TO THE EDITOR

Mechanical ventilation and respiratory syncytial virus infection

Sir,—Drs Lebel et al report weight and prematurity as risk indicators for the need for ventilation in bronchiolitis.1 In their case-control study a viral cause was determined in only 32% infants; with underlying cardiopulmonary abnormalities—who are known to be at increased risk for severe respiratory syncytial virus infections and mortality2 were excluded. Thus the results from their study may not readily be applied to all infants with bronchiolitis who need hospitalisation.

We would like to present the preliminary results of a prospective study (1987-9) of children admitted with a respiratory syncytial virus infection to the Sophia Children’s Hospital, The Netherlands. In all patients the infection was proved by direct immunofluorescent assay. Nineteen patients needed mechanical ventilation and 82 were admitted to the general ward.

Weight and age (corrected for prematurity) on admission were significantly related with mechanical ventilation (by χ², p<0.05). No relation between mechanical ventilation and risk factors for severe respiratory syncytial virus infections (prematurity, congenital heart disease, bronchopulmonary dysplasia, or immunodeficiency states) was found. Nine (47%) of the ventilated patients and 36 (43%) of the non-ventilated patients belonged to the risk group. In a stepwise logistic regression analysis (while controlling for the covariates: age, prematurity, and risk factors for severe respiratory syncytial virus infection) only weight appeared to be significantly related to the need for mechanical ventilation (coefficient --0.36682 (SE 0.1685), p<0.01), while none of the other variables did. Prematurity and age were not independent risk indicators for mechanical ventilation in this study. In contrast to Lebel et al, who report odds ratios as ‘relative risk’, from the data of our prospective study we are able to estimate the absolute risk of mechanical ventilation related to the weight on admission (figure). A low weight is related to an increased risk for mechanical ventilation. For infants less than 2kg, a relative risk for mechanical ventilation of 4·3 (95% confidence interval 1·3 to 13·9) was estimated.

H A VAN STEENKEL-MOLL
W J E TISSING
Department of Paediatrics, Sophia Children’s Hospital, Erasmus University Rotterdam, Gordelweg 160, 3083 GE Rotterdam, The Netherlands
J A HAZELZET
Department of Paediatrics, Subdivision of Paediatric Intensive Care, Sophia Children’s Hospital, Erasmus University Rotterdam, Gordelweg 160, 3083 GE Rotterdam, The Netherlands

3 MacDonald NE, Beece Hall C, Suffer SC, Alexson C, Harris PJJ, Manning JA. Respira- tory syncytial virus infection in infants.

Heliocobacter pylori and protein losing enteropathy

Sir,—We have demonstrated a high prevalence of anti-Heliocobacter pylori (formerly Campylobacter pylori) antibodies in children under the age of 3 years in The Gambia, West Africa.1 Using a serological test validated by histology and microbiology, 41/77 (53%) of children with chronic diarrhoea were shown to have significantly high anti-H pylori IgG antibody titres and, indeed, the prevalence of H pylori antibodies in healthy asymptomatic children of the same age was also high at 26%.

It was against the background of these findings that we were interested in the description, in 1987, by Hill and colleagues, of transient protein losing enteropathy in association with acute infection with H pylori.2 This observation might be due to chance but it should have important consequences in the nutritional rehabilitation of infants with chronic diarrhoea and severe protein energy malnutrition.

Therefore we undertook a study to establish whether or not H pylori infection was associated with protein losing enteropathy in Gambian children with chronic diarrhoea.

Fifty three subjects (25 boys, 28 girls; mean age 19 months) were studied and all had chronic diarrhoea (more than three loose stools per day for more than two weeks) and severe protein energy malnutrition (32 marasmus, 21 marasmic-kwashiorkor). After admission, three consecutive fresh stool samples were collected for serological, parasitological, and bacteriological investigation. Specific anti-H pylori IgG antibody was measured by enzyme linked immunabsorbent assay (ELISA) in all subjects. Gastroscopy and antral mucosal biopsy were performed in 20/53 children.

Protein losing enteropathy was estimated by random faecal α1-antitrypsin measurement. Whole single stools were collected ensuring that both the liquid and solid phase were obtained. These were frozen and lyophilised and α1-antitrypsin measured by a single radial immunodiffusion method. Random faecal α1-antitrypsin measurement (normal mean (SD) value in healthy Gambian children=1·54 (0·23) mg/g stool) has been shown to be a reproducible screening test for excessive enteric protein loss and has been validated against 31chromium labelled albumin excretion in the stool.3

Fifty six percent of this group of children had significantly raised anti-H pylori antibody titres and in 11/20 gastroscopped this was associated with recovery or the organism and histological gastritis. Strongyloides stercoralis, and Giardia lamblia were found in 11 and 38% of the patients respectively. Bacterial organisms occurring in children with S stercoralis was found to be associated with rash (p<0.001) with increased faecal α1-antitrypsin excretion (mean (SEM)2·47 (0·9) mg/g stool) whereas there was no difference between levels of faecal α1-antitrypsin found in children with (1·57 (0·2) mg/g stool) or without (1·62 (0·3) mg/g stool) evidence of H pylori infection. Therefore, in this study we failed to show that chronic infection with H pylori is associated with protein losing enteropathy.

P B SULLIVAN
Department of Child Health, Westminster Children’s Hospital, London W1P 2NP

J E THOMAS
E J EASTHAM
Department of Child Health, University of Newcastle, upon Tyne

1 Sullivan PB, Thomas JE, Wight DG, et al. Heliocobacter pylori in Gambian children with...