

Low Sodium Diet in Essential Hypertension Effect on Blood Cell Ions and Hemodynamic Parameters

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The influence of salt restriction for 3 months on blood pressure, peripheral vascular resistance (observed by occlusive plethysmography), erythrocyte sodium, platelet calcium, and pH, was studied in eight untreated essential hypertensive patients.

A low salt diet decreases blood pressure, vascular resistances, erythrocyte sodium, and platelet calcium, but not platelet pH. A strong positive correlation was noted between baseline platelet calcium

and vascular resistances ($r = 0.95, P < .01$). But during the salt restriction period, a negative correlation has been observed between the changes in these parameters, which casts doubt on the use of the platelet as a model of the smooth muscle cell. *Am J Hypertens* 1992;5:661-663

KEY WORDS: Sodium, calcium, hypertension, vascular resistances, erythrocyte, platelet.

Some controversy persists about the usefulness of low salt diet as a treatment for essential hypertension.^{1,2} Moreover, the mechanism by which such a diet could decrease blood pressure is controversial: does it decrease plasma volume and cardiac output or decrease the arterial vascular resistances?^{3,4}

This study was designed to measure the effect of a low salt diet on blood pressure (BP), vascular resistances, red blood cell sodium concentration (RBC Na), platelet calcium concentration (Pt Ca) and pH. We looked for the potential relationship between these ionic concentrations and hemodynamic values. Could the platelet be a good model for the study of smooth muscle cell calcium regulation during salt diet variation?

MATERIALS AND METHODS

Eight untreated (for at least the 2 months prior to the study) essential hypertensive patients, without cardiovascular complications, adhered to a low sodium chloride diet (<85 mol/L/day) for 3 months. This group

was composed of five women and three men with an average age of 41 ± 7 years.

These patients were examined in the morning, and under fasting conditions, at the start, after 1 month, and at the end of the study. At each visit, we measured weight, 24 h urinary sodium excretion, supine BP after 20, 25, and 30 min of rest (with a random zero sphygmomanometer), and leg muscular vascular resistances (PVR) by venous occlusive plethysmography at 10 cm below the patella. After these measurements, venous blood was drawn for blood cell ionic determinations (RBC Na, Pt Ca, and pH).

The RBC Na was determined after washing and lysing the cells at room temperature within 30 min after the blood was drawn.

The platelet Ca and pH were measured with the fluorescent probes FURA-2 and bis-carboxyethylcarboxy-fluorescein (BCECF), according to Cooper,⁵ and As-tarie,⁶ respectively.

RESULTS

Table 1 shows the data we noted during the study. Significant decreases were noted in weight, BP, PVR, urinary sodium excretion (showing the good diet compliance), RBC Na, and Pt Ca. No change was observed for heart rate or platelet pH.

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TABLE 1. CLINICAL AND BIOLOGICAL DATA DURING 3 MONTHS OF LOW SALT DIET IN 8 ESSENTIAL HYPERTENSIVE PATIENTS.

	t = 0	t = 1 month	t = 3 month	P
Heart rate (beats/min)	74 ± 7	72 ± 8	78 ± 8	NS
SBP (mm Hg)	160 ± 12	152 ± 8	148 ± 10	< .001
DBP (mm Hg)	99 ± 8	95 ± 8	90 ± 4	< .001
PVR (U)	67 ± 12	60 ± 9	52 ± 14	< .01
Weight (kg)	80 ± 7	79.5 ± 4	77.5 ± 6	< .05
Urinary sodium (mmol/24 h)	190 ± 25	130 ± 30	111 ± 18	< .001
RBC Na (mmol/L cells)	8.7 ± 1	8.5 ± 1	8.2 ± 1	< .01
Platelet Ca (nmol/L cells)	106 ± 8	102 ± 7	97 ± 7	< .05
Platelet pH	7.07 ± 0.02	7.08 ± 0.03	7.07 ± 0.05	NS

SBP, DBP, systolic and diastolic blood pressures, respectively; PVR, peripheral vascular resistance; RBC Na, red blood cell sodium concentration.

At the start of this study, positive baseline correlations were noted between systolic BP and Pt Ca ($r = 0.58$), diastolic BP and Pt Ca ($r = 0.69$, $P < .05$), diastolic BP and PVR ($r = 0.69$, $P < .05$), and PVR and Pt Ca ($r = 0.95$, $P < .01$).

Correlations were also noted during the diet period with changes between Pt Ca and systolic BP ($r = 0.78$, $P < .02$), diastolic BP and RBC Na ($r = 0.57$), weight and RBC Na ($r = 0.64$), and PVR and Pt Ca ($r = -0.61$).

PVR changes during the diet were positively correlated to weight ($r = 0.39$) or RBC Na changes ($r = 0.34$). Moreover, the latter has been positively correlated to diastolic BP changes ($r = 0.57$).

DISCUSSION

This study, in spite of being small, has been able to confirm that moderate sodium restriction can, in these salt-sensitive essential hypertensive patients, significantly decrease both the blood pressure and peripheral vascular resistances as measured by noninvasive venous plethysmography, in agreement with Egan.⁷

Systolic free calcium is important in the contraction of vascular smooth muscle cells. An increase in smooth muscle cell calcium concentration participates in active tension and in arteriolar resistances. Although calcium concentration has not been measured in human essential hypertensives, platelet calcium has been reported to be elevated for untreated patients,⁸ and it has been hypothesized that such an increase reflects a similar increase in vascular smooth muscle cell.

In the present study, before treatment, positive correlations were noted between blood pressure and blood cell intracellular calcium and sodium concentrations. Moreover, a very strong correlation was noted between platelet calcium concentration and vascular resistances, which could confirm that platelet calcium could be used as a good model for testing the smooth muscle cell calcium regulation.

However, during the low salt period, a negative correlation was noted between changes in peripheral vascular resistances and in platelet calcium, suggesting that platelet calcium regulation is somewhat different from that of smooth muscle level, in agreement with Standley.⁹

The observation that platelet calcium changes during the diet are related to systolic blood pressure changes would suggest that the platelet calcium concentration is influenced by cardiac output, compliance of large arteries, and blood turbulences at this level. On the other hand, RBC sodium changes have been correlated to changes in body weight, peripheral vascular resistances, and diastolic blood pressure.

We conclude, from their significant reduction level, that moderate sodium restriction has beneficial effects on blood pressure, peripheral vascular resistances, and RBC sodium and platelet calcium concentrations.

The negative correlation, between peripheral vascular resistance and platelet calcium concentration changes induced by the diet, suggests that platelets may have some limitations as a model of smooth muscle cells in studying calcium regulation.

REFERENCES

1. Cutler J, Follmann D, Elliott P, Suh I: An overview of randomized trials of sodium reduction and blood pressure. *Hypertension* 1991;17(suppl I):I-27-I-33.
2. Folkow B: Critical review of studies on salt and hypertension. *Clin Exper Hypertens [A]* 1992;A14:1-14.
3. Fujita T, Noda H, Ando K: Sodium susceptibility and potassium effects in young patients with borderline hypertension. *Circulation* 1984;69:468-476.
4. Sullivan J: Salt sensitivity, definition, conception, methodology, and long-term issues. *Hypertension* 1991;17(suppl I):I-61-I-68.
5. Cooper R, Shamsi N, Katz S: Intracellular calcium and sodium in hypertensive patients. *Hypertension* 1987;9:224-229.
6. Astarie C, David-Duflho M, Devynck MA: Direct characterization of the $\text{Na}^+\text{-H}^+$ exchanger in human platelets. *FEBS Lett* 1990;277:235-238.

7. Egan B, Petrin J, Hoffmann R: NaCl induces differential changes of regional vascular reactivity in salt-sensitive versus salt-resistant men. *Am J Hypertens* 1991;4:924-931.
8. Erne P, Bolli P, Bürgisser E, Bühler F: Correlation of platelet calcium with blood pressure. *N Engl J Med* 1984; 310:1084-1088.
9. Standley P, Gangasani S, Prakash R, et al: Human platelet calcium measurements, methodological considerations and comparisons with calcium mobilization in vascular smooth muscle cells. *Am J Hypertens* 1991;4:546-549.