Renal Excretion of Sodium in Arterial Hypertension

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Patients with arterial hypertension excrete a sodium load more rapidly than do individuals with normal blood pressure. The relationship of this abnormal sodium excretory response to blood pressure and such extrarenal factors as the central nervous system, dietary salt intake, body fluid volume and sodium content, and the adrenal glands has been studied. On the basis of this and other evidence, it is suggested that the exaggerated natriuresis is the result of a renal tubular defect which occurs after the development of hypertension.

It has been recognized since the studies of Farnsworth and Barker that there is an abnormally high renal excretion of chloride in hypertensive individuals. Most subsequent research has been concerned with sodium rather than chloride. There have been several reports in which subjects with essential hypertension were observed to excrete an intravenous sodium load more rapidly than individuals with normal blood pressure. The explanation has been offered that this excretory derangement is related to alterations in renal function and that the high salt excretors were those hypertensive subjects whose renal plasma flow was reduced and filtration fraction elevated. Others, however, have failed to observe such a relationship between increased sodium output and renal hemodynamics. The parallelism of the sodium clearance with both the level of the blood pressure and renal vascular resistance suggested to Cottier et al. an alternative possibility, namely, that the hypertensive natriuretic response is related to the increased renal intravascular pressure. Baldwin and his associates, on the other hand, interpreted their results to indicate an extrarenal basis for the abnormal sodium excretion in essential hypertension.

The present study was undertaken to characterize further the hypertensive pattern of sodium excretion and the factors contributing to it. Consideration was given to the following: (1) the relationship of sodium excretion to blood pressure in subjects with labile, essential, and secondary hypertension; (2) the influence of extrarenal factors, such as the central nervous system, dietary salt intake, body fluid volume and sodium content, and the adrenal glands.

Materials and Methods

The studies were performed on the following subjects: 16 with essential hypertension of varying severity, 4 with labile hypertension, 4 with secondary hypertension, and 11 with normal blood pressure. In the presentation of the data, the subjects with essential hypertension have been grouped in order of increasing blood pressure as follows: group I 130-160/90-110, group II 160-224/110-130, group III 236-240/134-140. Except for 2 patients with severe essential hypertension and diminished glomerular filtration there was no evidence of renal functional impairment. Cardiac decompensation was absent in all patients. The degree of retinopathy in the subjects with elevated blood pressure was grade II or less. There was no dietary restriction except when the effect of salt intake was being studied. No patient was receiving antihypertensive drugs.

Procedures, except when noted, were carried out at approximately the same time in the morning. Water and food were withheld from the preceding midnight until the completion of the study. The subjects were in the supine position. Whenever possible urines were collected through a soft rubber, multiholed, indwelling urethral catheter. In the remainder the specimens were voided and the
collections checked for completeness by comparing
the endogenous creatinine clearances for the sev-
eral periods in each study. In the occasional in-
stance when the correlation was poor the experi-
ment was discarded. After a control urine collect-
ion of 60 to 90 minutes, 100 ml. 5 per cent sodium
chloride per M.2 of body surface area (BSA) were
administered intravenously over a 25-minute pe-
riod. Urine specimens were then obtained 30, 60,
120, and 180 minutes after the start of the infusion
(for tabulation the last 2 periods have been aver-
age). Blood specimens were drawn during the
control period and at the midpoint of all subse-
quent urine collections. When inulin and paraami-
nohippurate clearances were measured, 200 ml.
water by mouth were given hourly beginning 2
hours before the start of the control period.

The possible influence of a center in the brain
on renal sodium excretion was studied in 4 indi-
viduals with essential hypertension. Except for
1 patient (J.L.) all were hydrated with intravenous
5 per cent glucose in water at 3 to 4 ml. per min-
ute. After a suitable baseline period, 5 per cent
saline was infused into the right carotid artery at
the rate of 1 ml. per minute for 25 minutes. This
amount was calculated to increase the carotid se-
rum sodium concentration 4 to 5 per cent without
significantly altering that of the rest of the body.6
Urine samples were collected 60 minutes after the
start of the infusion through an indwelling cath-
eter.

Serum and urinary sodium and potassium were
determined with an internal-standard flame pho-
tometer. Creatinine was measured by the method
of Polin and Wu12 as modified by Phillips.13 To
assure complete recovery of creatinine proteins
were precipitated at pH 2 by the procedure de-
scribed by Owen et al.14 Inulin was determined by
the Rolf, Surtshin, and White15 modification of the
method of Alving, Rubin, and Miller16 and paraami-
nohippurate by that of Smith et al.17 Blood pres-
ures were taken frequently throughout
each study with a mercury sphygmomanometer.

RESULTS

Subjects with Essential Hypertension. Our
results (table 1) show that subjects with
essential hypertension excrete sodium more
rapidly during the 3-hour period after the
start of the hypertonic saline infusion than
does a control group. A relationship can be
noted between the rate of excretion and the

*It was assumed that the cerebral blood flow was
0.9 L. per minute11 and that the right carotid artery
received approximately a fourth of this amount.

Fig. 1. The filtered load of sodium (ordinate) in
subjects with normal blood pressure and group II
essential hypertension is plotted against the time in
minutes (abscissa). The vertical solid and dashed lines
represent the standard deviation of the mean.
Filtered load of sodium (mEq. per minute per 1.73
M.2 BSA) = Endogenous creatinine clearance (ml.
per minute per 1.73 M.2 BSA) x serum sodium
(mEq. per ml.).

height of the blood pressure except in the 2
individuals comprising group III, who were
known to have diminished glomerular func-
tion. Nevertheless, their sodium output was
still increased over the normotensive levels.

The per cent of the infused sodium excreted
during the first hour in 2 of the mild hyper-
tensive subjects exceeded that observed in any
of the controls while in the other 2 and all
of the individuals with labile hypertension
the values were within the normal range.

There is no evident difference in filtered
sodium prior to or for the first hour following
the salt load between the individuals with
normal blood pressure and those in group II
who showed the maximum excretion (fig. 1).
During the second and third hours, however,
the values for the control subjects are higher.
Thus the sodium excretory response cannot
be correlated with the filtered load.

Subjects with Secondary Hypertension. In
table 2 the sodium excretory pattern following
hypertonic salt loading in 2 patients with
Cushing’s syndrome and 2 with pheochromocy-
toma is comparable to that observed in the
group II essential hypertensive subjects de-
scribed in table 1. No correlation is noted
between the natriuretic response and the
Table 1.—Sodium Excretion in Individuals with Normal Blood Pressure, Labile and Essential Hypertension Following the Intravenous Infusion of 5 Per Cent Sodium Chloride

<table>
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<tr>
<th></th>
<th>No. of subjects</th>
<th>Age</th>
<th>Control†</th>
<th>Sodium excretion (µ Eq./min.)</th>
<th>Per cent infused sodium excreted‡</th>
<th>Blood pressure (mm. Hg)</th>
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<td></td>
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<td></td>
<td></td>
<td>I</td>
<td>II</td>
<td>III</td>
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<td>Group II</td>
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<td>(25-59)</td>
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<td>(306-1475)</td>
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</table>

*The infused sodium load consisted of 100 ml. 5 per cent sodium chloride per M.² BSA.
†Control, 60-90 minutes prior to the start of the infusion; I, 0-30 minutes after the start of the infusion; II, 30-60 minutes after the start of the infusion; III, 60-180 minutes after the start of the infusion.
‡Per cent infused sodium excreted = \[
mEq. \text{ sodium excreted in post-infusion period} - \frac{mEq. \text{ sodium excreted per minute during control period} \times \text{no. of minutes in post-infusion period}}{mEq. \text{ sodium infused}} \times 100
\]
§The significance of the differences between the means for sodium excretion and per cent infused sodium excreted in the subjects with normal blood pressure and group II essential hypertension.
endogenous creatinine clearance. Fluctuations in urinary flow paralleled those for sodium excretion.

**Effect of 5 per cent Sodium Chloride Infusion into the Carotid Artery.** In 4 subjects with essential hypertension 5 per cent sodium chloride was infused into the right common carotid artery at a rate of 1 ml. per minute for 25 minutes. As previously explained, this procedure was designed to produce an increase in cerebral sodium concentration comparable to that achieved in this region by the intravenous administration of 100 ml. 5 per cent sodium chloride per M.² BSA, without, however, causing a significant elevation elsewhere in the body. As can be seen in table 3, no increase in sodium excretion similar to that following the usual intravenous hypertonic salt load was observed for the 60-minute period after the start of the infusion. The variation between the intravenous and carotid baseline sodium excretion in patients C.S. and S.T. may be explained by the fact that the studies were performed on different days on a free salt intake.

Except for J.L., the subjects were hydrated and their urinary flows were measured to determine whether the intracarotid hypertonic saline was being delivered to the cerebral osmoreceptors. In 1 of the hydrated subjects (W.C.) the apprehension and discomfort associated with carotid arterial puncture stimulated an antidiuretic response. However, in the 2 others (C.S. and S.T.) in whom satisfactory control urinary flows were obtained, the volumes fell during the infusion from 10.4 to 3.0 and 10.8 to 2.1 ml. per minute respectively, suggesting perfusion of the osmoreceptors.

**Effect of Changes in Sodium Intake on Renal Response to an Intravenous Salt Load.** As shown in table 4, a pronounced diminution in sodium excretion following the intravenous administration of 5 per cent saline is observed in 2 subjects with essential hypertension and 2 with Cushing’s syndrome after 5 to 7 days of a 200 to 800 mg. sodium diet. This can be correlated neither with a fall in blood pressure, since in only 2 of the 4 subjects is a decrease noted, nor with a reduction in the filtered load of sodium.
In a group I hypertensive patient, A.H., the extracellular fluid lost* during the period of sodium restriction was restored by the infusion of 1,500 ml. of isotonic saline over a period of 60 minutes. Immediately thereafter the sodium excretion following a salt load returned to that observed on a regular salt intake.

The effect of a high-sodium diet was studied in 1 normotensive individual. Increasing the intake of sodium from 4 to 8 Gm. per day did not alter the natriuretic response. However, on 12 Gm. per day the sodium excretion increased to borderline hypertensive levels. This was associated with a 5-pound gain in weight.

Effect of Acute Expansion of Body Fluid Volume on Sodium Excretion. To determine whether the abnormal natriuretic response observed in hypertensive patients following a 5 per cent saline load is the result of acute expansion of intravascular volume, 400 ml. of isosmotic albumin were administered to patient C.S. over a 25-minute period. This amount was estimated to expand the blood volume by an amount comparable to the 5 per cent saline. While sodium excretion rose (fig. 2) the peak value occurred during the second hour after the infusion and was less than half of that achieved with hypertonic salt loading when the maximum response is noted during the first hour (fig. 3). The endogenous creatinine clearance increased slightly during the infusion and then remained above the baseline, as did the urinary sodium, for the remainder of the study. It is possible that the increase in sodium excretion may be explained by the increase in glomerular filtration rate, whereas this would not appear to be the case for the excretory response portrayed in figure 3. Urine volume rose during the infusion, but then fell progressively to the control level.

Expansion of extracellular fluid with isotonic saline containing the same amount of salt as the 5 per cent solution also failed to produce a comparable natriuresis (fig. 4).

There was no significant change in blood pressure during any of these studies.

Discussion

Our results are consistent with those of others2-7 that individuals with essential hypertension excrete sodium more rapidly following a hypertonic salt load than do those with normal blood pressure. The degree of

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*Estimated on the basis of the fall in body weight.

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**Table 3.—The Effect on Sodium Excretion of Infusing 5 Per Cent Sodium Chloride into the Carotid Artery**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Blood pressure (mm. Hg)</th>
<th>Mode of administration</th>
<th>Sodium excretion† (mEq./hr.)</th>
<th>Total—Baseline</th>
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<tbody>
<tr>
<td>W. C.</td>
<td>39</td>
<td>M</td>
<td>194/112</td>
<td>Carotid Artery*</td>
<td>12.9</td>
<td>13.8</td>
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<td></td>
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<td></td>
<td></td>
<td>Intravenous†</td>
<td>23.9</td>
<td>13.0</td>
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<tr>
<td>J. L.</td>
<td>25</td>
<td>F</td>
<td>165/105</td>
<td>Carotid artery</td>
<td>8.3</td>
<td>10.3</td>
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<td></td>
<td>Intravenous</td>
<td>29.9</td>
<td>11.0</td>
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<tr>
<td>C. S.</td>
<td>29</td>
<td>F</td>
<td>160/110</td>
<td>Carotid artery</td>
<td>24.1</td>
<td>14.4</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Intravenous</td>
<td>32.1</td>
<td>3.4</td>
</tr>
<tr>
<td>S. T.</td>
<td>40</td>
<td>F</td>
<td>170/110</td>
<td>Carotid artery</td>
<td>11.1</td>
<td>12.2</td>
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<td></td>
<td></td>
<td>Intravenous</td>
<td>22.2</td>
<td>1.6</td>
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</table>

*The infusion of 5 per cent saline into the carotid artery was delivered at a rate of 1 ml. per minute for 25 minutes.
†The intravenous infusion consisted of 100 ml. 5 per cent sodium chloride per M.² BSA administered over a period of 25 minutes.
‡Total sodium excretion, mEq. sodium excreted during the 60-minute period following the start of the infusion of 5 per cent sodium chloride. Baseline sodium excretion, mEq. sodium excreted per minute during the control period preceding the infusion of 5 per cent sodium chloride multiplied by 60.
response roughly parallels the elevation of the blood pressure (until there is impairment of renal function). In the labile hypertensive subjects and 2 of the 4 group-I hypertensive subjects, as contrasted with all of the group-II patients, the salt excretion pattern resembles the normal. This would suggest the possibility that the actual duration of the hypertension could also be an important factor. Furthermore, because the abnormal natriuretic response is observed after the development of the hypertension, this response would appear to be related to the hypertension per se and not to some inherent defect peculiar to the individual who is destined to become hypertensive.

The abnormal renal response to salt loading in secondary hypertension (Cushing's syndrome and pheochromocytoma) is further support for the primary role of high blood pressure, since, as is discussed later, hormonal imbalance does not appear to be etiologically related.

To determine how the hypertensive excretory response might be influenced by sodium intake, 2 subjects with essential hypertension and 2 with Cushing's syndrome were studied while on a low-sodium diet. In all instances sodium excretion following the salt load reverted to or toward normal. There are 3 likely explanations for the diminished response:

1. Reduction in Filtered Load of Sodium. Neither a decrease in serum sodium nor filtration rate was observed in our patients while on a restricted sodium intake. However, current methods are unable to discern small changes in glomerular function that could be significant in terms of sodium excretion.

2. Increased Renal Tubular Reabsorption of Sodium. This factor could reduce sodium excretion independently or in conjunction with a decrease in filtered load. It has been demonstrated in both man18,19 and animals20 that body sodium conservation during salt depletion is associated with increased tubular sodium reabsorption. This may occur in the absence of a significant fall in filtration rate.21

Our studies would support these observations since no relationship was noted between filtered and urinary sodium when salt intake was restricted. Furthermore, in patient A.H., the replacement with isotonic saline of the extracellular fluid lost during the period of sodium restriction restored the abnormal salt load response without a significant change in glomerular function. This finding is supported by the studies of Black et al.19 on the regulation of sodium excretion in normal and salt-depleted individuals. They observed that the
### Table 4.—Effect of Changes in Sodium Intake on Sodium Excretion Following the Intravenous Infusion of 5 Per Cent Sodium Chloride

<table>
<thead>
<tr>
<th>Patient, age, sex, diagnosis</th>
<th>Sodium intake (Gm./day)</th>
<th>No. of days on diet</th>
<th>Weight (Lm.)</th>
<th>5% Sodium chloride infused (ml.)</th>
<th>Period (min.)</th>
<th>Endogenous clearance (ml./min./1.73 M.)</th>
<th>Serum sodium (mEq./L)</th>
<th>Filtration load sodium (mg./min./1.73 M.)</th>
<th>Effective sodium excretion (ml./min.)</th>
<th>Per cent infused sodium excreted</th>
<th>PAH clearance (ml./min./1.73 M)</th>
<th>Blood pressure (mm. Hg)</th>
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*The infused sodium load consisted of 100 ml. 5 per cent sodium chloride per M.² BSA.*

†This value was used as the control sodium excretion in calculating the per cent infused sodium excreted.
Table 4.—Effect of Changes in Sodium Intake on Sodium Excretion Following the Intravenous Infusion of 5 Per Cent Sodium Chloride* (Continued)

<table>
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<tr>
<th>Patient, age, sex, diagnosis</th>
<th>Sodium intake (Gm./day)</th>
<th>No. of days on diet</th>
<th>Weight (lbs.)</th>
<th>6% Sodium Intake (ml.)</th>
<th>Period (hr.)</th>
<th>Endogenous clearance (ml/min/1.73 M²)</th>
<th>Serum sodium (mEq./l.)</th>
<th>Filtration load (mEq./min)</th>
<th>Urine volume (ml/min)</th>
<th>Sodium excretion (mEq./min)</th>
<th>Per cent infused sodium excreted</th>
<th>PAH clearance (ml/min/1.73 M²)</th>
<th>Blood pressure (mm. Hg)</th>
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*The infused sodium load consisted of 100 ml. 5 per cent sodium chloride per M.² BSA.
increased sodium reabsorption noted while their subjects were on a rice diet persisted after the filtered sodium load was raised to normal levels, or higher, by the rapid infusion of saline. If, however, the period of infusion was lengthened from 20 minutes to an hour, as was the case in our patient, sodium reabsorption fell to within the normal range. They interpreted this delay in adjustment of renal function as indicating a hormonal mechanism and suggested that there may be an over-production of adrenal steroids. Corroborative evidence for this has been presented by Leaf and Couter and more recently by Bartter et al., who, with methods of steroid analysis previously unavailable, demonstrated a correlation between aldosterone secretion and extracellular fluid volume. Thus the reduction in volume associated with the low-salt diet would act to enhance the tubular reabsorption of sodium by an increased production of mineralocorticoid. The opposite might occur with increased salt intake as shown by the borderline hypertensive response to intravenous 5 per cent saline observed in the normotensive individual (C.R.) on a sodium intake of 12 Gm. per day.

3. Reduction in Blood Pressure. Although the blood pressure fell in 1 patient with essential hypertension and 1 Cushing’s patient during the period of sodium restriction, the absence of any change in the remaining subjects makes it unlikely that this contributed to the altered response.

As a possible explanation for the abnormal natriuretic response to a salt load in individuals with either essential or secondary hypertension, we postulated a cerebral sodium receptor exerting an effect on sodium comparable to that of the osmoreceptors on water. This could respond to alterations in cerebral sodium concentration and thereby control the renal tubular reabsorption of sodium. Such a center might become hyperactive in arterial hypertension, exerting a more pronounced effect on renal function. The literature contains reports of salt-losing syndromes associated with cerebral lesions. In a patient carefully studied by Cort interruption of the hypothalamic tracts by a pleomorphic glioma produced excessive loss of sodium from the body. This could not be explained by renal disease or abnormal pituitary-adrenal function. We were unable, however, to demonstrate the existence of abnormally reactive cerebral sodium receptors. The infusion of hypertonic saline into the right common carotid artery, which is known to supply the pituitary gland and the major portion of the hypothalamus including the osmoreceptors, had no effect on sodium excretion. It still remains to be demonstrated, however, that the postulated sodium receptors do not exist in the areas supplied by the vertebral vessels. Technical difficulties related to percutaneous arterial puncture of these vessels have not permitted further evaluation of this aspect of the problem.

Also considered was the possibility that the hypertensive sodium excretion pattern could result from the expansion of body fluid volume by the 5 per cent saline through an increase in filtered sodium load. As can be seen in figure 1, however, the subjects with elevated blood pressure did not show values higher than the normal. Further evidence against a volume mechanism was our failure to elicit the typical abnormal natriuretic response in a hypertensive individual when isosmotic albumin or isotonic saline was administered. It is also unlikely that an acute increase in body sodium was a contributing factor, since the isotonic saline that contained an amount of salt equivalent to the 5 per cent sodium chloride solution failed to produce a comparable natriuresis.

The underlying mechanism responsible for the abnormal natriuretic response to salt loading in arterial hypertension is still a matter of debate. Since the filtered load of sodium is not increased, attention must be directed to other mechanisms influencing renal tubular transport. The participation of neurogenic factors (including neurohumoral), while deserving of consideration, is not suggested by the evidence. We were unable to demonstrate a cerebral sodium receptor that is sensitive to changes in serum sodium con-
SODIUM EXCRETION IN HYPERTENSION

... body fluid volume or cerebral hypernatremia comparable to that produced by the hypertonic salt solution.

Catecholamines, because of their effect on electrolyte excretion, must be considered in an evaluation of this problem. It has been shown by Smythe et al.30 that the intravenous infusion of 1-norepinephrine, 1-epinephrine, and epinephrine promptly results in increased tubular reabsorption of sodium. Contrariwise, the chronic administration of epinephrine in oil is associated with increased sodium excretion.31 Since hypertension secondary to pheochromocytoma more closely resembles the latter situation, increased circulating medullary hormones could be a factor in the production of the accelerated natriuresis. This is unlikely, however, because the same load response pattern is observed in essential hypertension in which there is no conclusive evidence that these humoral agents are present in excess.32 Furthermore, in one of our patients with pheochromocytoma (J.E.), sodium excretion remained abnormal after removal of the tumor when urinary catecholamines had returned to normal levels.33

There is evidence that hypertension is associated with metabolic alterations in renal tubular cells.34, 35 Of particular interest are the histochemical studies of Shorr et al.36 in which it was demonstrated that the distribution pattern in the kidney tubule of certain intracellular dehydrogenases is altered in both essential hypertension and the hypertension associated with Cushing’s syndrome. The significance of these observations is enhanced by evidence suggesting that these enzymes may play a role in the renal transport of sodium.37-39 Further support for an intrinsic tubular lesion is the demonstration by Brodsky and Graubarth40 that osmotic loading with mannitol produced a 2 to 21/2 times greater sodium chloride excretion in hypertensive than normotensive individuals.

That the hypertensive subjects respond more normally to a salt load while on a low-salt diet in no way detracts from the concept of impaired tubular function, since Weston et al.41 have shown that the effect of mercurial...
diuretics on sodium reabsorption can be counteracted by such sodium-conserving agents as DCA.

It appears reasonable, therefore, that the abnormal renal excretory pattern of sodium in arterial hypertension could result from a tubular metabolic lesion produced by a chronically elevated blood pressure.

**Summary**

Subjects with essential hypertension and hypertension secondary to Cushing's syndrome and pheochromocytoma excrete sodium more rapidly following the intravenous administration of 5 per cent sodium chloride than do normotensive individuals. The abnormal sodium excretion pattern does not precede the development of hypertension. The degree of response to a salt load is roughly proportional to the elevation of blood pressure until there is impairment of renal function. It may also be related to the duration of the hypertension. Infusion of hypertonic saline directly into the carotid artery does not produce the abnormal renal excretion pattern in hypertensive subjects. Therefore, neither the hypothalamus, pituitary gland, nor other structures in the distribution of the carotid circulation respond to localized cerebral hypernatremia by altering the renal tubular transport of sodium. The accelerated natriuresis reverts toward normal in subjects with essential and secondary hypertension when dietary sodium is restricted. A borderline hypertensive response is observed in a normotensive individual while on a high-salt diet. The high sodium excretion in hypertension does not appear to be related to the effect of the intravenous saline on body fluid volume or sodium content. It is suggested that a renal tubular defect is responsible for the abnormal sodium excretory response to salt loading observed in hypertensive subjects.

**Acknowledgment**

We would like to express our thanks to Dr. Daniel Osher for his help with the carotid infusion studies. We would also like to express our appreciation to Mrs. Agnes Gallaway and Mrs. Yvonne Keenan, R.N., for their invaluable assistance. The insulin used in these studies was generously supplied by U.S. Standard Products Company, Mt. Prospect, Ill.

**Summario in Interlingua**

Le excretion de natrium post le administration intravenose de 5 pro cento de chloruro de natrium es accelerate in subjectos con hypertension essential o con hypertension secundari a syndrome de Cushing o pheochromocytoma in comparation con le responsa de subjectos normotensive. Le anormalitate del excretion de natrium non precede le diaspovimento de hypertension. Le grado del responsa a un carga de sal es grossiermente proportional al elevation del pression de sanguine usque il occurre un infraction del functional renal. Illo es etiam relationate possibilemente con le duration del hypertension. Le infusion de un hypertonic solution salin directemente in le arteria carotidica non evoca le mentionate anormalitate del excretion renal in subjectos con hypertension. Per consequente, ni le hypothalamo ni le corpore pituitari ni altere structuras in le distribution del circulation carotidica respond a localisate hypernatremia cerebral per effectuar un alteration del transporto de natrium in le tubulos renal. Le accelerate natriurese redeveni normal in subjectos con hypertension essential o secundari quando le ingestion dietaria de natrium es restringite. Un responsa limite de hypertension es observate in individuos normotensive quie ingere un dieta a alt e contento de sal. Le alte excretion de natrium in patientes hypertensive non pare esser relationate con le effecto del sal intravenose super le volumine o le contento de natrium del liquidos corpore. Es presentate le theses que un defecto reno-tubular es responsabile pro le anormalitate del responsa del excretion de natrium al cargation con sal que es observate in subjectos hypertensive.

**References**


Renal Excretion of Sodium in Arterial Hypertension

IRWIN B. HANENSON, HERTHA H. TAUSSKY, NATHAN POLASKY,
WILLIAM RANSOHOFF and BENJAMIN F. MILLER

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