

# Non-accidental salt poisoning

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## Abstract

The clinical features of 12 children who incurred non-accidental salt poisoning are reported. The children usually presented to hospital in the first six months of life with unexplained hypernatraemia and associated illness. Most of the children suffered repetitive poisoning before detection. The perpetrator was believed to be the mother for 10 children, the father for one, and either parent for one. Four children had serum sodium concentrations above 200 mmol/l. Seven children had incurred other fabricated illness, drug ingestion, physical abuse, or failure to thrive/neglect. Two children died; the other 10 remained healthy in alternative care. Features are described that should lead to earlier detection of salt poisoning; the importance of checking urine sodium excretion, whenever hypernatraemia occurs, is stressed.

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Non-accidental poisoning is a serious form of child abuse which is becoming recognised more frequently.<sup>1,2</sup> The usual perpetrator is the mother who commonly uses therapeutic and prescribed drugs such as laxatives, hypnotics, or anticonvulsants to make her young child ill.<sup>3</sup> Sometimes household products, including cooking ingredients, pest killers and horticultural agents, are given. One of the more common products used is common salt/table salt/sodium chloride.<sup>4-6</sup>

For many of the children the non-accidental poisoning is part of Munchausen syndrome by proxy child abuse.<sup>7,8</sup> During the past 15 years, nearly 300 such cases have been referred to, and studied by, the author. Altogether 20-25% of the children have incurred non-accidental poisoning as part of the factitious illness abuse. The details of 12 children who incurred salt poisoning are presented together with an analysis of the features and the investigations that may lead to earlier identification of salt poisoning.

## The poisoned children

### CRITERIA FOR DIAGNOSIS

Each child had:

- (1) Illness associated with high serum sodium concentrations and even higher urine sodium concentrations.
- (2) Normal renal, endocrinological, and other investigations during the course of extensive investigation for natural disease.
- (3) Cessation of hypernatraemia and good health when separated from probable perpetrator.
- (4) Circumstantial evidence indicating that either the child's mother or the father was poisoning the child with salt. (For seven children

Table 1 Presentation of 12 children from 10 families

Sex (M/F)	6/6
Age (months) of first confirmed hypernatraemia	1-5-9 (median 2.5)
11 children	41
1 child	1-45 (median 3)
Duration (months) of recurrent hypernatraemia	

the mother confessed to the poisoning and explained how she had done it.)

## PRESENTATION

The main features are outlined in table 1. Usually the child was presented to hospital within the first three months of life because of repetitive illness. Vomiting was the predominant feature, often associated with diarrhoea and failure to thrive. At times there was drowsiness which, on occasion, could amount to coma. Four children had markedly abnormal neurological signs including rigidity, hyper-reflexia, and seizures at the time of hypernatraemia. Six had at least one episode in which he/she became comatose. Unless the child was vomiting excessively, or was comatose, thirst was a usual feature. (One doctor recorded 'every time he came into hospital he drank like a fish'.)

Table 1 shows the age at which hypernatraemia was verified. In several cases there are strong reasons for believing that salt was being given to the child before the hypernatraemia was first identified. In all except two cases the children suffered repetitive poisoning. Poisoning usually recurred for about three months before it was detected, but in one case it continued over a period of 45 months. These periods are minimum times as several of the children had recurrent alleged illnesses before the serum sodium concentration was measured.

All the children were referred initially to hospital paediatricians and were admitted to children's wards. In five cases the degree of illness seemed mild to moderate. Seven children had severe illness, three of whom required ventilation in intensive care units.

## ASSOCIATED PROBLEMS

Table 2 shows that five of the children appeared to suffer only from salt poisoning. However the other seven suffered a combination of problems including other factitious illness and other forms of abuse. One child suffered salt poisoning during admission for genuine pertussis. The other children had no major genuine health problem.

## SODIUM CONCENTRATIONS IN BLOOD AND URINE

The children had repeated estimations of serum sodium concentrations, partly because the

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Table 2 Other problems for 12 poisoned children

Failure to thrive/neglect	4
Recurrent apnoea/seizures	3
Other fabricated illness	3
Other drug ingestion	2
Physical abuse	2
No other abuse/problems	5

clinicians tended to be incredulous at the high concentrations that were reported, and partly because of the detailed testing that the children underwent to find a cause for the recurrent hypernatraemia. The highest serum sodium concentration recorded for each child lay in the range 150–228 mmol/l (table 3). Five children had very high concentrations (190, 210, 213, 216, and 228 mmol/l); the other seven children had highest values in the range 150–175 mmol/l. The three children who required ventilation all had concentrations above 200 mmol/l and one died. But of the five with very high values four, including the one with the serum sodium of 228 mmol/l, survived without apparent brain damage or other long term injury; another child died during follow up, with a serum sodium of 200 mmol/l. The urine sodium concentration at the time of the hypernatraemia ranged from 150–360 mmol/l. Usually it was in the range 200–230 mmol/l. Otherwise the serum electrolytes were unexceptional. The serum chloride, when measured, was raised but the bicarbonate and urea concentrations were usually normal or low. The high urine sodium concentration was accompanied by high urine chloride concentration when that was measured. The high serum sodium concentration usually returned to normal within 24 hours, except in those cases of extreme hypernatraemia where the clinicians deliberately tried to lower the serum sodium concentration more slowly.

### The poisoning

In 10 cases there was circumstantial evidence implicating the mother. (In seven of which the mother confessed to her actions and explained how she had done it – usually adding table salt to the child's drink or putting salt in the child's mouth.) In one case there was circumstantial evidence implicating the father and, for one child, circumstantial evidence implicating the parents without it being clear which one was responsible.

Both the histories and the laboratory findings suggested repetitive poisoning for 10 children. Further information about the mode of poisoning was available for seven of the children: excess salt was identified in the milk that the mother was about to give the child in four cases and in fruit drink for one child. Two further children were found to have exceedingly high salt concentrations in their stomachs and, for one of these, the mother admitted to placing it there via a nasogastric tube. For at least nine of the 12

children there was either definite, or highly probable, evidence that the parent poisoned the child while the child was resident in hospital as well as at home. In addition to the other factitious illnesses and abuse that seven of the children incurred, a number of unusual happenings are worthy of note. Two children yielded extraordinarily high concentrations of sodium and chloride (above 1000 mmol/l) when subjected to sweat tests while in the care of their mothers. Two of the mothers, similarly tested, yielded extraordinarily high concentrations themselves; one of the mothers subsequently admitted to tampering with the test. One mother provided two samples of her breast milk which yielded sodium concentrations of 68 and 165 mmol/l. Subsequent samples collected under nursing supervision all had a sodium content in the range 1.9–3.5 mmol/l. For another child, a reliable and well maintained infusion set suddenly ran in an extra 100 ml of fluid inexplicably while the mother was alone with the baby in a hospital cubicle.

### Outcome for index child

All the children were investigated and assessed through standard child abuse procedures. One child died in hospital at the time of acute hypernatraemia. Ten children were taken into care away from the perpetrator. That served the dual purpose of ensuring the safety of the child and verifying the diagnosis (in that the children immediately became well when separated from the perpetrator, and thereafter had no further episodes of hypernatraemia or unusual illness). One child remaining with the perpetrator died suddenly a few months later (and had a serum sodium of 200 mmol/l shortly before death).

### Siblings

In six of the 10 families there were siblings in addition to the index child. Of the 12 siblings, five appeared healthy and there was nothing to suggest that they were being abused. However seven siblings were incurring, or had incurred, problems; these are listed in table 4.

### Psychopathology of perpetrators

Several of these mothers form part of a more detailed psychosocial study of mothers who fabricate illness and cause their child Munchausen syndrome by proxy abuse. Details of that study will be published. In common with that larger group of perpetrators, most of these were subject to psychiatric evaluation and were not found to have specific mental illness. For the seven cases in which a clear and reliable confession was received, the mother admitted to feelings of hatred and violence to the child in four cases. During extended follow up the mothers of three of these said that they had wished to kill the child.

At the time of detection of the poisoning it was usual for the perpetrators to deny the possibility, and for their partners to be outraged at the suggestion. During court proceedings or criminal investigation such denial usually con-

Table 3 Maximum serum\* sodium and urine sodium concentrations for the 12 children

No of children	Serum sodium (mmol/l)	Urine sodium (mmol/l)
4	200–228	
1	180–200	150–360
5	160–180	(mode 200–230)
2	150–160	

\* Throughout this paper the term 'serum' sodium has been used in relation to concentrations of sodium in the blood. It is probable that some of the samples, particularly for the smaller infants, may have been collected into heparinised containers and that plasma sodium was measured. However that additional fibrin content should not have affected significantly the reported sodium value.

Table 4 Problems found in seven of 12 siblings

Salt poisoning	2
Other fabricated illness	3
Failure to thrive/neglect	4
Physical abuse	1
Sudden unexpected death at age 1 year	1
No abuse	5

tinued. Confession, if it came, usually emerged slowly and in response to the therapeutic efforts of social workers and doctors. However three mothers began to admit part of the abuse during child care proceedings, though without true indication of motive. In one case the full details of the way in which the mother had poisoned the child did not emerge until two years later and, in another, eight years later.

#### Useful data for diagnosing salt poisoning<sup>9-12</sup>

- 1 g of sodium chloride contains 17 mmol of sodium.
- One level 5 ml teaspoon of table salt holds nearly 6 g of salt, that is approximately 100 mmol of sodium.
- One level 10 ml dessertspoon holds nearly 12 g of salt, that is approximately 200 mmol of sodium.

The usual serum sodium concentration is in the range of 135–143 mmol/l, subject to slight variation according to the laboratory and assay used. While there are at least two notorious causes (hyperlipidaemia and paraproteinaemia) of spurious hyponatraemia, hypernatraemia is most unlikely to be spurious or the result of laboratory error.

The body contains approximately 60 mmol of sodium per kg and 50 mmol of potassium per kg.

A child's sodium intake comes from food. Although salt intake varies considerably between different children, it is unlikely to exceed 4 g (65 mmol sodium) per day for children under the age of 2 years. From 4–6 years, daily intake ranges from 20–130 mmol. Excess salt is lost primarily by the kidneys, smaller amounts are lost in sweat and faeces (table 5).

Compared with an adult a child has a relatively smaller glomerular filtration rate, but the tubular reabsorption of sodium is similar to that of adults. This limits the child's capacity to excrete excessive sodium loads, particularly if deprived of water. Urinary chloride excretion follows, and is similar in amount to, sodium excretion.

If the urinary sodium excretion is known it is possible to calculate the sodium intake and the amount of salt that has been given to the child:

The child can be assumed to have a glomerular filtration rate (GFR) of 120 ml/min/1.73m<sup>2</sup> (a child under the age of 2 will have a GFR less than this). Thus an 18 month old child, with a surface area of 0.5 m<sup>2</sup> will have a GFR of 35 ml/min. As

Table 5 Sodium loss

Age (months)	Loss in mmol/kg body weight		Usual loss in mmol/24 hours	
	Sweat and faeces	Urine	Sweat and faeces	Urine
1–12	0.8	1–3	3–7	5–30
12–24	0.7	1–3	7–10	10–45

serum contains about 140 mmol/l (or 140 µmol/ml) the filtered load of sodium will be 35 ml × 140 µmol = 4900 µmol/min. Under usual circumstances the kidneys can only excrete 10% of the filtered load – that is 490 µmol/min or approximately 0.5 mmol/min. In 24 hours that is 490 × 60 × 24 = 705 600 µmol, or approximately 705 mmol of sodium; 705 mmol of sodium is contained in 705/17 = 41 g of sodium chloride. Thus the child has been given more than three dessert spoons of salt.

The quantity of salt needed to cause a specific level of hypernatraemia can be calculated as follows:

If an infant weighing 10 kg has a serum sodium of 216 mmol/l, that represents an excess of (216–140) = 76 mmol/l. A 10 kg infant has approximately 6 litres of water of which 2.5 litres is extracellular fluid. Therefore the excess sodium is represented by 76 × 2.5 = 190 mmol; 190 mmol of sodium is contained in (190/17) = 11 g of salt (just under two teaspoons full).

The usual urine sodium potassium ratio is 2.8 (range 1.4–5.2). The usual urine sodium:creatinine ratio is 39.

Gastric aspirate usually contains 50–60 mmol/l of sodium. If concentrations of over 200 mmol/l are identified, it is highly suggestive that salt has been ingested.

Doctors prescribe salt in the form of intravenous solutions and as oral rehydration therapy for ill children. The quantity of salt prescribed is relatively small with strong intravenous solutions, such as Plasma-Lyte (Baxter), containing 140 mmol of sodium per litre and five sachets of Dioralyte (Rorer) providing 60 mmol of sodium.

#### Discussion

There are two main reasons why salt poisoning is essentially a problem of infants and young children rather than older children or adults. Firstly the immature kidney has limited ability to excrete an excess sodium load and secondly an infant or young child can be denied access to water, which an older child or adult would obtain in order to quench thirst and thereby excrete the sodium load. Thus for young children salt poisoning tends to be a combination of excess salt intake plus restriction of fluid.<sup>13,14</sup> That same combination of circumstances accounts for the occurrence of salt poisoning in comatose older children or adults who are accidentally given too much sodium as part of intravenous therapies.<sup>15,16</sup> It is also similar to the sort of salt poisoning that is commonly seen in finishing pigs and other animals reared in farm factory conditions with limited supplies of drinking water and excess salt in their feeds.<sup>17,18</sup>

Whether or not water deprivation alone could cause very high sodium concentrations is questionable. It is most doubtful that it could in an older child – an adult prisoner on thirst strike for 13 days became extremely ill but had a serum sodium of only 164 mmol/l.<sup>19</sup> In 1970, Pickel and colleagues reported three children, in the age range 2.5 to 7.5 years, who were deprived of water by abusing parents and who became hypernatraemic.<sup>20</sup> Their highest serum sodium concentrations were 194, 201, and 183 mmol/l.

There is no doubt that the children had been abused and were deprived of water, but in retrospect one must hypothesise that the children may have been given excess salt also. Unfortunately the authors were not able to study urine sodium output at the time (M A Holliday, personal communication).

Though two teaspoons full of salt (needed to poison an infant) does not sound such a large amount, it does cause a strong and unpleasant taste in any drink to which it is added. Some mothers ensured that it was added to drinks that were flavoured strongly with banana or other fruit flavours; nevertheless it is, as we have found, very difficult to get young children to drink fluids with a strong salt flavour. That supports the contention that most of these children will have been deprived of other drinks in order to ensure that they would drink the unpleasant salty one. Alternative means of inducing salt ingestion included putting a sachet of salt in the mouth and using a nasogastric tube (the mother was an ex-nurse). In another case it is probable that salt solution was introduced by tube into the rectum.

In the cases reported here the clinicians sought, and excluded, many alternative diagnoses before alleging salt poisoning. It was common for hypernatraemic dehydration, as a result of gastroenteritis, to be considered. Hypernatraemic dehydration, which used to be common in Europe and North America 20 or 30 years ago, was thought to be due to a combination of hypotonic stool water loss, increased evaporative water loss, limited renal conservation of water, as well as inappropriately high solute feeds.<sup>21,22</sup> Even when hypernatraemic dehydration was a relatively common occurrence, it was exceptional for the serum sodium concentration to exceed 180 mmol/l. Moreover the hypernatraemia usually was associated with an acidosis and a raised plasma urea. The blood electrolyte values of the poisoned children reported in this paper showed disproportionately high sodium values with relatively little alteration of bicarbonate or urea. Similarly when there is excessive renal water loss in diabetes insipidus, serum creatinine and urea concentrations are raised – not just the sodium, and it is very rare for the sodium to reach a level of 180 mmol/l. For most of the poisoned children diabetes insipidus had been excluded by a water deprivation test. There have been reports of children with hypothalamic disorders and defective thirst centres who develop a chronic hyperosmolar state because of limited fluid intake. That rare condition is excluded if a child has normal thirst and if, in response to demand feeding, he/she achieves normal electrolyte values. In practice if a child presents with the isolated abnormality of a serum sodium of more than 160 mmol/l, for which there is no obvious explanation, evidence of excess sodium intake should be sought. Urine should be collected at once to establish whether an excess sodium load is being excreted; a random sample will suffice (both urine sodium concentration and the more quantitative sodium:creatinine ratio should be measured); a timed collection is the ideal. If there is high sodium excretion, then salt inges-

tion is the most likely cause. Common table salt, sodium chloride, is the likely agent though there have been examples of other sodium products, such as sodium bicarbonate, being the cause. It is important to remember that accidental salt poisoning can occur as a result of either parents or medical staff making mistakes with feeds or prescribed solutions.

Bearing in mind the very high serum sodium values recorded for these children, it is surprising that only two of them died at the time of recorded hypernatraemia. The others survived those high concentrations without apparent long term damage. Hypernatraemia is known to cause cellular dehydration, particularly within the brain.

Neurological symptoms and signs are common including irritability, hyper-reflexia, seizures and coma.<sup>23,24</sup> Both the morbidity and mortality from the hypernatraemic dehydration, associated with gastroenteritis, used to be high even though the serum sodium concentrations were only in the range 155–175 mmol/l. Presumably there were other factors contributing to that morbidity because children seem able to withstand the hypernatraemia of both accidental, and non-accidental, salt poisoning very much better. There is one report of an infant surviving a serum sodium of 274 mmol/l, after accidentally receiving excess salt in an infant formula.<sup>13</sup> For some children who receive excess salt, either accidentally or non-accidentally, the reason for their tolerance to very high sodium values may be because of the chronic or repetitive nature of the poisoning and the fact that their brain adapts to it. In experimental animals brain water increases rapidly during the first few hours of acute hypernatraemia, but after nine hours 'idiogenic osmoles' account for an increasing proportion of the intracellular osmolality.<sup>25</sup> After seven days of hypernatraemia the water content of the brain has returned to normal, though the osmolality remains raised, with up to 60% of that increase being the result of idiogenic osmoles, which may include amino acids.<sup>26</sup> These osmoles help to restore intracellular osmolality and cell volume in the brain. For this reason it is wisest to correct such hypernatraemia gradually and to aim to correct the electrolytes over a period of at least 24 hours. With acute poisoning the risks of over energetic treatment are less, though the clinician is likely to use hypotonic intravenous fluids carefully ensuring that there is good urine output and that fluid overload does not occur.

When infants have died as a result of salt poisoning, necropsy has revealed extensive haemorrhages particularly involving the brain.<sup>15</sup> Sometimes these have been described as a haemorrhagic encephalopathy and it has been presumed that the intracerebral bleeds, thromboses, and necrosis have been caused by changes in cell volume, tissue shrinkage and tearing of blood vessels. It is questionable whether specific areas of the brain are more at risk. A study by computed tomography of an infant who developed hypernatraemia as a result of salt poisoning showed low density lesions in the basal ganglia, especially the putamen.<sup>27</sup> The authors suggested that this was because the putamen was more vascular than the adjacent white matter and

that extracellular oedema and blood vessel engorgement could be accounting for the low density appearance. The appearance returned to normal once the hypernatraemia was corrected.

Most of the poisoned children suffered repetitive poisoning. Not only was that a reason why their brains may have withstood the high sodium concentrations without long term damage, but it was also one of the main reasons why the salt poisoning was detected. It is likely that many children who are merely poisoned once, or who are poisoned to a lesser extent and do not have such extraordinarily high serum sodium concentrations, go undetected. Although seven of these children, on detailed study, probably had suffered other abuse it was the salt poisoning that was the main form of abuse and the factor that invoked child abuse investigation procedures. Because of the author's long standing interest in Munchausen syndrome by proxy abuse, this report may under represent cases of salt poisoning that are single and cases that do not fit into the extended spectrum of factitious illness by proxy. The current epidemiological survey of Munchausen syndrome by proxy (including non-accidental poisoning and suffocation) conducted by the British Paediatric Surveillance Unit should provide useful information about the incidence of salt poisoning as well as the frequency of poisoning in children suffering factitious illness.

Some of the parents of these children are the subject of a more detailed psychosocial study which is nearing completion. As a group they fell within the personality types usually associated with Munchausen syndrome by proxy abuse. Similarly, as table 4 shows, there was the usual co-morbidity in the siblings of the index children, which is found in Munchausen syndrome by proxy abuse.<sup>28</sup> At the time of initial confrontation it was customary for the perpetrating parent to deny the abuse; the denial tended to extend during the time of any police investigation or child care proceedings. None admitted to hatred or intent to kill the child at that stage. The opportunity to meet certain mothers at periods ranging from 1–10 years after the initial poisoning led to the mothers of three children giving fuller versions of the poisoning and stating that they had wished to kill their children. It does seem that salt poisoning is an indicator of an extremely disturbed mother-child relationship, and may be combined with considerable hostility and violence to the child. A particular worry for those assessing the families was the fact that several mothers had poisoned their child again even though they had seen that excess salt caused their child to be extremely ill. In those cases the mother's need to poison the child completely outweighed the safety and welfare of the child. Those responsible for children who have been

poisoned with salt should address the cases with caution, ensure that appropriate psychosocial assessment takes place, and that the child's future is safeguarded.<sup>29</sup>

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- 1 Kempe CH. Uncommon manifestations of the battered child syndrome. *Am J Dis Child* 1975; **129**: 1265–8.
- 2 Rogers D, Tripp J, Bentovim A, Robinson A, Berry D, Goulding R. Non-accidental poisoning: an extended syndrome of child abuse. *BMJ* 1976; **i**: 793–6.
- 3 Meadow R. Poisoning. *BMJ* 1989; **298**: 1572–4.
- 4 Feldman K, Robertson WO. Salt poisoning: presenting symptom of child abuse. *Veterinary Medicine and Human Toxicology* 1979; **21**: 341–3.
- 5 Baugh JR, Krug EF, Weir MR. Punishment by salt poisoning. *South Med J* 1983; **76**: 540–1.
- 6 Nicol AR, Eccles M. Psychotherapy for Munchausen syndrome by proxy. *Arch Dis Child* 1985; **60**: 344–8.
- 7 Meadow R. Munchausen syndrome by proxy. The hinterland of child abuse. *Lancet* 1977; **ii**: 343–5.
- 8 Meadow R. Munchausen syndrome by proxy. *Arch Dis Child* 1982; **57**: 92–8.
- 9 Winters RW. *The body fluids in pediatrics*. Boston: Little Brown and Co, 1973.
- 10 Department of Health. *Report on health and social subjects No 41. Dietary reference values for food, energy and nutrients for the United Kingdom*. London: HMSO, 1991.
- 11 Allison ME, Walker V. The sodium and potassium intake of 3–5 year olds. *Arch Dis Child* 1986; **61**: 159–63.
- 12 De Courcy S, Mitchell H, Simmons D, MacGregor G. Urinary sodium excretion in 4–6 year old children: a cause for concern? *BMJ* 1986; **292**: 1428–9.
- 13 Finberg L, Kiley J, Luttrell CN. Mass accidental salt poisoning in infancy. *JAMA* 1963; **184**: 121–5.
- 14 Baars J, Hill L, Hill R, Martinez WM. Fatal poisoning from salt used as an emetic. *Am J Dis Child* 1973; **125**: 889–90.
- 15 Walter GF, Maresch W. Accidental saline poisoning in newborn infants. Morphologic findings and pathogenetic discussion. *Klin Padiatr* 1987; **199**: 269–73.
- 16 El-Dahr S, Gomez A, Campbell FG, Chevalier RL. Rapid correction of acute salt poisoning by peritoneal dialysis. *Pediatr Nephrol* 1987; **1**: 602–4.
- 17 Heinritz K. Poisoning in swine. *Tierarztl Prax* 1986; **14**: 219–30.
- 18 Barrs AJ, Spierenburg TJ, van Leengoed LA, Beersma PF, van Dijk KG, Groenland GJ. A case of common salt poisoning in slaughtering pigs. *Tijdschr Diergeneesk* 1988; **113**: 933–5.
- 19 Neeser M, Ruedin P, Restellini JP. 'Thirst strike': hypernatraemia and acute prerenal failure in a prisoner who refused to drink. *BMJ* 1992; **304**: 1352.
- 20 Pickel S, Anderson C, Holliday MA. Thirsting and hypernatremic dehydration – a form of child abuse. *Pediatrics* 1970; **45**: 54–9.
- 21 Finberg L, Harrison HE. Hyponatremia in infants: an evaluation of the clinical and biochemical findings accompanying this state. *Pediatrics* 1955; **16**: 1–12.
- 22 Weil WB, Wallace WM. Hypertonic dehydration in infancy. *Pediatrics* 1956; **17**: 171–81.
- 23 Finberg L. Pathogenesis of lesions in the nervous system in hypernatremic states. I. Clinical observations of infants. *Pediatrics* 1959; **23**: 40–4.
- 24 Finberg L, Luttrell C, Redd H. Pathogenesis of lesions in the nervous system in hypernatremic states. II. Experimental studies of gross anatomic changes and alterations in the chemical composition of the tissues. *Pediatrics* 1959; **23**: 46–53.
- 25 Arieff AI, Guisado R. Effects on the central nervous system of hypernatremic and hyponatremic states. *Kidney Int* 1976; **10**: 104–16.
- 26 Thurston JH, Hanhart RE, Dirgo JA. Taurine: a role in osmotic regulation of mammalian brain and possible clinical significance. *Life Sci* 1980; **26**: 1561–8.
- 27 Habbick BF, Hill A, Tchang SP. Computed tomography in an infant with salt poisoning: relationship of hypodense area in basal ganglia to serum sodium concentration. *Pediatrics* 1984; **74**: 1123–5.
- 28 Bools C, Neale B, Meadow R. Co-morbidity associated with fabricated illness (Munchausen syndrome by proxy). *Arch Dis Child* 1992; **67**: 77–9.
- 29 Neale B, Bools C, Meadow R. Problems in the assessment and management of Munchausen syndrome by proxy abuse. *Children and Society* 1991; **5**: 324–33.