

## Constituents of feeding regimens

Substance	Sodium content (mmol/l)	Carbohydrate content (g/l)	Hypernatraemia reported
Oral rehydration fluid	90	20	No <sup>3</sup>
Formula milk	49	50	Yes <sup>1</sup>
Lytren*	50	100	Yes <sup>4</sup>
Glucose mixture	<20	250	Yes <sup>5</sup>
Maxijul 20%†	4	200	This case

\*Mead Johnson; †Scientific Hospital Supplies.

On examination he was floppy and semiconscious, with poor skin texture, sunken eyes, and a doughy abdomen. His weight on admission was 10.6 kg (compared with 12.4 kg in the clinic one month before).

Plasma sodium concentration was 182 mmol/l, potassium 4.9 mmol/l, urea 6.2 mmol/l, and glucose 8.3 mmol/l. Serum osmolarity was 373 mmol/l, and serum ammonia concentration 36 µmol/l (reference range <50 µmol/l). Acid-base balance showed a pH of 7.44, bicarbonate 21 mmol/l, and base deficit 3. No pathogens were isolated from the stool.

He was resuscitated with plasma 20 ml/kg and then cautiously rehydrated with 5% dextrose and 0.18% saline. His serum sodium concentration returned to normal within five days. Oral protein was restarted after 24 hours to minimise endogenous protein breakdown. He made a good recovery with no obvious deterioration in his neurological condition.

### Discussion

Hypernatraemic dehydration is a common and potentially serious complication of gastroenteritis. It has been associated with artificial formula feeds, which have a high osmotic load, particularly if inappropriately made up.<sup>1</sup> It is probable that the high carbohydrate content of these feeds is poorly absorbed during an attack of gastroenteritis, with the resulting loss of

more water than sodium in the stools.<sup>2</sup> The sodium content of the feed is relatively unimportant; indeed, oral rehydration fluids containing high concentrations of sodium have been successfully used to treat hypernatraemic dehydration.<sup>3</sup> The sodium and carbohydrate contents of various feeds that have been reported to cause hypernatraemic dehydration is shown in the table, with that of the oral rehydration fluid for comparison. It is likely that the hyperosmolar feeds given to this child contributed to the development of hypernatraemia and his subsequent clinical deterioration.

The use of oral emergency regimens that include glucose polymers has undoubtedly improved the treatment of metabolic disease, but complications of such regimens are only now becoming apparent. One that has been recently reported was the catabolism of high carbohydrate feeds by gut bacteria in the colon to form propionate, that was readily absorbed. This caused a worsening encephalopathy in a child with methylmalonic acidaemia.<sup>6</sup> The occurrence of hypernatraemia is, however, related to osmotic load, and therefore children without metabolic disease are equally at risk. It is therefore important to emphasise the need to give adequate volumes of free water when using these feeds for nutritional support.

We thank Mrs A MacLean, senior paediatric dietician, St George's Hospital, for her helpful advice.

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## Correction of hypernatraemia with continuous arteriovenous haemodiafiltration

G D Moss, R J Primavesi, M E McGraw, T L Chambers

### Abstract

**Continuous arteriovenous haemodiafiltration was used successfully to achieve controlled correction of hypernatraemia in the presence of renal failure, when peritoneal dialysis was contraindicated, in a 4 year old girl.**

To reduce the risk of neurological complications resulting from the correction of hypernatraemia, therapeutic measures should produce a gradual fall in plasma sodium concentration.<sup>1</sup> This is difficult to achieve in the

presence of renal failure. Peritoneal dialysis has been successfully used,<sup>2</sup> but when this is contraindicated the alternative of haemodialysis results in a rapid, uncontrollable, fall in plasma sodium.

Since the development of haemofilters suitable for use in infants and children, continuous arteriovenous haemofiltration has become recognised as a useful technique for the correction of hypervolaemia, electrolyte imbalance, and metabolic acidosis secondary to renal failure in critically ill children.<sup>3</sup> Continuous arteriovenous haemodiafiltration is a modification of

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this technique, which—although primarily designed to increase urea clearance—will also improve the clearance of other dialysable substances. Theoretically, it could also be used to produce gradual, controlled, correction of hypernatraemia. We report the use of the technique in the management of a child with hypernatraemia and renal failure. To our knowledge this is the first report of its use for this purpose.

### Case report

A 4 year old girl who had previously been well was admitted in a semicomatose state that had developed suddenly after a two week history of lethargy and cough. Amoxicillin and clavulanic acid had been given for one week for a presumed respiratory infection. Initial assessment suggested a diagnosis of meningitis with pneumonia. Investigations showed the following plasma concentrations: sodium 189 mmol/l, potassium 2.8 mmol/l, glucose 22.1 mmol/l (no ketones detected), urea 69 mmol/l, and creatinine 549  $\mu$ mol/l; osmolality was 457 mmol/kg. The haemoglobin concentration was 89 g/l, and platelet count  $14 \times 10^9/l$ ; blood film and clotting studies were otherwise normal. Examination of the cerebrospinal fluid showed a protein content of 0.69 g/l but was otherwise normal. On computed tomography generalised cerebral shrinkage was visible, with no focal abnormalities. A chest radiograph was normal.

Platelets, packed cells, and crystalloid fluids were given. There was persistent oliguria; 30 hours after admission generalised and pulmonary oedema had developed. An endotracheal tube was inserted and intermittent positive pressure ventilation started. A McGaw peritoneal dialysis catheter was inserted percutaneously. Profuse intra-abdominal haemorrhage followed, leading to a hypovolaemic cardiac arrest. After initial resuscitation, the bleeding was controlled at laparotomy. Further peritoneal dialysis was technically impossible because of recurrent intraperitoneal bleeding.

Continuous arteriovenous haemodiafiltration was started to remove fluid, correct the hypernatraemia, and control the azotaemia. Vascular access was through a 22G radial arterial cannula and a 16G right internal jugular venous line. An Amicon Diafilter 20 haemofilter was used; the priming volume (including lines) was 60 ml. Dialaflex 61 peritoneal dialysis solution (Baxter Healthcare) with glucose 1.36% (sodium 132 mmol/l) was infused against the current through the haemofilter at 15 ml/kg/hour, and 10 U/kg of heparin were given at the start of haemodiafiltration followed by infusion of 10 U/kg/hour, adjusted to her coagulation studies.

In addition to maintenance fluids of 10% dextrose with 75 mmol/l sodium chloride, Dialaflex 61 peritoneal dialysis solution with glucose 1.36% was given intravenously as replacement fluid at a rate governed by the hourly ultrafiltrate production to produce a net sensible fluid loss of 500 ml/day. Hypotension necessitated a dopamine infusion of 8  $\mu$ g/kg/minute.

The total effluent was measured with a Ureofix 500 graduated urine collector attached

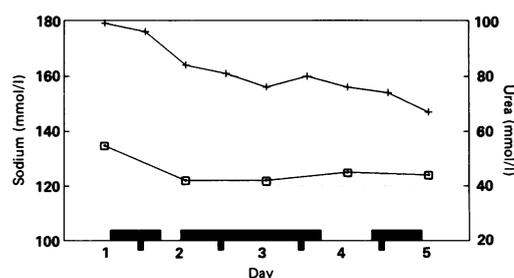


Figure 1 Falls in plasma sodium (upper curve) and urea (lower curve). Periods of continuous arteriovenous haemodiafiltration are shown as solid blocks.

to the ultrafiltrate collection line. Average ultrafiltrate production with systolic blood pressure of over 80 mm Hg was 104 ml/hour, 88 ml/hour with systolic blood pressure of 60 to 80 mm Hg, and 28 ml/hour (for a short period only) when the systolic blood pressure fell below 60 mmHg. The plasma sodium concentration gradually fell to 147 mmol/l over four days; plasma urea stabilised at 44 mmol/l (fig 1). Formal clearance studies were not done.

Technical problems were confined to manipulation of the coagulation studies in the presence of disseminated intravascular coagulation. Three haemofilters were used during the treatment period because of clotting of blood within the circuit. The haemodiafiltration was finally discontinued when ultrafiltrate production stopped as a result of irreversible hypotension.

The child died of diffuse alveolar damage and cardiogenic shock. Influenza A was isolated from the lungs at necropsy, and there was disseminated candidiasis. The kidneys were macroscopically normal; on microscopy there were changes consistent with both acute tubular necrosis and hypernatraemia. The aetiology of the hypernatraemia remains unknown. The source of the intra-abdominal bleeding could not be identified, but the child's hypocoagulable state is presumed to have played an important part.

### Discussion

In continuous arteriovenous haemodiafiltration an ultrafiltrate of plasma is produced by passing blood across the semipermeable membrane of a haemofilter in an extracorporeal circuit using the patient's blood pressure as the driving force. The rate of ultrafiltrate production is difficult to regulate and is dependent on the characteristics of the haemofilter, the blood flow, and the pressure across the membrane. The volume generated is often much more than is needed; replacement fluid in addition to maintenance requirements should be given at a rate governed by the patient's fluid balance and cardiovascular state.

Sodium and other electrolytes are removed but their concentration in the ultrafiltrate is the same as in the plasma. Plasma sodium concentration is therefore unchanged; an alteration is produced by selection of appropriate maintenance and replacement fluids.

In continuous arteriovenous haemodiafiltration, the dialysate fluid is run against the

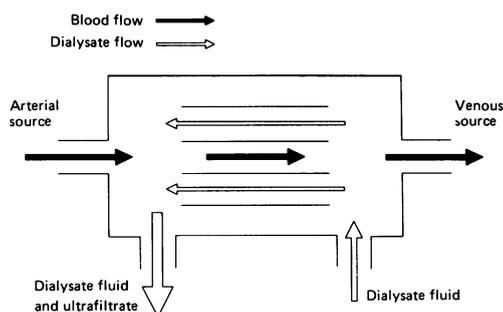


Figure 2 Diagrammatic representation of a haemofilter as used for continuous arteriovenous haemodiafiltration.

current through the haemofilter (fig 2). This achieves a twofold increase in urea clearance.<sup>4</sup> The use of a dialysis solution with a sodium concentration less than that of the plasma being dialysed would theoretically increase sodium clearance compared with that achieved with haemofiltration alone, although no relevant studies have been reported. Thus it removes sodium by the convective transport of haemofil-

tration plus the diffusive transport of haemodialysis. The rate of fall in plasma sodium concentration is regulated by judicious selection of the sodium concentration of dialysate, and maintenance and replacement fluids.

In our experience, it is a simple and safe technique, which does not require specifically trained personnel. A controlled, slow, fall in plasma sodium concentration with adequate removal of fluid was achieved. No complications were associated with its use, and it is a useful alternative in the management of hypernatraemia, particularly in the presence of renal failure.

We thank Dr J Broomhall for his permission to report this patient.

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## Cardiopulmonary bypass for resection of low tracheal haemangioma

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### Abstract

A 3 month old girl, weighing 4000 g, presented with a capillary haemangioma obstructing the lower trachea and left main bronchus; it was not responsive to steroids. Using cardiopulmonary bypass to maintain oxygenation the tumour was excised. We are not aware that this technique has been used before for the resection of such an obstructive haemangioma.

Capillary haemangiomata are common in preterm infants and usually require no treatment. We report a case where surgical excision of the tumour was needed.

### Case report

A 3 month old girl was admitted to hospital from home with a 10 day history of intermittent stridor and two cyanotic episodes during feeding in the previous 24 hours.

She had been born at a supposed 29 weeks' gestation (birth weight 1430 g) and had made excellent progress requiring no artificial ventilation. Feeding was initially by nasogastric tube but orally from 5 weeks of age. A 1 cm diameter capillary naevus was noted on the left side of the neck.

On admission to hospital after the cyanotic

episodes she weighed 4000 g and looked well; there was no cyanosis. There was minimal biphasic stridor, but no increased respiratory effort was evident.

Under general anaesthetic the epiglottis was noted to be short; the upper trachea was normal but a tumour occupied 75% of the lumen of the lower trachea just above the carina: this significantly occluded the left main bronchus. The lower limit of the mass was just visible and the right and left bronchi appeared normal beyond. A computed tomogram confirmed the bronchoscopic appearance of a hemispherical mass within the tracheal lumen that was restricted to the mucosa and was enhanced with contrast; all this suggested a haemangioma (see figs 1 and 2).

Recovery from the investigations was straightforward. She was extubated and breathed spontaneously without undue effort. In view of the preceding symptoms and the obvious risk of further airway obstruction treatment was commenced with 30 mg of prednisolone daily.

Initially her condition remained stable but 72 hours later there was a noticeable increase in respiratory effort and a transient rise in carbon dioxide pressure. Bronchoscopy showed some increase in tumour size. Stability returned but 24 hours later, relatively suddenly, her breathing became increasingly obstructed. Topical

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