Renal Excretion of Sodium During Oral Water Administration in Patients with Systemic Hypertension

By Robert A. Metzger, M.D., Liliana S. Vaamonde, M.D., Carlos A. Vaamonde, M.D., and Solomon Papper, M.D.

SUMMARY
The renal excretion of sodium and water was studied in 10 hypertensive subjects following an oral sustained 20 ml/kg water load. The study was performed under conditions controlled for sodium content of the diet, time of the day, and posture; the urine was collected by spontaneous voiding. The results were compared with those obtained from 10 normotensive subjects studied under similar conditions.

The increased urine flow following the water load in the hypertensive subjects was not accompanied by increased excretion of sodium, a pattern similar to the one observed in the normotensive subjects.

Comparing the group of hypertensive subjects with previously studied normotensives revealed the following additional information. The hypertensive subjects responded to water administration by increasing volume and C\text{H}_2\text{O} more rapidly than the normotensives; however, the maximal response was not significantly different. The fractional reabsorption of sodium was less in the hypertensive subjects despite a lower filtered load of sodium. This suggests a difference in the renal tubular handling of sodium between hypertensive and normotensive subjects.

Additional Indexing Words:
Sodium excretion  Exaggerated natriuresis  Water diuresis  Oral hydration

It is well established that patients with essential hypertension have an exaggerated natriuretic response to the rapid intravenous administration of sodium chloride solutions.\textsuperscript{1-4} This exaggerated natriuresis has also been reported following the infusion of glucose and mannitol solutions.\textsuperscript{5-8}

The pattern of excretion of sodium in hypertensive subjects following the oral ingestion of various solutions is less well documented.\textsuperscript{6, 9-11} It was, therefore, considered desirable to study the effects on sodium excretion of a standard acute oral water load in patients with elevated systemic blood

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pressure under circumstances controlled for sodium intake, posture, time of day, and without bladder catheterization. This information was also sought because in many of the intravenous salt-loading studies, an oral water load was administered at the same time in order to maintain a high urinary volume.

Our data demonstrate that sodium excretion in patients with systemic hypertension does not increase as urine flow increases following the water load. The hypertensive subjects responded to water administration more rapidly than a group of normotensives previously studied, although their maximal responses were not significantly different.

Methods

Ten hypertensive patients (HT), aged 37 to 66 years (mean, 49 years), were studied (table I). All subjects but four (V.A., V.M., D.V., and J.S.) were males. The patients were selected on the basis of a maintained resting diastolic blood pressure of at least 95 mm Hg. All but two were regarded as having essential hypertension on the basis of normal urinalyses, serum electrolytes, renal function studies, urinary vanilmandelic acid excretion and rapid sequence intravenous pyelogram (IVP). Patients J.L. and V.A. had unilateral renovascular hypertension diagnosed on the basis of abnormal rapid sequence IVP, radioactive renogram, angiotensin infusion test, renal arteriography, and differential renal function studies. This was confirmed by the return of the blood pressure to normal levels following removal of the involved kidney in patient V.A. Patient J.L. died 3 weeks after reparative surgery of the renal artery, but he remained normotensive during that period.

All had electrocardiographic evidence of left ventricular strain but were otherwise free of renal, cardiac, hepatic, or endocrine disease. Ocular fundi varied from normal to the presence of arteriolar narrowing. No hemorrhages, exudates, or papilledema was observed. All antihypertensive medication and diuretics were withheld for at least 2 weeks prior to the study day.

For at least 4 days prior to the study, all hypertensive subjects consumed a diet containing approximately 10 mEq of sodium daily which was supplemented with 160 mEq of sodium given in gelatin capsules. Daily urinary sodium excretion was measured throughout the experimental period, and seven patients were in sodium balance prior to the study. Patients J.D., V.M., and F.N. ingested more sodium than that contained in the experimental diet since their daily urinary sodium excretions exceeded the calculated sodium in the diet, but their weights remained stable or increased slightly.

The standard water test was done as follows: The subjects were instructed to proceed with their normal activities and fluid intake up to the morning of the study. On this day breakfast was withheld. All studies were done during the morning hours with the subjects in the recumbent position (standing only to void). At least two 60-min urine collections were obtained prior to the administration of the water load to all subjects except hypertensives J.L. and V.A. An oral water load of 20 ml/kg of body weight was then given in approximately 20 to 30 min and sustained throughout the study by replacing the voided urine with an equivalent amount of water plus 40 ml/hr to replace the insensible loss. Urine was collected every 30 min by spontaneous voiding. Blood samples were drawn immediately prior to the water load and after the last collection of urine.

Urine and serum were analyzed for sodium and potassium with an internal lithium standard flame photometer and for total solutes with a Fiske osmometer. Creatinine was measured in the urine by the method of Peters and in the serum by the method of Hare. Hematocrit values were estimated by the microcapillary technique. Endogenous creatinine (C_{CR}) and osmolar (C_{osm}) clearances were calculated by the conventional formulas. Solute-free water clearance (C_{H_2O}) represents the difference between the urinary flow per min (V) and C_{osm}. The filtered sodium (F_{Na}) was estimated using the formula: F_{Na} = serum concentration Na \times 0.95 \times C_{CR}. The amount of sodium delivered to the distal tubule was estimated as the sum of the urinary excretion of sodium (U_{Na}V) plus the sodium equivalent of C_{H_2O}. All data were corrected for body surface area of 1.73 m² and expressed as the mean ± 1 standard deviation (sd). Statistical evaluation of the data was done with Student’s t test for paired and unpaired data.

Although comparison with normotensive subjects is not necessary for the main purpose of
### Table 1

**Response to Oral Water Load in Ten Hypertensive Patients**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yr)</th>
<th>Blood Pressure (mm Hg)</th>
<th>V* (ml/min/1.73 m²)</th>
<th>U_{osm}† (mOsm/Kg H₂O)</th>
<th>C₆H₅O* (ml/min/1.73 m²)</th>
<th>Pre-load‡ C₆H₅O (ml/min/1.73 m²)</th>
<th>Post-load§ C₆H₅O (ml/min/1.73 m²)</th>
<th>V/CCr × 100* (ml/min/1.73 m²)</th>
<th>C₆H₅O/CCr × 100* (ml/min/1.73 m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.L.</td>
<td>43</td>
<td>200/112</td>
<td>14.5</td>
<td>48</td>
<td>11.1</td>
<td>101</td>
<td>101</td>
<td>16.3</td>
<td>13.5</td>
</tr>
<tr>
<td>V.A.</td>
<td>37</td>
<td>180/100</td>
<td>11.0</td>
<td>61</td>
<td>8.6</td>
<td>105</td>
<td>105</td>
<td>9.9</td>
<td>7.1</td>
</tr>
<tr>
<td>C.L.</td>
<td>55</td>
<td>150/100</td>
<td>13.2</td>
<td>54</td>
<td>10.7</td>
<td>107</td>
<td>108</td>
<td>12.5</td>
<td>10.1</td>
</tr>
<tr>
<td>L.D.</td>
<td>46</td>
<td>150/100</td>
<td>14.2</td>
<td>65</td>
<td>10.9</td>
<td>137</td>
<td>115</td>
<td>9.6</td>
<td>7.3</td>
</tr>
<tr>
<td>M.G.</td>
<td>54</td>
<td>190/120</td>
<td>10.9</td>
<td>50</td>
<td>8.8</td>
<td>71</td>
<td>70</td>
<td>17.7</td>
<td>14.6</td>
</tr>
<tr>
<td>V.M.</td>
<td>48</td>
<td>190/116</td>
<td>11.7</td>
<td>85</td>
<td>8.1</td>
<td>79</td>
<td>79</td>
<td>16.7</td>
<td>11.6</td>
</tr>
<tr>
<td>F.N.</td>
<td>66</td>
<td>174/112</td>
<td>13.8</td>
<td>70</td>
<td>10.4</td>
<td>82</td>
<td>71</td>
<td>17.7</td>
<td>13.3</td>
</tr>
<tr>
<td>D.V.</td>
<td>42</td>
<td>128/98</td>
<td>6.6</td>
<td>67</td>
<td>5.0</td>
<td>107</td>
<td>107</td>
<td>6.2</td>
<td>4.7</td>
</tr>
<tr>
<td>A.Z.</td>
<td>49</td>
<td>180/120</td>
<td>10.8</td>
<td>77</td>
<td>7.3</td>
<td>92</td>
<td>110</td>
<td>10.2</td>
<td>7.5</td>
</tr>
<tr>
<td>J.S.</td>
<td>46</td>
<td>170/110</td>
<td>12.0</td>
<td>66</td>
<td>9.0</td>
<td>80</td>
<td>88</td>
<td>14.5</td>
<td>10.5</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td></td>
<td>11.9</td>
<td>64</td>
<td>9.0</td>
<td>94</td>
<td>95</td>
<td>13.1</td>
<td>10.0</td>
</tr>
<tr>
<td>±σ</td>
<td>±2.3</td>
<td>±12</td>
<td>±1.9</td>
<td>±22</td>
<td>±17</td>
<td>±4.0</td>
<td>±3.3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Highest value following the water load.
† Lowest value following the water load.
‡ Value for the 60-min collection period immediately preceding the water load.
§ Mean value of 6 to 8 30-min periods immediately following the water load.
this study, we have included data on subjects with normal blood pressure from another study conducted in our laboratory because we believe some additional information was obtained in this fashion. These data were obtained with a comparable protocol to the present study in 10 healthy male normotensive subjects (NT), aged 24 to 36 years (mean, 29), who were consuming a regular diet containing 150 mEq of sodium daily.

**Results**

All the HT patients (mean BP, 171/109) had elevated blood pressures during the study period. Changes in diastolic blood pressure following the water load were inconsistent (−8 to +16 mm Hg) (table 1).

**Urine Flow**

All but one HT subject responded to the water load with urine flow (V) greater than 8 ml/min and all subjects had \( U_{\text{osm}} \) below 100 mOsm/kg H2O. A comparison of the pattern

*In our laboratory the normal response to water loading under these experimental conditions is a maximal flow of 8 ml/min or greater and a urinary osmolality below 100 mOsm/kg H2O.

**Figure 1**

The effect of a 20-ml/kg oral water load on urine flow (V), rate of sodium excretion (\( U_{\text{Na}} V \)), and creatinine clearance (\( C_{\text{CR}} \)) in 10 hypertensive subjects receiving a normal (170 mEq/day) salt intake. All values are corrected for 1.73 m² body surface area. The initial water load was given at the time marked by the solid black bar. The solid line represents the mean value and the shaded area plus or minus 1 standard deviation of the mean for each corresponding collecting period. The values for the control period (−60 to 0 min) are the means plus or minus the standard deviations for the eight patients in whom collections were made prior to the water load.
### Table 3

**Response to Oral Water Loading in Normotensive and Hypertensive Subjects**

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>V (ml/min)</th>
<th>C_H2O (ml/min)</th>
<th>C_V (ml/min)</th>
<th>U_NaV (μEq/min)</th>
<th>U_kV (μEq/min)</th>
<th>Frac. reab. Na%</th>
<th>C_H2O(100/C_V) (ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
</tr>
<tr>
<td>-60</td>
<td>1.1 ± 0.6</td>
<td>2.7 ± 1.9</td>
<td>-1.2 ± 0.4</td>
<td>148 ± 87</td>
<td>64 ± 19</td>
<td>99.0 ± 0.2</td>
<td>97.9 ± 1.9</td>
</tr>
<tr>
<td>30</td>
<td>2.5 ± 2.9</td>
<td>5.8 ± 3.9</td>
<td>-0.2 ± 2.3</td>
<td>171 ± 107</td>
<td>84 ± 21</td>
<td>98.9 ± 0.7</td>
<td>98.1 ± 1.4</td>
</tr>
<tr>
<td>60</td>
<td>6.3 ± 4.4</td>
<td>9.3 ± 3.4</td>
<td>3.5 ± 4.1</td>
<td>148 ± 77</td>
<td>90 ± 28</td>
<td>99.0 ± 0.5</td>
<td>97.9 ± 0.8</td>
</tr>
<tr>
<td>90</td>
<td>10.5 ± 4.6</td>
<td>10.6 ± 2.7</td>
<td>7.7 ± 4.1</td>
<td>147 ± 83</td>
<td>76 ± 17</td>
<td>99.0 ± 0.3</td>
<td>98.2 ± 0.9</td>
</tr>
<tr>
<td>120</td>
<td>11.0 ± 4.8</td>
<td>10.8 ± 2.9</td>
<td>8.6 ± 4.2</td>
<td>131 ± 74</td>
<td>64 ± 18</td>
<td>99.1 ± 0.5</td>
<td>98.3 ± 0.7</td>
</tr>
<tr>
<td>150</td>
<td>11.4 ± 5.0</td>
<td>11.0 ± 2.8</td>
<td>8.9 ± 4.3</td>
<td>124 ± 70</td>
<td>65 ± 31</td>
<td>99.1 ± 0.5</td>
<td>98.3 ± 0.8</td>
</tr>
<tr>
<td>180</td>
<td>10.5 ± 4.5</td>
<td>11.0 ± 2.5</td>
<td>8.1 ± 3.9</td>
<td>129 ± 79</td>
<td>65 ± 34</td>
<td>99.1 ± 0.5</td>
<td>98.4 ± 0.6</td>
</tr>
<tr>
<td>210</td>
<td>10.3 ± 4.7</td>
<td>10.6 ± 2.6</td>
<td>7.9 ± 4.1</td>
<td>143 ± 84</td>
<td>65 ± 32</td>
<td>99.1 ± 0.5</td>
<td></td>
</tr>
</tbody>
</table>

*All values are corrected for 1.73 m² body surface area and represent the mean ± sd.

†P > 0.05.

‡Frac. reab. Na = fraction of filtered sodium reabsorbed.
of diuresis with NT subjects may be found from examining tables 1 to 3 and figures 1 and 2. There was no difference in peak flow or in peak CH2O between the two groups; the urine of the hypertensives was diluted, as well as that of the normotensives. Although the mean maximal V and CH2O per unit of glomerular filtration rate were higher in hypertensive than in normotensive subjects, the differences were not statistically significant. In addition, no differences were observed in the excretion of CH2O per unit of urine flow between the two groups as found by Cannon,8 who compared hypertensive and normotensive subjects receiving intravenous glucose infusions. The mean time elapsed to reach the maximal V (estimated from the start of the water load to the midpoint of the period of highest V) was not different in HT (114 minutes) and NT (103 minutes).

It is apparent that the hypertensives responded more rapidly to water administration (table 3, figs. 1 and 2). At 30 min the mean V was double and mean CH2O was several times greater in the HT group than in the NT subjects. Correspondingly, at 30 min Uosm in the HT patients decreased from control values at a rate two and one-half times faster than in the NT subjects (per cent decrease in Uosm HT, 37% and NT, 14%). However, at 60 to 90 min the decreases in Uosm from control values were comparable (HT, 76 and 70%; NT, 69 and 88%).

Mean control creatinine clearance, although not significantly different (P < 0.1), was 17% lower in the hypertensives than in the normotensive subjects. However, CCR remained quite stable throughout the study in each group (figs. 1 and 2, table 3).

**Sodium Excretion**

It is clear that sodium excretion after oral water loading in HT patients does not increase as urine flow increases (fig. 1, table 3). Thus, in the presence of a stable CCR, UNaV did not increase despite increasing V from a control value of 2.7 ± 1.9 ml/min to a peak of 11.0 ± 2.8 ml/min at 150 min following water administration. The relatively large standard deviations in UNaV for the pre-load and the first 30 min periods as well as the apparently small decrease in UNaV for 60 to 90 min after water loading are due to the fact that two patients (V.M. and F.N.) had progressive natriuresis for the 180 min before loading and a rapid decrease in sodium excretion immediately after the water load. These two patients apparently consumed more sodium than that prescribed. The other HT subjects had minimal and inconsistent changes in UNaV in the 120 to 180 min before water loading.

It is evident that the NT subjects had a pattern of sodium excretion similar to that exhibited by the HT (fig. 2, table 3). The small transient increase in mean UNaV immediately following the water ingestion in this group was not statistically significant (P < 0.2).

Although the hypertensives as a group appeared to excrete more sodium at every period than the normotensive subjects, this difference was not of statistical significance.

Mean control serum Na in both groups was similar (HT, 140 ± 5; NT, 141 ± 5 mEq/L) and following water loading it decreased slightly, but not significantly, in both groups (HT, 137 ± 6; NT, 138 ± 5 mEq/L). As a
consequence of a lower $C_{cr}$ in all periods, the mean filtered load of sodium was lower in the HT group.

The fraction of the filtered sodium reabsorbed following oral hydration did not change significantly within each group from the control values. On the other hand, beginning 60 min after the water load the fractional reabsorption of sodium became significantly different between the two groups (table 3).

Figure 3 demonstrates that there is a spectrum in the change in $U_{Na}V$ following oral hydration in both the HT and NT subjects. Two hypertensives (A.Z. and J.S.) had greater responses than the others. Although the severity of their hypertension was not greater, they had the largest increases in diastolic blood pressure of the group during the one or two periods immediately prior to their peak $U_{Na}V$ (A.Z., + 16; J.S., + 12 mm Hg). Changes in $U_{Na}V$ were not related to changes in the blood pressure in the other HT subjects. It is of interest that G.S. and D.J. in the NT group also had larger increases in $U_{Na}V$ than the others, but these were not related to changes in the blood pressure. There were no significant changes in $U_{E}V$, serum K, serum osmolality, or hematocrit in either group following the water load.

**Discussion**

It is apparent from these data that patients with systemic hypertension, known to respond with an exaggerated natriuresis when acutely challenged with an intravenous salt load, do not increase sodium excretion as urine flow and $C_{H_{2}O}$ increases in response to the administration of a sustained 20-ml/kg oral water load.

The HT subjects clearly responded to water administration faster than the NT subjects, rapidly increasing $V$ and $C_{H_{2}O}$ and concomitantly decreasing $U_{osm}$. Similar results have been reported by Taquini and associates. However, in our patients the maximal response to water loading was not different from that observed in the normotensive subjects. Other investigators, reporting only the maximal response to oral or intravenous water loading, observed similar results.

Thus, this lack of enhanced sodium excretion following oral hydration apparently is not related to a lesser response to water administration in the HT patients.

The absence of significant changes in sodium excretion observed in our HT subjects is different from the results obtained by Ek and by Hanenson and associates. Ek reported that two 1,000-ml water loads given orally during consecutive 60-min periods to six patients who were in the sitting position resulted in a moderate increase in sodium excretion. However, closer analysis of his data reveals that $U_{Na}V$ appeared to increase only in three subjects, decrease in one, and change very little in two. Estimation of the glomerular filtration rate (GFR) was not available in this study, and proteinuria was present in four of the subjects.

Hanenson and associates reported that a sustained 1,000-ml oral water load produced a higher excretion of sodium in five patients.

![Figure 3](http://circ.ahajournals.org/DownloadedFrom)
with essential hypertension (352 μEq/min/1.73 m²) than it did in five normotensive subjects (97 μEq/min/1.73 m²). However, no control studies were available prior to the water load in either group, and the urines were collected through indwelling bladder catheters, a stimulus known to produce in man a natriuresis that is more prominent in hypertensive patients. Our results are comparable to those obtained by Miles and DeWardener who measured chloride excretion (UCl) in NT and HT subjects after ingestion of 1 liter of water, with urine being collected by spontaneous voiding. No apparent difference in the rate of chloride excretion was evident between the two groups, and UCl did not increase after water loading in either group. In these studies no detailed information is provided of the sequential changes in urine flow, electrolyte excretion, CH₂O, or GFR following administration of water.

Krück and associates recently reported that the response to a modest oral water-alcohol load was different in 14 hypertensive subjects from that of 12 normotensive subjects. While their NT subjects exhibited progressive natriuresis following water-alcohol administration, their HT patients showed a slight decline in sodium excretion associated with a decrease in the filtered load of sodium. These authors concluded that the initial relatively high mean UNaV (287 ± 166 μEq/min) observed in the HT subjects represented a hypernatriuretic response that was already maximal within 1 hr following the initial hydration. Although no basal observation of UNaV was made on their HT subjects, they assumed it was the same as that of 60 normotensive subjects previously studied in their laboratory (106 ± 38 μEq/min). Furthermore, no information is given on the status of the sodium balance prior to the study. Urinary bladder catheterization was employed in all subjects.

Although the maximal diuretic response to water-alcohol administration in their NT subjects was similar to that of a group of NT subjects previously studied in our laboratory with a similar protocol, our patients did not exhibit progressive natriuresis. A detailed discussion of these studies is reported elsewhere. The failure of a standard oral water load to increase the sodium excretion as urine flow and CH₂O increased in our hypertensive subjects may be the result of an ineffective expansion of the extracellular fluid volume. Martino and Earley, after demonstrating a natriuretic response to the volume expansion that occurs following the massive intake of water, suggested that volume expansion with water depresses proximal tubular sodium reabsorption in a manner qualitatively similar to infusions of saline. They further suggested that the extent to which sodium excretion is increased during water loading is dependent upon (1) the absolute extent to which proximal reabsorption is depressed, (2) the extent to which the filtered load of sodium is maintained, and (3) the extent to which increased distal reabsorption compensates for the depressed proximal reabsorption of sodium. Figure 4A demonstrates that this lack of an exaggerated natriuresis in our HT patients was not related to decreases in filtered sodium, as this increased in some hypertensives at the time of maximal V without significantly increasing sodium excretion. If it is assumed that the delivery of sodium to the distal diluting segment may be estimated by the sum of UNaV plus the sodium equivalent of CH₂O, it is apparent from figure 4B that in half of the hypertensives there was an increased (estimated) delivery of sodium to the distal site of 500 to 1500 μEq/min despite reductions in the filtered load of sodium, indicating suppression of proximal tubular reabsorption following this water load. Since UNaV did not increase, most of this proximal sodium was used in forming CH₂O. Figure 4 also indicates that these relationships were the same in hypertensive as in normotensive subjects. However, the difference between the two groups in the mean fractional reabsorption of sodium following the water load (table 3) may be an early indication of a quantitatively different response to water administration, and
further oral hydration would result in definite differences in $U_{Na}V$ between the two groups.

Since the serum sodium concentrations of all subjects in both groups were greater than 130 mEq/L and decreases following water ingestion were small, it is apparent that acute or chronic hyponatremia can be eliminated as a cause for enhanced tubular reabsorption and decreased sodium excretion during volume expansion as suggested by some authors.\(^{19, 20}\)

We conclude, therefore, that sodium excretion does not increase as urine flow increases after a standard oral water load under controlled conditions in hypertensive subjects.

The fractional reabsorption of sodium was less in the hypertensive subjects despite a lower filtered load of sodium. This suggests a difference in the renal tubular handling of sodium between hypertensive and normotensive subjects.

No apparent defect was observed in $C_{H_2O}$ formation in this study. Cannon\(^8\) has suggested that the augmented natriuresis in hy-
pertensive patients following volume expansion with intravenous glucose may be, in part, related to the formation of less CH₂O, resulting in increased fractional rejection of sodium in the distal tubule. Thus, it is possible that further massive oral hydration in hypertensive patients would result in impairment of distal sodium transport, exposing a defect in CH₂O formation and resulting in enhanced sodium excretion. This was not attempted in our study.

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