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 casual and excessive use of these techniques and then to dissociate themselves from their own misrepresentation.

References
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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.

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and urine sodium, and osmolality in nine healthy preterm neonates during the first five weeks of life (Sulyok et al, unpublished data).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.

References

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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, 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
Dr Shaw comments

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