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Mechanism Underlying the Changes in Plasma Potassium Concentration during Infusion of Isosmotic Nonelectrolyte Solution

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Generally, during infusion of an isosmotic nonelectrolyte solution that permeates the cell membrane, plasma K^+ concentration ($[K^+]_{pl}$) either does not change or it increases slightly. The mechanism underlying this $[K^+]_{pl}$ change has not been clarified. We continuously monitored the $[K^+]_{pl}$ and plasma Na^+ concentration ($[Na^+]_{pl}$) for 10 min during isosmotic mannitol infusion of 1.6 ml/100 g body weight in rats with intact kidney function (intact mannitol group). In addition, in nephrectomized rats, we compared the $[K^+]_{pl}$ change during infusion with isosmotic mannitol (which permeates the cell membrane; mannitol nephrectomized group) with that during infusion with isosmotic sucrose (which does not permeate the cell membrane; sucrose nephrectomized group) to evaluate the effect of cell volume regulation. In the intact mannitol group, $[Na^+]_{pl}$ decreased with dilution, and $[K^+]_{pl}$ remained relatively constant. In the sucrose nephrectomized group, $[K^+]_{pl}$ decreased by the same percentage as $[Na^+]_{pl}$ and gradually increased to greater than the control level. In the mannitol nephrectomized group, however, $[K^+]_{pl}$ increased immediately after the beginning of the infusion and reached the same level as that in the sucrose nephrectomized group. To confirm that the difference in $[K^+]_{pl}$ between the mannitol and sucrose nephrectomized groups was dependent on cell volume regulation, we investigated the changes in mean corpuscular volume of red blood cells, using a Coulter counter. This value remained constant during isosmotic sucrose infusion but increased during isosmotic mannitol infusion, returning to the original volume after the infusion. We kept $[HCO_3^-]$ and *p*H constant throughout the experiments. We conclude that cell volume regulation and dilutional effects, apart from dilutional acidosis, are responsible for the maintenance of constant $[K^+]_{pl}$ during the infusion of an isosmotic nonelectrolyte solution that permeates the cell membrane. (Fluid balance: electrolytes. Ions: potassium.)

INFUSION of isosmotic monosaccharide solution induces an increase in plasma K^+ concentration ($[K^+]_{pl}$), even when plasma Na^+ concentration ($[Na^+]_{pl}$) is reduced by dilution.[1,2] Similarly, intravascular absorption of isosmotic glycine irrigating fluid in patients undergoing transurethral resection of the prostate induces both an increase in $[K^+]_{pl}$ and hyponatremia, with no hemolysis.[3,4] Many investigators regard this phenomenon to be a result of K^+ efflux from the intracellular space due to ionic dilution in the extracellular space; *i.e.*, it is regarded as being due to dilutional acidosis[1,2,5] and low ionic strength effect.[6] However, the mechanism underlying this $[K^+]_{pl}$ change has not been clearly elucidated. We hypothesized that the change induced in $[K^+]_{pl}$ by infusion of an isosmotic nonelectrolyte solution, which permeates the cell membrane, depends on cell volume regulation, in addition to dilutional effects and renal excretion.

We have found no studies in which the effect of cell volume regulation[7–9] on $[K^+]_{pl}$ change during infusion of isosmotic nonelectrolyte solution was addressed. Since monosaccharides and glycine can permeate cell membranes (mannitol permeates hepatic[10] and red blood cell membranes[11]), isosmotic solutions of these compounds cause cell swelling, the swollen cells tending to return to their original volume. This process in cell volume regulation is called regulatory volume decrease and involves the efflux of K^+ and water from the intracellular fluid space.[7–9]

We performed experiments in rats with intact kidneys and in nephrectomized rats to separate the renal effect on $[K^+]_{pl}$ change. In the nephrectomized group, isosmotic sucrose infusion was used to evaluate the dilutional effects only, since sucrose does not permeate the cell membranes.[10] Comparing the effect of isosmotic mannitol infusion with that of isosmotic sucrose infusion thus excluded the dilutional effect from the effect on the regulatory volume decrease response. We also carried out microscopic observations of the shape of rat red blood cells *in vitro* in the isosmotic mannitol and sucrose solutions to confirm that isosmotic mannitol causes cell swelling, and we evaluated the changes in mean corpuscular volume of the red blood cells *in vivo* during isosmotic sucrose or mannitol infusion using a Coulter counter.

Materials and Methods

These studies were approved by the animal research committee of the Kyoto Prefectural University of Medicine. Experiments were performed on 39 male Wistar

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rats weighing 275.7 ± 7.3 g (mean ± SE). In the first series of experiments, isosmotic mannitol was infused in rats with intact kidney function (intact mannitol group, n = 7). In the second series of experiments, nephrectomized rats were infused with either isosmotic mannitol (mannitol nephrectomized group, n = 7) or isosmotic sucrose (sucrose nephrectomized group, n = 7). The remaining 18 rats were used to evaluate the changes in mean corpuscular volume of the red blood cells and the effect of nephrectomy on $[Na^+]_{pl}$ and $[K^+]_{pl}$.

All surgical interventions were carried out while the animals were anesthetized with pentobarbital (5 mg/100 g body weight, intraperitoneally). In the intact mannitol group, the urethra was ligated; the abdomen was opened by a midline incision; and the bladder was cannulated with a polyethylene tube (Clay Adams PE-90) for collecting the urine. In the mannitol nephrectomized and sucrose nephrectomized groups, the abdomen was opened in the same way as in the intact mannitol group, and both renal arteries and veins were ligated. After closure of the abdomen, the right carotid artery and jugular vein were cannulated with polyethylene tubes (Clay Adams PE-50). A polyethylene catheter (Terumo, Japan) was inserted into the trachea, and the rats breathed spontaneously.

Polyethylene tubes were used to establish an arteriovenous extracorporeal shunt between the right carotid artery and the right jugular vein to measure $[Na^+]_{pl}$ and $[K^+]_{pl}$ continuously, as previously reported by our laboratory.[12] Briefly, flow-through Na^+- (Horiba 2431A, Kyoto) and K^+- (Horiba 8411A) sensitive electrodes were installed with a reference electrode (Horiba 1411A) in the extracorporeal circuit, and electromotive force was transmitted to a high-impedance amplifier (Horiba HC-100). Flow in the circuit was maintained at 0.4 ml/min with a minipulse pump (Gilson Minipuls 2). The output of the electrodes was recorded (Rikadenki R64RS, Osaka) and analyzed by computer (NEC PC9801U, Tokyo) at 5-s intervals. To calibrate the electrodes, we computed a regression equation describing the relationship between the electromotive force and the log of the ion concentration, using two standard solutions ($[Na^+]$ = 140 and $[K^+]$ = 4.1, $[Na^+]$ = 61 and $[K^+]$ = 1.8 mEq/l) adjusted to *p*H 7.4 with Tris buffer. The 95% confidence limits were 1.5 mEq/l between 120 and 150 mEq/l for $[Na^+]$[12] and 0.05 mEq/l between 3.75 and 6.05 mEq/l for $[K^+]$. The 95% response time of each electrode was less than 5 s.

In the intact mannitol group, after $[Na^+]_{pl}$ and $[K^+]_{pl}$ had stabilized, 1.6 ml/100 g body weight of isosmotic mannitol (296 mOsm/kg H_2O) was infused over 10 min through the catheter in the jugular vein with an injection pump (Terumo ME-STC521). Urine was collected at 5-min intervals, and the volume (Chyo MP-300, Kyoto) and $[Na^+]$ and $[K^+]$ (Corning 480 Flamephotometer) were measured.

In the mannitol nephrectomized group, 1.6 ml/100 g body weight of isosmotic mannitol (296 mOsm/kg H_2O) was infused for 10 min, and in the sucrose nephrectomized group 1.6 ml/100 g body weight of isosmotic sucrose (297 mOsm/kg H_2O) was infused. Three blood samples were obtained, 0.4 ml 20 min before the infusion and 0.1 ml 12 min and 40 min after the infusion began. These blood samples were used to determine *p*H and $[HCO_3^-]$ (Corning 178 pH/Blood Gas Analyzer).

Five-milliliter blood samples were collected from anesthetized rats, and the red blood cells were immediately centrifuged (3,000 × g for 5 min at 4° C). Two milliliters of isosmotic sucrose or mannitol solution was then added. To confirm that isosmotic mannitol causes cell swelling *in vitro*, we observed the shape of the red blood cells under a microscope (Nikon Diaphoto TMD with Plan 100 DL).

Changes in the mean corpuscular volume of the red blood cells were also evaluated during isosmotic mannitol (n = 5) and sucrose infusion (n = 5) in nephrectomized rats. Under the same protocol as that in the mannitol nephrectomized and sucrose nephrectomized groups, four blood samples were obtained; 0.4 ml just before infusion and 0.4 ml 12, 25, and 40 min after the beginning of the infusion. Immediately after collection, mean corpuscular volumes were measured with a Coulter counter (Coulter Electronics S8/80).

The effect of nephrectomy on $[Na^+]_{pl}$ and $[K^+]_{pl}$ was evaluated in eight rats using the same protocol as in the mannitol nephrectomized and sucrose nephrectomized groups.

All values are reported as means ± SE. Two-way repeated analysis of variance was used to determine significant differences. The least significant difference method was used to identify significant differences between groups in various pairwise comparisons. The null hypothesis was rejected when $P < 0.05$.

Results

Control values of $[Na^+]_{pl}/[K^+]_{pl}$ just before infusion were 137.9 ± 5.5/3.95 ± 0.53 mEq/l in the intact mannitol group, 139.0 ± 1.2/4.45 ± 0.17 mEq/l in the sucrose nephrectomized group, and 137.1 ± 1.5/4.47 ± 0.17 mEq/l in the mannitol nephrectomized group. There were no significant differences between the groups. Figure 1 shows the changes in $[Na^+]_{pl}$ and $[K^+]_{pl}$ from the control values in the intact mannitol group. The urine volume, urinary Na^+ excretion, and urinary K^+ excretion in the intact kidney group are shown in figure 2. Figures 3 and 4 show the changes in $[Na^+]_{pl}$ and $[K^+]_{pl}$ from the control values in the mannitol nephrectomized and sucrose nephrectomized groups. Percent changes in $[K^+]_{pl}$ and $[Na^+]_{pl}$ respectively obtained every 5 min after infusion in the mannitol nephrectomized and sucrose ne-

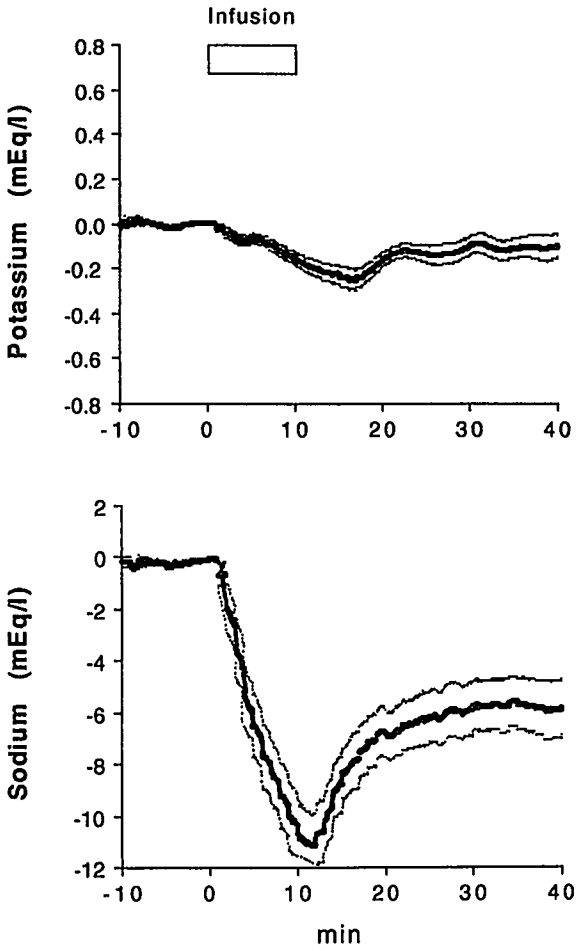

FIG. 1. Changes in plasma potassium concentration and sodium plasma concentration from control values after mannitol infusion in rats with intact kidneys (intact mannitol group, n = 7). Isosmotic mannitol, 1.6 ml/100 g body weight, was infused from 0 to 10 min. Heavy lines show mean values; fine lines represent ± SE.

phrectomized groups are shown in figure 5, for comparing the degree of dilution. After the infusion, we found no increase in plasma hemoglobin concentration determined spectrophotometricaly (Shimadzu UV-120-02, Japan), and there was no hemolysis.

In the intact mannitol group (fig. 1), $[K^+]_{pl}$ decreased slightly after the beginning of the infusion ($P < 0.05$). The nadir in this decrease followed, by about 10 min, the nadir of the decrease in $[Na^+]_{pl}$, after which $[K^+]_{pl}$ returned to the control level. $[Na^+]_{pl}$ decreased by 11 mEq/l during the infusion, increasing by about 4 mEq/l within 15 min after the end of the infusion. Urine volume, urinary Na^+ excretion, and urinary K^+ excretion increased sharply just after the beginning of the infusion ($P < 0.05$), decreasing rapidly after the end of the infusion (fig. 2). The maximum increases in urine volume, urinary Na^+ excretion, and urinary K^+ excretion occurred at the end of the infusion.

As shown in figures 3 and 5, $[K^+]_{pl}$ in the sucrose nephrectomized group decreased significantly during the infusion ($P < 0.05$) and then increased, exceeding the preinfusion level about 10 min after the end of the infusion ($P < 0.05$). In the mannitol nephrectomized group, $[K^+]_{pl}$ decreased transiently, by 0.04 mEq/l, for 1 min immediately after the beginning of the infusion, gradually increasing during and after the infusion ($P < 0.05$). $[K^+]_{pl}$ was significantly higher in the mannitol nephrectomized group than in the sucrose nephrectomized group from 5 to 20 min after the beginning of the infusion ($P < 0.05$; fig. 5). In both groups, $[Na^+]_{pl}$ decreased by 9–10 mEq/l, recovering, after the infusion recovered, to the same level as that in the intact mannitol group (fig. 4). The only significant difference in percent changes in $[Na^+]_{pl}$ between the mannitol nephrectomized and sucrose nephrectomized groups was after 5 min of infusion ($P < 0.05$; fig. 5).

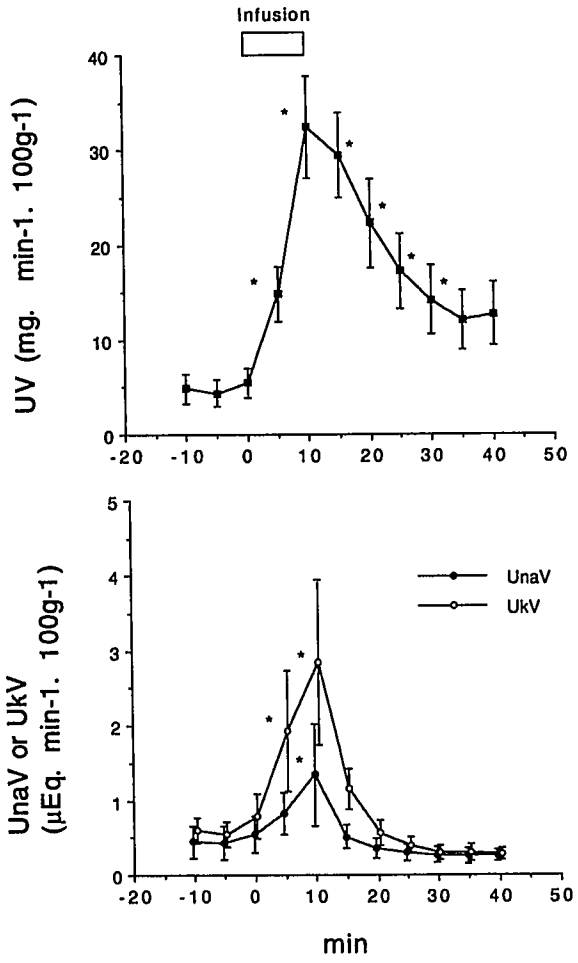

FIG. 2. Changes in urine volume (UV), urinary sodium excretion (UnaV), and urinary potassium excretion (UkV) in the intact mannitol group. Data represent mean ± SE. *Significantly ($P < 0.05$) different from control value at 0 min.

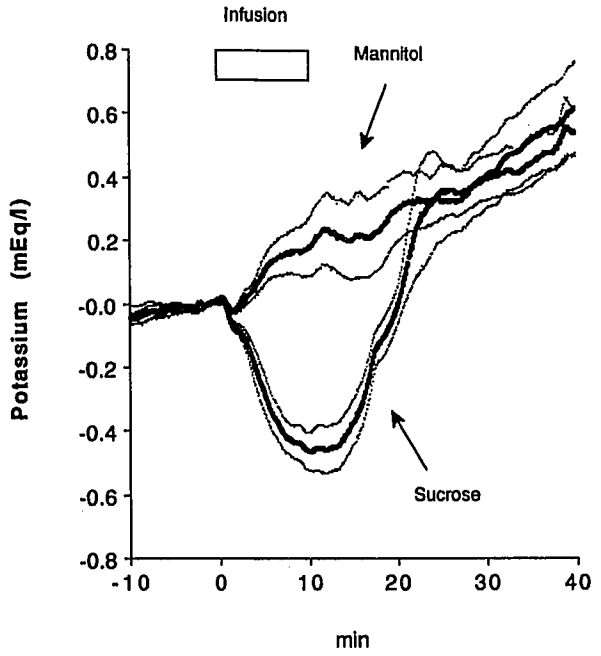

FIG. 3. Changes in plasma potassium concentration from control values in the mannitol nephrectomized (n = 7) and sucrose nephrectomized (n = 7) groups. Same format as in figure 1.

We observed that the shape of the red blood cells *in vitro* was spherocytic in the isosmotic mannitol solution although it showed no change in the isosmotic sucrose

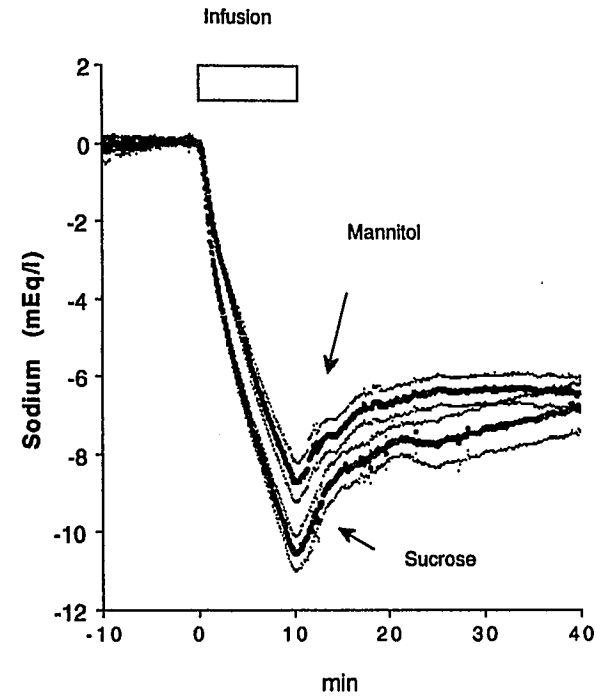

FIG. 4. Changes in plasma sodium concentration from control values in the mannitol nephrectomized (n = 7) and sucrose nephrectomized (n = 7) groups. Same format as in figure 1.

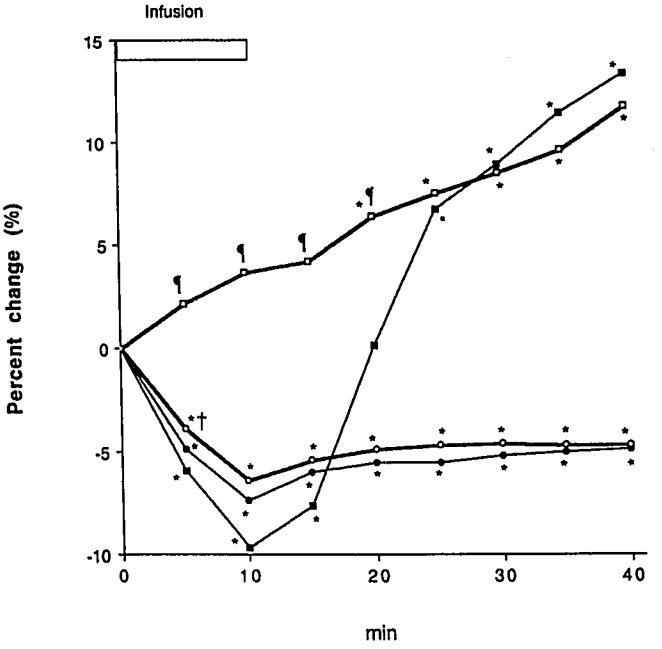

FIG. 5. Percent changes in plasma sodium concentration and plasma potassium concentration every 5 min after infusion in the mannitol nephrectomized (denoted by open circles and open squares with bold lines, respectively) and sucrose nephrectomized groups (denoted by closed circles and closed squares with fine lines, respectively). Data represent the mean, and each standard error was within 2.7%. *Significantly ($P < 0.05$) different from 0%. †Significantly ($P < 0.05$) different from percent change in plasma sodium concentration in the sucrose nephrectomized group. ¶Significantly ($P < 0.05$) different from percent change in plasma potassium concentration in the sucrose nephrectomized group.

solution. Figure 6 shows the percent changes in mean corpuscular volume of the red blood cells *in vivo* during isosmotic mannitol and sucrose infusions. This value showed no significant change with isosmotic sucrose infusion. With isosmotic mannitol, however, it increased significantly during infusion, returning to the original cell volume 25–40 min after the beginning of the infusion.

Ligation of the renal artery and vein caused $[K^+]_{pl}$ to increase, by 0.11 ± 0.05 mEq/l/h, while $[Na^+]_{pl}$ did not change. The values for pH/$[HCO_3^-]$ were 7.33 ± 0.04/24.3 ± 1.1 at −20 min, 7.34 ± 0.02/22.8 ± 1.3 at 12 min, and 7.35 ± 0.02/23.8 ± 2.1 at 40 min in the sucrose nephrectomized group. Also in the mannitol nephrectomized group, they were 7.31 ± 0.04/22.6 ± 0.8 at −20 min, 7.31 ± 0.02/22.6 ± 1.7 at 12 min, and 7.32 ± 0.03/22.9 ± 1.2 at 40 min. There were no significant changes in either group.

Discussion

The results of this study showed that the increase in $[K^+]_{pl}$ induced with isosmotic mannitol infusion in nephrectomized rats had two components, one component representing the difference in $[K^+]_{pl}$ change between the

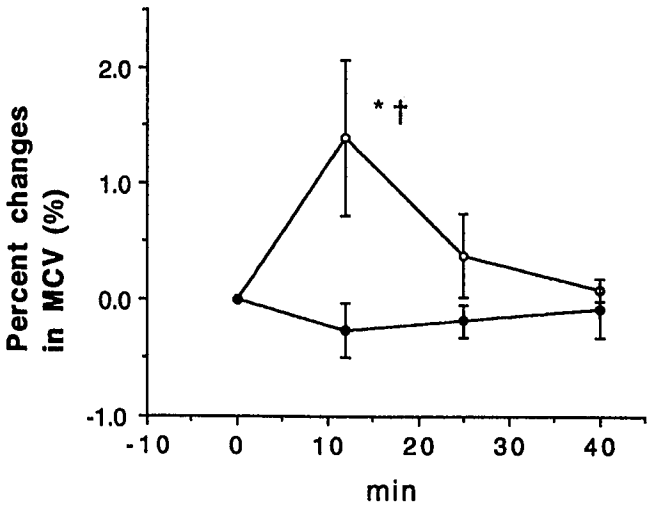

FIG. 6. Percent changes in mean corpuscular volume (MCV) of red blood cells. Isosmotic mannitol (open circles, n = 5) or isosmotic sucrose (closed circles, n = 5) was infused from 0 to 10 min in nephrectomized rats. Data represent mean ± SE. *Significantly ($P < 0.05$) different from the value in the sucrose infusion. †Significantly ($P < 0.05$) different from 0%.

mannitol nephrectomized and sucrose nephrectomized groups, and the other component representing the remainder of the increase in $[K^+]_{pl}$. The mean corpuscular volume of red blood cells increased with isosmotic mannitol infusion, its change paralleling the former component. Regulatory volume decrease response, low ionic strength effect, and dilutional acidosis may be involved in these two components of the increases in $[K^+]_{pl}$. In the present study, we examined the relationships among these factors to elucidate the mechanism underlying $[K^+]_{pl}$ change.

The decrease in $[Na^+]_{pl}$ in the intact mannitol group is explained by dilution and renal excretion, shown by the increase in urinary Na^+ excretion. $[K^+]_{pl}$, however, showed no change, and there was no hemolysis, although the extracellular fluid was diluted and urinary K^+ excretion increased. These results suggest that there may have been a K^+ efflux from the intracellular space.

The higher value of $[Na^+]_{pl}$ during mannitol than during sucrose infusion in the nephrectomized animals (fig. 5) appeared to depend on the movement of water into cells, and the disappearance of this difference after 10 min might be attributable to the efflux of water from the cells. In addition, investigation of the changes in mean corpuscular volume of the red blood cells provided direct evidence that the red blood cells *in vivo* were swollen by isosmotic mannitol infusion and returned to their original cell volume *via* the regulatory volume decrease response after the infusion (fig. 6). These results show that isosmotic mannitol infusion induced cell swelling due to extracellular hypotonicity[13] and that this induced the regulatory volume decrease response. Mannitol infusion may also induce the regulatory volume decrease response in rat hepatocytes, since it permeates hepatic cell membranes[10] and induces cell swelling, whereas sucrose cannot permeate these membranes.[10] The reflection coefficient of mannitol for the rat hepatic cell membrane is reported to be half that of sucrose.[14] The regulatory volume decrease response has been reported to begin within a few minutes in isolated perfused rat liver and kidney.[15–17]

In our experiment, dilutional effects could be excluded by comparing the mannitol nephrectomized group with the sucrose nephrectomized group. The difference in the changes in $[K^+]_{pl}$ between the mannitol nephrectomized and sucrose nephrectomized groups (fig. 3) does not include the changes induced in $[K^+]_{pl}$ due to dilutional effects, since isosmotic sucrose infusion induces only dilutional effects. We called this increase in $[K^+]_{pl}$ the first component, and its most possible cause may have been the regulatory volume decrease response, since it was observed in the mannitol nephrectomized group within a few minutes of the beginning of the infusion, and the changes in mean corpuscular volume of the red blood cells paralleled this component. After the end of the infusion, $[K^+]_{pl}$ gradually increased in both nephrectomized groups (fig. 3). This increase, which we called the second component, reflects the dilutional effects seen with a nonelectrolyte solution.

In the first component of $[K^+]_{pl}$ change, we assume that K^+ was released from the intracellular space concomitantly with the occurrence of the regulatory volume decrease response, as the toxicity of the extracellular space decreased as the infusion continued. The difference of $[K^+]_{pl}$ between the two nephrectomized groups gradually decreased after the end of infusion. This may indicate the recovery of intracellular K^+ after the regulatory volume decrease response, due to uptake of K^+ from the extracellular space.[18] The regulatory volume decrease response has been reported in rat thymic lymphocytes,[19] hepatocytes,[20,21] cerebrovascular endothelium,[22] renal cortex,[23] isolated kidneys,[15] and isolated livers.[16,17] In addition to red blood cells, these other cells may have been involved in the origin of the K^+ efflux in our study.

The second component of the $[K^+]_{pl}$ increase was observed in both the mannitol nephrectomized and sucrose nephrectomized groups and might be attributable to the dilution of the extracellular fluid by a large quantity of nonelectrolyte solution. Dilutional acidosis induced by a decrease in plasma $[HCO_3^-]$[2,5] and a low ionic strength effect[6] have been suggested as mechanisms underlying this increase. However, in this experiment, because the plasma $[HCO_3^-]$ and the *p*H did not change, it appears that dilutional acidosis did not cause this increase in $[K^+]_{pl}$. Bernhardt *et al.*[6] reported that decreases in extracellular ion concentrations stimulated Cl^--dependent Na^+–K^+ and

K^+–Cl^- cotransports and induced extracellular K^+ increases in human erythrocytes. This mechanism was called a low–ionic-strength effect, and it may have been involved in the second component of the $[K^+]_{pl}$ increase. Sympathetic activity, which may be increased by surgical stress, may be suppressed by cardiopulmonary baroreflex due to intravascular volume expansion; thus, changes in plasma noradrenaline and adrenaline concentration could affect $[K^+]_{pl}$.[24] These hormonal responses may be related to this second component.

In summary, we evaluated the changes in $[K^+]_{pl}$ induced by isosmotic mannitol infusion. Our results suggest that two components are involved in the increases in $[K^+]_{pl}$, the first possibly related to the regulatory volume decrease response and the second possibly due to dilutional effects, apart from dilutional acidosis. From a clinical viewpoint, these two components of $[K^+]_{pl}$ increases would counteract hypokalemia during the infusion of an isosmotic nonelectrolyte solution that permeates the cell membrane.

References

1. Kim WG, Brown EB Jr.: Potassium transfer with constant extracellular pH. J Lab Clin Med 71:678–685, 1968
2. Moreno M, Murphy C, Goldsmith C: Increase in serum potassium resulting from the administration of hypertonic mannitol and other solutions. J Lab Clin Med 73:291–298, 1969
3. Harn RG, Berlin T, Lewenhaupt A: Factors influencing the osmolality and the concentrations of blood haemoglobin and electrolytes during transurethral resection of the prostate. Acta Anaesthesiol Scand 31:601–607, 1987
4. Norlén H, Allgén L, Vinnars E, Bedrelidou-Classon G: Glycine solution as an irrigating agent during transurethral prostatic resection. Scand J Urol Nephrol 20:19–26, 1986
5. Asano S, Kato E, Yamauchi M, Ozawa Y, Iwasa M, Wada T, Hasegawa H: The mechanism of the acidosis caused by infusion of saline solution. Lancet 1:1245–1246, 1966
6. Bernhardt I, Erdmannn A, Vogel R, Glaser R: Factors involved in the increase of erythrocytes in low chloride media. Biomed Biochim Acta 46:S36–S40, 1987
7. Chamberlin ME, Strange, K: Anisosmotic cell volume regulation: A comparative view. Am J Physiol 257:C159–C173, 1989
8. Eveloff JL, Warnock DG: Activation of ion transport systems during cell volume regulation. Am J Physiol 252:F1–F10, 1987
9. Okada Y, Hazama A: A. Volume-regulatory ion channels in epithelial cells. News Physiol Sci 4:238–242, 1989
10. Alpini G, Garrick RA, Jones MJT, Nunes R, Tavoloni N: Water and nonelectrolyte permeability of isolated rat hepatocytes. Am J Physiol 251:C872–C882, 1986
11. Norlén H, Allgén L, Wicksell B: Mannitol concentrations in blood plasma in connection with transurethral resection of the prostate using mannitol solution as an irrigating fluid. Scand J Urol Nephrol 20:119–126, 1986
12. Nose H, Sugimoto E, Morimoto T, Usui S, Aomi T: Continuous recording of plasma sodium concentration and blood volume in awake rats. Jpn J Physiol 36:607–611, 1986
13. Rose BD: Physiology of body fluids, Clinical Physiology of Acid-Base and Electrolyte Disorders. New York, McGraw-Hill Information Services Company, 1989, pp 3–27
14. Haddad P, Thalhammer T, Graf J: Effect of hypertonic stress on liver cell volume, bile flow, and volume-regulatory K^+ fluxes. Am J Physiol 256:G563–G569, 1989
15. Joannidis M, Volkl H, Pfaller W, Lang F: Volume-regulatory potassium release from isolated perfused rat kidney. Renal Physiol Biochem 12:338–346, 1989
16. Haddad P, Graf J: Volume-regulatory K^+ fluxes in the isolated perfused rat liver: characterization by ion transport inhibitors. Am J Physiol 257:G357–G363, 1989
17. Lang F, Stehle T, Haussinger D: Water, K^+, H^+, lactate and glucose fluxes during cell volume regulation in perfused rat liver. Pflügers Arch 413:209–216, 1989
18. Guggino WB: Potassium and cell volume, The Regulation of Potassium Balance. Edited by Seldin DW, Giebisch G. New York, Raven Press, 1987, pp 121–137
19. Grinstein S, Smith JD: Calcium-independent cell volume regulation in human lymphocytes. J Gen Physiol 95:97–120, 1990
20. Christine EB: A nonselective cation channel in rat liver cells is activated by membrane stretch. Am J Physiol 258:C421–C428, 1990
21. Corasanti JG, Gleeson D, Boyer JL: Effects of osmotic stresses on isolated rat hepatocytes: I. Ionic mechanisms of cell volume regulation. Am J Physiol 258:G290–G298, 1990
22. Kempski O, Spatz M, Valet G, Baethmann A: Cell volume regulation of cerebrovascular endothelium in vitro. J Cell Physiol 123:51–54, 1985
23. Marin R, Ayes G, Proverbio T, Proverbio F: The effect of K^+ on the volume regulation in rat kidney cortex cells. Biomed Biochim Acta 48:303–315, 1989
24. DeFronzo RA, Bia M: Extrarenal potassium homeostasis, The kidney. Edited by Seldin DW, Giebisch G. New York, Raven Press, 1985, pp 1179–1206