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A retrospective study of hyponatremia in tetraplegic/paraplegic patients with a review of the literature

B M Soni MBBS MS,[1] S Vaidyanthan MS MCh PhD,[2] J W H Watt FFARCS,[3] K R Krishnan MB FRCS[4]

[1]*Associate Specialist,* [2]*Registrar,* [3]*Consultant Anaesthetist,* [4]*Director, Mersey Regional Spinal Injuries Centre, District General Hospital, Town Lane, Southport, Merseyside, PR8 6PN, England.*

The aetiology of hyponatremia in tetraplegic patients is multifactorial and includes not only general factors such as the use of diuretics and the intravenous infusion of hypotonic fluids, but also certain mechanisms which operate in the spinal cord injured: decreased renal water excretion due to both intrarenal and arginine vasopressin dependent mechanisms (resetting of the osmostat), coupled with habitually increased fluid intake, and the ingestion of a low salt diet. Between 1984 and 1993 we treated 28 episodes of hyponatremia in 19 patients (males: 10; females: 9). Fourteen were tetraplegic and five paraplegic (thoracic lesion in four and lumbar lesion in one). Six patients were asymptomatic during seven episodes of hyponatremia which were detected during routine blood tests. Seven patients were suffering from an acute chest infection, three had an acute urinary tract infection, one had an infected ischial pressure sore and a 69 year old paraplegic patient had bronchopneumonia as well as sepsis from a gangrenous pressure sore in the supraanal region. The time interval between the onset of paralysis and occurrence of the first episode of hypnoatremia was less than a month in only four of the patients. The lowest plasma sodium level observed was less than 100 mmol/l in two, between 100 and 110 mmol/l in four, between 111 and 120 mmol/l in eight patients, and between 121 and 128 mmol/l in 14 cases. Six patients also had hypokalemia ($K^+ < 3$ mmol/l). Only one patient had an elevated plasma creatinine (201 umol/l). Treatment of sepsis and fluid restriction were the mainstay of treatment with only two patients receiving hypertonic saline. All patients with underlying sepsis were treated with antibiotics, usually administered intravenously. The outcome was good in 26 of the 28 episodes.

Two patients died: a 68 year old tetraplegic patient with consolidation of the left lung, cystadenocarcinoma of both ovaries and squamous cell carcinoma of the forehead who presented with generalised oedema, with a plasma sodium level of 118 mmol/l, and potassium of 2.4 mmol/l and who was treated with 2 N saline + postassium + frusemide; she died 1 day later. The only other death was that of a 78 year old female tetraplegic patient who 2 days after sustaining cervical trauma developed hyponatremia because of intravenous infusion of hypotonic fluids given at another hospital, presumably to correct hypotension. She recovered from hyponatremia with fluid restriction, but 3 days later she succumbed to bronchopneumonia and respiratory insufficiency. No patient developed central pontine myelinolysis. No patient with a severe degree of hyponatremia (sodium < 100 mmol/l) had respiratory involvement requiring ventilatory assistance. In conclusion, hyponatremia is seen in tetraplegic patients often in association with sepsis either in the lungs or in the urinary tract, and is best managed by treament of the predisposing factor(s) along with fluid restriction.

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Introduction

Hypoosmolar hyponatremia has been observed in spinal cord injury patients with a prevalence rate of approximately 5–10% (serum or plasma sodium of less than 130 mmol/l).[1] Apart from the aetiological factors which cause hyponatremia in the general population, such as diuretics and intravenous infusion of hypotonic fluids,[2] it appears that some special risk factors operate in spinal cord injured patients. These include forced high fluid intake, low solute intake/excretion, decreased glomerular filtration rate, resetting of the osmostat and either an absolute increase in circulating ADH levels or heightened renal sensitivity to ADH. Leehey *et al*[1] hypothesised that hypoosmolar hyponatremia in tetraplegic patients is related to decreased renal water excretion due to both intrarenal and arginine vasopressin dependent mechanisms (resetting of the osmostat) coupled with a habitually increased fluid intake. Both mechanisms of impaired water excretion may be related to a decrease in central or effective blood volume. We undertook a retrospective study of patients with hyponatremia treated at this centre over the past 9 years in order to determine the predisposing factors for hyponatremia in tetraplegics/paraplegics and to review the current trends in prevention and management of hyponatremia in this special category of patients.

Methods

This is a retrospective study of patients admitted to the Regional Spinal Injuries Centre, Southport between 1984 and 1993 who had plasma sodium levels of less than 130 mmol/l. The details regarding their underlying illness, precipitating factors, if any, for the hyponatremic episodes, presenting clinical features, laboratory investigations, treatment provided for hyponatremia and the outcome were noted.

Nineteen patients (10 males, nine females) developed hyponatremia, of whom six developed more than one episode (31.6%). Four of these people developed two episodes of hyponatremia and one patient developed four episodes of hyponatremia (Tables I, II). Five of these six people with more than one episode were females. The time interval between these successive episodes of hyponatremia was less than a month in two people, and between 6 months and 5 years in the remaining four. Of the 19 patients with hyponatremia, 14 were tetraplegic and only five were paraplegic. Amongst those who were paraplegic, four had a thoracic lesion and only one had a lumbar lesion. Thus hyponatremia is infrequently seen in paraplegic patients and is very uncommon in a paraplegic patient with a lumbar lesion. The time interval between the onset of paralysis and the occurrence of the first episode of hyponatremia varied from 1 week to 37 years, but it was less than a month in only four (21%). Thus the majority of these patients developed hyponatremia after they recovered from the acute effects of their initial illness. Six people amongst the 19 were asymptomatic during the first episode of hyponatremia. In contrast, only one of the six who developed more than one episode of hyponatremia was asymptomatic during the second or successive episode of hyponatremia.

Acute chest infection was the main precipitating factor for the development of hyponatremia in eight episodes, an acute urinary tract infection was responsible for three episodes of hyponatremia, and an infected/gangrenous pressure sore for two episodes. Thus sepsis appears to trigger excessive secretion of ADH in these people. Although patients were asymptomatic during seven of the 28 (25%) episodes of hyponatremia, they presented with symptoms related to central nervous system dysfunction, such as confusion, incoherent speech, lethargy and drowsiness, during nine of the 28 (32%) episodes (Tables I, II). The lowest plasma sodium level observed was 93 mmol/l. It was less than 120 mmol/l in six episodes of hyponatremia. A female tetraplegic patient had four episodes of hyponatremia between 1984 and 1990 and she recovered fully from each episode of hyponatremia without any sequalae.

The mainstay of management was treatment of sepsis with antibiotics. Ancillary measures included fluid restriction, admin-

Table I Clinical profile of patients with hyponatremia

Serial No.	Dob	Sex	Diagnosis	Date injury/ onset paralysis	Neurology level	Date hypoatremia observed	Presenting clinical symptoms
1	10.01.27	M	Paget's disease	07.12.80	T12	12.10.84	Asymptomatic
2	10.07.22	F	Cystadeno carcinoma in both ovaries. Squamous cell carcinoma in forehead	01.07.82	C4	02.06.88	Incoherent speech, confused Generalised oedema
3	"	"	"	"	"	20.07.90	Generalised oedema
4	02.03.24	F	Poliomyelitis	14.12.86	C5	29.08.88	Confusion +
5	24.02.18	F	Traumatic paraplegia	17.11.67	T10	24.02.87	Pyrexia, cough
6	09.01.22	M	Traumatic tetraplegia	03.11.80	C6	26.11.90	Abdominal distension
7	06.08.34	M	Traumatic tetraplegia	22.11.91	C4	13.12.91	Asymptomatic
8	13.03.47	F	Multiple sclerosis (incomplete tetraplegia)	1975	C5	26.07.93	Asymptomatic
9	"	F	"	"	"	03.09.88	Vomiting
10	21.07.38	M	Traumatic tetraplegia	07.06.93	C7	17.06.93	Fever with chills
11	19.02.29	M	Traumatic paraplegia	15.08.56	L1	29.07.93	Nil
12	04.07.42	M	Traumatic tetraplegia	10.02.90	C4	28.07.91	Features of acute chest infection Collapse consolidation of left lung
13	30.03.76	M	Traumatic tetraplegia	05.09.92	T9	04.03.93	Lethargic
14	"	"	"	"	"	25.03.93	"
15	09.08.20	F	Post cervical discectomy, ventilator dependent	March 1993	C4	06.08.93	Asymptomatic
16	"	F	"	"	"	23.08.93	Generalised oedema
17	11.02.43	M	Traumatic tetraplegia	04.09.81	C4	28.09.87	Incomprehensive speech, drowsy

Table I (*cont*)

Serial No.	Dob	Sex	Diagnosis	Date injury/ onset paralysis	Neurology level	Date hypoatremia observed	Presenting clinical symptoms
18	30.09.27	F	Traumatic tetraplegia	23.04.76	C6	13.08.91	Confused, febrile
19	16.07.23	M	Traumatic tetraplegia	08.11.74	C5	19.08.87	Asymptomatic
20	18.07.20	F	Post-irradiation myelopathy tetraplegia	Dec 1959	T6	22.03.84	Breathlessness, features of acute chest infection
21	"	"	"	"	"	16.05.87	"
22	"	"	"	"	"	17.08.90	Acute chest infection
23	"	"	"	"	"	09.11.90	Infected pressure sore
24	08.12.15	F	Traumatic tetraplegia	06.08.93	C5	08.08.93	Agitated, pulling out nasogastric tube
25	25.09.50	M	Traumatic tetraplegia	10.02.93	C7	23.08.93	Convulsions, probably not related to hyponatremia. Known case of epilepsy on phenytoin and carbamazepine
26	28.05.50	F	Traumatic tetraplegia	24.05.91	C6	30.05.91	Asymptomatic
27	"	"	"	"	"	2.11.92	Asymptomatic
28	"	"	"	"	"	01.05.93	Drowsy, confused, incoherent speech, conjunctival oedema

Table II Lowest plasma sodium, corresponding potassium level, precipitating factors if any for hyponatremia, treatment and outcome of patients with hyponatremia

Serial No.	Lowest plasma sodium level observed (mmol/l)	Corresponding potassium (mmol/l)	Serum creatinine (μmol/l)	Precipitating factors	Management	Outcome
1	125	4.8	201	Bronchopneumonia	Antibiotics Amoxycillin 500 mg q.i.d. Frusemide 40 mg IV	Recovered, discharged 24.10.84
2	115		Urea 2.6	Acute urinary tract infection	Fluid restriction and antibiotic (ceftazidime)	Recovered, discharged 22.6.88
3	118	2.4	46	Consolidation left lung—lower lobe. Diuretic prescribed by GP	2N saline 500 ml + potassium Frusemide 400 mg	Died 21.7.90
4	110	3.5	52	Acute urinary tract infection Asked to drink mugs of water	Normal saline & potassium Intravenous antibiotic (ceftazidime)	Recovered, discharged 4.11.89
5	119	3.7	Urea 9.8	Bronchopneumonia, sepsis from gangrenous pressure sore—supra anal area	Intravenous antibiotic. Cefuroxine 750 mg t.d.s. No fluid restriction	Recovered, 4 months later pressure sore repaired by gluteal myocutaneous flap
6	112	2.9	Urea 2.7	Nil	Intravenous normal saline with potassium	Recovered, discharged 3.12.90
7	128	3.5	Urea 5.9	Nil	Nil	Recovered spontaneously, discharged 10.4.92
8	126	3.8	41	Nil	Nil	Recovered, discharged 28.7.93
9	125	1.7	Urea 3.2	Peritonitis due to perforation of sigmoid colon	2N saline + KCl. Resection of sigmoid colon on 8.9.88	Recovered, discharged 19.12.88
10	119	3.8	66	Acute urinary tract infection Low salt intake	Antibiotics—Claforan 1 g × 8 h Increased salt intake in diet	Plasma sodium gradually returned to normal, otherwise asymptomatic
11	123	3.6	73	Large fluid intake. Diuretic	Fluid restriction. Increased salt intake in diet	Recovered, discharged home.
12	126	3.8	Urea 2.7	Bronchopneumonia left lung	Bronchoscopy. Antibiotics—cefotaxime and aminophylline	Recovered, discharged home 21.10.91

Table II (*cont*)

Serial No.	Lowest plasma sodium level observed (mmol/l)	Corresponding potassium (mmol/l)	Serum creatinine (μmol/l)	Precipitating factors	Management	Outcome
13	125	4.2	43	Head injury, hypopituitarism. ?Side effect of desmopressin	Nil specific	Plasma sodium rose to 131 mmol/l 18.03.93 but hyponatremia recurred 25.3.93
14	123	4.1	62	Head injury, hypopituitarism. ?Side effect of desmopressin	Started on hydrocortisone 20 mg mane and 10 mg nocte	Recovered, plasma sodium rose to 140 mmol/l 24.04.93
15	127	4.6	48	Nil	Fluid restriction	Recovered
16	128	4.2	58	Nil	Administered 20% albumin for hypoalbuminemia	Recovered
17	106	4.1	51	Nil	Hypertonic saline desmopressin 4 mg IM. Restricted oral fluids	Recovered, discharged 12.10.87
18	117	4.0	Urea 0.7	Acute chest infection	Fluid restriction Cefuroxime 1.5 g/dl IV	Recovered, discharged
19	114	4.1	66	Nil	Added table salt. Fluid restriction DDVAP	Recovered, discharged home
20	118	3.7	54	Diuretic, bronchopneumonia	Diuretic continued (Navidrex-K) Slow sodium tablet 1 t.i.d. Fluid (1500 IV & 3400 oral). Antibiotic	Recovered, discharged 15.7.84
21	93	2.4	42	Acute chest infection, diuretic, drinking large volumes of water	Amoxycillin, chest physiotheraphy with terbutaline. Fluid restriction, normal saline	Recovered, discharged home 4.6.87
22	105	4.0	41	Bronchopneumonia, left lower lobe	Fluid restriction, normal saline IV Hydrocortisone 100 mg t.i.d. Cerufoxime 1 ml 1.5 g IV	Recovered, discharged home 12.11.90
23	125	4.1	Urea 4.1	Infected ischial pressure sore—cavity down to bone	Blood transfusion. Dressing	Discharged home 12.11.90

Table II (*cont*)

Serial No.	Lowest plasma sodium level observed (mmol/l)	Corresponding potassium (mmol/l)	Serum creatinine (μmol/l)	Precipitating factors	Management	Outcome
24	121	5.0	96	Fluid overload in another hospital when hypotension was noticed after cervical spinal injury	Fluid restriction Dopamine infusion for hypotension	Recovered from hyponatremia. Plasma sodium rose to 132 mmol/l on 10.8.93. She died 13.8.93 due to bronchopneumonia & respiratory insufficiency
25	124	4.4	47	Carbamazepine	Rectal diazepan for convulsions	Recovered
26	103	2.9	Urea 5.1	Nil	Fluid restriction	Recovered
27	125	4.1	48	Nil	Nil	Recovered
28	98	1.8	48	Large fluid intake. Diuretic prescribed by GP	Fluid restriction + potassium Developed grand mal seizure—administered IV diazepam & phenytoin IV infusion. Frusemide 10 mg IV	Recovered fully, discharged home

DDAVP = Desamino-D-arginine vasopressin.

istration of frusemide, normal saline, potassium supplement and withdrawal of the offending agent such as a diuretic. Hypertonic saline (2N) was administered in two instances only.

The outcome was excellent for all but two episodes of hyponatremia. A 60 year old female with a C4 lesion presented with generalised oedema and a sodium level of 118 mmol/l. She had consolidation of the left lower lobe of the lung and died on the second day of hospitalisation. The other death occurred in a 78 year old tetraplegic patient who developed CNS features (e.g. agitation) of hyponatremia (plasma sodium level of 121 mmol/l). She recovered from hyponatremia but later succumbed to bronchopneumonia and respiratory insufficiency.

Thus the philosophy of management of sepsis (if present)—fluid restriction, frusemide and cautious use of hypertonic saline—was successful, with a mortality rate of only 3.5% for hyponatremia and 7% hospital mortality for all these patients. No patient developed central pontine myelinolysis. No patient developed respiratory insufficiency requiring ventilatory assistance.

Discussion

Asymptomatic hyponatremia generally does not require aggressive treatment. If the patient is receiving drugs which might contribute to hyponatremia, they should be discontinued if possible. Fluid restriction to less than 1 litre/day will result in a negative water balance but will achieve only a slow increase in the serum sodium concentration, rarely exceeding 1.5 mmol/24 hours. In patients with symptomatic hyponatremia the most appropriate therapeutic regimen is hypertonic (usually 514 mmol/l) sodium chloride (3%), often given in conjunction with a loop acting diuretic such as frusemide.[2] In patients with raised antidiuretic hormone concentrations, simultaneous administration of frusemide may be necessary to prevent circulatory overload. Patients with arterial hypoxaemia or respiratory insufficiency should be intubated and mechanically ventilated. Hypertonic (514 mmol/l) sodium chloride (3%) should be delivered by a constant infusion pump with the absolute increase in the serum sodium concentration limited to 25 mmol/l within the initial 48 hours of treatment. The endpoint is a plasma sodium concentration which is increased by 20–25 mmol/l, or has reached 130 mmol/l, or resulted in an asymptomatic patient. The serum sodium concentration should not be corrected to normal values, nor should hypernatremia be allowed to develop. There has been controversy regarding the rate of correction of symptomatic hyponatremia. It was suggested that development of central pontine myelinolysis might be the result of rapid correction of chronic hyponatremia.[3] Virtually all hyponatremic patients in whom cerebral palsy developed after active correction had suffered a hypoxic episode or had their serum sodium concentration corrected to either normonatremic or hypernatremic levels or increased by more than 25 mmol/l during the first 48 hours.[4] We have not exceeded 0.5 mmol/l/hour of increase in serum sodium concentration when treating symptomatic hyponatremia and we did not encounter any case of central pontine myelinolysis. Figure 1 gives the rate of correction of hyponatremia in one of our patients who had profound hyponatremia, with a serum sodium of less than 100 mmol/l. However, there are reports of over 160 patients who have undergone rapid correction (mean 1.6 mmol/l/hour) of symptomatic hyponatremia without morbidity, clearly documenting the safety and efficacy of this approach.[2]

Barter & Schwartz[4] suggested that when patients with renal failure, adrenal failure and saline depletion were excluded, there was a group of patients with hyponatremia in whom there was indirect evidence of continuing ADH secretion. They suggested the term 'syndrome of inappropirate secretion of anitdiuretic hormone' for this condition and defined it as a combination of: (1) hyponatremia with corresponding hypoosmolality of the serum and extracellular fluid; (2) continued renal excretion of sodium; (3) absence of clinical evidence of fluid volume depletion; (4) urine less than maximally dilute; (5) normal renal function; and (6) normal adrenal function.

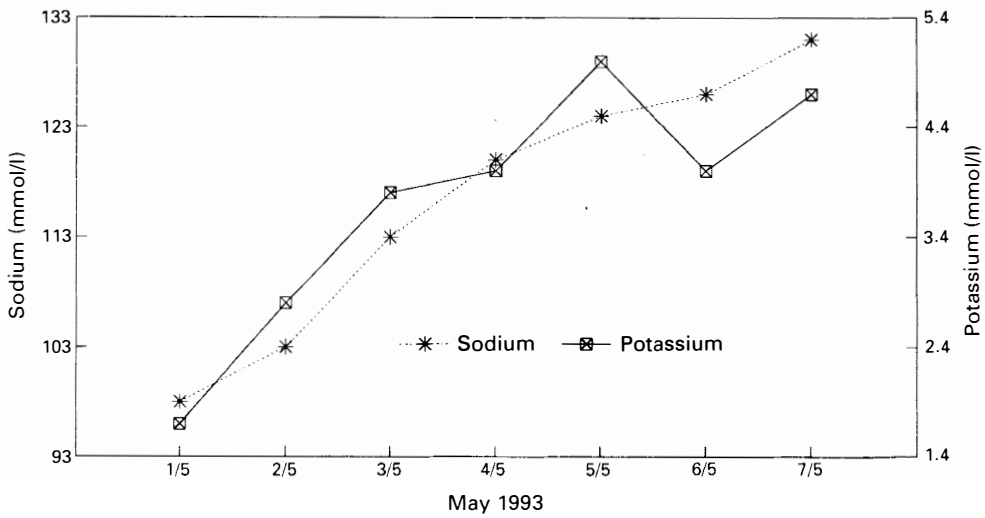

Figure 1 Rate of correction of hyponatremia in one patient.

Thomas *et al*[6] described hyponatremia in four patients with chest infection with plasma arginine vasopressin (AVP) levels of 22.1, 0.8, 3.0 and 46 pg/ml respectively which were excessive compared with the values expected, i.e. < 0.3 pg/ml (by extrapolation in healthy people for a plasma sodium of less than 125 mmol/l). In patients with chest infection, the return of plasma sodium to normal was associated with a reduction in plasma arginine vasopressin levels. In one of the patients the AVP level was 5.6 pmol/l on 2 May 1993 when she had severe hyponatremia and a month later (9 June 1993), the AVP level was < 0.3 pmol/l, when the plasma sodium had returned to normal. Thomas *et al*[6] concluded that the hyponatremia of chest infection was transient and the increase in plasma sodium in patients with chest infection was too rapid to be produced by water-deprivation treament and was due to the return of plasma-ADH to normal. Schrier[7] suggested that the release of arginine vasopressin from the pituitary due to nonosmotic stimuli is an appropriate response to stress reaction. In this study of hyponatremia in tetraplegics/paraplegia, eight patients had an acute chest infection, three had an acute urinary infection and three suffered from sepsis either from a gangrenous pressure sore or from a perforated sigmoid colon. Hyponatremia reverted to normal, concomitant with treatment of sepsis and fluid restriction. As suggested by Thomas *et al*,[6] instead of calling this a syndrome of inappropriate secretion of antidiuretic hormone, a simpler, more descriptive terminology such as 'hyponatremia with chest infection in a tetraplegic' would be more useful and less confusing in the clinical situation.

Sibley[8] reported a case of a C5 tetraplegic patient with symptoms of acute urinary tract infection, complaints of restlessness and confusion. His serum sodium level was 118 mEq/l. In this study, three patients had an acute urinary infection when they developed hyponatremia. As in hyponatremia of chest infection, these patients had probably 'inappropriate' release of ADH, which may be an appropriate response to stress related to sepsis. With treatment of the acute urinary infection, hyponatremia also subsided in these patients.

Another mechanism which contributes to hyponatremia in cervical spinal cord injury patients is resetting of the osmostat. The osmotic threshold is defined as the plasma osmalility at which ADH release is initiated. When the osmotic threshold is lowered, ADH is released before plasma sodium or osmolality has reached normal levels. This

mechanism has been termed 'resetting the osmostat', in which long term intake of large volumes of fluid may be implicated. Another possibility is that the lowered threshold for ADH release may be due to decreased sympathetic vasomotor tone.

The ability to excrete a normal fraction of an administered water load depends on the state of sodium balance. Sica & Culpepper[9] performed a water load test of 20 ml/kg bodyweight in a C6 complete male tetraplegic, who had persistent chronic hyponatremia. While on a 46 mmol sodium diet, the minimum urinary osmolality that could be attained was only 232 mOsm/kg water and just 43% of the water load was excreted over 5 hours. The maximum free water clearance during the test was 0.43 ml/minute. With a sodium intake of 150 mmol/day, the urinary osmolality declined to 60 mOsm/kg water and 95% of the ingested water load was excreted in the 5 hour period. Maximum free-water clearance on a high sodium diet alone was 4.73 ml/minute.

In our study of a tetraplegic patient, who had developed severe hyponatremia, only 14.6% of the water load was excreted in 5 hours and the lowest urine osmolality was 338 mOsm/kg water. The maximum free-water clearance was 0.44 ml/minute. Thus, ingestion of a low salt diet results in a decreased effective extracellular fluid volume, persistent ADH release, and a distinctly abnormal water load test. This case illustrates the important role of sodium intake and effective extracellular volume in suppressing ADH release in spinal cord injury. Chronic haemodynamic instability leading to altered regulation of plasma ADH levels, when combined with a patient-specific predilection for high fluid intake and low salt intake, prediposes to the development of hyponatremia.

Leehey *et al*[1] demonstrated that hypoosmolar hyponatremia in quadriplegic patients was associated with impaired water excretion. Intrarenal defects in water excretion may occur secondary to decreased osmolal clearance and/or a decrease in delivery of glomerular filtrate to the distal diluting segments of the nephron. The cause of these intrarenal defects in water excretion is not clear. Decreased osmolar clearance

Table III Water load test (20 ml/kg of water taken by mouth in 10 min) performed on patient 19 in sitting posture on 5th July 1993

Time	Plasma				Urine					
	Sodium mmol/l	Potassium mmol/l	Creatinine umol/l	Osmolality mOs/kg	Sodium mmol/l	Potassium mmol/l	Creatinine mmol/l	Osmolality mOs/kg	Output ml per h	Free water clearance ml/min
09.30	132	—	52	276	68	49	3.2	357	—	—
(Water load at 14.30 pm)										
15.30	129	4.4	45	268	16	99	6.6	492	15	0.21
16.30					19	66	3.8	363	75	0.44
17.30	128	4.0	33	276	12	59.7	3.5	338	30	0.11
18.30					13	58.7	3.7	353	30	0.14
19.30					22	60.7	4.1	420	25	0.21

may be related to a low renal solute load, occurring either because of reduced solute intake and/or a decrease in endogenous solute production secondary to decreased muscle mass.

The reduction in the percentage of glomerular filtrate delivered to the diluting sites of the nephron could occur because of a decrease in central or effective blood volume; the resultant alterations in intrarenal haemodynamics would then cause increased fluid reabsorption in the proximal nephron segments.

We observed that the sodium intake in patient 19 was 60, 75, 70 and 45 mmol/day. Thus decreased salt intake was certainly a contributory factor to the development of hyponatremia. We also performed a water load test (20 ml/kg body weight) on her, the result of which is given in Table III. After ingestion of 1200 ml water, the urine output for the next 5 hours was only 175 ml. She was taking normal breakfast and lunch prior to the test; thus she was not in a state of dehydration at all. Even then she excreted only 175 ml of urine during 5 hours while she was sitting up in the chair. The phenomenon of decreased urine production in the sitting posture by tetraplegics has been observed frequently by us. The contributory factors are pooling of blood in the venous system, resulting in decreased central blood volume because of interruption of descending sympathetic pathways and absence of pumping action of leg muscles. The decrease in central or intrathoracic blood volume acts as a haemodynamic stimulus for arginine vasopressin release. Tetraplegic patients (as well as paraplegic patients with lesions above the T6 neurotome) have labile blood pressures in the low normal range and tend to develop hypotension when placed in the seated or upright tilted position. Hypotension occurring as a result of tilting in such patients not unexpectedly results in a marked elevation of plasma arginine vasopressin levels. Thus a decrease in central or effective blood volume leads to impaired water excretion due to both arginine vasopressin dependent and intrarenal mechanisms and could be the cause of both the resetting of the osmastat and the intrarenal defects in water excretion. Therefore, measures to increase effective blood volume such as salt and/or mineralocorticoid supplements and elastic leg stockings, in combination with decreased fluid intake should be beneficial in correcting the hyponatremia in spinal cord injury patients.

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