Decline of hypernatraemia as a problem in gastroenteritis

P D MANUEL AND J A WALKER-SMITH

Academic Department of Child Health, Queen Elizabeth Hospital for Children, London

SUMMARY During the 5-year period 1973–77 there was a considerable change in infant feeding practice (the use of low solute cows' milk formulae instead of high solute formulae and the later introduction of solid foods into the diet) which should have led to a reduced incidence of hypernatraemia among infants admitted to hospital with gastroenteritis. We have compared the incidence of hypernatraemia in such infants admitted during three 12-month periods between January 1973 and December 1977. Our results show there was such a reduction; they also show that hypernatraemia did not occur in infants fed a low solute milk formula alone.

Until recently hypernatraemia was a common biochemical abnormality in infants with gastroenteritis in Western countries, and it is not unknown in developing ones. This abnormality has been associated with a high mortality rate and particularly with cerebral damage. This damage is generally due to cerebral oedema or intracranial haemorrhage and is the most serious consequence.

The high incidence of hypernatraemia in infants with gastroenteritis in Western countries has been accompanied by a high protein intake which stresses the homeostatic mechanisms controlling plasma osmolality. It has also been associated with hyperosmolar glucose solutions used in the treatment of gastroenteritis, and with excessive stool water loss caused by intestinal carbohydrate malabsorption together with slow treatment of volume deficits.

Davies called for a return to breast feeding as a protection against hypernatraemia, but this has happened only to a very limited extent. However, the use of low solute cows' milk formulae is now widespread and this has been accompanied by later introduction of solids into the diet. (P D Manuel and J A Walker-Smith, 1978, unpublished observations).

There is now evidence that overconcentration of feeds produced by faulty reconstitution is becoming less common, possibly as a result of propaganda aimed at discouraging high solute feeds although this is not the experience of some workers.

This study was undertaken to find out if the change to low solute feeding has affected the incidence of hypernatraemic dehydration in a population of infants presenting with acute gastroenteritis.

Methods

A retrospective study was made of all infants admitted with hypernatraemia to the gastroenteritis unit of this hospital during two 12-month periods—1 October 1975 to 30 September 1976 and 1 January 1977 to 31 December 1977. Plasma electrolyte concentrations were estimated on admission. Hypernatraemia was defined as plasma sodium concentration of at least 150 mmol/l. Clinical details particularly those relating to reason for admission, feeding history, physical state on admission, and results of investigation and treatment were compared between the two periods and were also compared with results obtained in a prospective study undertaken in the same unit from 1 January 1973 to 31 December 1973 when mainly high solute milks were used in infant feeding.

Results

Total admissions during the three periods were comparable and show no decrease in incidence of gastroenteritis between 1973 and 1977, Table 1. The sex ratio was similar with slightly more boys...
(54%). The incidence of hypernatraemia however, fell from 4.9 to 0.9% during this period. This fall is significant \( P<0.01 \).

The ethnic origin of infants with hypernatraemia in 1975–76 was European 5, Asian 5, and West Indian 1, while in 1977 it was 3, 1, and 1 respectively. Mean age at admission of these infants was 10.6 months in 1975–76 and 12.6 months in 1977. Most infants with hypernatraemia were admitted suffering from gastroenteritis—20 in 1973, 11 in 1975–76, and 5 in 1977. Three infants in 1973 had surgical conditions.

In 1975–76 the mean duration of symptoms before admission was 2–8 days (range 2–4) and in 1977 2.6 days (range 2–3), suggesting no delay in seeking treatment. Seven (64%) infants in 1975–76 and 5 (100%) infants in 1977 had been seen and treated by a doctor (Table 2). ‘Appropriate’ treatment was considered to be maintenance of hydration with clear fluids. ‘Inappropriate’ treatment was the use of such drugs as kaolin, antiemetics, lomotil, or antibiotics. The parents of one infant were advised to add one teaspoonful salt and one teaspoonful sugar to a pint of water; this was considered dangerous. 62.5% infants were treated inappropriately.

None of the infants admitted with hypernatraemia was being fed low solute milks alone (Table 3). No infant was breast fed; 82% in 1975–76 and 100% in 1977 of infants with hypernatraemia were fed solids. Three infants on low solute milks in 1975–76 were also taking significant quantities of solids, while one 6-day-old infant had been fed both a low solute and a high solute milk.

In infants with gastroenteritis dehydration was not generally the main reason for admission. For example, in 1973 only 31.5% infants admitted were dehydrated. Dehydration however, was pronounced in most infants admitted with hypernatraemia (Table 4).

Other investigations in infants with hypernatraemia admitted in 1975–76 and 1977 showed various abnormalities: 14 of 16 had high concentrations of blood urea; 4 of 16 had significant hypokalaemia (K<3.0 mmol/l); 5 of 13 had haemoglobin levels < 11 g/dl on admission at a time when they were dehydrated; 6 of 8 had acidosis (bicarbonate <15 mmol/l or pH <7.2); 3 of 9 had high WBC counts (>10 x 10⁹/l). Bacterial pathogens were found in the stools of none of the 16 infants. Rotavirus was looked for systematically only in 1977 and 2 of the 5 infants were found to have rotavirus on electron microscopical examination of their stools.

14 of the 16 infants required immediate intravenous treatment, generally given as 0.45% saline in 5% dextrose to correct the fluid deficit over 48 to 72 hours and 0.18 saline in 5% dextrose to provide maintenance fluids (100 ml/kg for infants below 3 months old, less for older ones). Intravenous treatment was stopped in these infants as soon as possible. Two infants were initially given a glucose electrolyte solution orally, although one developed sugar intolerance and subsequently required treatment intravenously.

The most severe complication, cerebral oedema, occurred in one infant in 1975–76 and one in 1977. Both infants were unconscious and convulsing on admission and both died. None of the other 14 infants suffered from convulsions; other complications are listed in Table 5. Two infants died with hypernatraemia in 1973, one as a direct result, the other also suffered from intussusception and peritonitis.

Table 1 Incidence of hypernatraemia in gastroenteritis admissions

<table>
<thead>
<tr>
<th></th>
<th>1973</th>
<th>1975–76</th>
<th>1977</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex ratio M:F</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infants with hypernatraemia</td>
<td>57%</td>
<td>51%</td>
<td>56%</td>
</tr>
<tr>
<td>Infants with (Na&lt;sup&gt;+&lt;/sup&gt;) 160 mmol/l</td>
<td>23 (4.9%)</td>
<td>11 (2.5%)</td>
<td>5 (0.9%)</td>
</tr>
<tr>
<td>Infants without (Na&lt;sup&gt;+&lt;/sup&gt;) 160 mmol/l</td>
<td>8 (1.7%)</td>
<td>3 (0.7%)</td>
<td>1 (0.2%)</td>
</tr>
</tbody>
</table>

Table 2 Outpatient treatment

<table>
<thead>
<tr>
<th></th>
<th>1975–76</th>
<th>1977</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean duration of symptoms before admission</td>
<td>2–8 days</td>
<td>2–6 days</td>
</tr>
<tr>
<td>Appropriate treatment</td>
<td>3</td>
<td>1*</td>
</tr>
<tr>
<td>Inappropriate treatment</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Dangerous treatment</td>
<td>1*</td>
<td>0</td>
</tr>
<tr>
<td>No outpatient treatment</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

*Infants who died.

Table 3 Feed at onset of illness

<table>
<thead>
<tr>
<th></th>
<th>1975–76</th>
<th>1977</th>
</tr>
</thead>
<tbody>
<tr>
<td>'Doorstep' milk</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>High solute milk</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Low solute milk</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Breast milk</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Solid food</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>Low solute milk without solid food</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 4 Severity of dehydration

<table>
<thead>
<tr>
<th>Degree of dehydration</th>
<th>1973</th>
<th>1975–76</th>
<th>1977</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not dehydrated</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Slight (3% or less)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Moderate (4–6%)</td>
<td>22</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Severe (7% or more)</td>
<td>8</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Total dehydrated</td>
<td>22 (96%)</td>
<td>11 (100%)</td>
<td>4 (80%)</td>
</tr>
</tbody>
</table>
Table 5  Complications of infants admitted with hypernatraemic dehydration

<table>
<thead>
<tr>
<th>Complications</th>
<th>1975-76</th>
<th>1977</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebral oedema leading to death</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Convulsions (excluding above)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sugar intolerance (transient)</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Late presenting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebral damage</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Delayed recovery (postenteritis syndrome)</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Anaemia</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Further recovery with gastroenteritis</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>No follow-up</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

**Discussion**

The incidence of hypernatraemia at this hospital has dropped markedly from 4.9% of admissions in 1973 to 0.9% in 1977. The main change likely to have influenced the incidence of hypernatraemia during this period was the widespread change from high solute to low solute milks after DHSS recommendations. In 1973 most infants were fed high solute milk formulae, by 1977 infants under 6 months were generally taking low solute milks (P D Manuel and J A Walker-Smith, 1978, unpublished observations), while 1975–76 was a transitional period.

None of the 16 infants with hypernatraemia in 1975–76 or 1977 was found to be excreting a recognised bacterial pathogen in stools, although the infant who died wholly from hypernatraemia in 1973 was excreting an enteropathogenic *Escherichia coli*. In 1977, 2 of 5 infants were excreting rotavirus in their stools, this being a similar incidence to that found among all infants admitted with gastroenteritis to this unit, but providing further evidence that rotavirus gastroenteritis can be severe.

**Addendum**

The incidence of hypernatraemia as a complication of gastroenteritis has declined, probably as a result of the change to low solute milks in the feeding of infants. Where it did occur the infants tended to be older and were being fed a high solute milk formula or unaltered cows' milk together with solids.

Half the number of infants admitted with hypernatraemia (compared with 56% of all infants in 1975–76 and 1977 with gastroenteritis), were from the indigenous population.

Management during hospital admission was similar in the three periods, as was the outcome. Three deaths occurred wholly as a result of hypernatraemia, one in each of the 12-month periods, an incidence of major complications of 7.7% (similar to that reported by Ironside et al.). Previous studies reported a high (30%) incidence of convulsions during rehydration, but no infant had convulsions in this study.

Significant hypokalaemia, hyperglycaemia, acidosis, and anaemia were not major problems in management. None of the infants admitted in 1975–76 or 1977 was found to be excreting a recognised bacterial pathogen in stools, although the infant who died wholly from hypernatraemia in 1973 was excreting an enteropathogenic *Escherichia coli*. In 1977, 2 of 5 infants were excreting rotavirus in their stools, this being a similar incidence to that found among all infants admitted with gastroenteritis to this unit, but providing further evidence that rotavirus gastroenteritis can be severe.

Transient sugar intolerance occurred in 5 (31%) of 16 infants in 1975–76 and 1977, while delayed recovery (postenteritis syndrome) occurred in only one (6.67%) of 16 infants, but may reflect the older age group from which the hypernatraemic infants were taken and their good nutritional state.

In conclusion, the incidence of hypernatraemia as a complication of gastroenteritis has declined, probably as a result of the change to low solute milks in the feeding of infants. Where it did occur the infants tended to be older and were being fed a high solute milk formula or unaltered cows' milk together with solids.

**References**

Decline of hypernatraemia as a problem in gastroenteritis


Correspondence to Dr P D Manuel, Academic Department of Child Health, Queen Elizabeth Hospital for Children, Hackney Road, London E2 8PS.

Received 24 April 1979
Decline of hypernatraemia as a problem in gastroenteritis.

P D Manuel and J A Walker-Smith

Arch Dis Child 1980 55: 124-127
doi: 10.1136/adc.55.2.124

Updated information and services can be found at:
http://adc.bmj.com/content/55/2/124

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/