

of all the factors linked to hydration, whether over- or underhydration. In the era of delayed feeding in the 1950s neurological and intellectual handicap were the all too frequent outcome of a regimen which often led to underhydration. Necrotising enterocolitis and patent ductus arteriosus are not the sole hazards of low birthweight.

Our paper refers to the ordered and selective use of central and peripheral venous lines, nasogastric, and nasoduodenal feeding. It is difficult to understand why Dr Clarke and his colleagues seem anxious to imply a causal and excessive use of these techniques and then to dissociate themselves from their own misrepresentation.

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Hyponatraemia in preterm infants—arginine vasopressin secretion

Sir,

We read with interest the report by Rees *et al* on the role of excessive arginine vasopressin secretion and subsequent water retention in the development of early hyponatraemia in sick preterm infants.¹

We would like to point out that the increased rate of arginine vasopressin secretion may also be implicated in the aetiology of late hyponatraemia frequently seen in healthy low birthweight preterm infants, which was thought to be due to renal salt wasting.

In a recent study we measured simultaneously the urinary excretion of aldosterone and arginine vasopressin along with sodium balance, plasma and urine sodium, and osmolality in nine healthy preterm neonates during the first five weeks of life (Sulyok *et al*, unpublished data). It could be shown that both urinary aldosterone and arginine vasopressin excretion increased with advancing postnatal age from the initial values of mean (SD) 0.94 (0.16) µg/day and 0.38 (0.08) ng/day in the first week to a maximum of 4.30 (0.76) µg/day (P<0.01) and 1.19 (0.26) ng/day (P<0.01), respectively in weeks 4 and 5, in spite of the declining plasma sodium concentration. Moreover, significant positive correlation was found between urinary aldosterone and arginine vasopressin excretion in seven of the nine infants studied.

On the basis of these observations it seems to be relevant to assume that in salt-losing preterm infants the increasing rate of aldosterone and arginine vasopressin secretion

occurs in response to the same stimulation, that is to the protracted contraction of extracellular fluid compartments. The higher arginine vasopressin secretion rate results in more efficient water reabsorption and may contribute to restoring body fluids to normal. In support of this assumption antipyrine and bromide space studies by Roy *et al* did not show any difference in the fluid compartments in preterm infants with or without late hyponatraemia.²

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Dr Shaw comments:

Drs Kovács and Sulyok draw attention to an important point. It has been generally believed for many years that in sodium depletion the development of hyponatraemia represents an appropriate secretion of arginine vasopressin due to a fall in blood volume and mediated through atrial volume receptors. There have, however, been few published data to support this proposition, and none in preterm babies. It is therefore valuable that their data support the current theories. It is true, as they say, that an 'increased rate of arginine vasopressin secretion is implicated in the aetiology of late hyponatraemia . . .', but it must be emphasised that the increased secretion of arginine vasopressin is, as far as we know, an entirely physiological response to volume contraction and it should not be thought that late hyponatraemia is caused by water overload due to inappropriate release of arginine vasopressin. It is usually due to sodium depletion and should be treated with sodium supplements, not water restriction.

Urinary albumin excretion in school children

Sir,

We read with interest the recent paper on urinary albumin excretion in normal children,¹ which has provided valuable normal ranges for this measurement. The authors quote their results both as albumin excretion rates and urine albumin:creatinine ratio. We note that the authors had to

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reject 12% of their samples, even in well motivated children. This further emphasises to us the difficulty, which we have found in our own studies, in the collection of accurately timed and measured volumes of urine in children. The use of urine albumin:creatinine ratio obviates the need for timed or measured samples and correlates well with albumin excretion rates,² and we would suggest this as an ideal method for further study of albuminuria in children. In our own studies of diabetic children, a group that require repeated assessment of renal function, it has become evident that some form of stress is necessary to unmask latent glomerular damage. Using urine albumin:creatinine ratio urine specimens taken before and after exercise we have identified 20 out of 60 diabetic children (compared with none in a control non-diabetic group) with abnormal albumin excretion, but who have normal values for albumin excretion rates and urine albumin:creatinine ratios on random and timed 24 hour split urine collections.

We would also echo the plea for standardisation of units. The authors chose to use SI units of mg/mmol for their urine albumin:creatinine ratio rather than the original mg/mg, which has the merit of not mixing units and is comparable with most previous data without a difficult conversion (1 mmol=113.1 mg creatinine). We would suggest continuing with mg/mg for the measurement of urine albumin:creatinine ratio.

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Congenital diaphragmatic hernia: association between pulmonary vascular resistance and plasma thromboxane concentrations

Sir,

The report by Ford *et al*¹ of an association between plasma prostanooids and pulmonary vascular tone was extremely interesting. In the initial paragraph of the 'Results' section, however, they note an alveolar-arterial difference in oxygen tension (A-a Do₂) of 550 mm Hg. The alveolar air equation has undergone numerous revisions since first suggested by Benzinger,² but in its simplest form alveolar Po₂ is derived by first subtracting water vapour pressure from atmospheric pressure and then further subtracting the result of the arterial PCO₂ divided by the respiratory exchange ratio. Assuming a P_aCO₂ of 30 mm Hg, the A-a Do₂ would then become 446 mm Hg. Furthermore, infusion of tolazoline did not seem to eliminate the right to

left shunt, or venous admixture, but the infusion did succeed in reducing the shunt to somewhat less than that noted five hours after surgery, which might be roughly estimated as about 20%. Had tolazoline completely eliminated the shunt and produced a P_aCO₂ near 675 mm Hg, a rather new and more startling explanation would seem to have been required. These points are offered in clarification; the work is noteworthy and potentially quite important.

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Dr Ford and co-workers comment:

We calculated our alveolar-arterial difference in oxygen tension (A-a Do₂) using the umbilical artery as the source of arterial oxygen rather than the radial artery. Thus we derived an A-a Do₂ of approximately 550 mm Hg. This has been used previously to attempt to determine prognosis in these neonates.³

Obviously we have failed to clarify the degree of shunt at the various points in time. At the time of surgery, as the radial artery P_aO₂ was less than the expected level on an F_IO₂ of 1.0, we assumed there was a shunt at the cardiac level plus or minus a ventilation-perfusion mismatch. At the same time point, the difference between the radial and umbilical artery was assumed to represent ductal shunting. When the radial and umbilical oxygenation levels became the same at 15 hours after surgery and then both dropped, we assumed that shunting was occurring at the foramen ovale or there was a gross ventilation-perfusion mismatch, or both. Tolazoline improved this situation dramatically and at the same time thromboxane concentrations fell. We at no stage attempted to quantify the shunt.

References

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Medical contribution to the management of dyslexia

Sir,

What Drs Gordon *et al* allude to in their paper,¹ but perhaps should have clarified, is the way in which parents involve doctors—which is often as follows. Their child is not doing well at school. The head teacher has a talk with them and may mention referral to the educational psycho-



Urinary albumin excretion in school children.

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