Fortnightly Review

Management of hyponatraemia

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Hyponatraemia occurs in many different systemic disease states and is the most frequent electrolyte abnormality seen in a general hospital population, with an incidence of about 1%.[1,2] Hyponatraemia is usually the result of dilution, although both total body water and extracellular volume may be high, low, or normal. Asymptomatic hyponatraemia is often benign, but when patients have central nervous system symptoms treatment is mandatory to prevent permanent brain damage. Almost all of the morbidity associated with hyponatraemia is due to brain damage, and recent studies show that the age and sex of the patient are major determinants of such brain damage. The incidence of symptomatic hyponatraemia is similar among men and women,[2] but most patients who develop permanent brain injury are children and menstruant women (tables I and II).[2,3] Earlier studies suggesting that severe hyponatraemia was often benign had generally evaluated only postmenopausal women and older men,[4] groups not generally susceptible to hyponatraemia induced brain injury.[2,3]

Summary points

- The morbidity associated with hyponatraemia is most closely related to the age or sex of the affected patient (highest in children and menstruant women) and is not related to either the magnitude or duration of the hyponatraemia
- When hyponatraemia is accompanied by central nervous system manifestations (hyponatraemic encephalopathy) there is substantial morbidity, whereas asymptomatic hyponatraemia is often benign
- A major cause of hyponatraemic encephalopathy and subsequent morbidity is hypotonic fluids given to postoperative patients
- Symptomatic hyponatraemia requires treatment, usually hypertonic sodium chloride infusion, limiting the magnitude of correction to about 25 mmol during the initial 24-48 hours; a loop diuretic or intubation is often indicated as adjunctive treatment
- The morbidity associated with hyponatraemic encephalopathy is primarily due to brain oedema, respiratory insufficiency, and hypoxaemia, with resultant hypoxic brain damage

Brain damage and hyponatraemia

When symptomatic hyponatraemia occurs there is usually brain oedema. If adaptation of the brain is not adequate pressure of the swollen brain on the skull can lead to a decrease in cerebral blood flow and pressure necrosis. Initial adaptation of the brain to hyponatraemia is by loss of blood and cerebrospinal fluid, followed by cellular extrusion of osmotically active cations (initially sodium, then potassium and possibly amino acids), which tends to lower the osmolality without substantial gain of water.[5] If symptomatic hyponatraemia is not corrected oedema may increase with possible tentorial herniation, often leading to respiratory arrest and cerebral hypoxia and ischaemia.[6,7]

The above sequence has been verified by computed tomography, magnetic resonance imaging, and postmortem studies in over 40 hyponatraemic patients.[2,6-8] Recent evidence suggests that contributory factors to hyponatraemic brain injury may also include (*a*) systemic hypoxaemia; (*b*) a direct vasoconstrictive effect of antidiuretic hormone on cerebral blood vessels; (*c*) female sex; (*d*) physical factors; and (*e*) preexisting liver disease, alcoholism, or structural lesions in the central nervous system.[2,3] Neither magnitude of fall nor rate of fall in serum sodium concentration is important in the genesis of brain damage (table III).[2]

TABLE I—*Distribution of cases of permanent brain damage among men and women who suffered postoperative hyponatraemia*

	All postoperative patients (n=76 678)	All hyponatraemic controls (n=674)	All hyponatraemic encephalopathy cases (n=65)	All cases with brain damage (n=34)
No (%) of men	37 626 (49)	307 (46)	25 (68)	1 (3)
No (%) of women	39 052 (51)	367 (54)	40 (62)	33 (97)

Data from Ayus *et al.*[2]
Statistical comment: Sex distribution was not significantly different among the hyponatraemic controls or the 65 cases. But of the 34 patients who died or suffered permanent brain damage, 33 were women ($p<0.001$). Relative risk of dying or developing permanent brain damage was 28 times greater in women than men (95% confidence interval 5 to 141).

TABLE II—*Menstruant states of women with brain damage resulting from asymptomatic postoperative hyponatraemia*

	All postoperative female patients (n=39 052)	All female hyponatraemic controls (n=367)	All female cases with brain damage (n=33)
No (%) of menstruant patients	21 088 (54)	39 (11)	25 (76)
No (%) of postmenopausal patients	17 964 (46)	328 (89)	8 (24)

Data from Ayus *et al.*[2]
Statistical comment: Among female controls distribution of menstruant and postmenopausal patients was significant ($p<0.001$). Distribution of menstruant and postmenopausal patients was also significant among patients with brain damage ($p<0.001$). Relative risk of dying or developing brain damage from postoperative hyponatraemia was 26 times as great among menstruant women than among postmenopausal women (95% confidence interval 11 to 62).

Causes of hyponatraemia

POSTOPERATIVE HYPONATRAEMIA

Postoperative hyponatraemia is a frequent and potentially dangerous complication among adults in

TABLE III—*Effects of rate of fall of plasma sodium concentration and magnitude of postoperative symptomatic hyponatraemia in men and women*

	Duration of hyponatraemia (hours)		Plasma sodium concentration (mmol/l)	
	<24	≥24	86-115	116-128
No (%) of men	14 (56)	11 (44)	15 (60)	10 (40)
No (%) of women	13 (42)	27 (68)	19 (48)	21 (52)

Data from Ayus *et al.*[2]
Statistical comment: Whatever the grouping, mortality was significantly greater in women than in men ($p<0.001$). Differences in mortality between women whose plasma sodium concentration was greater than on up to 115 mmol/l or whose duration of hyponatraemia was greater than or up to 24 hours were all not significant.

the United States and United Kingdom.[1,2] In the United States the incidence of postoperative hyponatraemia is about 1%, or about 250 000 cases among the roughly 25 million inpatient operations that are performed each year.[2] Raised plasma antidiuretic hormone concentrations with impaired excretion of free water occur in almost all patients in the first two to six days after operation[1] in response to multiple non-osmotic stimuli—for example, pain, fear, blood loss, anaesthesia, anxiety, vomiting, volume depletion, and narcotics or sedative-hypnotics.[9] During certain operations—for example, transurethral prostate resection and endometrial ablation—hypotonic solutions used to irrigate the operative site may be rapidly absorbed through opened veins, with an effect similar to intravenous administration.[10,11] Thus any patient in the postoperative period should be considered at risk of hyponatraemia and be given appropriate prophylaxis. Of critical importance is the choice of intravenous fluids.

INTRAVENOUS FLUIDS

The most common cause of in hospital hyponatraemia in the United States and United Kingdom is intravenous hypotonic fluids. Apparently based on anecdotal data and recommendations made before 1950, some physicians still infuse hypotonic solutions postoperatively, often glucose in water (280 mmol/l).[7] The rationale for using hypotonic fluids in the postoperative period is unclear, as few objective data support the practice. Before 1950 there were suggestions that postoperative infusion of isotonic sodium chloride might lead to complications,[12] including worsening of glomerulonephritis,[13] with vague references to postoperative "salt intolerance".[14] However, data published after the early 1950s all suggest that the practice is probably without scientific justification. Since the 1930s a profusion of studies have shown the propensity of intravenous hypotonic solutions to cause death or permanent brain damage in the postoperative period.[9,15] Since the 1960s most textbooks of surgery, gynaecology, medicine, and nursing have emphasised the dangers of postoperative hypotonic fluids.[9,16] Permanent brain damage from hyponatraemia is very often a direct consequence of improper fluid administration.[2,3,5-7,17]

AIDS

AIDS is a major cause of hyponatraemia.[18] The hyponatraemia in AIDS may be secondary to inappropriate secretion of antidiuretic hormone, often associated with pulmonary or intracranial lesions; to volume deficiency (due to vomiting or diarrhoea) and replacement by hypotonic fluids[18,19]; or to mineralocorticoid deficiency, often with intact glucocorticoid secretion.[18,20] In the presence of mineralocorticoid deficiency the result of the corticotrophin stimulation test may be normal, possibly because AIDS often affects the zona glomerulosa of the adrenal gland.[20] In such patients fludrocortisone acetate is indicated if renal salt wasting can be shown in the presence of hyponatraemia.

ROLE OF HORMONES

The plasma antidiuretic hormone concentration is often "inappropriately" raised in hyponatraemia.[6] Associated clinical conditions include volume depletion, secretion of antidiuretic hormone by certain malignant tumours, and certain brain and pulmonary lesions.[9] Symptomatic hyponatraemia may occur during labour and delivery or during treatment for gastrointestinal haemorrhage in patients receiving hypotonic fluid and either vasopressin or oxytocin. These agents should be given in isotonic sodium chloride. Desmopressin given with excess free water has been associated with symptomatic hyponatraemia.[8] Both adrenal insufficiency and hypothyroidism may contribute to hyponatraemia. Finally, oestrogen may impair and testosterone augment brain adaptation to hyponatraemia.[8]

PHARMACOLOGICAL AGENTS

Many pharmacological agents may interfere with the ability of kidney to excrete free water. They include sedatives, hypnotics, analgesics, oral hypoglycaemics, tranquillisers, narcotics, antineoplastic drugs, antipsychotic agents, and diuretics. In most instances there is retention of ingested free water. In the case of thiazide associated hyponatraemia there is often an idiosyncratic reaction to thiazides, with a combination of massive loss of sodium and potassium in the urine and associated polydipsia.[21] Often hyponatraemia which occurs as a side effect of a drug will respond to discontinuation of the offending agent. If such patients have symptoms hypertonic sodium chloride should be instituted in order to prevent respiratory insufficiency and permanent brain damage.[21,22]

PSYCHOGENIC POLYDIPSIA

Another common setting in which symptomatic hyponatraemia may occur is psychogenic polydipsia.[23] Maximal free water clearance in adults is around 700 ml/h (17 l/day) or more. Thus to develop hyponatraemia in the absence of raised plasma concentrations of antidiuretic hormone a 60 kg adult would need to drink over 20 l/day. Most patients who have psychogenic polydipsia and hyponatraemia associated with oral water intoxication have actually ingested less water than the maximal daily renal excretion. Instead, they have a smaller fluid intake but abnormal urinary dilution with excessive antidiuretic hormone secretion.[24] Beer potomania somewhat resembles psychogenic polydipsia, but the hyponatraemia is associated with massive ingestion of beer and carries a high mortality.[25]

Treatment

ASYMPTOMATIC HYPONATRAEMIA

Asymptomatic hyponatraemia generally does not require aggressive treatment with hypertonic sodium chloride, as pharmacological measures combined with water restriction are often sufficient, particularly if the plasma sodium concentration exceeds 120 mmol/l. In patients who are obviously volume depleted isotonic (154 mM) sodium chloride is usually the fluid of choice. When adrenal insufficiency or hypothyroidism has been identified appropriate hormone replacement is warranted. If the patient is receiving drugs which might contribute to hyponatraemia they should be discontinued if possible. Water restriction is theoretically important in patients without symptoms, but from practical considerations, particularly compliance, it is generally not useful. Fluid restriction of less than 1 l/day will result in a negative water balance but only a

slow increase in the serum sodium concentration, rarely exceeding 1·5 mmol/l/24 h.

Several medical regimens have been used for the long term management of patients with asymptomatic hyponatraemia. With chronic "inappropriate" increase in the antidiuretic hormone concentration lithium will often induce nephrogenic diabetes insipidus, but generally produces an erratic response. Lithium toxicity may affect kidneys, central nervous system, heart, haemopoietic system, and thyroid.[15] Demeclocycline, a tetracycline antibiotic, may be used to induce nephrogenic diabetes insipidus in doses above 600 mg/day. It has been used successfully to treat patients with raised antidiuretic hormone concentrations, but acute renal failure and renal tubular toxicity have been reported in hyponatraemic patients with heart failure or cirrhosis.[15] Other possible pharmacological agents for chronic hyponatraemia include urea and inhibitors of antidiuretic hormone. Finally, correction of the functional state of intravascular volume depletion that exists in heart failure and decompensated cirrhosis are often associated with improvement of hyponatraemia. In cirrhosis with ascites this can sometimes be achieved after placing a peritoneal-jugular shunt.

SYMPTOMATIC HYPONATRAEMIA

In patients with symptomatic hyponatraemia the most frequent presenting symptoms are headache, nausea, vomiting, and weakness, the presence of at least one of these defining encephalopathy.[7] Less frequent and more severe symptoms are shown in box 1. Respiratory arrest with hypoxia is a persistent feature in symptomatic hyponatraemic patients who suffer brain damage.[2,3] Thus the therapeutic objective in such patients is reduction of brain oedema by increasing the serum sodium concentration such that the patient becomes asymptomatic with adequate ventilation. In patients with symptomatic hyponatraemia the morbidity and mortality associated with treatment by water restriction are unacceptably high.[26-28] The most appropriate therapeutic regimen for such patients is hypertonic (usually 514 mM) sodium chloride,[26] often given in conjunction with a loop acting diuretic such as frusemide (box 2).[29] In some patients, particularly those with raised antidiuretic hormone concentrations, hyponatraemia, and volume expansion or circulatory insufficiency, simultaneous administration of frusemide may be necessary to prevent circulatory overload.[29] Isotonic (154 mM) sodium chloride is indicated only if the patient is volume and sodium chloride depleted. Such patients include those with volume depletion due to vomiting, sweating, or diarrhoea, who have ingested free water.

The most appropriate setting for correction of symptomatic hyponatraemia is the intensive care unit, where neurological, respiratory, and haemodynamic function can be monitored. Patients with arterial hypoxaemia or respiratory insufficiency should be intubated and mechanically ventilated. Total body water volume should be estimated (figure); the mean in hospitalised adults is about 50%.[9]

Hypertonic (514 mM) sodium chloride should be delivered by a constant infusion pump, with correction planned over 24 to 48 hours at a rate set to increase the serum sodium concentration by about 1 mmol/l/h (box 2). The end point is a plasma sodium concentration that is increased by 20-25 mmol/l or has reached 130 mmol/l, or a patient who has become asymptomatic

Box 1—Signs and symptoms of hyponatraemia

Early hyponatraemic encephalopathy

- Headache
- Nausea
- Vomiting
- Weakness

Advanced hyponatraemic encephalopathy

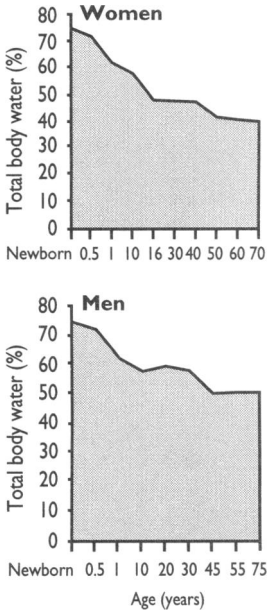
- Impaired response to verbal stimuli
- Impaired response to painful stimuli
- Bizarre (inappropriate) behaviour
- Visual hallucinations
- Auditory hallucinations
- Obtundation
- Urinary incontinence
- Faecal incontinence
- Hypoventilation

Very advanced hyponatraemic encephalopathy (manifestations secondary to increased intracranial pressure)

- Decorticate or decerebrate posturing, or both
- Unresponsiveness
- Bradycardia
- Hypertension
- Altered temperature regulation (hypothermia or hyperthermia)
- Dilated pupils
- Seizure activity (focal or grand mal or both)
- Respiratory insufficiency
- Respiratory arrest
- Coma
- Polyuria (secondary to central diabetes insipidus)

Box 2—Treatment of symptomatic hyponatraemia: basic outline

- Active treatment (infusion of hypertonic sodium chloride) is needed only if the hyponatraemia is symptomatic
- Target of treatment is a serum sodium concentration of about 130 mmol/l, but correction by no more than 25 mmol/48 h
- Determine patient's total body water volume (litres) as a percentage of body weight (kg)
- Subtract patient's serum sodium concentration (mmol/l) from 130 mmol/l. Difference is the needed correction of the serum sodium concentration (in mmol/l)
- Needed correction of the serum sodium concentration (in mmol/l) is the same as the number of hours over which the serum sodium concentration should be corrected
- Multiply the total body water volume (litres) by the needed correction of the serum sodium concentration (mmol/l). This gives the number of mmol of sodium needed to correct the patient's serum sodium concentration to 130 mmol/l
- Number of mmol of sodium needed for correction is then divided by 514 (the number of mmol of sodium in 1 litre of 514 mM sodium chloride). This number times 1000 gives the number of ml of 514 mM sodium chloride needed to correct the serum sodium value to 130 mmol/l
- Divide the number of ml of 514 mM sodium chloride to be given by the number of hours needed for correction of the serum sodium value. This gives the infusion rate of 514 mM sodium chloride in ml/h
- For patients with circulatory impairment, or hypervolaemia with raised plasma concentrations of antidiuretic hormone, give frusemide concomitantly with hypertonic sodium chloride such that there is a net free water diuresis without a net loss of sodium in the urine



Total body water volume as percentage of body weight in women and men throughout life

TABLE IV—*Change in plasma sodium concentration with rapid correction of severe symptomatic hyponatraemia in 167 paediatric and adult patients from three different countries and six different states in the United States*

	Initial plasma sodium (mmol/l)	Final plasma sodium (mmol/l)	Absolute change (after 24-48 hours) (mmol/l)	Rate of correction (mmol/l/h)
Mean (SD)	112 (8)	132 (5)	20 (5)	1·6 (0·8)

Data compiled from Arieff and Ayus,[10] Worthley and Thomas,[17] Ayus *et al*,[22] Cheng *et al*,[23] Ayus *et al*,[26] Hantman *et al*,[29] and Sarnaik *et al*.[30]

(table IV). The serum sodium concentration should not be corrected to normal values, nor should hypernatraemia be allowed to develop. The regimen may require modification in patients with severe hepatic, renal, or cardiac disease. The absolute increase in the serum sodium concentration must be limited to 25 mmol/l within the initial 48 hours of treatment,[26] but the rate of correction of hyponatraemia is not important in the outcome.[2 17 30] Initially the patient's total body water volume should be estimated (figure). Total body water volume varies with age, sex, and weight from about 72% in infants to 35% in elderly obese women.

COMPLICATIONS OF CORRECTING HYPONATRAEMIA

Circulatory congestion is a potential complication of correcting hyponatraemia with intravenous sodium chloride solutions. Such a complication is rare and may be forestalled by giving hypertonic sodium chloride and frusemide.[29] In the past there was controversy regarding the rate of correction of symptomatic hyponatraemia. It was suggested that development of a rare neurological syndrome, central pontine myelinolysis[31] (sometimes called "osmotic demyelination"), might be the result of "rapid" correction of "chronic" hyponatraemia.[4] It had been proposed that if the increase in serum sodium concentration did not exceed some arbitrary rate, often said to be 0·6 mmol/l/h, such complications could be prevented.[4 15] Virtually all hyponatraemic patients in whom cerebral lesions developed after active correction had suffered a hypoxic episode or had their serum sodium concentration corrected to either normonatraemic or hypernatraemic levels or increased by more than 25 mmol/l during the first 48 hours.[26] The vast majority of patients with central pontine myelinolysis have not had hyponatraemia but, rather, severe associated medical conditions, such as advanced liver disease, alcoholism, extensive burns, sepsis, or malignancies.[15 31]

The diagnosis of central pontine myelinolysis requires either histological confirmation or radiological studies with computed tomography or magnetic resonance imaging.[8] With such criteria central pontine myelinolysis is almost never observed in patients who have been hyponatraemic. Rather, the observed lesions are diffuse areas of cerebral infarction with secondary cerebral demyelinating lesions.[6 8] Multiple clinical conditions occur in the absence of hyponatraemia but which are associated with brain lesions which resemble central pontine myelinolysis—for example, subcortical arteriosclerotic encephalopathy, radiotherapy, multiple ischaemic lesions, and sequelae of head trauma.[15] Furthermore, cerebral lesions similar to those sometimes called "osmotic demyelination" are found in untreated hyponatraemic patients.[6] Thus use of terms such as "central pontine myelinolysis" or "osmotic demyelination syndrome" to describe patients with hyponatraemia and brain damage seems unwarranted. The rate of correction is not a factor in the genesis of hyponatraemic brain injury. There are worldwide prospective reports of over 160 patients who have undergone "rapid" correction (mean 1·6 mmol/l/h) of symptomatic hyponatraemia without morbidity,[10 17 22 23 26 29 30] clearly documenting both the safety and the efficacy of this approach (table IV).

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Correction

Guidelines for the management of spontaneous pneumothorax

An authors' error occurred in figure 1 of this article by A C Miller and J E Harvey on behalf of the standards of care committee of the British Thoracic Society (10 July, pp 114-6). In section 4 of figure 1, on simple aspiration, the cannula is described as being of French gauge 16 or larger; this should read standard wire gauge 16.