Hypernatremia in Hospitalized Patients

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Objective: To determine the incidence, clinical characteristics, and outcome for general medical–surgical hospital patients with hypernatremia.

Design: A prospective cohort study.

Setting: A 942-bed urban university hospital.

Patients: All patients who developed a serum sodium concentration of 150 mmol/L or greater during a 3-month observation period.

Measurements: Daily fluid balance, mental status, and serum and urine electrolytes and osmolality.

Results: 103 patients were identified. Eighteen patients were hypernatremic on hospital admission, and 85 developed hypernatremia during hospitalization. Patients who developed hypernatremia during hospitalization were younger than patients who developed hypernatremia before hospital admission (mean age ± SD, 58.9 ± 19.2 years compared with 76.6 ± 16.6 years; $P < 0.01$) but did not differ in age from the patients of the general hospitalized population. Eighty-nine percent of patients who developed hypernatremia during hospitalization had urine concentrating defects, primarily as the result of the use of diuretics or of solute diuresis, whereas only 50% of patients who were hypernatremic on admission could be shown to have concentrating defects ($P < 0.01$). Fifty-five percent of all hypernatremic patients had increased insensible water losses, and 35% had increased enteral water losses. Eighty-six percent of patients with hospital-acquired hypernatremia lacked free access to water, 74% had enteral water intake of less than 1 L/d, and 94% received less than 1 L of intravenous electrolyte-free water per day during the development of hypernatremia. No supplemental electrolyte-free water was prescribed during the first 24 hours of hypernatremia in 49% of patients. The duration of hypernatremia was shorter in patients who were hypernatremic on admission (median duration, 3 days) than in patients with hospital-acquired hypernatremia (median duration, 5 days; $P < 0.05$). Mortality was 41% for all patients, but hypernatremia was judged to have contributed to mortality in only 16% of patients.

Conclusions: Although the development of hypernatremia before hospital admission occurs primarily in geriatric patients, hospital-acquired hypernatremia was more common in our cohort and had an age distribution similar to that of the general hospitalized population. Hospital-acquired hypernatremia was primarily iatrogenic, resulting from inadequate and inappropriate prescription of fluids to patients with predictably increased water losses and impaired thirst or restricted free water intake or both. Treatment of hypernatremia is often inadequate or delayed. Efforts to manage hypernatremia better and altogether avoid hospital-acquired hypernatremia should focus on both physician education and the development of hospital systems to prevent errors in fluid prescription.

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Hypernatremia in adults is a common problem that has been associated with mortality rates ranging from 42% to 60% (1–4). Most studies of this condition have focused on its occurrence in the geriatric population, for which it has been proposed as a marker for concomitant infection and an indicator of neglect in nursing home care (1, 2). An increased incidence of hypernatremia in mentally handicapped patients has also been reported (5). However, the epidemiologic factors and pathogenesis of hypernatremia in the general hospitalized population have not been well defined.

We did a study to determine the incidence, clinical characteristics, and outcome of hypernatremia in patients in a general medical–surgical hospital. We reviewed patient records for the period before the onset of hypernatremia to assess factors contributing to the development of hypernatremia, and we followed its treatment prospectively. Our results show that hospital-acquired hypernatremia is not a disease that occurs only in the elderly. Rather, the age distribution of persons with this condition is similar to that of all hospitalized patients. Furthermore, because it is primarily iatrogenic, resulting from the inappropriate prescription of fluids to patients with predictably increased electrolyte-free water losses, it should be preventable.

Methods

Patients

We prospectively studied all adult (≥16 years of age) inpatients at the Presbyterian and Montefiore University Hospital divisions of the University of Pittsburgh Medical Center for the 3-month period from 1 July 1993 to 30 September 1993. The facility is a 942-bed urban medical–surgical university hospital that had an average occupancy of 72.8% during this period. All patients with a serum sodium concentration greater than or equal to 150 mmol/L during the preceding 24 hours were identified daily. We reviewed records of each patient thus identified, and we followed each patient prospectively until his or her hypernatremia resolved. We recorded volume status, fluid prescription, and laboratory findings

daily. When possible, we measured urine osmolality and urine electrolyte content. In this noninterventional study, the therapy and management of each patient was determined by the patient's primary service without direct intervention from the investigators. However, in the course of the study, the primary physician of each patient was notified of patient enrollment and informal management suggestions were offered. The study was approved by the Institutional Review Board for Biomedical Research of the University of Pittsburgh.

A serum sodium concentration greater than or equal to 150 mmol/L was used as the entry criterion for inclusion in the study. This concentration was selected because it is clearly abnormal and reflects moderate-to-severe hypernatremia. In patients identified as hypernatremic, the onset, duration, and resolution of hypernatremia were determined on the basis of an upper limit of normal of 145 mmol/L.

Evaluation

The medical records of all patients identified as hypernatremic were evaluated by one reviewer (RB). Primary and concomitant diagnoses and medications prescribed at the onset of hypernatremia were recorded on a standardized abstracting form. Clinical variables including weight, temperature, blood pressure, central venous pressures, physical findings pertinent to assessment of volume status (skin turgor, membrane hydration, edema), and fluid balance were recorded retrospectively for 3 to 5 days before the onset of hypernatremia and prospectively through its resolution. For patients with hypernatremia on admission to the hospital, data collection began at admission. The volume and composition of intravenous fluids, enteral and parenteral nutrition, and oral water intake were recorded. Daily urine volume, based on the intake and output record, was recorded. Values for serum sodium, potassium, chloride, total carbon dioxide, glucose, blood urea nitrogen, creatinine, and calcium, measured plasma osmolality, and urine specific gravity, osmolality, and sodium and potassium concentrations were recorded daily when obtained by the patient's managing physician.

State of consciousness, based on recorded data in nurses' notes and physicians' progress notes, was assessed on the basis of the following definitions: 1) alert: patient responds immediately and appropriately to stimulation; 2) obtunded: patient requires increased stimulation to evoke a response; 3) stuporous: patient can be aroused only by vigorous and continuous stimulation; and 4) comatose: patient exhibits minimal or no response to vigorous stimulation (6). When the required information to assess state of consciousness could not be ascertained during the daily review of the patient's record, the patient was examined by one investigator (RB). State of consciousness was not recorded for intubated patients because these patients do not have free access to water and their state of consciousness has no bearing on free water intake.

The causal relation of hypernatremia to morbidity and mortality was determined during data analysis by the consensus of all three investigators. Factors considered by the investigators included the severity of the patient's underlying diagnosis, the severity of his or her comorbid conditions, and the temporal relation of hypernatremia to the eventual outcome of hospitalization. For example, hypernatremia was not considered a contributing factor to mortality in a patient who developed hypernatremia during the course of septic shock and who died of multiorgan system failure, but it was considered as such in a patient who became obtunded as a result of hypernatremia, developed aspiration pneumonia, and died of resulting respiratory failure.

Serum electrolytes, glucose, urea nitrogen, and creatinine concentrations were measured using an Ektachem 700XRC autoanalyzer (Eastman Kodak, Rochester, New York), with direct potentiometry used for the sodium assay. Urine sodium and potassium concentrations were measured using flame emission photometry (Instrumentation Laboratory, Lexington, Massachusetts). Plasma and urine osmolality were determined by freezing-point depression (Advanced Instruments, Inc., Medham Heights, Massachusetts).

Statistical Analysis

Data were analyzed using the SigmaStat statistical software package (Jandel Scientific Software, San Rafael, California). Nominal data were analyzed by chi-square test and ordinal data by the Mann–Whitney rank sum test. Multiple comparisons were made using Dunn's method following Kruskal-Wallis one-way analysis of variance on ranks. Censored data were analyzed using the log-rank test. A P value less than 0.05 was considered significant. All data are expressed as mean $\pm$ SD.

Results

A serum sodium concentration greater than or equal to 150 mmol/L was reported for 117 patients during the 3-month period of 1 July 1993 through 30 September 1993. In 14 patients, hypernatremia was considered spurious because it could not be confirmed on repeat measurement and because of the absence of clinical evidence of disturbances of water homeostasis to account for the development

Table 1. Demographic Characteristics of Patients*

Variable	Patients with Hypernatremia on Admission (n = 18)	Patients with Hospital-Acquired Hypernatremia (n = 85)
Age (range), *y*	76.6 ± 16.6 (23–98)	58.9 ± 19.2† (16–101)†
Male sex, *n (%)*	4 (22)	46 (52)‡
Transferred from nursing home, *n (%)*	11 (61)	8 (9)†
Peak sodium concentration (range), *mmol/L*	157.3 ± 5.4 (151–168)	154.9 ± 4.1 (151–168)‡
Duration of hospital stay before onset of hypernatremia, *d*		25.2 ± 35.7 (1–224)
Duration of hypernatremia, (median; range), *d*	2.8 ± 1.4 (3; 1–6)	5.1 ± 4.2 (5; 1–24)‡

* Data are presented as mean ± SD.
† $P < 0.01$
‡ $P < 0.05$.

and resolution of hypernatremia. The remaining 103 patients constituted the study population. Eighteen were hypernatremic on hospital admission, and 85 became hypernatremic during the course of their hospitalization. The eighteen patients whose serum sodium concentrations were 150 mmol/L or greater on admission accounted for 0.2% of the 7836 patients admitted to the Presbyterian and Montefiore University Hospital divisions of the University of Pittsburgh Medical Center during the 3-month study period. The incidence of hospital-acquired hypernatremia in the 8517 patients at risk (669 patients admitted before the start of the study and 7818 patients admitted without hypernatremia) was 1%. The daily prevalence of hypernatremia was 0.8% Demographic data for the patients are shown in Table 1, and the age distributions of the two patient groups are shown in Figure 1. Patients who developed hypernatremia before admission were significantly older than the general population of patients admitted to the hospital during the same 3-month period (mean age, 76.6 ± 16.6 years compared with 53.6 ± 19.1 years; $P < 0.01$). In contrast, the age of patients with hospital-acquired hypernatremia was not significantly different from that of the population of hospitalized patients at risk (mean age, 58.9 ± 19.2 years compared with 57.4 ± 23.4 years, $P = 0.59$). Eleven patients (61%) who were hypernatremic on admission were transferred from nursing homes compared with only 8 patients (9%) who developed hypernatremia during their hospitalization ($P < 0.01$).

The primary diagnoses for patients with hypernatremia are listed in Table 2. In 15 of the 18 patients (83%) who were hypernatremic on hospital admission, underlying infections were the primary reason for hospitalization. Seven patients (39%) had pneumonia, 5 patients (28%) had urinary tract infections or urosepsis, and 3 patients (17%) had bacteremia of unidentified origin. In contrast, only 17 patients (20%) with hospital-acquired hypernatremia were admitted for a primary infectious process ($P < 0.01$).

Fluid Balance during Development of Hypernatremia

To assess factors contributing to the development of hypernatremia, urine-concentrating ability during hypernatremia was assessed on the basis of measured urine volume and osmolality. Twenty-four-hour urine volume was recorded in 95 patients (Figure 2), and urine osmolality was recorded in 48 patients (Figure 3). A urinary concentrating defect was defined as a urine osmolality less than 700

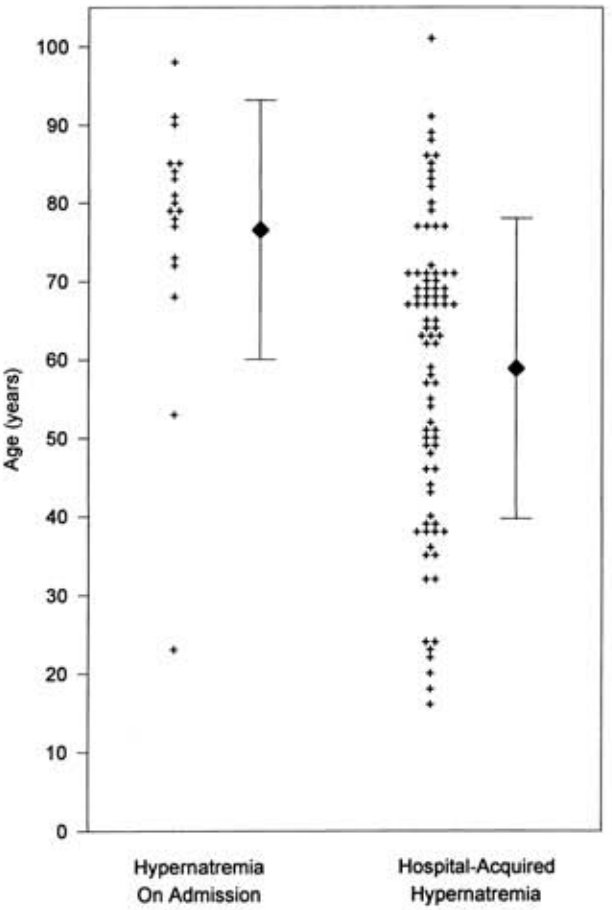

Figure 1. Age distribution of patients with hypernatremia. Ages of 18 patients who were hypernatremic at the time of hospitalization and 85 patients who developed hypernatremia during hospitalization. Individual patient ages (crosses) and group mean age ± SD (filled diamond and bar) are shown; $P < 0.01$.

mmol/kg or a urine volume greater than 1000 mL/24 h during hypernatremia. Concentrating defects were present in 85 patients: 9 (50%) with hypernatremia on admission and 76 (89%) with hospital-acquired hypernatremia ($P < 0.01$). In 3 patients in each group, concentrating ability could not be assessed. The causes of the concentrating defects are shown in Table 3. Concentrating defects were multifactorial in 23 patients. The most common factors contributing to impaired renal concentrating ability were diuretic administration and solute diuresis caused by hyperglycemia, mannitol administration, or protein loading (urea diuresis). Only 6 patients had diabetes insipidus: 2 with hypothalamic diabetes insipidus after pituitary surgery, 2 with hypothalamic diabetes insipidus secondary to head trauma, 1 with hypothalamic diabetes insipidus after a cerebrovascular accident, and 1 with nephrogenic diabetes insipidus related to lithium toxicity.

Increased enteral fluid losses due to diarrhea, nasogastric drainage, fistulas, or biliary drains were observed in 2 patients (11%) who were hypernatremic on admission and in 34 patients (40%) with hospital-acquired hypernatremia. Pyrexia (>38 °C) resulted in increased insensible free-water losses in 9 patients (50%) with hypernatremia on admission and in 48 patients (56%) with hospital-acquired hypernatremia.

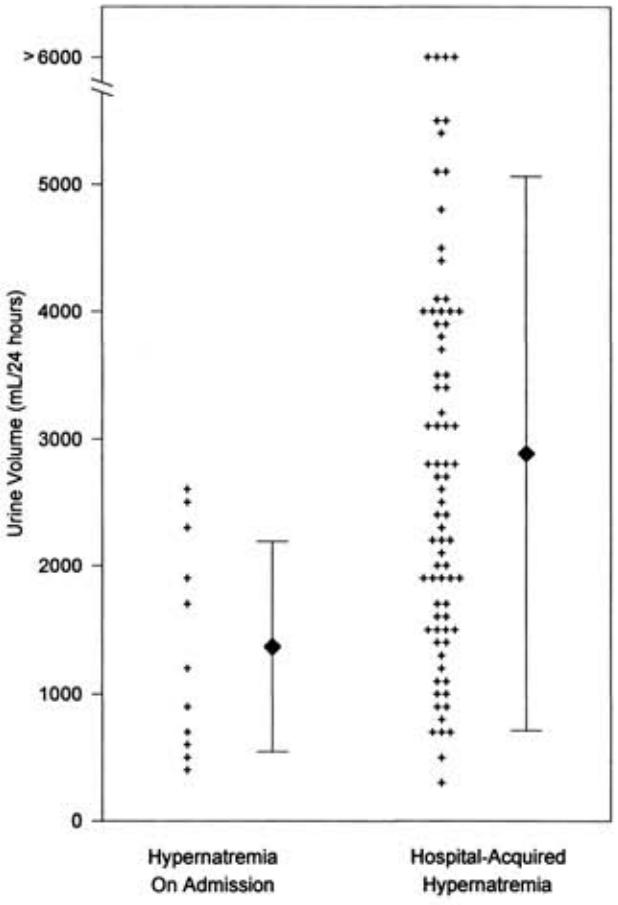

Figure 2. Urine volume during hypernatremia. Twenty-four-hour urine volume during hypernatremia in 11 patients who were hypernatremic on admission and in 82 patients with hospital-acquired hypernatremia. For patients who had multiple measurements of urine volume, the value reported is for the measurement for the first 24-hour period. Individual patient values (crosses) and group mean ± SD (filled diamond and bar) are shown; $P < 0.01$.

Table 2. Primary Diagnoses for Patients with Hypernatremia

Variable	Patients with Hypernatremia on Admission (n = 18)	Patients with Hospital-Acquired Hypernatremia (n = 85)
	← n →	
Infection	15	17
Malignancy		14
Hepatic failure/transplantation		14
Trauma		9
Gastrointestinal disease		9
Cardiovascular disease		8
Neurologic disease	1	6
Pulmonary disease		5
Pituitary surgery		2
Lithium toxicity		1
Diabetes mellitus	1	
Drug overdose	1	

Seventy-three patients (86%) with hospital-acquired hypernatremia did not have free access to water or had mental status changes that impaired thirst perception during the development of hypernatremia. Forty-two patients were intubated; of the remaining 43 patients, 31 were obtunded, stuporous, or comatose. Enteral fluid intake during the day preceding the onset of hypernatremia was recorded in 83 patients (98%) with hospital-acquired hypernatremia. Twenty-five patients with hospital-acquired hypernatremia received no enteral fluids, and only 20 patients had a recorded intake that exceeded 1000 mL/24 h (Table 4) during the day preceding the onset of hypernatremia. Sixty patients (71%) received intravenous fluids during the day preceding the development of hypernatremia (Table 4). Although the average daily volume of intravenous fluids administered was 1500 ± 1000 mL, the mean electrolyte-free water content of these fluids (calculated as follows) was only 300 ± 500 mL:

$$V \times \left[1 - \frac{[Na^+] + [K^+]}{154}\right]$$

where V is fluid volume and $[Na^+]$ and $[K^+]$ are the fluid sodium and potassium concentrations, respectively.

Seven patients receiving total parenteral nutrition were administered solutions containing a cation (sodium and potassium) concentration greater than 154 mmol/L.

Fluid intake during the development of hypernatremia could not be reliably assessed in the 18 patients who were hypernatremic on hospital admis-

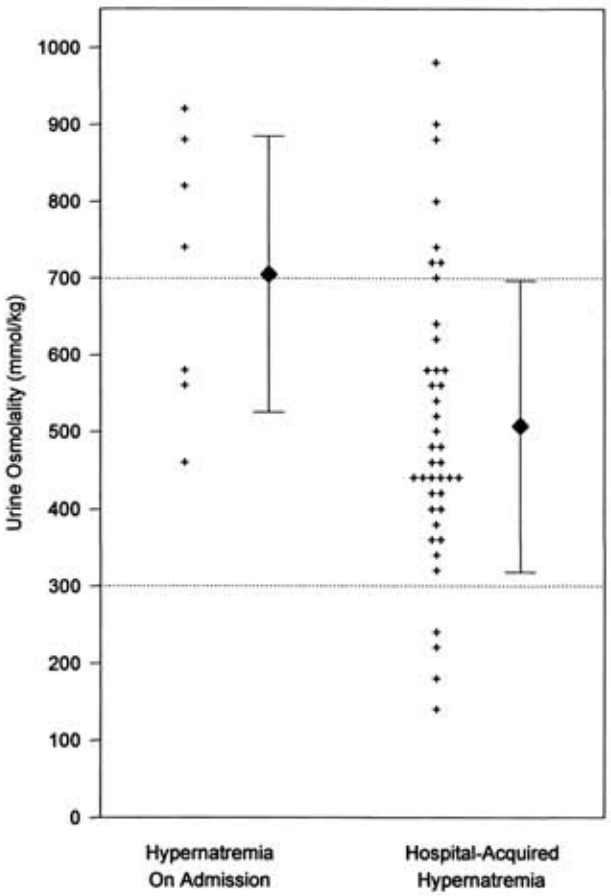

Figure 3. Urine osmolality during hypernatremia. Urine osmolality during hypernatremia in 7 patients who were hypernatremic on admission and 41 patients with hospital-acquired hypernatremia. Individual patient values (crosses) and group mean ± SD (filled diamond and bar) are shown; $P < 0.05$. For patients who had multiple measurements of urine osmolality during hypernatremia, the value reported is the first measurement. A urine osmolality of greater than 700 mmol/kg during hypertonicity was considered to indicate normal urinary concentration, and a urine osmolality of less than 300 mmol/kg during hypertonicity was considered diagnostic of diabetes insipidus.

sion. Only 2 patients in this group had a normal sensorium on admission, and 15 were obtunded or stuporous and would therefore be expected to have had impaired thirst perception. One patient was intubated at the time of transfer to the University of Pittsburgh Medical Center after a brief (<24 hours) hospitalization at a referring hospital. Overall, 16 of the 18 patients (89%) were presumed to have had impaired access to electrolyte-free water before hospitalization.

Treatment and Outcome

Hypernatremia persisted for a median of 4 days (10th to 90th percentiles, 1 to 10 days). In 50 patients (49%), the fluid prescription for the first 24 hours after the report of a serum sodium concentration of 150 mmol/L or greater did not include any supplemental electrolyte-free water. Hypernatremia corrected within 72 hours of onset in only 48 patients (47%). The duration of hypernatremia in patients who were hypernatremic on admission was approximately half that of patients with hospital-acquired hypernatremia, despite similar severity of water deficits (patients who were hypernatremic on admission: median, 3 days; 10th to 90th percentiles, 1 to 4 days; patients with hospital-acquired hypernatremia: median, 5 days; 10th to 90th percentiles, 1 to 11 days; $P < 0.05$).

Outcome data are shown in Table 5. The overall hospital mortality was 41% (42 patients); however, most of these deaths could be attributed to the patients' underlying illnesses. Although no deaths resulted directly from the hypernatremia itself (for example, from intracranial bleeding caused by hypernatremic cerebral dehydration), the hypernatremia was judged to be a contributing factor in the deaths of 17 patients (16%). The difference in mortality between the patients who were hypernatremic on admission and the patients with hospital-acquired hypernatremia was not statistically significant. In 14 patients (14%), hypernatremia was judged to have contributed to associated morbidity and decreased functional status at hospital discharge.

Discussion

Disorders of water homeostasis are among the most common fluid and electrolyte disturbances in hospitalized patients. Studies of the incidence of hyponatremia have shown that 4.1% of patients are hyponatremic on admission (7). The incidence and prevalence of hyponatremia among hospital inpatients has been reported as 1.0% and 2.5%, respectively, with approximately two thirds of cases developing during hospitalization (8).

The incidence and prevalence of hypernatremia in the general population of hospitalized patients in the United States are unknown. Studies from the United Kingdom have reported serum sodium con-

Table 3. Causes of Renal Concentrating Defects in Patients with Hypernatremia

Cause	Patients with Hypernatremia on Admission	Patients with Hospital-Acquired Hypernatremia
	n (%)	
Diuretic use	3 (17)	47 (55)
Solute diuresis		
Glucose	2 (11)	14 (16)
Urea		13 (15)
Mannitol		6 (7)
Renal insufficiency (creatinine >176 μmol/L)	4 (22)	13 (15)
Diabetes insipidus		
Hypothalamic		5 (6)
Nephrogenic		1 (1)

Table 4. 24-Hour Fluid Intake Immediately before the Onset of Hypernatremia in Patients with Hospital-Acquired Hypernatremia

Intake	Enteral Fluids	Intravenous Fluids	
		Total Volume	Electrolyte-Free Water
		n (%)	
None	25 (29)	24 (28)	48 (56)
<500 mL	27 (32)	12 (14)	18 (21)
500–1000 mL	11 (13)	12 (14)	8 (9)
>1000 mL	20 (24)	36 (42)	4 (5)
Hypertonic fluids			7 (8)
Unknown	2 (2)	1 (1)	1 (1)

centrations of greater than 150 mmol/L in 0.3% of hospitalized patients (4) and sodium concentrations of greater than 154 mmol/L in 0.12% of patients being admitted to the hospital (9). In the United States, studies have focused on hypernatremia as a geriatric disease (1–3, 10) and have not examined its incidence in the general population. Thus, we designed our study to evaluate the incidence, causes, and treatment of moderate-to-severe hypernatremia (serum sodium concentration > 150 mmol/L) in a general medical–surgical university hospital.

Our findings show two distinct populations of patients with hypernatremia. The first, composed of patients who had serum sodium concentrations of 150 mmol/L or greater on hospital admission, accounted for 0.2% of all hospital admissions. These patients were substantially older than the general hospitalized population of patients, and more than 60% had been transferred from nursing homes. As previously described, most patients with hypernatremia on admission also have serious concomitant infections (2).

The second group of patients developed hypernatremia during the course of hospitalization, with an overall incidence of 1%. They constituted a distinct population because their mean age was almost 20 years less than that of patients who were hypernatremic on admission and was not significantly different from the mean age of all hospitalized patients. Our results indicate that although hypernatremia that precedes hospitalization is a geriatric disease, hospital-acquired hypernatremia is not.

Hospital-acquired hypernatremia resulted from inadequate fluid prescription for patients who had increased free-water losses combined with an inability to increase oral water intake freely in response to hypertonicity. Increased free-water losses were caused by impaired renal concentrating capacity in 89% of patients as well as by increased extrarenal fluid losses, with increased enteral losses in 40% of patients and increased insensible losses in 56% of patients.

The normal defense against the development of hypernatremia is the stimulation of thirst and increased water consumption. Almost 50% of our patients with hospital-acquired hypernatremia were intubated. Regardless of whether they had intact thirst perception, they were unable to regulate their water intake. More than two thirds of the patients who were not intubated had impaired mental status during the development of hypernatremia and would therefore be expected to have impaired thirst perception (11). Thus, most patients with hospital-acquired hypernatremia had both increased free-water losses and impaired ability to regulate water consumption. Thus, tonicity homeostasis in these patients depended greatly on the appropriateness of prescribed fluids.

In most patients in this cohort, hypernatremia developed as the result of inadequate fluid prescription. Almost 30% of patients were receiving no enteral fluids, and an additional 45% received less than 1 L/d during the development of hypernatremia. Of the 72% of patients who received intravenous fluids, nearly 40% received only 0.9% saline, and therefore were receiving no intravenous electrolyte-free water. In addition, more than 10% of the patients who received intravenous fluids were prescribed hypertonic electrolyte solutions as part of their total parenteral nutrition. These observations indicate that most hospital-acquired hypernatremia is iatrogenic and preventable, resulting from inadequate provision of electrolyte-free water to patients who have predictably increased electrolyte-free water losses and impaired ability to defend against hypertonicity through thirst and free intake of water.

Despite frequent measurement of the serum sodium concentration, treatment of hypernatremia was frequently inadequate or delayed. The standard recommendation for the treatment of hypernatremia is that, in addition to replacement of ongoing water losses, approximately half of the water deficit be replaced during the first 24 hours and that the remainder of the deficit be replenished during the subsequent 2 to 3 days (12–14). Despite these recommendations, approximately 50% of the patients

Table 5. Outcome Data

	All Patients	Patients with Hypernatremia on Admission	Patients with Hospital-Acquired Hypernatremia
	← *n* (%) →		
Died	42 (41)	4 (22)	38 (45)
Discharged	28 (27)	3 (17)	25 (29)
Transferred to nursing home or rehabilitation facility	28 (27)	10 (56)	18 (21)
Transferred to acute care hospital	5 (5)	1 (6)	4 (5)

in our study did not receive any supplemental electrolyte-free water during the first 24 hours after serum sodium concentrations of 150 mmol/L or greater were observed, and hypernatremia was corrected within 72 hours in only 36% of patients. Of interest, patients who were hypernatremic on admission received treatment much earlier and with more appropriate fluids than did patients with hospital-acquired hypernatremia, despite similar severity of water deficits. It is likely that these patients were treated earlier because water deficits are recognized more promptly on initial presentation than when they develop during hospitalization.

The mortality we observed is similar to that reported in other studies of hypernatremia (1–4). As has been stressed in these studies, however, much of the excess mortality cannot be directly attributed to the hypernatremia itself. Based on careful review of the medical records, we believe that hypernatremia partially contributed to mortality in only 16% of patients.

In summary, although hypernatremia that develops in adults before hospitalization is primarily a geriatric disease, hospital-acquired hypernatremia is more common and has an age distribution similar to that of the overall hospitalized population. Hospital-acquired hypernatremia results primarily from inadequate and inappropriate prescription of fluids to patients with predictably increased water losses and impaired thirst or restricted free water intake or both. In addition, modifications in fluid prescription necessary for the treatment of hypernatremia are often inadequate or delayed.

We believe that efforts to manage hypernatremia and altogether avoid hospital-acquired hypernatremia are required. Although improved physician education about fluid management in patients who are either at high risk for hypernatremia or have overt hypernatremia is necessary, studies of medication errors in hospitals suggest that physician education alone is inadequate to eliminate errors (15, 16). Rather, efforts must also be directed at developing a systems approach to minimize the risk for errors in fluid management and ensuring the adequate treatment of hypernatremia when it occurs.

Our findings suggest several approaches. Pharmacy review of parenteral nutrition fluids containing hypertonic electrolytes could be readily instituted, but this would have only a small effect on prevention of hypernatremia, because intake of hypertonic electrolytes contributed to hospital-acquired hypernatremia in only 7 of 85 patients. More broad-based checks to ensure adequate provision of water are more difficult to design because fluid prescription and administration tend to be highly decentralized.

Maneuvers designed to hasten institution of therapy and ensure its adequacy may be more effective. Because most of the patients in our cohort received inadequate treatment after developing hypernatremia, prompt notification by the laboratory, along with delivery to the chart of recommendations for treatment, could bring about earlier recognition of hypernatremia and improve its management. However, whether such an approach will improve treatment or alter the outcome from hypernatremia remains to be seen.

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References

1. **Himmelstein DU, Jones AA, Woolhandler S.** Hypernatremic dehydration in nursing home patients: an indicator of neglect. J Am Geriatr Soc. 1983;31: 466-71.
2. **Mahowald JM, Himmelstein DU.** Hypernatremia in the elderly: relation to infection and mortality. J Am Geriatr Soc. 1981;29:177-80.
3. **Snyder NA, Feigal DW, Arieff AI.** Hypernatremia in elderly patients: a heterogeneous, morbid, and iatrogenic entity. Ann Intern Med. 1987;107: 309-19.
4. **Long CA, Marin P, Bayer AG, Shetty HG, Pathy MS.** Hypernatraemia in an adult in-patient population. Postgrad Med J. 1991;67:643-5.
5. **Macdonald NJ, McConnell KN, Stephen MR, Dunnigan MG.** Hypernatraemic dehydration in patients in a large hospital for the mentally handicapped. Br Med J. 1989;299:1426-9.
6. **Plum F, Posner JB.** Diagnosis of Stupor and Coma. Philadelphia: F.A. Davis; 1966.
7. **Tierney WM, Martin DK, Greenlee MC, Zerbe RL, McDonald CJ.** The prognosis of hyponatremia at hospital admission. J Gen Intern Med. 1986;1: 380-5.
8. **Anderson RJ, Chung HM, Kluge R, Schrier RW.** Hyponatremia: a prospective analysis of its epidemiology and the pathogenetic role of vasopressin. Ann Intern Med. 1985;102:164-8.
9. **Daggett P, Deanfield J, Moss F, Reynolds D.** Severe hypernatraemia in adults. Br Med J. 1979;1:1177-80.
10. **Beck LH, Lavizzo-Mourey R.** Geriatric hypernatremia. Ann Intern Med. 1987;107:768-9.
11. **Fitzsimons JT.** The physiological basis of thirst. Kidney Int. 1976;10:3-11.
12. **Palevsky PM, Singer I.** Hypernatremia. In: Glassock RJ, ed. Current Therapy in Nephrology and Hypertension. 3rd ed. St. Louis: Mosby–Year Book; 1992.
13. **Brennan J, Ayus JC.** Acute versus chronic hypernatremia: how fast to correct ECF volume? J Crit Illness. 1990;5:330-3.
14. **Gullans SR, Verbalis JG.** Control of brain volume during hyperosmolar and hypoosmolar conditions. Annu Rev Med. 1993;44:289-301.
15. **Leape LL.** Error in medicine. JAMA. 1994;272:1851-7.
16. **Leape LL, Bates DW, Cullen DJ, Cooper J, Demonaco HJ, Gallivan T, et al.** Systems analysis of adverse drug events. JAMA. 1996;274:35-43.