The effect of potassium and bicarbonate ions on the rise in blood pressure caused by sodium chloride

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Summary

1. A group of eight patients with mild hypertension, sensitive to sodium intake, were studied.
2. Sodium chloride (70 mmol daily) caused their blood pressure to rise by 19/14 mmHg.
3. Sodium bicarbonate (70 mmol daily) caused their blood pressure to rise by 12/5 mmHg.
4. Sodium chloride given together with potassium chloride (70 mmol of each daily) caused their blood pressure to rise by 9.6 mmHg.
5. These results suggest that sodium bicarbonate causes a smaller rise in blood pressure than sodium chloride does and that potassium chloride reduces the blood pressure raising effect of sodium chloride.
6. A low sodium, high potassium and an alkaline diet may therefore be a more effective dietary method to reduce blood pressure than a diet low in sodium alone.

Key words: bicarbonate, potassium, renin, sodium.

Introduction

Most uncultured populations have a blood pressure lower than Western populations and a relative absence of vascular disease [1–3]. Hypertension develops in these people when they move into Western society and this has been attributed to an increase in sodium intake. However, their diet changes in many other ways, including a reduction in potassium content and in alkalinity. In this study we examined the influence of extra potassium and bicarbonate on the hypertensive effect of extra sodium.

Methods

The patients were eight males, age 55–70 years, who were sodium sensitive in that they had a fall in blood pressure of 10 mmHg or more when their sodium intake was reduced. The patients were maintained on this reduced sodium intake throughout the study. After an equilibration period of 2 weeks the patients were given a placebo tablet for 2 weeks. They were then randomly allocated to groups given either 70 mmol of NaCl or 70 mmol of NaHCO₃ daily for 2 weeks and were then crossed over to the other treatment. All patients were then given 70 mmol of NaCl together with 70 mmol of KCl daily for 2 weeks and finally were given placebo for 2 weeks.

On days 13 and 14 of each study period patients were seen and blood pressures (phase I and IV) were recorded in duplicate with an amplified mercury sphygmomanometer after lying for 10 min and again after 5 min standing. Only the supine values are given here. Patients were weighed and a 24 h urine collection was made on each patient; this was analysed for Na⁺, K⁺ and creatinine. Blood was taken for the estimation of electrolytes, creatinine and plasma renin activity.

The results in each study period were compared with those in the control period and with each other by using a paired t-test, the values obtained on day 13 and on day 14 of each study period being used.

Results

The patients in this study had all previously reduced their sodium intake to half of their initial intake. They had each had a fall in supine diastolic blood pressure of 10 mmHg or more and the average blood pressure in the group had fallen from 161/105 mmHg to 142/89 mmHg. The average values for weight, blood pressure and
urinary electrolytes did not differ significantly at the entry point or during either placebo periods. There was an increase of about 70 mmol/day in urinary Na⁺ excretion in each study period and a rise of about 70 mmol/day in urinary K⁺ excretion in the KCl supplemented period, suggesting that patients complied with the dietary restriction and with the test procedures (Table 1).

Blood pressure rose by 19/14 mmHg ($P < 0.001$) when patients took 70 mmol of NaCl extra each day. This blood pressure was similar to the blood pressure measured 18 months earlier before the patients were placed on a reduced sodium intake. When NaHCO₃ was added to their diet blood pressure rose by 12/5 mmHg ($P < 0.05$) but this rise was significantly less than that obtained with NaCl supplements. When KCl was given as well as NaCl the blood pressure fell by 10/8 mmHg ($P < 0.01$) compared with that in the NaCl period, but was higher than the value obtained in the control period.

When NaCl was given weight rose in all patients and there was a mean gain of 1 kg. When NaHCO₃ was given the weight rose significantly but the rise was less than that seen with NaCl. The weight when the patients were on KCl as well as NaCl was similar to the weight on NaCl.

Serum potassium fell when NaCl was added and fell even further when NaHCO₃ was ingested. Potassium chloride caused the serum K⁺ to rise to a value similar to that of the controls.

There were no significant changes in sodium, chloride, bicarbonate, urea or creatinine. Plasma renin activity was reduced by sodium loading and was reduced further by potassium loading (Table 1).

Discussion

The patients in this study were selected because a reduction in sodium intake had caused a significant fall in blood pressure [4]. Sodium chloride added to their diet increased blood pressure to a value similar to that present before dietary restriction of sodium. When the same amount of sodium was given as sodium bicarbonate blood pressure rose less than it did after sodium chloride. Body weight rose after each supplement but less after sodium bicarbonate. The distal nephron is relatively impermeable to HCO₃⁻ and this means that less sodium is reabsorbed and its excretion is aided. The greater fall in serum potassium with sodium bicarbonate supports this suggestion. Thus the increase in total body sodium after sodium bicarbonate would be less than after sodium chloride and the mechanism leading to hypertension, whether this be humoral [5–7] or volume [8] dependent, would be activated less.

Potassium chloride administration reduced the rise in blood pressure due to sodium chloride and this effect has also been observed in rats with genetic hypertension [9]. The mechanism is unclear. It could be due to suppression of plasma renin or a potassium chloride load, like the bicarbonate load, may cause sodium to be excreted more readily and thereby reduce body sodium or volume so that the mechanisms leading to hypertension are not activated. Alternatively the potassium supplements may have specific effects on cell transport of Na⁺ and K⁺, which might ameliorate any effect on sodium transport that is subsequent to sodium loading in people with hypertension [5].

If these effects of sodium, potassium and bicarbonate are shown to be separate and additive then a more effective dietary treatment of hypertension would be not only to reduce sodium intake but also to increase potassium and bicarbonate intake. This could be achieved by eating less processed foods and moving towards a more vegetarian diet.
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References