

Oral Water Intoxication in Infants

An American Epidemic

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- Between 1975 and 1990, a total of 34 patients with water intoxication were treated at St Louis (Mo) Children's Hospital, 24 of these in the last 3 years, indicating a marked increase in incidence of this previously rare condition. Thirty-one were infants living in poverty who ingested excessive amounts of water offered at home by their caretakers. Exhaustion of the supply of infant formula was the most common reason given for this substitution. Infants were treated by a single infusion of hypertonic saline or a slow infusion of isotonic saline. Central pontine myelinolysis was not observed as a complication of hypertonic saline therapy. Modification of the Special Supplemental Food Program for Women, Infants, and Children to provide sufficient formula for the growing infant and better education of mothers as to the hazards of excessive water ingestion might reduce the incidence of this preventable and life-threatening condition.

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In 1922, Larson et al¹ described the acute neurologic disturbance that results from rapid, excessive water intake. Water intoxication has been considered a rare clinical event that follows excessive parenteral or enteral water administration by medical personnel,² malicious forcing of water on a child,³ repeated immersion (infant swimming lessons),⁴ or voluntary ingestion of iced water

See also p 981.

to control toothache⁵ or occurs during marathon runs,⁶ after drug testing,⁷ or as a manifestation of psychosis.⁸ In 1967, Dugan and Holliday⁹ provided the first description of infants who developed water intoxication after the voluntary ingestion of dilute formula. During the following decade and a half, 21 additional examples of healthy in-

fants suffering from water intoxication by the oral route in institutions in five US cities appeared in the medical literature.¹⁰⁻¹⁴ In 1987, Medani¹⁵ described 19 such infants at a single Baltimore (Md) hospital. The possibility that an epidemic is occurring is suggested by these reports and our own experience in the years 1975 through 1990. The details of the clinical syndrome are provided to guide clinicians in the recognition and treatment of the illness. Preventive interventions applicable to the population at risk are discussed.

PATIENTS AND METHODS

St Louis (Mo) Children's Hospital is a 235-bed facility with 50 000 emergency room visits per annum that serves the health needs of a large geographic region. Approximately 60% of patients have private insurance, and 40% are enrolled in Medicaid. There was a modest increase in the number of urban poor cared for when two small pediatric services in municipal hospitals closed 5 to 10 years ago. A second children's hospital (Cardinal Glennon Memorial Children's Hospital) also serves the region's pediatric needs, but no fluctuation in the acute care activities of either children's hospital occurred during the study period of a magnitude that would explain the first appearance and recent increase in the number of infants with oral water intoxication described in this report.

The records of all patients diagnosed as having water intoxication (*International Classification of Diseases, Ninth Edition*, 276.6) were obtained from the Medical Records Department and the Pediatric Gastroenterology Registry at St Louis Children's Hospital for the period from January 1975 through July 1990. All records that contained a description of an acute neurologic syndrome occurring in association with hyponatremia were selected for detailed review and analysis. Information was abstracted from notes of physicians, social workers, emergency medical technologists, and nurses. Follow-up information was sought by telephone interview with each child's current physician or parent if adequate information was not present in the child's chart.

The 34 patients identified by this process were divided into group 1 (infants) (n = 31) and group 2 (children) (n = 3) for further analysis and to facilitate discussion. The infants were further subdivided by the treatment given (hypertonic saline [group 1a] or a slow infusion [1.0 L/m² per day] of isotonic fluid [group 1b]). A patient's treatment was chosen by the clinicians at the time rather than by randomized assignment as would be done in a prospective study of the efficacy of the therapies. Infants in group 1b were more likely to have been recognized to have water intoxication late in their course; a brisk water diuresis had already occurred. Infants in group 1a received a single infusion of 3% saline given during 30 to 90 minutes. The dose was calculated

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Patient/Age, mo	Treatment	Serum Sodium					Respiratory Failure	
		Admission, mmol/L	Rate of Increase, mmol·L ⁻¹ ·h ⁻¹	Time Until Normal, h	Temperature, °C [†]			
					Group 1a (n=17)	Group 1b (n=14)		
1/3	3%	116	10.0	2	36.0		—	
2/9	3%	114	2.5	8	35.4		—	
3/6	3%	114	2.0	8	36.7		+	
4/1	3%	116	1.7	14	35.0		—	
5/3	3%	114	3.0	6	35.0		+	
6/4	3%	116	2.5	12	36.5		—	
7/6	3%	115	1.7	14	35.0		+	
8/4	3%	119	1.8	10	35.0		+	
9/4	3%	112	1.7	13	34.0		+	
10/3	3%	112	1.7	15	35.2		+	
11/4	3%	115	2.8	15	35.2		—	
12/3	3%	112	2.2	8	35.0		—	
13/4	3%	118	3.6	5	36.4		—	
14/4	3%	116	4.4	5	35.4		+	
15/4	3%	117	4.4	5	34.2		+	
16/4	3%	114	2.7	8	36.7		+	
17/10	3%	115	3.3	6	34.0		—	
Mean ± SD (range)	4.5 ± 2.2 (1-10)	... 115.0 ± 2.0 (112-119)	3.05 ± 2.0 (1.7-10.0)	9.06 ± 4.1 (2-15)	35.3 ± 0.9		9 (53%)	
Group 1b (n=14)								
18/4	NS	118	3.0	5	34.0		+	
19/4	0.25 NS	117	2.0	10	35.4		—	
20/10	RL	119	1.8	10	36.0		—	
21/3	RL	112	1.2	16	34.0		+	
22/2	RL	122	2.0	2	36.7		—	
23/4	NS	116	3.0	15	36.0		—	
24/4	NS	125	1.5	10	36.4		—	
25/3	NS	119	1.4	11	35.5		—	
26/2	RL	117	2.0	12	36.5		—	
27/5	NS	119	1.2	10	35.4		+	
28/4	PO	123	3.0	4	36.4		+	
29/5	RL	116	1.5	12	37.0		+	
30/2	PO	127	1.0	8	36.0		—	
31/2	NS	111	1.8	12	35.5		+	
Mean ± SD (range)	3.86 ± 2.07 (2-10)	... 118.6 ± 4.5 (111-127)	1.89 ± 0.68 (1.0-3.0)	9.79 ± 3.95 (2-16)	35.8 ± 0.9		6 (43%)	
Group 2 (n=3)								
32/8 y	0.5 NS	120	3.0	5	36.2		—	
33/3 y	5%	114	2.6	8	34.7		+	
34/2 y	3%	112	7.0	30	<34.0		+	

*Group 1a consisted of infants intoxicated by the oral route, treated by infusion of 3% saline. Group 1b consisted of infants intoxicated by the oral route, treated with normal saline (NS), Ringer's lactate (RL), or infant formula (PO). Group 2 consisted of children intoxicated by the parenteral or oral route. The difference (1a vs 1b) in initial serum sodium level (115.0 vs 118.6 mmol/L) was statistically significant ($P<.05$). The differences (1a vs 1b) in age, sex, race, rate of serum sodium level increase, time until sodium level was normal, temperature, and proportion of patients with respiratory failure failed to reach statistical significance.

[†]Temperatures are given as rectal. In seven infants, rectal temperatures were not measured; the number given is their axillary temperature transformed to rectal. In the three infants with temperatures of 34°C, that was the lowest reading possible on the thermometer; they may have been colder.

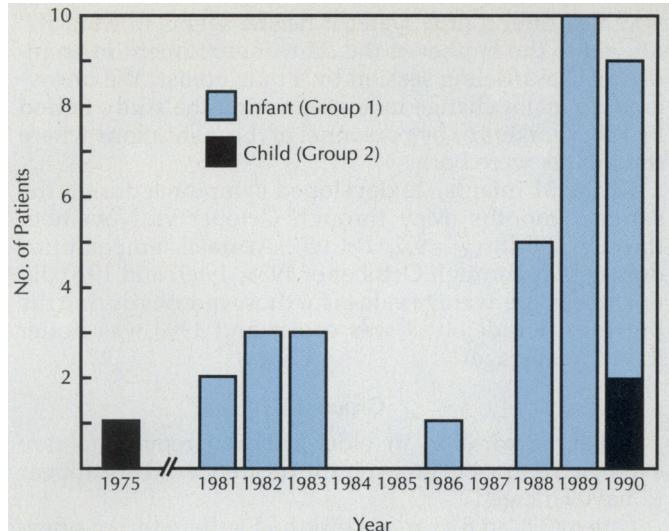


Fig 1.—Frequency distribution by calendar year of all patients diagnosed as having water intoxication between January 1975 and December 1990.

as follows: (body weight in kilograms)(125 mEq/L – initial serum sodium level)(0.6), or about 10 mL of 3% saline per kilogram of body weight.

We obtained records of ambient temperature in St Louis during the years 1988, 1989, and 1990 from the National Oceanic and Atmospheric Administration, US Department of Commerce (Asheville, NC) and compared these with the average temperatures in this area during the last three decades.

The local offices of the Supplemental Food Program for Women, Infants, and Children (WIC) of the State of Missouri/US Department of Agriculture were visited to gather data concerning the availability of food supplements and nutritional advice provided to families in our area during the period of the study. The instructions provided to new mothers by physicians and nurses at St Louis Regional, St Louis Jewish, and Barnes Hospitals (hospitals where most of the infants had been born) were reviewed. Particular attention was paid to changes in allotments of formula and to any advice that might have fostered the substitution of water for formula.

Availability of a clinical microchemistry laboratory and the practice of measuring serum electrolytes in the first sample taken from a convulsing child had not changed during the period of the study.

Descriptive and comparative statistics were calculated by conventional methods, and comparison of means was carried out by *t* test. Comparison of ratios of discrete variables was carried out with the χ^2 test. All tests were two tailed. $P < .05$ was considered statistically significant.

RESULTS

Thirty-four patients were treated for water intoxication, the first infant in 1982 and 24 during the last 3 years (1988 through 1990) (Fig 1). Selected data from each patient are seen in Table 1.

Group 1

The mean \pm SD age of the 31 infants was 4.2 ± 2.1 months; 17 were male. The illness started suddenly with convulsions or apnea. Seizures occurred in all infants and usually (28 of 31 cases) consisted of generalized tonic-clonic activity. Opisthotonos posturing was common (10 of 31 cases). Seizures persisted from 15 minutes to 6 hours, often intermixed with periods of reduced responsiveness. The possibility of continuing, clinically inapparent seizures, the postictal state, and the depressant

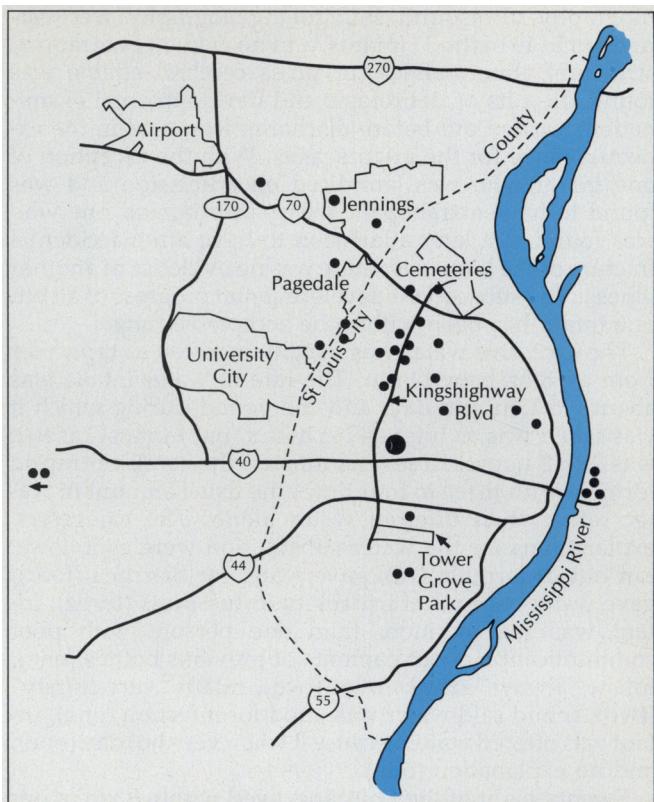


Fig 2.—Map of the St Louis (Mo) metropolitan area. St Louis Children's Hospital (star) is within the city limits. The dots to the extreme right represent infants from East St Louis, Ill. The two dots to the extreme left are two white infants, one from a small city 112 km from St Louis, the other from a suburb 24 km from the hospital. Kingshighway Boulevard is the main north-south traffic artery through a densely populated, poor, residential area.

effect of anticonvulsant medications made determination of causality of the depressed level of consciousness difficult. In several infants, physicians noted rapid improvement in neurologic signs as hypertonic saline was administered. Bulging of the fontanelle and radiologic evidence of increased intracranial pressure were notably absent.

Respiratory failure, neither preceded nor obviously caused by seizure, occurred in six patients, either at home (five patients) or in the radiology suite (one patient). Others stopped breathing in the emergency room (nine patients). The overall rate of respiratory failure was 15 of 31 (48%). Except in one infant who developed noncardiologic pulmonary edema, mechanical ventilation was withdrawn within 24 hours of admission.

Mean \pm SD laboratory values (Table 1) included the following: urine specific gravity (31 of 31 patients), 1.004 ± 0.001 ; serum urea nitrogen (31 of 31 patients), 1.4 ± 0.4 mmol/L; serum calcium (31 of 31 patients), 2.40 ± 0.07 mmol/L; serum magnesium (29 of 31 patients), 0.88 ± 0.03 mmol/L; and serum glucose (31 of 31 patients), 5.5 ± 0.67 mmol/L. Cerebrospinal fluid (29 of 31 patients) was sterile and contained a nucleated cell count of less than 8×10^6 /L. The serum cortisol level (24 patients) was within the normal reference range, as was the blood ammonia level (25 patients). A urine toxic screen (26 patients) failed to reveal substances capable of causing seizures, respiratory failure, or hyponatremia. The mean platelet count (31 of 31 patients) was 615×10^9 /L (range, 250 to 1300×10^9 /L). Imaging studies of the brain (computed to-

mography, ultrasound, skull roentgenography) were carried out in 19 of the 31 infants, and no evidence of trauma, structural abnormalities, or gross cerebral edema was found. Results of neurologic and developmental examinations carried out before discharge were within the expected range for the infants' ages. With the exception of one infant who was jaundiced on admission and was found to have extrahepatic biliary atresia, and one who was found at a later admission to have a nonaccidental fracture of the humerus, there was no evidence of another illness. The subsequent developmental progress of all but one infant has been within the acceptable range.

The excessive water was usually ingested as tap water from an 8-oz baby bottle. The rate of water intake was about 7.5 L/m² per day, and the period during which it was taken was as brief as 1½ hours, but in most cases it was 2 to 8 hours. In several infants, the caregiver mixed formula with three to four times the usual amount of water rather than offering water alone. The caregivers' explanations for the water substitution were as follows: ran out of formula (16), gave water for diarrhea (four), gave water because of irritability or fussiness (three), infant was fed by more than one person, with poor communication about contents of previous bottles (one), infant "always" drinks a lot (two), infant "very thirsty" (two), friend said water was good for infection (one), infant was offered water because it was a very hot day (one), and no explanation (one).

Twenty-eight of the 31 infants lived within 8 km of our hospital (Fig 2), most in residential areas known to have high lead exposure risk, high crime rates, and poverty. Twenty-seven infants were black. Twenty-seven were enrolled in WIC, Aid to Families With Dependent Children, and the food stamp program. Only one mother was married; three were addicted to crack cocaine and alcohol. Most of the mothers were in their 20s and 30s and had older children (one had seven).

Since the WIC program began in 1976, the maximum provision of infant formula has been one can of formula per day per infant (403 fl oz of concentrated liquid formula per month) through the first 12 months of life. In some areas this is provided as an equivalent amount of powdered or ready-to-feed formula. This allotment provides 2293 kJ, or a little more than three 8-oz bottles per infant per day. Based on the average body weight of 4-month-old infants, this provides 378 kcal/kg per day, which is approximately the 50th percentile of observed intake of healthy 4-month-old infants. A small amount of juice and cereal is provided after age 4 months. The mothers of many of our patients found themselves using more than one can per day and exhausted the WIC-supplied formula before the end of the month. The WIC nutritionists, and our hospital social workers, reported a disappearance of alternate (non-WIC) sources of free infant formula and a sharp increase in the cost of formula in retail outlets during the last decade. Sources of additional formula, which in the past were manufacturers' representatives, food pantries, and local churches, appear to have been eliminated. The WIC workers emphasize the word *supplemental* in the program title, but they recognize that many mothers presume that WIC provides everything the infant needs and are reluctant or unable to use their food stamps or cash to buy infant formula. Advice concerning water feeding has been the same since the program began in 1976: "After the infant has taken the breast or formula,

you may offer a little water if he/she seems to want it," offered to the mother at the time of enrollment in an individual counseling session by a nutritionist. We uncovered no major change in advice during the study period by WIC workers or by personnel of the institutions where the infants were born.

Of the 31 infants, 22 developed symptoms during the summer months (May through October vs November through April) ($\chi^2=9.2$, $P<.05$). Average temperatures during May through October of 1988, 1989, and 1990 did not exceed the average values for those months during the previous decade; 1989 was cooler and 1990 was hotter than the average.

Group 2

Water intoxication in older children remains a rare event; in contrast to group 1, the incidence did not appear to have changed.

Patient 32, an 8-year-old girl, had suffered from physical abuse as an infant and had openly stated her intention to stab to death a foster parent. Her foster parents, after much counseling, chose water drinking as a method of punishment. When forced to stand in the kitchen and drink 12 glasses of water, the patient lost consciousness. Her recovery was complete.

Patient 33, a 3-year-old girl, was admitted to an outlying hospital with severe dehydration (serum urea nitrogen level, 23.6 mmol/L) due to gastroenteritis. Fluid resuscitation (40 mL/kg) was carried out with the use of 5% glucose in water instead of the intended 5% glucose in saline. As the 4-hour infusion ended, the patient lost consciousness, serum sodium level decreased from 133 to 114 mmol/L, and apnea and coma ensued; hypertonic saline and nonspecific measures were of no avail, and the patient died. Autopsy showed only brain swelling.

Patient 34, who was 2 years old, became comatose 4 hours after elective hip surgery. Serum sodium level had decreased to 112 mmol/L while dilute intravenous fluids were infused. Again, therapy had no apparent effect, and autopsy showed brain swelling. The vulnerability of postoperative patients to water intoxication has been emphasized,^{2,16} and the devastating course in such patients has recently been described.¹⁷

COMMENT

Oral water intoxication is a recognizable clinical syndrome. The victim is usually 3 to 6 months old, comes from a poor family, and presents to the ambulance service or emergency room with apnea or seizures. Body temperature is low, even in hot weather, and the majority of episodes occur in the summer months. Respiratory failure, if not the presenting complaint, may occur as the seizures are treated. After documentation of hyponatremia and the infusion of hypertonic saline, the neurologic disturbance abates. In some infants, spontaneous water diuresis occurs so rapidly that treatment with hypertonic saline may be unnecessary. Ventilatory support is seldom needed for more than 12 hours, and recovery appears complete within a few days. The infants' salt and water homeostatic mechanisms are intact, and, with appropriate feeding instructions, recurrence can be avoided. There is clearly some risk of death or hypoxic organ damage, although this did not occur in our patients (group 1).

The controversy^{18,19} that surrounds the treatment of chronically hyponatremic adults and the belief that ex-

Table 2.—Oral Water Intoxication in 85 Healthy Infants

Source, y	City	No. of Patients	Age, mo	Comments
Dugan and Holliday, ⁹ 1967	Pittsburgh, Pa	1	3	Errors in mixing; 1/6 strength formula
Dugan and Holliday, ⁹ 1967	Oakland, Calif	1	5	Ten 8-oz bottles of water over 20 h
Nickman et al., ¹⁰ 1968	Philadelphia, Pa	2	4, 14	Fed water
Crumpacker and Kriel, ¹¹ 1973	Minneapolis, Minn	5	5.8±4.1	Short of money; gave 12 8-oz bottles of tap water in 1 d; "Hunger is probably the main force that would compel a baby to accept a solute-poor diet . . ."
Schulman, ¹² 1980	Albany, NY	2	5, 6	Ran out of milk; gave 64 oz of tap water
David et al., ¹³ 1981	Pittsburgh	8	3.4±1.0	Authors focused on antidiuretic hormone secretion
Partridge et al., ¹⁴ 1981	Cincinnati, Ohio	4	4.1±1.5	Two sibling pairs; water given after each feeding for irritability; physical abuse in one family
Lipsitz, ²⁶ 1984	Pueblo	1	2.5	Water and tea substituted due to coryza
O'Connor, ²⁷ 1985	Wilmington, Del	1	2	Water fed because of diarrhea
Corneli et al., ²⁸ 1985	Cincinnati	7	6.7±1.8	All occurred in 6-wk period, summer 1983
Gold and Koenigsberg, ²⁹ 1986	Chicago, Ill	1	4	Tap water given because of summer heat
Borowitz and Rocco, ³⁰ 1986	Nashville, Tenn	2	5, 3	Considered previous salt restriction important
Medani, ¹⁵ 1987	Baltimore, Md	19	5.1±4.3	Water supplements
Schaeffer and Ditchek, ³¹ 1991	Brooklyn, NY	3	4, 6, 10	...
Present report	St Louis, Mo	31	4.2±2.1	...

cessively rapid administration of hypertonic saline may cause central pontine myelinolysis should not unduly influence physicians caring for acutely hyponatremic children. The rate of increase in serum osmolality/sodium recommended in the former group of patients (less than 0.5 mmol/h)¹⁸ is inappropriate in acutely hyponatremic patients, such as those described in this report. For symptomatic patients with acute hyponatremia, the rate of increase should be at least 1 mmol/L per hour¹⁹; the rate of 2 to 3 mmol/L per hour that occurred in our patients (Table 1) was associated with an excellent overall outcome. The rate of increase in serum osmolality in the infants who had spontaneous diuresis (group 1b) was threefold faster than the maximal recommended rate of correction in the chronically hyponatremic adult. The safety and efficacy of prompt infusion of hypertonic saline in the amounts recommended by current pediatric authorities²⁰⁻²² is supported by our experience. We recognize the limits of our retrospective study and cannot reject the possibility that isotonic fluids at a restricted infusion rate may be effective therapy for many infants, although we think that a more rapid reversal of the movement of water into the brain may be lifesaving in some.

Incidence figures for water intoxication are not available. Since 1935, when the first report²³ of fatal water intoxication appeared, until 1958, only seven patients, of all ages, were noted in the literature.²³⁻²⁵ In 1959, Crawford and Dodge² described five children, three intoxicated by parenteral fluids, one by gastrostomy, and one by enemas. After the first report of infants who took excessive water by mouth appeared in 1967,⁹ only seven similar infants were observed in the next decade,^{10,11} but, during the last 5 years, 33 examples^{15,26-30} have appeared. Table 2 summarizes 85 reported cases of water intoxication.^{9-15,26-31} An article in the lay press in 1981 mentioned 17 Milwaukee (Wis) infants who were not described in the medical litera-

ture.³² We found only three reports³³⁻³⁵ of infants with oral water intoxication from other countries. The ages of those infants were more evenly distributed through the 1st year of life than in the American patients; withdrawal of formula or breast before excessive water ingestion was a consistent theme in these reports.

The reason for the striking increase in the occurrence of oral water intoxication is not clear. Although other investigators have suggested that abusive caretakers,¹⁴ excessive secretion of antidiuretic hormone,¹³ or previous salt restriction may have played a role in their patients, we share Crumpacker and Kriel's view¹¹ that hunger overwhelms the infants' innate protective mechanisms and is the most important cause of infantile water intoxication. Three facts strongly suggest that unavailability of formula plays a major causative role: (1) the current volume of formula provided by WIC meets the needs of most 3-month-olds and fails to satisfy many 4- and 5-month-old healthy infants; (2) in our study, and in most US reports (Table 2), the ages of the infants are 4 to 6 months; and (3) the majority of mothers said that exhaustion of their supply of formula was the reason they were feeding water. Even when another explanation for water feeding was recorded in the patient's chart, specific history concerning the family's formula supply may not have been sought or offered. We also speculate that the summer clustering may be due to the added burden placed on the families' limited resources by the interruption of the school lunch program for the older siblings and other children in the home.

We are convinced that oral water intoxication is a new, probably underreported, entity. A prospective, national, epidemiologic study is needed to ascertain the incidence of and causative factors responsible for this illness. Immediate consideration should be given to increasing the availability of formula to infants living in poverty. Increased education concerning the dangers of excessive water intake in infants who are denied formula or breast-

feeding is also needed,³⁶ both for medical personnel and for infant caregivers.

Our gratitude to the pediatric interns, residents, nurses, and staff physicians in the emergency room, pediatric intensive care unit, and floors of St Louis Children's Hospital, and to the medical record room staff, the social workers, WIC nutritionists, and the child neurology staff, all of whom saw the infants' needs and met them, documenting what they saw and did. The suggestion concerning the cause of summer clustering came from Rebecca Graves, the able director of the St Louis Children's Hospital social work department. Thanks to Aileen Derhake, whose transformation of handwritten gibberish into clean prose raises the skill of typing to a profession.

References

1. Larson EE, Rountree LG, Weir JF. Studies in diabetes insipidus, water balance, and water intoxication. *Arch Intern Med.* 1922;29:306-330.
2. Crawford JD, Dodge PR. Complications of fluid therapy in patients with neurologic disease with special emphasis on water intoxication and hypertonic dehydration. *Pediatr Clin North Am.* 1959;6:257-279.
3. Mortimer JG. Acute water intoxication as another unusual manifestation of child abuse. *Arch Dis Child.* 1980;55:401-403.
4. Kropp RM, Schwartz JF. Water intoxication from swimming. *J Pediatr.* 1982;101:947-948.
5. Pickering LK, Hogan GR. Voluntary water intoxication in a normal child. *J Pediatr.* 1971;78:316-318.
6. Frizzell RT, Lang GH, Lowance DC, Latham SR. Hyponatremia and ultramarathon running. *JAMA.* 1986;255:772-774.
7. Klonoff DC, Jurow AH. Acute water intoxication as a complication of urine drug testing in the workplace. *JAMA.* 1991;265:84-85.
8. Cheng JC, Zikos D, Skopicki HA, Peterson DR, Fisher KA. Long-term neurologic outcome in psychogenic water drinkers with severe symptomatic hyponatremia: the effect of rapid correction. *Am J Med.* 1990;88:561-566.
9. Dugan S, Holliday MA. Water intoxication following the voluntary ingestion of excessive fluids. *Pediatrics.* 1967;39:418-420.
10. Nickman SL, Buckler JM, Weiner LB. Further experience with water intoxication. *Pediatrics.* 1968;41:149-151.
11. Crumpacker RW, Kriel RL. Voluntary water intoxication in normal infants. *Neurology.* 1973;23:1251-1255.
12. Schulman J. Infantile water intoxication at home. *Pediatrics.* 1980;66:119-120.
13. David R, Ellis D, Gartner JC. Water intoxication in normal infants: role of antidiuretic hormone in pathogenesis. *Pediatrics.* 1981;68:349-353.
14. Partridge JC, Payne ML, Leisgang JJ, Randolph JF, Rubenstein JH. Water intoxication secondary to feeding mismanagement, a preventable form of familial seizure disorder in infants. *AJDC.* 1981;135:38-40.
15. Medani CR. Seizures and hypothermia due to dietary water intoxication in infants. *South Med J.* 1987;80:421-425.
16. Arieff AI. Hyponatremia, convulsions, respiratory arrest, and permanent brain damage after elective surgery in healthy women. *N Engl J Med.* 1986;314:1529-1535.
17. Arieff AI, Fraser CL. Detrimental consequences of untreated hyponatremia in children. *Kidney Int.* 1987;31:290. Abstract.
18. Sterns RH. The treatment of hyponatremia: unsafe at any speed? *Am Kidney Found Nephrol Lett.* 1989;6:1-10.
19. Cluitmans FHM, Meinders AE. Management of severe hyponatremia: rapid or slow correction? *Am J Med.* 1990;88:161-166.
20. Fleisher GR, Ludwig S. *The Textbook of Pediatric Emergency Medicine.* Baltimore, Md: Williams & Wilkins;1983:419.
21. Black JA. *Paediatric Emergencies.* 2nd ed. London, England: Butterworths;1987:125.
22. Feld LG, Kaskel FJ, Schoeneman MJ. The approach to fluid and electrolyte therapy in pediatrics. In: Barness LA, DeVivo DC, Morrow G, Osaki F, Rudolph AM, eds. *Advances in Pediatrics.* Chicago, Ill: Mosby Year Book Medical Publishers Inc; 1988:526.
23. Helwig FC, Schutz CB, Curry DE. Water intoxication, report of a fatal human case. *JAMA.* 1935;104:1569-1575.
24. Swanson AG, Iseri OA. Acute encephalopathy due to water intoxication. *N Engl J Med.* 1958;258:831-834.
25. Baskin JL, Keith HM, Scribner BH. Water metabolism in water intoxication. *AJDC.* 1958;83:618-627.
26. Lipsitz DJ. Herbal teas and water intoxication in a young child. *J Fam Pract.* 1984;18:933-937.
27. O'Connor RE. Water intoxication with seizures. *Ann Emerg Med.* 1985;14:71-73.
28. Corneli HM, Cormley CJ, Baker RC. Hyponatremia and seizures in the first two years of life. *Pediatr Emerg Care.* 1985;1:190-193.
29. Gold I, Koenigsberg M. Infantile seizures caused by voluntary water intoxication. *Am J Emerg Med.* 1986;4:21-27.
30. Borowitz SM, Rocco M. Acute water intoxication in healthy infants. *South Med J.* 1986;79:1156-1158.
31. Schaeffer AV, Ditchek S. Current social practices leading to water intoxication in infants. *AJDC.* 1991;145:27-28.
32. Heyman K. Baby doctors warn of diluted formula. *Milwaukee J.* August 11, 1981:1.
33. Etzioni A, Benderly A, Levi Y. Water intoxication by the oral route in an infant. *Arch Dis Child.* 1979;54:551-553.
34. Lingh HF, Hsu CH, Chyou SC, Lee YJ, Chang KL. Neonatal water intoxication secondary to feeding mismanagement. *Chung Hua I Hsueh Tsa Chih.* 1987;39:131-134.
35. Vanaprucks V, Prapaitvakul K. Water intoxication and hyponatremic convulsions in neonates. *Arch Dis Child.* 1989;64:734-735.
36. Finberg L. Too little water has become too much. *AJDC.* 1986;140:524.

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Comorbidity of Psychiatric Diagnoses in Anorexia Nervosa

Katherine A. Halmi, MD; Elke Echert, MD; Peggy Marchi, PhD; Vincent Sampognaro, MA; Robin Apple, MA; Jacob Cohen, PhD (*Arch Gen Psychiatry.* 1991;48:712-718)