Effect of Potassium Solution on Rehydration in Rats: Comparison with Sodium Solution and Water

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Abstract Thermally dehydrated rats were given isotonic KCl, NaCl solution, or tap water ad libitum for 17h and the differences of the restoration rate in fluid and cation were compared between the groups to elucidate the effect of Na⁺ and K⁺ ions on the replacement of each body fluid compartment during rehydration. When rats were provided with NaCl solution, the gains of fluid and Na⁺ exceeded the amounts lost during the dehydration period, while in the isotonic KCl and tap water groups fluid gain was 70% of the fluid lost during the dehydration period. The recovery of extracellular fluid (ECF) volume was 178% of the loss in the NaCl group and 50% in the KCl group. The Na⁺ concentration of the ECF was regulated closely in all groups. The recovery in intracellular fluid (ICF) volume did not differ significantly between groups and never exceeded the control level, but tended to be higher in the KCl group than in the NaCl group. These results indicated that in the ECF the regulation of Na⁺ concentration preceded that of volume while in the ICF, volume regulation had priority. In addition, the effect of K⁺ supplementation on the recovery of ICF volume after thermal dehydration was shown to be modest, unlike the effect of Na⁺ on the recovery of ECF volume.

Key words: thermal dehydration, cation balance, fluid balance, ECF, ICF.

Previous studies have shown that during the rehydration process after thermal dehydration, Na⁺ replacement is indispensable for the full restoration of lost body fluid (Nose et al., 1985; Okuno et al., 1988). When rats were provided with a choice of water and NaCl solution to drink, they consumed hypotonic Na⁺ solution to correct hypertonicity in the early stages of rehydration, and then corrected the volume deficit by consuming almost isotonic fluid thereafter (Yawata et al., 1987). Similar responses have been reported for polyethylene glycol induced hypovolemia (Stricker et al., 1969, 1981). These findings indicate that under hypertonic or isotonic dehydration, the behavioral control of extracellular osmolality precedes.

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volume regulation. During dehydration, fluid shift from the intracellular fluid space is also known to take place (Nose et al., 1983, 1985), but the recovery process of intracellular dehydration remains unclear, although volume-regulatory behavior at cell level has been reported (Grinstein et al., 1984). In the present experiment, the effects of K⁺ supplementation on the rehydration process were studied by providing isotonic KCl solution *ad libitum* to thermally dehydrated rats. The changes of water balance and cation levels during rehydration were compared with rats given isotonic NaCl solution or tap water. The changes in volume and cation content in extracellular fluid (ECF) and intracellular fluid (ICF) were also determined before and after rehydration, and the effects of Na⁺ and K⁺ ions on the replacement of each body fluid compartment during rehydration were analyzed.

**METHODS**

Thirty-nine adult Wistar male albino rats weighing 320 ± 6 g were used for the 4 series of experiments. The rats were housed in mesh cages in a room maintained at 23–25°C and illuminated from 700 to 1800. They were provided with laboratory rat chow (Oriental, Osaka) and tap water *ad libitum* except during the experiments. They were deprived of food for 12 h prior to and during the experiments to avoid any influence of food intake on the fluid and electrolyte balance.

*Rehydration experiment.* The water and ionic balances were determined during the rehydration process for 17 h after thermally induced dehydration. Twenty rats were dehydrated by exposure to a temperature of 36°C (dry bulb temperature) with a 20% relative humidity. Dehydration was performed by placing the metabolic cages containing rats into a thermostatically controlled box (Tabai, Osaka LC-100) for about 7 h. No water was given during this period. When the rats weighed about 9% less than their initial body weight, they were removed from the hot environment and allowed 30 min at room temperature (25°C) to regain a normal colonic temperature (36–37°C). They were then divided into 3 groups and given isotonic KCl solution (*n* = 7), isotonic saline (*n* = 7), or tap water (*n* = 7) *ad libitum* starting at 1800. Fluid intake was measured by a drop counter (Muromachi Kikai, Tokyo) and recorded with a data logger (Thermodac 32, Etoh Electronics, Tokyo). Urine was collected at 30-min intervals using a fraction collector (Micro Fractionator, Gilson, WI). The urinary excretion of Na⁺ and K⁺ was calculated from the urine volume and the Na⁺ and K⁺ concentrations were determined using a flame photometer (480, Corning Medical). To determine the cation loss during heat exposure, the cage was washed down with distilled water and the Na⁺ and K⁺ levels were determined.

* Determination of plasma and ECF parameters. ECF volume, plasma volume (PV), plasma, Na⁺ and K⁺ concentrations ([Na] and [K]), and plasma osmolality were determined in the control group (*n* = 6), the dehydrated group (*n* = 6), and 3 rehydrated groups after 17 h of the free intake of each solution (*n* = 7 in each group). The rats were anesthetized with pentobarbital Na (4.0 mg/100 g i.p.), and the right
jugular vein was cannulated with a catheter. Both renal arteries and veins were ligated to avoid loss of $^{51}$Cr-EDTA into the urine. And 5 μCi of $^{51}$Cr-EDTA was injected (Daichi Pure Chemicals, Tokyo) through the jugular catheter, and allowing 50 min for equilibration, 3 μCi of $^{125}$I-radiiodinated human serum albumin (Amersham, Tokyo) was similarly injected. After 10 min, 2 ml of blood was obtained through the jugular catheter, and the rat was killed by the injection of pentobarbital Na (7 mg/100 g, i.v.) through the catheter. After centrifugation, the blood samples were used for the determination of ECF and PV, as well as plasma [Na] and [K], as previously described (Nose et al., 1983).

The changes in ICF volume after rehydration were calculated as follows:

$$\Delta IC F = F_{gain} - \Delta ECF,$$

where $F_{gain}$ is the fluid gain determined by subtracting urine volume from fluid intake, and $\Delta ECF$ is the difference in ECF volume compared with the mean volume of the dehydrated group. The electrolyte content of each fluid compartment was calculated as follows:

$$E_{ECF} = [E]_{PI} \times PV + [E]_{PI} \times 0.95 \times (ECF - PV);$$

$$\Delta E_{ICF} = E_{gain} - \Delta E_{ECF},$$

where $E$ is the electrolyte content (Na⁺ or K⁺), $[E]_{PI}$ is the plasma concentration, and 0.95 is the Gibbs-Donnan equilibrium ratio between serum and interstitial fluid.

Plasma K⁺ concentration after 1.5 h of rehydration with KCl solution was determined on 6 rats. Dehydrated rats were given 154 mEq/l KCl solution for 1.5 h and blood was sampled by cardiac puncture.

**Tissue water and cation content.** Pieces (1–2 g) of thigh muscle and liver were sampled to determine their fluid volume and electrolyte content. The volume of each fluid compartment was determined in these tissues from the radioactivities as previously described (Nose et al., 1983). Tissue water content was determined by drying tissues at 105°C to a constant weight for 24 h. Electrolyte levels in each tissue sample were determined by flame photometry after ashing them at 550°C (Operusen, Toyo, Tokyo). ICF was determined by subtracting the ECF from the tissue water content.

The results were normalized for 100 g of body weight except for the tissue values which were normalized for dry weight. Results are given as the mean ± S.E. Comparisons between groups were made using analysis of variance (Sokal and Rohlf, 1981), and the level of significance was set at $p < 0.05$.

**RESULTS**

*Rehydration experiment*

Figure 1 shows the time courses of cumulative fluid intake and urine volume during rehydration. Dehydrated rats drank vigorously when they were provided
with any kind of fluid and excreted little urine up to about 4 h, and then they continued to drink small amounts till the 11th h while urine output became almost equal to fluid intake. Figure 2 shows the time course of fluid balance during rehydration. The amount of fluid loss during dehydration was $8.9 \pm 0.1 \text{ml/100 g body weight}$ ($n = 28$), and is indicated by a broken line. The fluid balance remained in virtually a steady state from the 4th h; and after 17 h of rehydration, the NaCl group had regained $9.5 \pm 0.5 \text{ml/100 g body weight}$, 108% of the fluid lost, whereas both the KCl and tap water groups had regained only 70%. To demonstrate the relationship between fluid intake and urine output, cumulative urine volume was plotted against the cumulative fluid intake volume measured every 30 min (Fig. 3). In the NaCl group, the fluid intake was $9.8 \pm 1.8 \text{ml/100 g body weight}$ during the initial 4 h of rehydration, and only 13% of the fluid intake was excreted as urine, while thereafter about 70% of the fluid intake was excreted. In the tap water group, the fluid intake was $5.7 \pm 0.3 \text{ml/100 g body weight}$ in the first 2.5 h and 9% of the fluid intake was excreted, but thereafter urine output almost equalled fluid intake. In the KCl group, the fluid intake before the shift (observed in the 4th h) was $6.1 \pm 0.3 \text{ml/100 g body weight}$ and 21% of the intake was excreted. Thus, fluid retention was less in the KCl group than in the other groups, although it later
Fig. 2. Cumulative fluid balance determined by subtracting urine volume from fluid intake during the recovery from thermal dehydration. Amount of fluid loss during dehydration is shown by (---). Values are the mean and S.E. of 7 rats for the KCl and NaCl groups and 6 rats for the tap water group.

Fig. 3. Relationship between fluid intake and urine volume. Each point indicates cumulative mean value measured every 30 min. Regression lines were calculated so as to obtain the highest correlation coefficient.
increased gradually to become similar to that in the tap water group.

As for the cations, the relationship between cation intake and loss into urine during rehydration was similar to that between fluid intake and urine volume. In the NaCl group, the amount of Na⁺ retained during the initial 4 h was 1,000 μEq/100 g body weight, and the Na⁺ balance remained steady until the 12th h, and then it gradually decreased. The Na⁺ retention after 17-h rehydration was 822 ± 40 μEq/100 g body weight. The K⁺ loss in the NaCl group was minimal and the cumulative loss was 190 μEq/100 g body weight during the 17-h rehydration period. Rats provided with KCl solution retained 400 μEq/100 g body weight of K⁺ during the initial 1.5 h, and then excreted an equal amount to that ingested. Final retention of K⁺ was 479 ± 47 μEq/100 g body weight. The Na⁺ loss in the KCl group was 200 μEq/100 g body weight in the initial 2.5 h and almost nil thereafter. In the tap water group, Na⁺ loss was 65 μEq/100 g body weight and K⁺ loss was 90 μEq/100 g body weight during the 17-h rehydration period.

**Plasma and ECF parameters**

Table 1 shows the plasma parameters, fluid volume, and cation content in ECF and ICF after dehydration and rehydration. Dehydration caused increases in plasma [Na⁺] and osmolality and a decrease in [K⁺]. Plasma [Na⁺] recovered to the control level after rehydration, except in the KCl group where it remained significantly lower. Plasma [K⁺] was significantly lower than the control level in the NaCl and tap water groups and higher in the KCl group. Plasma osmolality recovered to the control level in each group.

ECF volume decreased 17% from the control value with dehydration. After the 17-h rehydration period, the gain was 178% of the lost fluid in the NaCl group, 75% in the tap water group, and 50% in the KCl group. ICF volume calculated by subtracting the changes in ECF from the total fluid gain decreased 5.3 ml/100 g body weight with dehydration and recovered by 83% in the KCl group, 64% in the tap water group, and 58% in the NaCl group. The change in extracellular Na⁺ content after dehydration (293 μEq/100 g body weight) was almost equal to the Na⁺ loss during dehydration, while the extracellular K⁺ level showed little change, indicating that K⁺ loss occurred from the ICF.

After rehydration, 85% of the Na⁺ gained by rats from the NaCl solution was retained in the ECF. In contrast, the increase in extracellular K⁺ content was only 6% of the K⁺ gained from the KCl solution and the remainder entered the ICF. The decrease in intracellular Na⁺ content in the KCl group amounted to 216 μEq/100 g body weight. Although electrolytes were not given to the tap water group, there was a slight increase of extracellular cations, and a decrease of intracellular cations.

Figure 4 shows the relationship between changes in ECF or ICF volumes (ΔECF or ΔICF) and total fluid gain (F_gain) determined using the data obtained on each rat after the 17-h rehydration. A significant positive correlation was observed between total fluid gain and the change in ECF in the NaCl group ($r=0.95, p<0.01$, J. Physiol. Osaka)
Table 1. Plasma parameters, fluid volume, and cation content.

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 6)</th>
<th>Dehydration (n = 6)</th>
<th>Rehydration with</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>KCl (n = 7)</td>
<td>NaCl (n = 7)</td>
<td>Tap water (n = 7)</td>
</tr>
<tr>
<td><strong>Plasma</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Volume (ml/100 g body wt.)</td>
<td>2.66 ± 0.05</td>
<td>2.46 ± 0.06</td>
<td>2.53 ± 0.06</td>
<td>3.15 ± 0.14*</td>
<td>2.80 ± 0.08</td>
</tr>
<tr>
<td>[Na] (mEq/l)</td>
<td>141.8 ± 0.7</td>
<td>152.9 ± 0.7*</td>
<td>139.5 ± 0.3*</td>
<td>141.2 ± 0.5</td>
<td>141.1 ± 0.3</td>
</tr>
<tr>
<td>[K] (mEq/l)</td>
<td>3.55 ± 0.06</td>
<td>3.31 ± 0.08</td>
<td>4.63 ± 0.13*</td>
<td>3.13 ± 0.08*</td>
<td>2.95 ± 0.09*</td>
</tr>
<tr>
<td>Osmolality (mOsm/kg)</td>
<td>292.2 ± 1.2</td>
<td>312.2 ± 2.0*</td>
<td>292.6 ± 1.2</td>
<td>287.9 ± 1.4</td>
<td>291.0 ± 1.6</td>
</tr>
<tr>
<td><strong>Fluid volume (ml/100 g body wt.)</strong></td>
<td></td>
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<tr>
<td>ΔTotal</td>
<td></td>
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<tr>
<td>ECF</td>
<td>20.5 ± 0.3</td>
<td>16.9 ± 0.3*</td>
<td>18.7 ± 0.3</td>
<td>23.3 ± 0.8*</td>
<td>19.6 ± 0.2</td>
</tr>
<tr>
<td>ΔECF</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>ΔICF</td>
<td>-5.3</td>
<td>4.4</td>
<td></td>
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<tr>
<td>Na (μEq/100 g b.w.)</td>
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<tr>
<td>ΔTotal</td>
<td>-330 ± 16</td>
<td>-203 ± 29*</td>
<td>+822 ± 40*</td>
<td>-65 ± 15*</td>
<td></td>
</tr>
<tr>
<td>ECF</td>
<td>2776 ± 36</td>
<td>2483 ± 45</td>
<td>2496 ± 42</td>
<td>3176 ± 127*</td>
<td>2644 ± 30</td>
</tr>
<tr>
<td>ΔECF</td>
<td>-293</td>
<td>13</td>
<td>693</td>
<td>+161</td>
<td></td>
</tr>
<tr>
<td>ΔICF</td>
<td>-37</td>
<td>-216</td>
<td>+129</td>
<td>-225</td>
<td></td>
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<tr>
<td>K content (μEq/100 g body wt.)</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>ΔTotal</td>
<td>-170 ± 7</td>
<td>+479 ± 47*</td>
<td>-192 ± 14*</td>
<td>-90 ± 8</td>
<td></td>
</tr>
<tr>
<td>ECF</td>
<td>69.5 ± 1.4</td>
<td>54 ± 2*</td>
<td>83 ± 3*</td>
<td>74 ± 4</td>
<td>55 ± 2</td>
</tr>
<tr>
<td>ΔECF</td>
<td>-16</td>
<td>29</td>
<td>+16</td>
<td>+2</td>
<td></td>
</tr>
<tr>
<td>ΔICF</td>
<td>-154</td>
<td>450</td>
<td>-209</td>
<td>-92</td>
<td></td>
</tr>
</tbody>
</table>

ΔTotal: total balance, (intake) − (loss); ΔECF: change in extracellular compartment during dehydration or rehydration; ΔICF: change in intracellular compartment, (ΔTotal) − (ΔECF). Values are mean ± S.E. *Significant difference from the control value at *p* < 0.05. †Significant difference from other rehydration groups at *p* < 0.01.
Fig. 4. Relationship between fluid gain and ECF (top) or ICF (bottom) replenishment after 17 h of rehydration with isotonic KCl solution (○), NaCl solution (●), or tap water (×). Each point indicates the value from each rat.

$\Delta$ECF = 1.6$F_{\text{gain}}$ - 9.0, and between fluid gain and the change in ICF in the KCl group ($r=0.91, p<0.025$, $\Delta$ICF = 1.7$F_{\text{gain}}$ - 6.2). The mean total fluid loss and the ECF and ICF losses during dehydration are shown by broken lines. In the NaCl group, both total fluid and ECF gain were higher than the amount lost, and the relationship between total fluid gain and ECF gain indicated that the fluid gained in excess of the control level was retained mainly in the ECF. Although the ECF loss was not regained in the KCl group, the ICF volume increased in proportion to the total fluid gain. However, no significant difference in the recovery of the ICF was found between the groups, and the ICF did not increase above the control level except in one rat. In addition, the ICF in the NaCl group tended to diminish as total fluid gain increased.

Figure 5 shows the relationship between the changes in ECF volume and extracellular Na$^+$ after rehydration. There was a positive correlation between these 2 parameters ($r=0.98$). The volume of 1.6 ml, the point where the line crosses the abscissa, equals the amount of water required to restore to normal the increase in Na$^+$ concentration which occurred during dehydration.

Plasma [K] was 6.7 ± 1.2 mEq/l after 1.5 h of rehydration when rats drank 3.5 ± 1.6 ml/100 g body weight of KCl solution, while the value increased to 8.1 mEq/l.
Fig. 5: Relationship between ECF replenishment and extracellular Na⁺ content after 17h of rehydration with isotonic KCl solution (○), NaCl solution (●), or tap water (×). Each point indicates the value from each rat.

when a rat drank 6.0 ml/100 g body weight in 1.5 h.

Tissue water and cation content

The changes in water and cation content in liver and muscle are summarized in Table 2. After dehydration, water content decreased in both tissues, and there was a significant decrease of K⁺ in muscle. After rehydration, trends similar to those for the whole body were observed. In the NaCl group, Na⁺ exceeded the control value and K⁺ was less than in the control value in both tissues, with hepatic K⁺ being significantly lower than in the dehydrated state. In the KCl group, both fluid and cations recovered to the control level in both tissues. The muscle K⁺ level did not exceed the control level, in contrast to the result for the whole body balance. In the tap water group, a significant difference from the control values was found for the K⁺ content in both tissues.

DISCUSSION

During thermal dehydration, rats lose Na⁺ in the urine and saliva and cannot
Table 2. Water and cation content of liver and muscle.

<table>
<thead>
<tr>
<th></th>
<th>Control (n=6)</th>
<th>Dehydration (n=6)</th>
<th>Rehydration with</th>
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<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>KCl (n=7)</td>
<td>NaCl (n=7)</td>
<td>Tap water (n=7)</td>
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<tr>
<td>Liver</td>
<td></td>
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<tr>
<td>Total water (ml/g dry wt.)</td>
<td>2.43±0.04</td>
<td>2.21±0.04*</td>
<td>2.40±0.03</td>
<td>2.57±0.07</td>
<td>2.38±0.04</td>
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</tr>
<tr>
<td>ECF volume (ml/g dry wt.)</td>
<td>0.80±0.03</td>
<td>0.69±0.03</td>
<td>0.79±0.01</td>
<td>0.89±0.07</td>
<td>0.73±0.03</td>
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</tr>
<tr>
<td>ICF volume (ml/g dry wt.)</td>
<td>1.63±0.03</td>
<td>1.53±0.02</td>
<td>1.62±0.02</td>
<td>1.68±0.04</td>
<td>1.65±0.02</td>
<td></td>
</tr>
<tr>
<td>Na (µEq/g dry wt.)</td>
<td>107±7</td>
<td>102±5</td>
<td>98±3</td>
<td>133±6*</td>
<td>107±6</td>
<td></td>
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<tr>
<td>K (µEq/g dry wt.)</td>
<td>295±7</td>
<td>290±5</td>
<td>296±4</td>
<td>244±11*</td>
<td>228±7*</td>
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<tr>
<td>Muscle</td>
<td></td>
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<tr>
<td>Total water (ml/g dry wt.)</td>
<td>3.15±0.01</td>
<td>2.74±0.06*</td>
<td>3.13±0.03</td>
<td>3.22±0.01</td>
<td>3.18±0.01</td>
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</tr>
<tr>
<td>ECF volume (ml/g dry wt.)</td>
<td>0.35±0.01</td>
<td>0.32±0.02</td>
<td>0.34±0.01</td>
<td>0.43±0.01*</td>
<td>0.34±0.01</td>
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<tr>
<td>ICF volume (ml/g dry wt.)</td>
<td>2.80±0.02</td>
<td>2.42±0.06*</td>
<td>2.79±0.03</td>
<td>2.78±0.01</td>
<td>2.84±0.02</td>
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<tr>
<td>Na (µEq/g dry wt.)</td>
<td>74±2</td>
<td>72±2</td>
<td>66±2</td>
<td>100±3*</td>
<td>82±2</td>
<td></td>
</tr>
<tr>
<td>K (µEq/g dry wt.)</td>
<td>473±2</td>
<td>446±2*</td>
<td>460±6</td>
<td>438±5*</td>
<td>449±3*</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean±S.E. * Significant difference from the control value at p<0.05.
REHYDRATION AND K⁺ SOLUTION

restore the fluid loss without replacement of the lost Na⁺ (Nosé et al., 1985; Okuno et al., 1988), although correction of hypertonicity of the ECF precedes volume expansion (Yawata et al., 1987; Sugimoto, 1988).

In this study, such trends were observed in the tap water and NaCl groups. Rats given tap water drank and retained more than the NaCl group for the first 2.5 h, and then showed a urine output almost equal with fluid intake. In the NaCl group, the increase in urine output was observed after 4 h of rehydration (cf. Fig. 3), and the delay of start of urine output caused a significantly greater fluid gain in the NaCl group. On the other hand, rats given KCl solution regained fluid more slowly than the other groups. An increase in plasma [K⁺] is known to have a powerful kaliuretic effect (Young, 1982), and the increase in plasma [K⁺] to 6.7 mEq/l observed at 1.5 h of KCl consumption explains the larger early urine output in this group.

Within 4.5 h of rehydration, the water diuresis in the tap water group seemed to become comparable with kaliuresis in the KCl group, and fluid balance in the KCl group was almost the same as in the tap water group after 4.5 h.

A concentration of 154 mEq/l of KCl was chosen because of the isotonicity, and also to compare the results with those of isotonic NaCl solution. The concentration seems to be rather high in comparison with normal foods. For example, a commercially available liquid diet for rats contains about 40 mEq/l K⁺, but the K⁺ concentration in vegetables is in the range of 20–140 mEq/l, and in the case of potatoes, the value is known to be 143 mEq/kg H₂O. When 154 mEq/l KCl solution was provided, plasma [K⁺] increased to 6.7 mEq/l at the 1.5 h, and the fluid intake was almost the same as in the tap water group. From these results, it appears that ingestion of K⁺ at this concentration of K⁺ can be regulated.

It is known that insulin and catecholamines play an important role in the disposal of an acute potassium load by transfer from the extracellular to the intracellular compartment (DeFronzo, 1985). These factors might be involved in the regulation of the K⁺ concentration in the body fluid during the initial rehydration period when urine output was at a minimum. After the first 1.5 h, most of the K⁺ ingested was excreted and Na⁺ excretion was negligible. Consequently the urinary Na⁺ to K⁺ ratio was very low, which suggested an increase in the secretion of aldosterone. The time when the ratio reached a steady state coincides with the delay before aldosterone would have started to act. Thus, it is considered that the K⁺ load taken in after dehydration was disposed of by transfer into the intracellular compartment in the early phase and by renal excretion after aldosterone secretion, consequently lowering the increased plasma [K⁺] after 17-h rehydration.

ECF volume expanded when Na⁺ was provided, while the deficit due to dehydration was not regained when Na⁺ was not provided. In addition, Fig. 5 indicates that even when Na⁺ was not given, as in the tap water and KCl groups, the changes in ECF volume closely paralleled the change in ECF Na⁺ content and the Na⁺ concentration of the fluid gained by the ECF was regulated closely.

On the other hand, K⁺ was lost during dehydration, mainly from muscle,
which coincides with our previous findings (Nose et al., 1985). The differences in the ICF between the groups were not so large as those in the ECF and the ICF K⁺ level never exceeded the control level even with the high intake of K⁺ after rehydration. However, the degree of ICF rehydration tended to be higher in the KCl group than in the NaCl group, and further increased with the increase in total fluid balance. These results suggest the importance of K⁺ supplementation to allow recovery of ICF volume. The Na⁺ level in the ICF also changed considerably, which suggested some influence of Na⁺ on the recovery of ICF volume. In the tap water group, fluid volume, plasma [Na], and the tissue Na⁺ content showed a good recovery, despite the absence of cation supplements, which could presumably be attributed to the buffering action of the ICF and the production of unknown osmotic substances in response to dehydration (Pollock and Aliff, 1980).

Tissue analysis did not show any K⁺ gain in excess of the amount lost during dehydration in the KCl group, which suggested differences in the distribution of the ingested K⁺. Edmonds (1967) reported that the concentration of K⁺ in fecal water in the distal colon was usually over 10-fold greater than in the ECF. Active secretion of K⁺ into the colon has also been reported (Smith and McCabe, 1984). Rabinowitz et al. (1986) reported that the gut K⁺ level after ingestion of a liquid diet containing 150 mEq/l of K⁺ for 3 days was 150 μEq/100 g body weight. Although the cation content of the gastrointestinal tract was not determined in our experiment, it is possible that K⁺ was retained in the colon, and that the difference of K⁺ concentration between the ECF and the colon resulted in a higher ICF K⁺ level.

In summary, when isotonic Na⁺ solution was provided to dehydrated rats, the recovery of fluid and Na⁺ content was in excess of the control level and expansion of the ECF was prominent. When isotonic K⁺ solution or tap water was provided, the water loss was not regained, and this was attributable to inadequate replacement of the ECF. The ICF volume never exceeded the control level, even with K⁺ supplementation. These findings suggest that the regulation of Na⁺ concentration in the ECF precedes that of volume and the regulation of ICF volume precedes that of K⁺ concentration. It also appears that the effect of supplementary K⁺ on the recovery of the ICF after thermal dehydration is not so large as that of Na⁺ on the ECF.

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