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Low-Sodium Diet Versus Low-Sodium/High-Potassium Diet for Treatment of Hypertension*

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Summary. In 21 patients with essential hypertension the effects of moderate sodium restriction from the usual 200 mmol Na/day to 80 mmol/day over 4 weeks were compared with the effects of a combined low-sodium/high-potassium intake (80 mmol Na/120 mmol K) which was also given over 4 weeks in an open crossover trial. Systolic blood pressure in nine untreated patients with essential hypertension decreased significantly by between 5 and 7 mm Hg during moderate sodium restriction; no further decrease of blood pressure occurred during the combined low-sodium/high-potassium diet. In 12 treated patients with essential hypertension moderate sodium restriction or the combined low-sodium/high-potassium diet enabled a marked reduction of anti-hypertensive therapy in eight patients without impairment of blood pressure control (β-blockers by an average of 32%, saluretics by an average of 27% and vasodilators by an average of 24%). Here there were also no substantial differences between low-sodium intake and the combined low-sodium/high-potassium intake. It is concluded that sodium restriction to 80 mmol/day is effective in lowering systolic blood pressure but that a combined low-sodium/high-potassium diet does not further improve blood pressure control if the usual potassium intake is at least 80 mmol/day. Considerable reduction of anti-hypertensive therapy may be achieved by practical moderate sodium restriction.

Key words: Hypertension – Treatment – Sodium restriction – Potassium supplementation

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Introduction

It appears that the efficiency of moderate salt restriction for lowering blood pressure in hypertensive patients has been established beyond any doubt [1–4]. It remains controversial, however, whether an additional potassium intake provides any further benefit [5–7]. In a crossover trial we compared the efficiency of a low-sodium diet with a combined low-sodium/high-potassium diet. Since a reduction of sodium intake should in our opinion be a mainstay in the treatment of essential hypertension, the study was not designed to investigate the efficiency of a high-potassium intake in the presence of a high-sodium intake. Therefore our study is not comparable to previous studies in which the efficiency of a high-potassium intake in the presence of a high-sodium intake was evaluated [7, 12].

Methods

Nine patients with untreated essential hypertension (eight males, one female, mean age 32 years, age range 21–46 years, four with borderline hypertension, WHO I 3, WHO II 2) and 12 patients with drug-treated essential hypertension (nine males, three females, mean age 45 years, age range 28–69 years) participated in the study. All secondary forms of hypertension were excluded by the usual procedures including sonography of the kidneys, plasma renin, plasma aldosterone and urinary catecholamine measurements.

All subjects were already under weekly blood pressure controls in our out-patient clinic. After a 4-week period without dietary advice during which measurements of blood pressure were taken when supine, sitting and standing and 24-hour urinary electrolyte and creatinine excretion were controlled, the subjects were allocated randomly either to a low-sodium diet or a combined low-sodium/high-potassium diet. After a further 4 weeks the subjects were then changed to the other group. Dietary advice was given verbally and with an instruction sheet. The patients on moderate salt restriction were advised to avoid food products with added salt and to use no salt in the kitchen

or at the table. Patients on the combined low-sodium/high-potassium diet were additionally advised to increase their intake of potassium-containing foods, such as vegatables and fruit, and to take 40 mmol potassium day as a potassium supplement. Blood pressure measurements were carried out using the auscultatory method and a sphygmo manometer after the patient had been lying down for 5 min, sitting for 1 min and standing for 1 min. Urinary sodium, potassium and creatinine excretion was measured weekly through out the 12 weeks.

As blood pressure changed during the modification of the diet, we adjusted the dosage of the anti-hypertensive drugs according to blood pressure in order to maintain systolic blood pressure below 150 mm and diastolic blood pressure below 100 mm mercury. Of the 12 patients receiving anti-hypertensive drugs, four received β-blockers as monotherapy, three received β-blockers combined with a saluretic, four received β-blockers, saluretics and vasodilators and one patient received β-blockers combined with a vasodilator. For the calculation of total drug dosage during the two dietary periods in any individual patient, the total drug dosage received during the control period was taken as 100% and the drug dosage during the dietary periods was expressed as a percentage of the control period.

Results

Results of urinary sodium, potassium, creatinine, urinary sodium/potassium ratio, systolic, diastolic and mean blood pressure in the sitting, standing and lying position of the nine untreated patients during the three dietary periods are given in Table 1. The same parameters of the 12 patients with essential hypertension receiving drug therapy are given in Table 2.

Untreated Patients

As can be seen, mean urinary sodium decreased significantly from 214 to 82 mmol during the low-sodium diet and to 83 mmol during the low-sodium/high-potassium diet. Mean urinary potassium during the usual diet and the low-sodium diet were identical; it increased significantly from 80 to 117 mmol during the combined low-sodium/high-potassium diet. Only systolic blood pressure decreased by between 5 and 7 mm mercury during the low-sodium diet, whereas diastolic and mean blood pressure did not change significantly. During the combined low-sodium/high-potassium diet systolic blood pressure taken when the patient was lying, sitting and standing decreased similarly but not significantly; other blood pressures remained unchanged. Hence, blood pressure reduction achieved with the combined low-sodium/high-potassium diet was certainly not greater than that obtained with the low-sodium diet (Table 1).

Table 1. Urinary sodium, potassium, creatinine and blood pressure in nine untreated hypertensive patients without dietary advice, with low-sodium diet and with low-sodium/light-potassium diet. Figures represent $\bar{x} \pm SD$, $n=9$

		0 (No diet)	I (Low Na^+)	II (Low Na^+/high K^+)
Na^+ mmol/24 h		213.5 ± 83.2	81.5 ± 33.5[a]	83.0 ± 33.2[a]
Na^+/creatinine ratio		120.8 ± 52.2	50.9 ± 28.0[a]	53.5 ± 26.6[a]
K^+ mmol/24 h		79.8 ± 34.9	84.4 ± 45.4	116.5 ± 47.4[b,g]
K^+/creatinine ratio		42.7 ± 14.7	47.9 ± 23.3	71.3 ± 26.7[a,f]
Na^+/K^+ ratio		3.26± 2.02	1.18± 0.66[a]	0.85± 0.54[a,i]
Creatinine mg/24 h		1,915.4 ±644.7	1,867.0 ±687.2	1,695.5 ±469.8
Blood pressure (supine)	systolic	152.9 ± 9.7	147.3 ± 11.0[e]	148.6 ± 13.9
	diastolic	91.0 ± 10.6	90.7 ± 13.6	92.7 ± 12.7
	mean	117.0 ± 8.3	114.6 ± 10.9	116.2 ± 11.7
Blood pressure (sitting)	systolic	149.8 ± 8.7	142.1 ± 10.7[d]	144.1 ± 13.4
	diastolic	92.1 ± 11.2	91.1 ± 13.8	95.0 ± 14.8
	mean	116.4 ± 7.5	112.6 ± 11.0	115.7 ± 11.8
Blood pressure (standing)	systolic	146.9 ± 10.6	139.1 ± 13.7[d]	141.7 ± 16.1
	diastolic	95.4 ± 10.7	98.0 ± 12.3	97.4 ± 12.6
	mean	117.0 ± 8.5	115.4 ± 11.1	116.2 ± 11.7

Significancies for Tables 1 and 2

Unpaired t-test diet 0 vs diet I or diet II:
[a] ($P<0.001$), [b] ($P<0.005$), [c] ($P<0.01$).

Paired t-test diet 0 vs diet I or diet II:
[d] ($P<0,02$), [e] ($P<0.05$).

Unpaired t-test diet I vs diet II:
[f] ($P<0.001$), [g] ($P<0.005$), [h] ($P<0.01$), [i] ($P<0.02$)

Table 2. Urinary sodium, potassium, creatinine and blood pressure in 12 hypertensive patients on drug treatment without dietary advice, with low-sodium diet and with low-sodium/high-potassium diet. Figures represent $\bar{x} \pm SD$, $n = 12$

		0 (No diet)	I (Low Na^+)	II Low Na^+/High K^+)
Na^+ mmol/24 h		165.9 ± 58.9	82.2 ± 39.4[a]	84.4 ± 33.6[a]
Na^+/creatinine ratio		119.7 ± 46.0	66.1 ± 35.2[a]	70.9 ± 38.5[a]
K^+ mmol/24 h		64.8 ± 17.4	67.2 ± 19.5	82.1 ± 32.4[c,g]
K^+/creatinine ratio		47.1 ± 17.8	53.8 ± 22.1	67.4 ± 33.9[b,h]
Na^+/K^+ ratio		2.67± 0.94	1.32± 0.71[a]	1.17± 0.72[a]
Creatinine mg/24 h1,		465.5 ±384.7	1,332.2 ±379.9	1,291.2 ±369.4
Blood pressure (supine)	systolic	153.7 ± 30.7	149.1 ± 20.1	154.1 ± 22.9
	diastolic	101.0 ± 18.4	98.0 ± 14.6	98.0 ± 14.1
	mean	123.2 ± 22.0	119.5 ± 15.6	121.6 ± 16.7
Blood pressure (sitting)	systolic	150.7 ± 32.3	144.5 ± 18.3	148.9 ± 21.7
	diastolic	101.5 ± 19.0	100.2 ± 16.1	100.3 ± 11.7
	mean	121.1 ± 21.6	118.8 ± 15.4	120.8 ± 14.4
Blood pressure (standing)	systolic	146.5 ± 30.2	143.8 ± 17.7	148.4 ± 19.9
	diastolic	103.1 ± 18.8	103.2 ± 14.6	103.1 ± 10.8
	mean	121.4 ± 22.1	120.4 ± 14.6	122.2 ± 13.2

Symbols see Table 1

Table 3. Relative drug dosages, without dietary advice, during low-sodium diet and during low-sodium/high-potassium diet in 10 hypertensive subjects. (B = β-blockers, D = Diuretics, V = Vasodilators)

Patient	No diet			Low Na^+			Low Na^+/High K^+		
	B(%)	D(%)	V(%)	B(%)	D(%)	V(%)	B(%)	D(%)	V(%)
1	100	–	–	50	–	–	0	–	–
2	100	–	–	75	–	–	100	–	–
3	100	–	–	46	–	–	58	–	–
4	100	–	–	90	–	–	100	–	–
5	100	100	–	0	0	–	30	30	–
6	100	100	–	84	84	–	67	67	–
7	100	100	100	100	100	19	30	50	42
8	100	100	100	100	100	100	100	100	126
9	100	100	100	66	100	100	75	100	100
10	100	–	100	93	–	21	100	–	100
Total amount of drugs used (%)	100	100	100	70.4	76.8	60.0	66.0	69.4	92.0
Reduction in total amount of drugs used (%)				29.6	23.2	40.0	34.0	30.6	8.0

Treated Patients

In patients with essential hypertension who were receiving drug therapy, mean urinary sodium decreased from 166 mmol during the usual diet to about 80 mmol during the low-sodium diet and the low-sodium/high-potassium diet. Mean urinary potassium increased from about 65 mmol during the usual and low-sodium diets to 82 mmol during the low-sodium/high-potassium diet. There were no significant differences in systolic, diastolic and mean blood pressure during any of the dietary periods (Table 2).

In the 12 treated patients the low-sodium diet and/or combined low-sodium/high-potassium diet enabled a marked reduction of anti-hypertensive therapy in eight patients without impairment of overall blood pressure control (Table 2). During the low-sodium diet β-blocking drugs could be reduced by 30% and by 34% during the low-sodium/high-potassium diet. Diuretics were reduced by 23% during the low-sodium diet and by 31% dur-

Table 4. Blood pressure values and drug dosage in two initially unsatisfactorily controlled hypertensive patients. (B = β-blockers, D = Diuretics, V = Vasodilators)

Pat.	Blood pressure (mm Hg, $\bar{x}$)			Total drug dosage (%)		
	supine	sitting	standing	B	D	V
No diet						
11	220/137.5	205/132.5	205/137.5	100	100	–
12	215/125	207.5/127.5	192.5/125	100	100	–
Low Na^+						
11	200/140	190/150	190/155	200	200	–
12	174/114	165/116	168/119	113	100	–
Low Na^+/High K^+						
11	187.5/125	180/125	180/120	200	200	–
12	184.4/113.9	177.2/105.6	172.8/111.1	116	56	100

ing the low-sodium/high-potassium diet. Vasodilators could be reduced by 40% during the low-sodium diet and by 8% during the low-sodium/high-potassium diet (Table 3). Two patients are not included in Table 3 and are shown separately since their blood pressure control was unsatisfactory during the usual diet and only became better after modification of the diet combined with an increase of drug dosage. Blood pressure values and drug dosage of these two patients are shown in Table 4.

Discussion

The present study was designed to demonstrate whether the blood pressure lowering effect of a low-sodium diet can be further improved by a high-potassium intake. Further more we tried to evaluate whether significant reductions of anti-hypertensive therapy can be achieved through these dietary regimens. Instead of designing a long-term study, during which a reduction of sodium intake is usually not achieved to the intended degree, we preferred to do a short-term study with close supervision of the patient and consequently better dietary control. As can be seen from Tables 1 and 2, highly significant reductions of sodium intake could be achieved during this short-term study by informing the subjects each week whether the dietary goal of sodium reduction as measured by urinary sodium excretion, had been achieved the week before. Thus, better compliance in keeping to the diet was obtained.

Untreated Patients

As can be seen from Table 1, significant reductions in systolic blood pressure taken when the patient was lying, sitting and standing could be achieved during the low-sodium diet. Similar reductions of blood pressure were achieved during the low-sodium/high-potassium diet (Table 1); due to a greater variability of blood pressure this change was not significant. Thus it appears that the blood pressure reduction, which can be achieved through dietary advice, is achieved from the low-sodium diet alone and that there is no further improvement of blood pressure control during the combined low-sodium/high-potassium diet. This is perhaps not surprising considering that one of the actions of potassium is a saluretic effect on the kidney [8]. Furthermore, it must be assumed that potassium intake was probably already adequate during the control period judging from the urinary potassium excretion of 80 mmol/day. We cannot exclude that an increased potassium intake following a diet deficient in potassium (less than 40 mmol/day) [5] may well have shown a beneficial effect.

Furthermore, if indeed the sodium/potassium ratio of the diet does play an important role in the reduction of blood pressure [9], it must be considered that the major reduction in urinary sodium/potassium ratio to about 1 had already been achieved during the low-sodium diet and that little further reduction was obtained during the combined low-sodium/high-potassium diet. The comparatively small reduction in blood pressure during either diet is probably explained by the fact that many borderline hypertensives were included and because probably some salt-insensitive subjects [6, 10, 11] were included in the study group.

Treated Patients

In subjects with treated essential hypertension it has been possible to reduce significantly the total dosage of anti-hypertensive drugs, especially of β-blocking agents, during the low-sodium diet and the low-sodium/high-potassium diet (Table 3) without impairment of blood pressure control. Here there was also no substantial difference between a low-sodium diet and a combined low-sodium/high-potassium diet. The reduction in anti-hypertensive therapy of about 30% is of considerable economic significance. When one considers the high percentage of hypertensive subjects in the population, it appears that the amount of money spent on anti-hypertensive therapy could be reduced enormously.

We are aware that our study has several shortcomings, e.g. that the study was open, that the groups of treated and untreated patients were small, that in the group of treated patients the com-

bination of drugs used was not changed to uniform regimen, and that after dietary intervention no further control period was possible. These shortcomings, however, should not affect the main purpose of our paper, namely to compare the efficiency of a low sodium diet with that of a combined low sodium/high potassium diet. With the above reservations in mind, we would conclude that a practical lowering of sodium intake to 80 mmol/day is effective in lowering systolic blood pressure in untreated hypertensive subjects and in reducing the dosage of drugs in patients with treated essential hypertension. The low sodium diet combined with an increase in potassium intake did not further improve blood pressure control. If sodium restriction is guaranteed there appears to be at present no justification to advise a high-potassium diet for patients with hypertension, at least not in societies where potassium intake is higher than 80 mmol/day.

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