Apparent disappearance of hypernatraemic dehydration from infant deaths in Sheffield

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Summary and conclusions

The death certificates and necropsy reports of the 1115 Sheffield infants who died under 2 years of age in 1969-78 were examined. This study showed the apparent disappearance of deaths with hypernatraemia and in particular deaths presenting as cot deaths.

The fall in incidence may be as much the result of an intensive local campaign on child care and infant feeding as of the change in the composition of dried milk for baby feeding.

Introduction

Hypernatraemia has been recognised as a complication of infantile diarrhoea since 1911.¹ In 1972 it was suggested that hypernatraemia might be potentiated by overconcentrated feeds.² Further studies have shown that babies fed on cows' milk have a higher plasma osmolality³ and higher urea concentration than breast-fed babies,⁴ and that 172 out of 302 (57%) Sheffield mothers prepared overconcentrated feeds.⁵

In 1974 it was shown that 12 out of 25 infants who died unexpectedly had vitreous humour concentrations of sodium and urea that indicated severe electrolyte disturbance.⁶ It was postulated that the hypernatraemia was partly due to the infants being fed concentrated feeds and that high-solute feeding and water deficiency might be a factor in some unexpected infant deaths. This was supported by a contemporaneous study in this country⁷ but not by a recent study in Denmark.⁸ We have noted a very great reduction of vitreous hyponatremic states in infants who have died recently in Sheffield and thought that the local epidemiology of the condition needed study. We attempted to ascertain the importance of hypernatraemia as a factor in the deaths of Sheffield children during the years 1969 to 1978.

Methods

Copies of the death certificates of all infants aged under 2 years who died within the Sheffield city boundary from 1969 to 1978 were obtained from the Sheffield Office of Population Censuses and Surveys. Details of the Sheffield infants who died outside the city boundary were added and a total of 1735 certificates scrutinised. The home addresses on the certificates were checked, and we found that 620 infants of non-Sheffield residents had died in the city, mostly in teaching hospitals. There were, therefore, 1115 infants of Sheffield residents who died in 1969-78.

The records of the laboratories in the city where infant necropsies are performed were searched and the reports scrutinised. No child under 1 week had died of gastroenteritis or dehydration. Necropsies

had been carried out on 65% of the total infants and on 77% of the infants dying between 1 week and 2 years; 73% of these latter necropsies were carried out at the Sheffield Children's Hospital, where the vitreous electrolytes have been examined routinely since October 1972. There has been no change in the selection policy for necropsies, and all coroners' necropsies on children have been carried out at the Children's Hospital throughout the period.

The vitreous sodium was measured by integrated flame photometry and the urea by the urease-Barthelot method. Before October 1972 details of biochemistry were too scanty for statistical validity. The variation in the proportion of postperinatal deaths in which a vitreous examination was performed (table I) is accounted for by deaths from major congenital abnormalities. In all cases of cot death a vitreous examination was performed.

We used a clinical definition of hypernatraemia, where a sodium concentration in excess of 150 mmol(mEq)/l was taken as abnormal, although at the Children's Hospital laboratory, a level of 146 mmol/l is greater than two standard deviations above the mean.

Results

Table I shows the complete figures obtained in this study. Columns six and seven are not mutually exclusive: infants who died with gastroenteritis and hypernatraemia are recorded in both columns (see table II).

At the Children's Hospital from October 1972 to December 1978 228 necropsies were performed on Sheffield infants dying under 2 years and over 1 week of age. We found that 23 of these infants had vitreous hyponatremia. The principal causes of death of these children as given on the necropsy record are listed in table II.

The decline in hypernatraemia as a factor in these deaths was dramatic: by the regression method outlined in Maxwell,⁹ the downward trend over the period 1972-78 in the proportion of all deaths attributable to hypernatraemia was statistically significant at the 0.1% level. The steady decline in 1969-78 in the proportion of all deaths attributable to gastroenteritis was statistically significant at the 1% level.

Feeding histories were obtained from the mothers of 20 of the 23 infants who died with hypernatraemia and from age-matched living controls drawn from the Sheffield birth register.¹⁰ Equal numbers of cases and controls received unmodified milks but four controls were breast-fed beyond six weeks whereas none of the infants who died were. There was no difference between cases and controls in the introduction of cereals or solids into the diet in the last three weeks of life, nor was there any difference in the number receiving cereals or solids by the date of death. The most striking difference was that seven of the infants who died had complete changes of milk formula whereas no controls did.

Discussion

There are many possible explanations for the disappearance of hypernatraemia, none of which are mutually exclusive. The four most important factors probably were: (a) the gradual reduction in the incidence of gastroenteritis; (b) the removal of unmodified (high sodium) milk preparations; (c) an increased incidence of breast-feeding; and (d) a local campaign to increase awareness of the dangers of overconcentrated feeds and promote increased care in the reconstitution of dried milk feeds.

The fall in the number of deaths from gastroenteritis cannot wholly explain the fall in the number of hypernatraemic deaths because gastroenteritis was not present in 15 of the 23 infants

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TABLE I—Complete data for the areas covered by Sheffield County Borough, 1969-74 and Sheffield Metropolitan Borough 1974-8

	Total live births	Total deaths (0-2 yr)	Postperinatal deaths (1 wk-2 yr)	% of postperinatal deaths with vitreous examination	No of deaths with hypernatraemia (per 1000 live births)	No of deaths from gastroenteritis (per 1000 live births)	No of cot deaths (per 1000 of live births)
1969	8465	150	73			14 (1.7)	28 (3.3)
1970	8214	170	83			12 (1.5)	34 (4.1)
1971	7875	168	71			8 (1.0)	27 (3.4)
1972	6994	126	64			8 (1.1)	30 (4.3)
	1551*	34*	12*	63*	6*	1*	9*
1973	6100	104	37	79	10 (1.6)	7 (1.1)	20 (3.3)
1974	6810	103	22	63	5 (0.7)	6 (0.9)	11 (1.6)
1975	5739	83	19	59	1 (0.2)	2 (0.3)	13 (2.3)
1976	5525	78	23	74	1 (0.2)	1 (0.2)	11 (2.0)
1977	5450	78	19	51	0 (0)	1 (0.2)	12 (2.2)
1978	5536	55	15	56	0 (0)	2 (0.4)	7 (1.3)

*October-December 1972 only.

who died with hypernatraemia (table II). Of these 15 deaths, 12 would previously have been classified as unexpected infant deaths with minimal pathology.

The fall in the number of hypernatraemic deaths in Sheffield began before the DHSS working party had published its findings in 1974¹¹ and before unmodified milks, especially National Dried Milk, were removed in 1975. Breast-feeding in Sheffield has increased gradually, and by 1977 44% of babies were receiving breast milk at one month, 32.3% being wholly breast-fed and 11.5% receiving breast and some bottle feeds (E W Pursall, personal communication). This trend is being led by the most educated mothers.¹²

TABLE II—Indexed cause of death of infants dying with hypernatraemia (October 1972-December 1978)

	Total	Year of death					
		1972†	1973	1974	1975	1976	1977-8
Gastroenteritis	8	1	4	2		1	
Meningitis	1		1				
CNS abnormality	2		1	1			
Laryngotracheobronchitis ..	5*	2	2	1			
Malabsorption	1*		1				
Endocardial fibroelastosis ..	2*	1		1			
Unexplained sudden death	4*	2	1		1		
Total	23	6	10	5	1	1	0

*Presented as sudden unexpected infant deaths.

†October to December only.

A local campaign to encourage the correct dilution of dried milk feeds may have played a major part in improving infant care. This campaign started as soon as the vitreous abnormalities were discovered in 1972 and reached a climax during the spring of 1974, with intensive press and radio coverage. The campaign was co-ordinated by a small working party with the specialist in community medicine (child health) as chairman. This committee took four main avenues of action. Firstly, every mother in the lying-in wards who did not want to breast-feed was individually taught how to make up powdered feeds correctly. She was also given a leaflet which outlined the dangers of overconcentrated feeds and advised that only boiled water should be offered between feeds. Secondly, meetings were arranged with every midwife and health visitor and many family doctors, when the importance of hypernatraemia and Smith's results⁵ were presented. Thirdly, there was a detailed press handout, followed up by many interviews being given by medical staff. Finally, the coroner commented publicly on some cases in which hypernatraemia was a factor in the death. The resulting public interest generated further local press and radio coverage.

A decline in "hypernatraemic" deaths has apparently occurred in another area.¹³ In an earlier letter from the same area¹⁴ it was noted that nine out of 11 infants admitted with hypernatraemia in 1975-6 had been fed on low solute milks. The measures taken in Sheffield to ensure the correct dilution of milk powder

may have had a greater effect than those taken elsewhere to modify the solute load of the milk. Biering-Sorensen *et al*⁸ in a detailed article on infant feeding and the sudden infant death syndrome reported that, in Denmark, many babies are fed on diluted fresh cows' milk and very few on powdered milks. Unfortunately, they did not report on the sodium or osmolar loads of these milks but they concluded that there was no evidence that infants who suffered unexpected deaths got more concentrated feeds than controls who did not die.

Hypernatraemia related to cot death is important because of apparently conflicting reports on the subject. There has been no case of hypernatraemia in a Sheffield-born infant presenting as a cot death from December 1976 to the time of writing (July 1979). Thus the report in 1974 that hypernatraemia was present in a significant number of cot deaths⁶ is no longer true for Sheffield children. This reduction in "hypernatraemic" deaths was associated with a reduction in cot deaths in the same period. Deaths with hypernatraemia have disappeared while cot deaths have continued at a lower rate. There are many contributory factors to cot death, and hypernatraemia seems to have been one of these in Sheffield. Thus the importance of hypernatraemia in unexpected infant deaths is likely to vary greatly between different places, and reports from areas with a low cot death rate will probably show a picture such as that which exists in Sheffield today and not that of 1973-4. Possibly the incidence of deaths or hospital admissions with hypernatraemia could be used as an index of the state of the infant feeding in the community.

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