Sodium Excretion and Blood Pressure

SUMMARY The urinary excretion of sodium, potassium, and water during three 24-hour periods were related to blood pressure in a sample of 49-year-old men. Of 3205 49-year-old men living in Göteborg, 2376 (74%) took part in the blood pressure screening, and of these, 120 subjects having blood pressures from the lowest to the highest were selected by systematic sampling based on diastolic blood pressure. Only subjects who were not on antihypertensive treatment were included. Results showed a marked day-to-day variation in sodium excretion, with between-days values of 0.23-0.64. The variation was less for two consecutive days than for two nonconsecutive days. The mean urinary excretion and the diurnal rhythm of sodium, potassium, and water did not differ significantly among normotensive, borderline, and hypertensive subjects. Over a wide range of blood pressures, no correlation was found between blood pressure and the urinary excretion of sodium, potassium, or water. In the low blood pressure range, however, there was a significant positive correlation between blood pressure and urinary sodium excretion (R = 0.46, p < 0.01; R = Spearman's coefficient of rank correlation).

The large intraindividual variation in sodium excretion and blood pressure makes it difficult to estimate an individual's mean values. Furthermore, there are limitations in drawing longitudinal conclusions from cross-sectional data. With these methodological problems in mind, our findings indicate that salt intake influences blood pressure in a part of the normotensive population, but do not lend support to the hypothesis that habitual salt intake might be of major importance for the blood pressure level in mild to moderate essential hypertension. (Hypertension 3: 318-326, 1981)

KEY WORDS: • blood pressure • essential hypertension • sodium excretion • potassium excretion • body size • epidemiology

A positive relationship between salt intake and the prevalence of hypertension in both primitive and more developed societies has been found in a great number of investigations. In societies with extremely low sodium intake (< 10 mmole/24 hr) and high potassium intake, hypertension seems to be absent and blood pressure does not seem to rise with age. In societies with high sodium intake (> 350 mmole/24 hr), 30% or more of the adult population have been shown to be hypertensive. However, the relationship between salt intake and blood pressure within populations is not clear. A positive association between salt excretion and blood pressure has been found in some recent population studies while in others no such association has been found. In a previous small population study of middle-aged men from our group in Göteborg, a positive correlation between the 24-hour urinary sodium excretion and the basal blood pressure was found only in the normotensive blood pressure range. In the hypertensive blood pressure range, however, a negative correlation was found between sodium excretion and blood pressure. The cause of this negative correlation was not revealed, but one possibility that was suggested was a decrease in salt appetite as hypertension develops. A high potassium content of the diet has been claimed to give protection against hypertension in subjects on a high-sodium diet, but in most population studies no association between urinary potassium output and blood pressure has been found. However, the ratio between salt intake and potassium...
intake may be of importance for the blood pressure level; a positive correlation between the urinary Na/K-ratio and blood pressure has been reported in some population studies but denied in others. Thus, there are many questions unsolved before we know how salt intake influences blood pressure.

The objective of this study was to determine the urinary excretion of sodium, potassium, and water and relate it to blood pressure and body size in men of the same age, representing all blood pressure levels.

Methods

Study Population

During 1975 and 1976, a random half of all 49-year-old men living in Göteborg were invited to a screening examination of blood pressure. Of the invited 3205 subjects, 2376 (74%) attended; they represented the entire blood pressure range. Based on the subject’s diastolic blood pressure (DBP) at screening, we randomly selected 1/30 of those with a DBP < 95 mm Hg (n = 23); 1/15 of those with a DBP 95-104 mm Hg (n = 15); 1/6 of those with a DBP 105-114 mm Hg (n = 15); and all those with a DBP ≥ 115 mm Hg (n = 67).

Subjects fulfilling the criteria and who were not on antihypertensive treatment were asked to participate in the investigation. The expected number of subjects in the various DBP groups had been calculated from the DBP distributions of earlier screening examinations of middle-aged men in Göteborg. Of those fulfilling the selection criteria, 36 subjects were not included in the study because they were on antihypertensive treatment. Another eight were excluded because of diseases (valvular heart disease, n = 4; previous myocardial infarction, n = 1; previous stroke, n = 1; chronic glomerulonephritis, n = 1; and severe alcoholism, n = 1), and 15 subjects refused to participate in further investigation. There were 120 subjects who remained for the study, i.e., 88.9% (120/135) of those asked to participate.

Screening Protocol

The screening examination took place from March to May and from September to November, 1975 and 1976, between 4:30 and 7:00 pm. The subjects were on unrestricted diet and remained outpatients during the study. Initially, a 3-day urine collection for determination of blood pressure. Of the invited 3205 subjects, 2376 (74%) attended; they represented the entire blood pressure range. Based on the subject’s diastolic blood pressure (DBP) at screening, we randomly selected 1/30 of those with a DBP < 95 mm Hg (n = 23); 1/15 of those with a DBP 95-104 mm Hg (n = 15); 1/6 of those with a DBP 105-114 mm Hg (n = 15); and all those with a DBP ≥ 115 mm Hg (n = 67).

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Blood Pressure

Blood pressure (BP) was measured in the right arm in three different situations: casual blood pressure, after supine rest, and as an outpatient. The DBP was recorded when the Korotkoff sounds disappeared (phase five). Mean arterial pressure (MAP) was calculated as the diastolic blood pressure + 1/3 of the pulse pressure.

Casual blood pressure at screening was measured once using a mercury manometer with the subject in the seated position after a few minutes’ rest. The BP was determined to the nearest 2 mm Hg, to avoid digital preference.

Blood pressure after rest was measured twice during very quiet conditions after 45 minutes of recumbent rest in a sound-protected room using a rubber cuff that was automatically inflated and deflated. Simultaneous registrations were made of cuff pressure, Korotkoff sounds, and electrocardiogram. The blood pressure was calculated to the nearest 1 mm Hg.

Blood pressure at the outpatient hypertension clinic was measured on three different occasions within 1 month after the urine collection. The BP was measured with the subject supine after 5 minutes of rest, using the same methods as at screening.

Heart Rate

At screening and at the hypertension clinic, heart rate was determined by pulse palpation, but after the 45 minutes of rest the heart rate was calculated from the blood pressure recordings.

Body Cell Mass and Body Fat

Radioactive potassium (40K) as a measure of body cell mass was measured in a whole-body counter in all subjects except one, who refused due to claustrophobia. Body cell mass was calculated from total body potassium according to the formula of Moore et al., and body fat according to that of Forbes et al.

Urine Collection

Urine was collected for three consecutive 24-hour periods divided into day (7 am–7 pm) and night (7 pm–7 am). Detailed written and verbal instructions were given. Determination of urine sodium, potassium, creatinine (autoanalyzer), and urine volume was done. The creatinine content of each portion was used as an index of the completeness of the urine collection. We used the same arbitrary criteria for acceptable collection that we had used in an earlier study. Thus, a 24-hour collection was not accepted if the urinary creatinine content was less than 8.8 mmoles (1 g), and the separate day and night portions were not accepted if the day/night ratio of the urinary creatinine content exceeded 2, or if the night/day ratio exceeded 1.5. In total, 96.1% of the 24-hour periods (346/360) and 80.8% (291/360) of the 12-hour night and day collections were considered to be acceptably collected. All 120 subjects collected urine acceptably for one 24-hour period, 118 subjects for two 24-hour periods, and 107 subjects for all three 24-hour periods.

The mean 24-hour creatinine excretions did not differ significantly between the three 24-hour periods. This indicates that the collection was approximately
the same on the three separate days. Correlation coefficients for the 24-hour creatinine excretions were 0.57–0.58 between any 2 of the 3 days.

**Diagnostic Work-Up**

A detailed history was obtained and physical examination performed at the outpatient hypertension clinic. Ocular fundi were examined. Chest x-ray was taken and heart volume calculated according to Jonsell. Serum concentrations of sodium, potassium, calcium, creatinine, urate, and aldosterone were measured. Urine tests for glucose, hematuria, and bacterial culture were also done. The 24-hour urinary excretions of protein, catecholamines, and cortisol were determined. Renal concentrating capacity was measured using the vasopressin test. Isotope renography was performed using a standard method and equipment. In patients with abnormal radiorenograms, further investigation with intravenous pyelography or renal aortography was performed.

**Characteristics of the Study Group**

None of the subjects had a history of, or showed signs of, previous or recent myocardial infarction, angina pectoris, cardiac decompensation, intermittent claudication, or stroke. One subject had occasionally had glucosuria prior to the investigation, but he had received no treatment for diabetes. Those with fundoscopic changes were rated Grade I or II according to the Keith-Wagener-Barker classification. None in the study group had secondary hypertension.

Three clinical groups were defined, to enable comparisons with more clinically oriented studies. Subjects having a DBP ≥ 105 mm Hg (the mean of three BP measurements) at the outpatient hypertension clinic were regarded as hypertensives (n = 24). Subjects having a DBP < 95 mm Hg at screening and a DBP < 90 mm Hg (mean of three recordings) at the hypertension clinic constituted the normotension group (n = 21). The remaining subjects were allocated to the borderline group (n = 75).

In all three groups, the BP at screening was considerably higher than after rest and also significantly higher than at the outpatient hypertension clinic (table 1). The blood pressure after rest was regarded as almost basal, and the studied variables were related to this blood pressure.

Both borderline subjects and hypertensives weighed significantly more than normotensives, apparently due to more body fat. Height, body surface area, and body cell mass did not differ significantly between the normotension and hypertensives (table 1).

The prevalence of hypertensive heart and kidney involvement is seen in table 2. Left ventricular hypertrophy on electrocardiogram (ECG) was diagnosed

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**TABLE 1.** Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), and Heart Rate (HR) in Three Different Situations and Height, Weight, Body Surface Area (BSA), Body Cell Mass (BCM), and Body Fat in Three Clinical Groups (Mean ± Standard Deviation)

<table>
<thead>
<tr>
<th></th>
<th>Normotensive group (n = 21)</th>
<th>Borderline group (n = 75)</th>
<th>Hypertensive group (n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BP at screening (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>130 ±11</td>
<td>163 ±15</td>
<td>190 ±16</td>
</tr>
<tr>
<td>DBP</td>
<td>84 ±7</td>
<td>113 ±10</td>
<td>128 ±10</td>
</tr>
<tr>
<td>MAP</td>
<td>99 ±8</td>
<td>130 ±11</td>
<td>148 ±12</td>
</tr>
<tr>
<td>HR</td>
<td>76 ±13</td>
<td>86 ±12</td>
<td>88 ±13</td>
</tr>
<tr>
<td><strong>BP at outpatient hypertension clinic (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>127 ±7</td>
<td>147 ±12</td>
<td>175 ±16</td>
</tr>
<tr>
<td>DBP</td>
<td>76 ±6</td>
<td>93 ±8</td>
<td>114 ±7</td>
</tr>
<tr>
<td>MAP</td>
<td>93 ±5</td>
<td>111 ±9</td>
<td>135 ±9</td>
</tr>
<tr>
<td>HR</td>
<td>70 ±11</td>
<td>74 ±10</td>
<td>77 ±12</td>
</tr>
<tr>
<td><strong>BP after 45 minutes' rest in sound-protected room (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>112 ±11</td>
<td>133 ±13</td>
<td>157 ±24</td>
</tr>
<tr>
<td>DBP</td>
<td>63 ±9</td>
<td>79 ±11</td>
<td>96 ±15</td>
</tr>
<tr>
<td>MAP</td>
<td>79 ±8</td>
<td>97 ±11</td>
<td>116 ±17</td>
</tr>
<tr>
<td>HR</td>
<td>62 ±10</td>
<td>67 ±11</td>
<td>69 ±8</td>
</tr>
<tr>
<td><strong>Height (cm)</strong></td>
<td>176.4 ± 5.2</td>
<td>177.2 ± 6.7</td>
<td>176.0 ± 6.9</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td>75.9 ± 9.1</td>
<td>82.6 ± 11.7*</td>
<td>84.8 ± 14.0*</td>
</tr>
<tr>
<td><strong>BSA (m²)</strong></td>
<td>1.93 ± 0.12</td>
<td>2.00 ± 0.16</td>
<td>2.01 ± 0.18</td>
</tr>
<tr>
<td><strong>BCM (kg)</strong></td>
<td>33.2 ± 3.9</td>
<td>34.6 ± 4.7</td>
<td>34.2 ± 3.1</td>
</tr>
<tr>
<td><strong>Body fat (kg)</strong></td>
<td>17.4 ± 6.2</td>
<td>21.3 ± 7.8*</td>
<td>24.6 ± 10.5†</td>
</tr>
</tbody>
</table>

Levels of significance when body size variables are compared with the corresponding variables in the normotension group (t test) = *p < 0.05; †p < 0.01.
TABLE 2. Prevalence of Hypertensive Organ Involvement in the Clinical Normotensive, Borderline, and Hypertensive Groups

<table>
<thead>
<tr>
<th></th>
<th>Normotensive group (n = 21)</th>
<th>Borderline group (n = 75)</th>
<th>Hypertensive group (n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart enlargement on x-ray</td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>(&gt; 500 ml/m² BSA)</td>
<td>1 (4.8)</td>
<td>3 (4.0)</td>
<td>4 (16.7)</td>
</tr>
<tr>
<td>Left ventricular hypertrophy on ECG</td>
<td>0</td>
<td>4 (5.3)</td>
<td>6 (25.0)</td>
</tr>
<tr>
<td>S-creatinine &gt; 114 µmoles/liter (&gt; 1.3 mg/100 ml)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>U-albumin &gt; 200 mg/24 hrs</td>
<td>0</td>
<td>2 (2.7)</td>
<td>4 (16.7)</td>
</tr>
</tbody>
</table>

according to the Minnesota code (3:1 or 3:3 + 4:1–3 or 5:1–3). In the hypertension group, three subjects with cardiac enlargement on x-ray also had left ventricular hypertrophy on ECG. Thus, while usual clinical signs of heart and kidney involvement were rare in the borderline group, a substantial proportion of the hypertension group had signs of such organ involvement.

Statistical Methods

Standard methods were used for calculation of mean (x), standard deviation (SD), Pearson’s correlation coefficient (r), and Spearman’s coefficient of rank correlation (R). The hypothesis of no difference in means between the two groups was tested using Student’s t test. Only two-tailed tests were used. The hypothesis of no linear correlation between two variables was tested using the correlation coefficient. Values of p < 0.05 were regarded as statistically significant.

In addition, a “sliding mean value” method was used to describe the relationship between blood pressure and urinary sodium excretion. The subjects were ranked 1 to 120 by increasing blood pressure after rest (MAP) and were then subgrouped. The first subgroup included Subjects 1 to 40 having the lowest blood pressure. The second subgroup included Subjects 2 to 41, the third subgroup Subjects 3 to 42, and so on. The mean values of MAP and urinary sodium were calculated in each subgroup. The successive points were then joined to form continuous curves. The mean values changed very slowly, since one subgroup differed from the next by only two subjects. Two extra subgroups including the 20 subjects with the lowest MAP and the 20 subjects with the highest MAP were compiled to illustrate the extreme values. These curves thus describe how the mean value of a variable on the y-axis changes when blood pressure on the x-axis increases. This method was used to give a more dynamic description of how a variable changes with increasing blood pressure.

The shape of the curves was tested with conventional stratification into deciles. The correlation between the variables studied is not demonstrated by these curves and is expressed in the usual way, i.e., by the correlation coefficient (R). The blood pressure axis was arbitrarily divided into three ranges (MAP < 90, 90–99, and ≥ 100 mm Hg) before data analyses, to make correlation analysis between BP and sodium excretion possible in different parts of the BP distribution. The same ranges were used in analyses of other variables in the same study group. The correlations in the three MAP ranges as well as in the whole study group were calculated using the Spearman’s coefficient of rank correlation, since this test does not require normally distributed variables. All significant correlations with Spearman’s coefficient of rank correlation were also found to be significant with the usual linear regression analysis. Calculations were made by computers (IBM 360/65, or Olivetti P 652).

Results

Urinary Sodium Excretion

A marked day-to-day variation was seen in the individual sodium excretion during the 3 examination days (fig. 1). This variation was less for two consecutive than for nonconsecutive days. The mean sodium excretion did not differ significantly between the three separate 24-hour periods. Sodium excretion during working days did not differ significantly from that during days off work.

Whole Study Group

When mean sodium excretion of the three 24-hour periods was plotted against resting blood pressure, a wide scatter along the blood pressure axis was found (fig. 2, upper panel). As demonstrated by the curves in figure 2, the mean sodium excretion did not change much over a wide range of blood pressures. There was no correlation between blood pressure and sodium excretion in the whole study group (R = 0.01).

Three Ranges of Resting Blood Pressure

A significant positive correlation between sodium excretion and blood pressure was found in the lowest blood pressure range (R = 0.46, p < 0.01), but no correlation was found in the middle or highest range (fig. 2).
**FIGURE 1.** Relationship between urinary sodium excretions during three consecutive 24-hour periods in the 107 subjects having three acceptable collections.
Three Clinical Groups (Normotensives, Borderline Cases, and Hypertensives)

Mean urinary sodium excretion was highest in the borderline group, but did not differ significantly from that in the normotensive (0.05 < p < 0.10) or hypertensive group (0.05 < p < 0.10). The sodium excretion varied more than four times within all three groups, and in each group some subjects excreted more than 300 mmoles/24 hrs.

The ratio between day and night sodium excretion did not differ significantly among the groups. The daytime portion of the 24-hour excretion amounted to 54.3% in normotensives, 51.8% in borderline subjects, and 53.4% in hypertensives. Thus, no tendency of a reversed diurnal rhythm of sodium excretion was found in the hypertensive group.

Sodium Excretion Corrected for Body Size

To analyze the influence of body size on salt intake, sodium excretion was related to body weight, height, body surface area, and body cell mass. In the whole study group, there was a significant correlation only to body cell mass (R = 0.18, p < 0.05). Therefore, body cell mass was used to correct for body size. Also, after correction for body size, there was a wide scatter in sodium excretion in relation to blood pressure (fig. 2, lower panel). There was no significant correlation between these variables in the whole study group (R = 0.02), but in the lowest blood pressure range a positive significant correlation was found (R = 0.45, p < 0.01).

Potassium Excretion

The urinary potassium excretion also varied a great deal from day to day, but the variation tended to be less than that of the sodium excretion. We found no significant correlation between potassium output and MAP in the whole study group (R = 0.03), in the middle (R = —0.15), or upper blood pressure range (R = 0.18), but in the lowest blood pressure range there was a tendency toward a negative correlation (R = —0.265, 0.05 < p < 0.10).

Also, within each clinical group there was a considerable variation in potassium excretion (table 3), but the mean potassium excretion did not differ significantly among the clinical groups. Among the body size variables, potassium excretion was only significantly correlated with body cell mass (R = 0.23, p < 0.02), as was the sodium excretion. However, correction of the potassium values for body cell mass did not change the results significantly.

Na/K Ratio

In the whole study group, no significant correlation between the Na/K-ratio and blood pressure was found (R = 0.06), but a significant positive correlation was seen in the lowest blood pressure range (R = 0.46, p < 0.01) and a tendency toward a negative correlation in the highest blood pressure range (R = −0.28, 0.05 < p < 0.10). The mean Na/K-ratio did not differ significantly among the clinical groups (table 3).

Urine Volume

Urine volume was not significantly correlated to blood pressure in the whole study group (R = 0.05). It varied greatly within the clinical groups (table 3), but the means did not differ significantly. The mean daytime portion of the 24-hour urine volume varied between 50% and 52% in the clinical groups.

Table 3. Mean of Three 24-Hour Urinary Excretions of Sodium and Potassium, With and Without Correction for Body Cell Mass (BCM), the Ratio of Urinary Na/K, and Urine Volume in the Three Clinical Groups

<table>
<thead>
<tr>
<th>Urinary excretion</th>
<th>Normotensive group (n = 21)</th>
<th>Borderline group (n = 75)</th>
<th>Hypertensive group (n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>x ± sd (range)</td>
<td>x ± sd (range)</td>
<td>x ± sd (range)</td>
</tr>
<tr>
<td>Na/24 hrs</td>
<td>173 ± 64 (61 - 358)</td>
<td>197 ± 50 (78 - 331)</td>
<td>176 ± 55 (73 - 332)</td>
</tr>
<tr>
<td>(mmole)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Na/24 hrs/BCM</td>
<td>5.3 ± 1.8 (1.8 - 9.2)</td>
<td>5.8 ± 1.6 (2.5 - 11.8)</td>
<td>5.1 ± 1.4 (2.1 - 8.6)</td>
</tr>
<tr>
<td>(mmole/kg BCM)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>K/24 hrs</td>
<td>79 ± 22 (19 - 132)</td>
<td>76 ± 18 (46 - 129)</td>
<td>78 ± 20 (30 - 114)</td>
</tr>
<tr>
<td>(mmole)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>K/24 hrs/BCM</td>
<td>2.4 ± 0.67 (0.55 - 3.4)</td>
<td>2.2 ± 0.48 (1.4 - 3.4)</td>
<td>2.2 ± 0.58 (0.92 - 3.3)</td>
</tr>
<tr>
<td>(mmole/kg BCM)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Na/K ratio</td>
<td>2.23 ± 0.62 (1.30 - 3.6)</td>
<td>2.67 ± 0.81 (0.8 - 5.6)</td>
<td>2.37 ± 0.59 (1.5 - 3.7)</td>
</tr>
<tr>
<td></td>
<td>(1.30 - 3.6)</td>
<td>(0.8 - 5.6)</td>
<td>(1.5 - 3.7)</td>
</tr>
<tr>
<td>Urine volume/24 hrs</td>
<td>1464 ± 305 (985 - 2160)</td>
<td>1482 ± 529 (733 - 3347)</td>
<td>1362 ± 351 (803 - 2227)</td>
</tr>
</tbody>
</table>

Discussion

The results of this study could be generalized to a large background population due to the use of stratified random samples. Although the whole study group is not a random sample, each stratum of blood pressure was selected at random. The successive enrichment of subjects with increasing blood pressure...
was made to enable a better description of changes occurring in the borderline and hypertensive range. Since only 49-year-old men were included, the results are not influenced by age and sex. Only subjects who had never been on antihypertensive treatment were included, since most hypertensive drugs influence the sodium balance.

The results showed no correlation between blood pressure and urinary excretion of sodium, potassium, or water, over a wide range of blood pressures. In the normotensive blood pressure range, however, there was a significant but weak positive correlation between blood pressure and urinary sodium excretion, and urinary sodium/potassium ratio respectively.

The relationship between salt intake and renal ability to excrete sodium has been suggested to be of major importance for the long-term blood pressure level. Such a relationship should ideally be studied prospectively. However, this would entail repeated measurements in a large group of subjects over many years, and would therefore be extremely difficult to carry out. We have made observations in a cross-sectional study where the subjects are representative of a wide blood-pressure range.

Urinary sodium excretion was used as a measure of sodium intake, which equals urinary excretion under normal circumstances except for a negligible loss of sodium in the sweat and feces. Day-to-day variation in sodium excretion was probably due to real variations in sodium intake, since the variation in sodium excretion was greater than that of creatinine excretion. This interpretation is also strengthened by the finding that urinary sodium excretion on 2 consecutive days was better correlated than excretions on 2 nonconsecutive days, indicating an influence of sodium intake of 1 day on the urinary sodium excretion of the following day.

The day-to-day variation in salt intake makes it difficult to determine a subject's true mean level of salt intake if only consecutive collections were used. It has been claimed that at least seven 24-hour collections are required for a proper classification of individuals into groups with respect to salt intake. This conclusion was based, however, on two sets of 2-day collection 3 months apart, which may give other results than if only consecutive collections were used as in our study. Furthermore, good correlation \( (r = 0.90) \) has been shown between sodium excretions of the first three and a total of six consecutive home 24-hour urine collections.

Also, blood pressure shows a large biological variation. A near basal blood pressure has been shown to best reflect the long-term pressure load on the heart and kidneys. We have therefore used the blood pressure recorded after prolonged rest in a sound-protected room as the blood pressure to which electrolyte excretions have been related.

One yet unsolved problem when studying sodium intake (via sodium excretion) is whether a correction should be made for body size. We have chosen to present the data both with and without correction for body size, using the body cell mass for correction, since this body size variable was best correlated to both sodium and potassium excretion.

When interpreting the results from this study and other studies of this kind, three problems must be considered. One is that the sodium intake, which we estimate by the sodium excretion, is assumed to be representative of the level of salt intake of that person during days and months preceding our examination. The second problem is whether the sodium intake is determined solely by habits or if counter-regulating mechanisms influence sodium appetite as hypertension develops. The third is whether the same salt intake has the same effect on blood pressure in all subjects or whether the sensitivity to a certain salt intake may vary from individual to individual or may differ between the hypertensive and normotensive blood-pressure range. Some studies with very small study groups have been devoted to the last problem, but all these three problem areas are largely unclarified, although insight into these problems is a necessary prerequisite for proper interpretation of these kinds of studies.

The finding in our study of no relationship between sodium intake and blood pressure in the total population is consistent with the findings of some other population studies, but at variance with others. The explanation for the conflicting results between the studies mentioned may be due to differences in composition of the study groups with regard to severity of hypertension, age, sex, and race, and may also be due to methodological differences in the accuracy and number of urine collections and standardization of the blood pressure recordings, as discussed above.

In the strictly normotensive blood-pressure range in our present study, we found a significantly positive correlation between sodium intake and blood pressure in accordance with some previous studies. The relationship was almost the same after correction for body size. In the hypertensive blood pressure range, on the other hand, we could not confirm the finding of a significant negative correlation between the urinary sodium excretion and blood pressure that had been previously described, although there was a weak tendency toward such a relationship. The lack of a significant negative correlation in our present study might be explained by a somewhat milder hypertension in the hypertensives in this study than in the previous one.

The mean urinary sodium excretion did not differ significantly among the clinical groups of normotensives, borderline hypertensives, and definite hypertensives. These findings and the possible curvilinear shape of the salt-blood pressure relationship imply that group comparisons of mean values from normotensives and hypertensives are of limited value in studying this relationship.

The finding of no correlation between urinary potassium excretion and blood pressure in the whole study group is consistent with the results of some other studies. In hypertensives, a negative correlation
between urinary potassium output and blood pressure has been reported in a population study of 60-year-old men. This finding could not be confirmed in the present investigation.

The Na/K-ratio was not correlated to blood pressure in the whole study group, which is in accordance with other population studies. The 24-hour urine volume and day/night ratio did not differ between normotensives and hypertensives, which is in accordance with the findings of Stibrna et al. and Berglund et al. In Brod's study, a reversed diurnal rhythm was found, which is possibly due to a more advanced hypertension in the subjects of that study than in ours.

One can only speculate about whether the positive correlation between sodium excretion and blood pressure in the lowest MAP range is causal or whether it is an expression of a parallel phenomenon caused by a common underlying factor. If the salt-blood pressure relationship was a causal one, the causality would only apply to the strictly normotensive part of the population, as no significant positive correlation was found in the rest of the population. The situation is probably much more complicated than a simple increase in blood pressure when salt intake increases. If such a straightforward relationship were to exist, it would be confined to a minority of the normotensive population. The relative weak salt-blood pressure relationship (R = 0.46, R² = 0.21) points to that possibility.

The lack of correlation between salt and blood pressure in the higher blood pressure range might be explained in at least two ways. Either there is no relationship in this blood pressure range, or the basic relationship is disturbed by salt-appetite depressing, counter-regulatory mechanisms coming at play as the blood pressure increases. If a high salt intake should increase the blood pressure, it would have to be accompanied with salt retention at some point in the time during the development of high blood pressure. Salt retention should expand extracellular volume. However, extracellular volume seems to be unchanged and plasma volume somewhat contracted in essential hypertension. Thus, hypothetical sodium retention would be placed intracellularly. Increased severity of hypertension is associated with decreased activity of the renin-angiotensin system, a system that might well be one of the counteracting systems for the salt appetite mentioned above. Studies of the dynamic changes of these and other factors accompanying changes in salt intake are needed to further clarify these interrelationships.

Conclusions

In 120 unselected middle-aged men, no correlation between blood pressure and urinary excretions of sodium, potassium, or water was found over a wide range of blood pressures. Mean sodium excretion was not higher in hypertensives than in normotensives. In the normotensive blood pressure range, however, there was a significant positive correlation between blood pressure and the urinary sodium excretion and urinary Na/K-ratio respectively. Due to the large intra-individual variation in both urinary sodium excretion and blood pressure, it is difficult to estimate an individual's mean values. Furthermore, there are limitations in drawing longitudinal conclusions from cross-sectional data. With these methodological problems in mind, our findings indicate that salt intake influences blood pressure in a part of the normotensive population but do not lend support to the hypothesis that habitual salt intake might be of major importance in affecting the blood pressure level in mild-to-moderate essential hypertension.

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