The effectiveness and side effects of dexamethasone in preterm infants with bronchopulmonary dysplasia

EDITOR.—In his comprehensive review of dexamethasone treatment for bronchopulmonary dysplasia Dr Ng proposes that improvement in lung function is mediated by the anti-inflammatory action of dexamethasone and describes changes in plasma and urine amino acid concentrations as metabolic 'complications' of treatment that reflect protein catabolism in muscle.¹ An alternative hypothesis is that, far from being complications, these changes explain the rise in lung compliance induced by dexamethasone.

Animal studies have demonstrated that high dose steroid treatment impairs lung growth and suppresses lung protein synthesis.² Steroid treatment also significantly increases pulmonary venous efflux of glutamine and alanine in rats,³ indicating induction of lung catabolism. Thus animal data suggest that protein metabolism in lung parenchyma is as sensitive to corticosteroids as in skeletal muscle. There is also evidence that lung protein deposition affects lung function. Matsui *et al* studied rats fed isocaloric diets sufficient or deficient in protein and found that protein deficiency both increased lung compliance and reduced elastic recoil.⁴ Both could be beneficial to the baby being weaned from mechanical ventilation in whom the time course of changes in pulmonary function parallels closely that of change in plasma and urinary urea and amino acid concentrations.^{5 6}

Measurement of organ specific rates of protein turnover in preterm babies are not yet technically feasible. However, I believe they will prove eventually both that increased lung compliance is a property of acute lung parenchymal protein wasting and

that changes in concentrations of inflammatory mediators are merely epiphenomena.

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Delayed umbilical cord separation in alloimmune neutropenia

EDITOR.—The timing of umbilical cord separation remains largely unexplained despite many studies during the last decade. It is mainly of interest in view of the strong relation between delayed cord separation and a genetically determined deficiency of leucocyte adherence glycoproteins.¹ The time of cord separation is also influenced by other factors such as neonatal infections, gestational age, and method of delivery.²

Kemp and Lubitz's report of markedly

delayed separation of the umbilical cord at the age of 55 days in association with alloimmune neutropenia is very interesting as it represents an as yet unreported cause of delayed cord separation.³ In a study of neonates who died within the first week of life we found that cord separation came about by infiltration of neutrophils progressing from the edges of the umbilical area towards the centre.⁴ This infiltration finally forms a continuous band producing a demarcation zone between the mummified cord stump and the vital tissues of the umbilical area of the abdominal wall from which the cord is in the process of separating. No cells other than the neutrophils are present and no bacteria were seen. An association between delayed cord separation and neonatal neutropenia can perhaps be explained on the grounds of these histological findings.

Even though the exact reason why the cord separates at a particular time is not always completely clear it is important to note the time of cord separation. A cord that separates within the normal time limit for the population may be reassuring and delayed cord separation should always raise the question 'why is it delayed?'.

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