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Isotonic hyponatremia and cerebrospinal fluid sodium during and after transurethral resection of the prostate

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Abstract: We examined the effects on the central nervous system of hyponatremia during transurethral resection of the prostate (TURP). Initially, a prospective study was done on 165 consecutively treated patients undergoing TURP, to evaluate symptoms related to the serum osmolality. There were ten patients with hyponatremia below 120 mEq·L^{-1}, and in whom the serum sodium decreased to 111.9 ± 6.4 mEq·L^{-1} (mean ± SD) postoperatively, the measured serum osmolality remained near normal. The calculated osmolality decreased to 237.4 ± 11.9 mOsm·kg^{-1} and the estimated osmolar gap was 33.5 ± 10.4 mOsm·kg^{-1} due to absorption of the irrigating sorbitol. Neurological symptoms were mild and complications such as seizures or loss of consciousness never occurred. There were five other patients with hyponatremia (serum sodium 118.0 ± 6.7 mEq·L^{-1}) from whom lumbar cerebrospinal fluid (CSF) was collected before and after TURP through a single puncture. CSF sodium did not decrease throughout 1.5 h after TURP, and there was a CSF-to-serum sodium gradient. Our study shows that in cases of acute dilutional hyponatremia during and after TURP, symptoms are mild because the serum osmolality remains near normal and CSF sodium does not decrease despite severe postoperative hyponatremia.

Key words: Complication, Transurethral resection of the prostate, Isotonic hyponatremia, Osmolar gap, Cerebrospinal fluid sodium

Introduction

Transurethral resection of the prostate (TURP) may lead to acute dilutional hyponatremia due to the intravascular absorption of large amounts of non-electrolyte irrigating fluids. Severe clinical symptoms related to neurological or cardiovascular functions can occur when serum sodium levels fall below 120 mEq·L^{-1} [1–4]. We experienced a patient with asymptomatic hyponatremia during TURP in whom the serum sodium was 102 mEq·L^{-1}. To better understand the pathophysiology of asymptomatic hyponatremia, the present prospective study was conducted to evaluate symptoms associated with hyponatremia and related to the serum osmolality. We measured serum osmolality and calculated the osmolar gap. Cerebrospinal fluid (CSF) sodium, which decreased in animal experiments with dilutional hypotonic hyponatremia with cerebral edema [4], was also measured to estimate brain interstitial fluid (ISF) sodium and to evaluate the effect of hyponatremia on the central nervous system during and after TURP.

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Materials and methods

Experiment A

A prospective study was undertaken on 165 consecutively treated Japanese patients undergoing TURP for benign prostatic hyperplasia (BPH), from January 1992 to March 1993 at Kumamoto Chuo Hospital. One hour prior to TURP, patients were premedicated with atropine sulfate 0.25–0.5 mg and hydroxyzine 25–50 mg given intramuscularly. To all these patients, spinal anesthesia was administered through a 23-gauge needle at the L2–3 or L3–4 level, using 1.5–2.0 ml of Percamine S (0.3% dibucaine and 5% NaCl) with 0.2 ml of epinephrine. The anesthesia reached a sensory level of approximately T8–10. During the surgery, lactated Ringer's solution was administered intravenously. The irrigating fluid was 3% sorbitol solution (osmolality 169 mOsm·kg^{-1}) and infused with a hydrostatic pressure of 60–70 cm H_2O. The intermittent bladder-filling technique with suction drainage of irrigating fluid was used.

Blood pressure, heart rate, electrocardiogram (ECG) and oxygen saturation by fingertip pulse oximeter were monitored during TURP. Sedation was not given so that the level of consciousness could be observed.

Blood samples were collected immediately before and after the surgical procedures and serum electrolytes (sodium, potassium), serum osmolality, serum glucose, blood urea nitrogen (BUN), and arterial blood gas values were measured. We calculated the "osmolar gap" as follows:

$$\text{Osmolar Gap} = (\text{measured osmolality}) - (\text{calculated osmolality})$$

$$\text{calculated osmolality} = (2 \times \text{serum sodium}) + (\text{glucose}/18) + (\text{BUN}/28)$$

When the serum sodium level fell below 120 $mEq \cdot L^{-1}$ during TURP, additional samples were taken 30, 90, and 180 min after the procedure.

Experiment B

After receiving approval from the Hospital Committee on Human Research and after obtaining informed consent, ten other patients were entered into this study. In five patients with hyponatremia, arterial blood and lumbar CSF were collected before, immediately after, and 30, 60, and 90 min after the procedure to measure electrolytes (sodium, potassium) and osmolality. In five patients who did not develop hyponatremia, arterial blood and lumbar CSF were also collected before, immediately after, and 90 min after the procedure. Lumbar CSF was collected through a single puncture, at each sampling time with patient's permission.

Serum sodium and potassium were measured by flame photometry, osmolality by the freezing point depression method, glucose and BUN by an enzymatic method, and blood gas analysis by the electrode method. Student's t-test was used for statistical evaluation and a $P < 0.05$ value was considered significant.

Results

Experiment A

Data on the 165 patients are presented in Table 1. No patient required blood transfusion. Table 2 summarizes the medical histories. These patients were divided into two groups based on the levels of postoperative serum sodium: Group A, serum sodium above 120 $mEq \cdot L^{-1}$; group B, serum sodium below 120 $mEq \cdot L^{-1}$.

Of the 165 patients, 10 (6.0%) showed a serum sodium value below 120 $mEq \cdot L^{-1}$. There was no significant difference in the two groups with regard to age, body weight, weight of resected prostate, resection time, or volume of irrigating fluid (Table 1). Medical histories included hypertension ($n = 4$), arrhythmia ($n = 3$), pulmonary emphysema ($n = 1$), and cerebral infarction ($n = 1$).

In group A, the serum sodium was 139.0 ± 3.3 $mEq \cdot L^{-1}$ preoperatively and decreased to 132.7 ± 4.3 $mEq \cdot L^{-1}$ postoperatively. The measured serum osmolality decreased from 282.0 ± 4.4 to 277.8 ± 4.6 $mOsm \cdot kg^{-1}$, and the calculated osmolality decreased from 290.1 ± 6.7 to 278.2 ± 9.0 $mOsm \cdot kg^{-1}$; there was no osmolar gap (Fig. 1). Arterial blood gas analysis showed no significant change (pre- and postoperative HCO_3^- were 24.7 ± 3.3 and 23.9 ± 3.2 $mEq \cdot L^{-1}$, Pco_2 were 37.5 ± 5.2 and 36.9 ± 5.4 mmHg).

In group B, the volume of irrigating fluid absorbed into the circulation was 2.7 ± 1.0 L, as calculated from the following equation:

$$\text{volume absorbed} = \left[\frac{(\text{preoperative serum sodium})}{(\text{postoperative serum sodium})} \times \text{ECF}\right] - \text{ECF}$$

The extracellular fluid (ECF) was estimated to be 20% of body weight (in kg).

The serum sodium decreased from 138.9 ± 4.0 to 111.9 ± 6.4 $mEq \cdot L^{-1}$ postoperatively. With administration of furosemide 10–20 mg and 10% NaCl 20–80 ml infusion, the serum sodium gradually recovered to

Table 1. Patient background information

	Overall	Group A (Na > 120 $mEq \cdot L^{-1}$)	Group B (Na ≦ 120 $mEq \cdot L^{-1}$)
No. of patients	165	155	10
Age (years)	71.3 ± 8.7	71.2 ± 8.9	72.8 ± 5.1
Body weight (kg)	57.7 ± 8.0	57.8 ± 8.1	55.9 ± 5.2
Weight of resected specimen (g)	16.7 ± 11.7	16.6 ± 11.4	18.5 ± 16.4
Operation time (min)	57.5 ± 20.5	57.8 ± 20.4	53.1 ± 21.9
Volume of irrigating fluid (L)	24.4 ± 9.2	24.4 ± 9.3	23.5 ± 8.2

Table 2. Medical history of 165 patients

Hypertension	52	31.5%
Heart disease and/or arrhythmia	38	23.0%
Lung disease	24	14.5%
Diabetes mellitus	22	13.3%
Liver dysfunction	19	11.5%
Cerebrovascular disease	16	9.6%

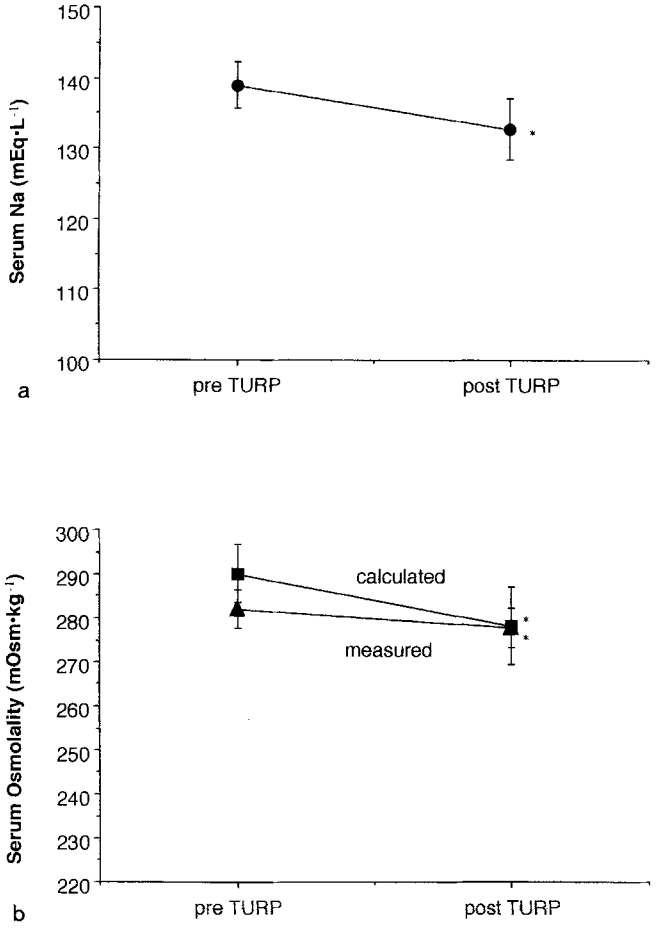

Fig. 1 Changes in serum sodium (**a**), mesured and calculated osmolalities (**b**) of group A ($Na > 120$ mEq·L^{-1}, $n = 155$). Values are mean ± SD. *$P < 0.05$. TURP, transurethral resection of the prostate

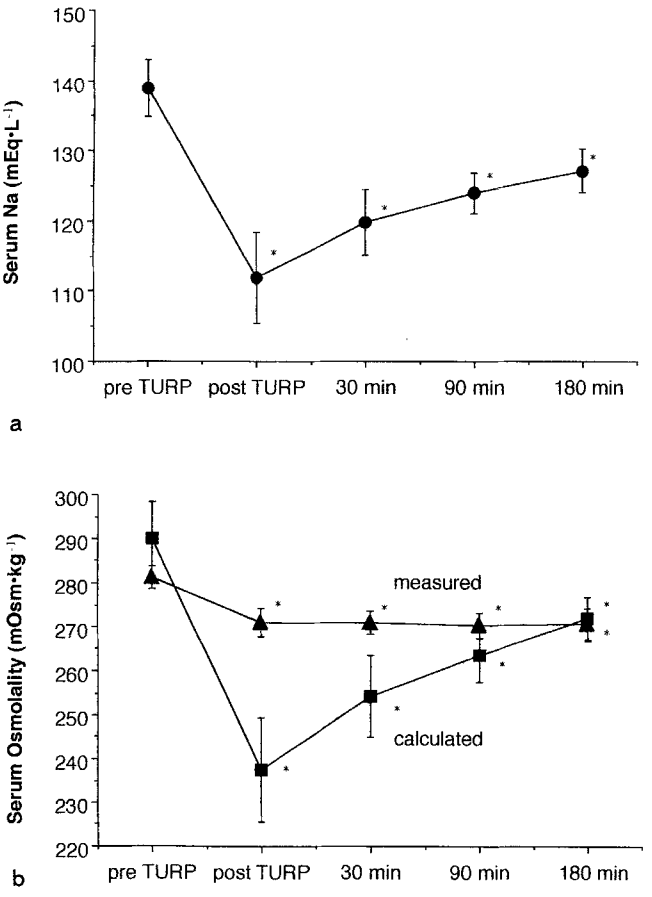

Fig. 2 Time course of changes in serum sodium (**a**), measured and calculated osmolalities (**b**) of group B ($Na \leqq 120$ mEq·L^{-1}, $n = 10$). The distance between the two curves in the *bottom panel* represents the "osmolal gap." Values are mean ± SD. *$P < 0.05$ compared with preoperative value

127.1 ± 3.2 mEq·L^{-1}, at 180 min postoperatively. The measured serum osmolality decreased from 281.3 ± 2.6 to 271.0 ± 3.3 mOsm·kg^{-1}, and the calculated osmolality from 290.0 ± 8.4 to 237.4 ± 11.9 mOsm·kg^{-1}. The osmolar gap increased to 33.5 ± 10.4 mOsm·kg^{-1} at the end of the procedure (Fig. 2) and thereafter the osmolar gap gradually decreased. In the arterial blood gas analysis, HCO_3^- significantly decreased from 26.2 ± 3.1 to 22.5 ± 3.0 mEq·L^{-1}, but Pco_2 showed no significant change (Table 3).

For eight patients, heart or cerebrovascular diseases, diabetes mellitus, and liver and renal dysfunction were absent but two patients had pulmonary emphysema or cerebral infarction. Signs and symptoms in group B, postoperatively, were ST depression on ECG, cardiac arrhythmia, and restlessness during the surgery (1 case each), nausea and vomiting ($n = 7$), chest oppression ($n = 4$), confusion ($n = 2$), and headache and bradycardia (1 case each) (Table 4). In all but one, the systolic blood pressure fell to 80–100 mmHg, and dopamine 3–7 γ·kg^{-1}·min^{-1} infusion was required. In seven patients, hypotension rapidly occurred in the postoperative period. Two patients became confused for a few minutes, in the presence of hypotension (65/30 mmHg) or bradycardia (40 beats·min^{-1}) immediately and 1 h after the procedure.

Experiment B

In patients with hyponatremia, the serum sodium decreased from 140.2 ± 1.3 to 118.0 ± 6.7 mEq·L^{-1} postoperatively, and after 90 min it gradually reverted

Table 3. Time course changes in serum sodium, osmolality, and blood gas analysis (group B, Na ≦ 120 mEq·L^{-1}, $n = 10$)

Time	Na (mEq·L^{-1})	Osm (mOsm·kg^{-1})			Blood gas analysis		
		Measured	Calculated	Osmolar gap	pH	Pco_2 (mmHg)	HCO_3 (mEq·L^{-1})
Pre-TURP	138.9 ± 4.0	281.3 ± 2.6	290.0 ± 8.4	−8.9 ± 7.8	7.42 ± 0.03	39.9 ± 5.1	26.2 ± 3.1
Post-TURP	111.9 ± 6.4*	271.0 ± 3.3*	237.4 ± 11.9*	33.5 ± 10.4	7.40 ± 0.06	36.5 ± 6.5	22.5 ± 3.0*
30 min	119.7 ± 4.6*	271.0 ± 2.5*	254.1 ± 9.3*	16.9 ± 8.8	7.39 ± 0.04	36.9 ± 5.9	22.2 ± 3.0*
90 min	123.9 ± 3.0*	270.3 ± 3.0*	263.4 ± 6.0*	7.3 ± 8.7	7.41 ± 0.04	36.2 ± 5.9	22.7 ± 2.9*
180 min	127.1 ± 3.2*	270.7 ± 3.5*	271.8 ± 5.1*	−0.1 ± 4.8	7.43 ± 0.03	37.5 ± 3.8	24.8 ± 2.6

Values are means ± SD.
TURP, transurethral resection of the prostate
* $P < 0.05$ (compared with preoperative value)

Table 4. Data on ten patients with hyponatremia

Case no.	Weight of resected specimen (g)	Operation time (min)	Signs and symptoms	Postoperative		Osmolar gap (mOsm·kg^{-1})
				Na (mEq·L^{-1})	Osm (mOsm·kg^{-1})	
1	8	65	Nausea, vomiting, hypotension	116	275	37
2	4	33	Chest oppression, hypotension	113	272	32
3	15	72	Chest oppression, nausea, hypotension	109	266	34
4	60	100	Restlessness, nausea, hypotension	115	269	23
5	25	25	Chest oppression, nausea, hypotension	114	276	34
6	6	38	Arrhythmia	116	270	29
7	7	56	Chest oppression, confusion nausea, vomiting, hyptension	107	271	31
8	6	28	ST depression, nausea, hypotension	115	274	30
9	24	55	Hypotension	119	271	23
10	30	59	Confusion, headache nausea, vomiting, hypotension	95	266	62
mean	18.5	53.1		111.9	271.0	33.5

to 127.4 ± 6.5 mEq·L^{-1}. The CSF sodium increased from 147.2 ± 3.6 to 159.2 ± 8.5 mOsm·kg^{-1} postoperatively, which was attributed to spinal anesthesia with 5% NaCl in Percamine S, and after 90 min it recovered to the preoperative level of 149.0 ± 3.5 mOsm·kg^{-1}. In the control patients, the change in CSF sodium was similar to that seen in those with hyponatremia, but with no significant difference (Fig. 3).

Discussion

Excessive intravascular absorption of irrigating fluid can cause hyponatremia and overloading of the circulatory system. Sodium is one of the essential electrolytes for proper function of excitatory cells, particularly for those of the brain and heart. Severe hyponatremia can lead to serious neurological symptoms, including seizures and loss of consciousness [4]. Hypotension and suppressed myocardial contractility can occur when the serum sodium falls below 120 mEq·L^{-1}, and bradycardia and ECG changes (widening QRS complex [5], ventricular ectopic beats and T-wave inversion [6]) are likely to be present when the level falls below 115 mEq·L^{-1}. A level below 100 mEq·L^{-1} may cause cardiac arrest [5].

For irrigating fluid during TURP, distilled water was first used because of its excellent optical qualities. However, it was extremely hypotonic, and caused hemolysis, shock, renal failure, and cerebral edema. Thus, isotonic solutions such as sorbitol, glycine, mannitol, and glucose became the irrigating solution of choice. Glycine is widely used but it has toxic effects on the heart and retina [7–9], and its metabolites, including ammonia, may cause CNS depression [10]. We use 3% sorbitol, which is non-toxic and is rapidly metabolized in the liver to fructose and glucose [11]. In the group B patients (Na ≦ 120 mEq·L^{-1}), the calculated osmolality decreased but the measured osmolality remained near normal. Although there was an osmolar gap between them, our cases are representative of isotonic hyponatremia. The osmolar gap is attributed to sorbitol in

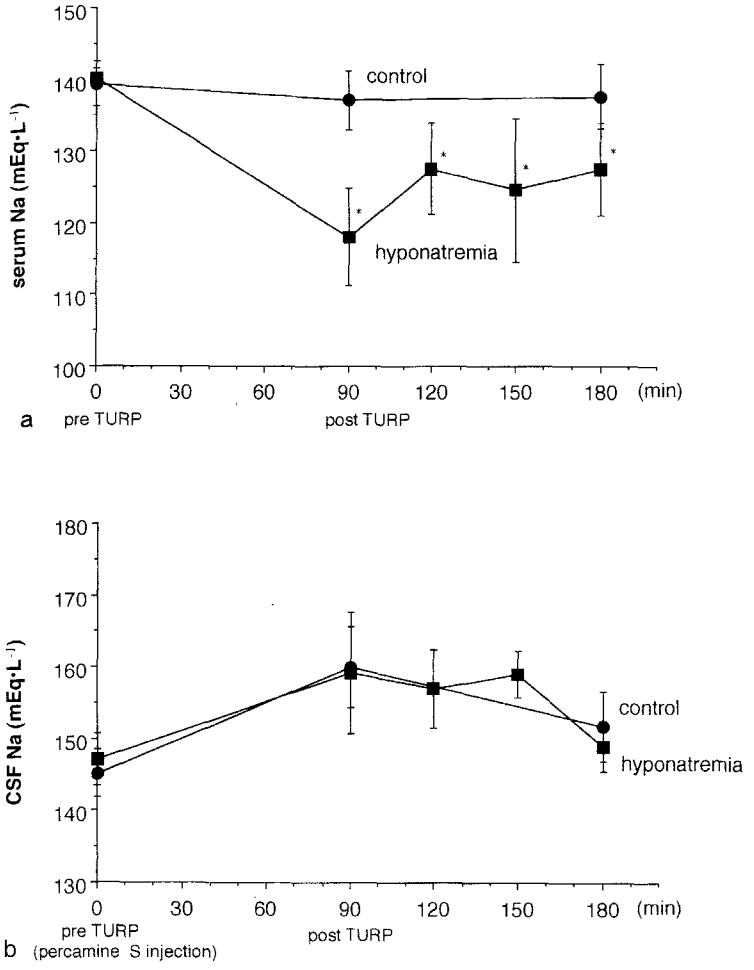

Fig. 3 Time course of changes in sodium of serum **(a)** and cerebrospinal fluid (CSF) **(b)** in both control and hyponatremia. Values are mean ± SD. $^*P < 0.05$ compared with control

the serum, since sorbitol itself contributes to the measured serum osmolality. According to Arieff et al. [4], 2 h after induction of dilutional hypotonic hyponatremia in laboratory animals, an osmotic gradient between plasma and the brain of at least 30 mOsm·kg^{-1} was required to induce acute cerebral edema, with a high mortality rate. Therefore, cerebral edema was not likely to occur in our patients.

Animal experiments related to dilutional hypotonic hyponatremia showed that the plasma sodium decreased 23 mEq·L^{-1} and the CSF sodium decreased 28 mEq·L^{-1} [4]. In one study, isotonic hyponatremia was produced by continuous hemodialysis over 2 h; plasma sodium progressively decreased from 145 to 78 mEq·L^{-1} and CSF sodium decreased from 152 to 140 mEq·L^{-1} [12]. After 2–3 h of isotonic hyponatremia in another group of animals, the decreases in plasma and CSF sodium were 16 and 11 mEq·L^{-1}, respectively [13]. In cases of isotonic hyponatremia, CSF sodium decreases slightly but the change is significantly less than the change seen in plasma and it remains within a normal range.

We first measured the CSF sodium of the patients with hyponatremia during and after TURP. The CSF sodium slightly increased postoperatively (1.5 h after Percamine S injection), and it reverted to the preoperative level approximately 1.5 h after TURP (3 h after injection of the anesthetic). Percamine S is a hyperbaric solution that contains 5% NaCl. Five percent NaCl is equal to 855 mEq·L^{-1} and the volume of the total spinal subarachinoid space is around 30 ml, so the 1.5–2.0 ml of Percamine S we used is considered to be enough to increase the CSF sodium level. However, Percamine S has little effect on CSF sodium levels postoperatively because injected Percamine S spreads to the lower spinal subarachinoid space where it is quickly absorbed by nerve fibers. There was no significant difference between the control and the hyponatremia group, with regard to the CSF sodium level. Therefore, the CSF sodium did not decrease and there was a CSF-to-serum sodium gradient in isotonic hyponatremia during and after TURP. These findings are important to understand the pathophysiology of asymptomatic hyponatremia.

Brain interstitial fluid (ISF) and CSF are not always in equilibrium. In laboratory animals, the ^{22}Na uptake curve from the plasma into the CSF was resolved into two components: a fast component (half-life 0.18 h) that represents isotope movement across the blood-CSF barrier (the choroid plexus), and a slow component (half-life 1.2 h) that reflects isotope distribution from brain ISF into the CSF [14]. Because of the rapid exchange of isotopes between the brain ISF and CSF, CSF sodium is considered to be a good approximation of the brain ISF sodium.

Patients with benign prostatic hyperplasia (BPH) are generally elderly men, many of whom may have either heart disease or little cardiac reserve. Among our patients, there were six cases of old myocardial infarction, ten of angina pectoris, and three of valve disease. When the left heart handles fluid overload, the fluid should move rapidly into the interstitial space. A study of a 65-year-old man who was given 1.3 L of 5% dextrose intravenously over a 20-min period suggests that the circulatory blood volume did not change [15]. The incompetent heart, however, cannot handle this load and pulmonary edema follows left heart failure. In one of our patients with ST depression, the intravascular load was not handled well, as 1.5 L of irrigating fluid was absorbed, and in such cases intracardiac pressure can increase and lead to ischemia. According to Agius and Cutajar [16], a 77-year-old man with angina pectoris became confused, pulmonary edema occurred after TURP, and serum sodium was 120 $mEq \cdot L^{-1}$; he died on the 2nd postoperative day from biventricular failure. When left heart failure occurs, the absorbed fluid will remain in the intravascular space, and the fall of serum sodium and osmolality secondary to metabolism of the solute will give rise to cerebral edema. Serum sodium and osmolality should be closely monitored especially for the patients with heart and renal disease.

For an early diagnosis of hyponatremia, anesthesiologists should watch for changes in blood pressure, pulse rate, ECG, and the level of consciousness in patients during TURP. Blood pressure first increases with absorption, unless there is a significant loss of blood. The central venous pressure (CVP) should be monitored in the poor-risk patient. CVP rises faster than blood pressure [17], and a statistically significant correlation was seen between the degree of hyponatremia and the rise in CVP [18].

Six of the ten patients ($Na \leqq 120\ mEq \cdot L^{-1}$) were in stable condition during surgery, and the blood sample taken immediately after TURP suggested the presence of hyponatremia. There was a delay in the onset of symptoms following TURP and hypotension rapidly occurred postoperatively. Thus, estimation of the electrolyte balance should be done immediately after TURP to diagnose and treat hyponatremia as early as possible.

If the patient is asymptomatic with isotonic hyponatremia ($Na > 120\ mEq \cdot L^{-1}$) and has adequate renal and cardiac function, no treatment is indicated. The hyponatremia will correct itself as the excess water is excreted and the sorbitol is metabolized [19], but when the sodium falls below 120 $mEq \cdot L^{-1}$, brain and heart functions can be affected. Fluid that is moved to the interstitial space will later return to the vascular compartment, resulting in difficulty in correcting the serum sodium. In addition, serum osmolality may fall several hours after TURP if clearance of sorbitol (half-life 35 min [11]) is more rapid than that of free water. Hypotonic hyponatremia will lead to irreversible brain damage and administration of hypertonic saline is often prescribed for such patients. However, if the osmolality is in the normal range, a hyperosmolar state may occur. There is also the problem of central pontine myelinolysis with a rapid and excess correction of hyponatremia [20]. Accordingly, when the sodium falls below 120 $mEq \cdot L^{-1}$, we recommend administration of hypertonic saline and diuretics with frequent measurement of the serum electrolytes and osmolality until the sodium level reaches 125–130 $mEq \cdot L^{-1}$. One hundred milliliters of 5% NaCl over a period of 1–2 h is feasible [21].

We conclude that in the case of acute, dilutional, and isotonic hyponatremia during TURP, the symptoms are mild and CSF sodium is well preserved, despite severe postoperative hyponatremia. The human body can tolerate even severe hyponatremia for a short time if the serum osmolality remains normal.

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