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

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and urine sodium, and osmolality in nine healthy preterm neonates during the first five weeks of life (Sulyok et al, unpublished data).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

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
Correspondence

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 obvi-
ates the need for timed or measured samples and correlates
well with albumin excretion rates, 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.

References
1 Davies AG, Posthethwaite RJ, Price DA, Bium JL, Houl-
ton CA, Fielding BA. Urinary albumin excretion in school
2 Barratt TM, McLaine PN, Soothill JF. Albumin excretion as
a measure of glomerular dysfunction in children. Arch Dis Child

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

Sir,
The report by Ford et al of an association between plasma
prostanoids 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 Benzinginer, 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 Paco2 divided by the respiratory
exchange ratio. Assuming a Paco2 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 Paco2 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.

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 PaO2 was less than the expected level on an
FiO2 of 1.0, we assumed there was a shunt at the cardiac
level plus or minus a ventilation-perfusion mismatch. At
the same point in time, 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
1 Ford WDA, James MJ, Walsh JA. Congenital diaphragmatic
hernia: association between pulmonary vascular resistance and
2 Benzinginer T. Untersuchungen uber die Atmung und den
Gastrogwegewechsel insbesondere bei Sauerstoffmandel und
Unterdruck, mit fortlaufend unmittelbar aufzeichnenden
Methoden. Ergebnisse der Physiologie biologischen Chemie
3 Raphael RC, Downes JJ, Jr. Congenital diaphragmatic hernia:

Medical contribution to the
management of dyslexia

Sir,

What Drs Gordon et al allude to in their paper, 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-

References
1 Ford WDA, James MJ, Walsh JA. Congenital diaphragmatic
hernia: association between pulmonary vascular resistance and
2 Benzinginer T. Untersuchungen uber die Atmung und den
Gastrogwegewechsel insbesondere bei Sauerstoffmandel und
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