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Sodium and potassium intake and blood pressure

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Summary and conclusions

Sodium and potassium intakes were increased in normotensive volunteers to assess the effects on their blood pressures. An approximately threefold increase in sodium intake for eight days had no effect on the blood pressures of seven volunteers, while a two-stage increase in potassium intake, by about 40% for eight days and a further 55% for 14 days, had no effect on the blood pressures of 21 volunteers. Renal electrolyte excretions and the blood pressures of all 28 subjects showed no statistically significant correlations between either sodium or potassium excretion and blood pressure. A weak negative correlation was found between the sodium: potassium ratio and systolic pressure.

The small reductions in sodium intake and increases in potassium intake that might be achieved through propaganda and changes in food processing are unlikely to lower mean blood pressure in Western societies.

Introduction

Most authors of review articles have condemned the high-sodium, low-potassium diets of Western man.[1-3] Sasaki blames the hypertension of the Japanese on their high sodium intake and suggests that areas in Japan whose populations have less hypertension than average may be protected by their high potassium intakes.[4] Nevertheless, several authors have failed to find any relation between sodium intake and blood pressure in five different countries,[5-10] and very recent reviews have tended to agree that the case against sodium has not been completely proved.[11-13] On the other hand, the antihypertensive effect of potassium has been shown only in children consuming a great deal of sodium[14] and in rats with salt-induced hypertension.[15]

Raised intakes of sodium may cause high blood pressures in man,[16 17] but this may not be a general response of the whole population.[18 19] So far the protective effects of potassium have been shown only in cases of salt-induced hypertension. Nevertheless, it has been suggested that small changes in sodium[20] and potassium intake[21] may cause changes in the blood pressures of normal individuals. We have observed the effects of increasing the sodium intake and the potassium intake on the blood pressures of two groups of normotensive volunteers.

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Subjects and methods

Twenty-eight normotensive volunteers took part in the study. Seven subjects (three women, four men aged 20-35 years) increased their sodium intake for eight days. The three women and two of the men took 25 Ciba Slow Sodium tablets (600 mg or 10 mmol NaCl each) daily. The two remaining men, whose renal sodium excretion was higher than that of the others, took 30 tablets daily. The experimental period was preceded and followed by eight-day control periods. All urine produced during the 24 days was collected, the volume and time of each micturition noted, and a small sample preserved for analysis. The urine samples were analysed for sodium and potassium using a Corning 435 flame photometer. The results were expressed as mean daily excretions of sodium and potassium for the various periods. Changes in all variables during the experimental period were compared with control values using the paired *t* test.

Twenty-one subjects (11 women, 10 men aged 20-47 years) increased their potassium intake for 22 days. For eight days the subjects replaced their table salt with a mixture of equal parts of commercial table salt and potassium chloride. This was followed by 22 days during which the 11 women took eight Ciba Slow K tablets (600 mg or 8 mmol KCl) daily while the 10 men took 10 tablets daily. The experimental periods were preceded by a 14-day control period and followed by a 10-day control period. Urine was collected as above, but only for four 48-hour periods, one towards the end of each control and experimental period. Analysis was carried out as above.

During the study all the subjects had their blood pressure measured two or three times a week. They also refrained from strenuous activity, which might have resulted in a large electrolyte loss in the sweat, during the experiment. All blood pressures were measured using a Hawksley random zero sphygmomanometer, using the fifth Korotkoff sound as the diastolic pressure. The subjects were seated quietly for 10 minutes before the measurements. Each blood pressure measurement was the mean of three cuff inflations. Subjects were asked to clench their fist gently between inflations to reduce the possibility of diastolic pressure distortion through the pooling of venous blood.

Results

In the first experiment sodium intake was increased sufficiently to raise the renal sodium excretion by 190% for eight days. This change in sodium intake had no significant effect on either systolic or diastolic blood pressure (table I). While sodium intake was raised there was a slight increase in renal potassium excretion.

In the second experiment the potassium intake was increased in two stages. The first stage increased renal potassium excretion by 40% for eight days while the second stage caused a further increase of 55% for 14 days. Although the renal sodium excretion was expected to diminish slightly during the first stage, when table salt was replaced by a mixture of sodium chloride and potassium chloride, this did not occur. Neither change in potassium intake had any effect on either systolic or diastolic blood pressure (table II). There was a small statistically significant fall in the pulse pressure during both periods of increased potassium intake.

TABLE I—*Effects of dietary sodium supplementation on the blood pressure and renal electrolyte excretion of 7 volunteers. Values are means ± SE*

Period	Na^+ excretion (mmol/day)	K^+ excretion (mmol/day)	Blood pressure (mm Hg)	
			Systolic	Diastolic
Control	142·7±6·8	66·2±5·5	120·1±1·8	79·4±1·6
	p<0·001			
NaCl supplements	414·1±14·4	72·8±5·1	117·2±2·6	76·3±2·3
	p<0·001			
Control	157·6±10·1	65·6±5·9	117·7±1·9	77·6±2·0

TABLE II—*Effects of substituting mixture of equal parts sodium chloride and potassium chloride for table salt for eight days and of adding "SlowK" for 14 days on blood pressures and renal electrolyte excretions of 21 volunteers. Values are means ± SE*

Period	Na^+ excretion (mmol/day)	K^+ excretion (mmol/day)	Blood pressure (mm Hg)	
			Systolic	Diastolic
Control	171·3±16·4	53·4±4·2	115·3±1·6	79·5±0·8
		p<0·01		
$Na^+ + K^+$ ..	188·6±13·9	75·5±5·8	114·7±1·6	80·2±0·8
		p<0·001		
Slow K^+	173·3±12·0	117·0±5·1	114·8±1·4	80·2±0·9
		p<0·001		
Control	160·7±13·5	59·0±3·8	115·1±1·2	80·5±0·8

Correlation coefficients (r) calculated for the mean renal electrolyte excretion values and systolic and diastolic blood pressures of all 28 subjects during their control periods showed no statistically significant interdependence between the arterial pressure and the renal sodium or potassium excretion (table III). Systolic pressure was negatively correlated with the sodium to potassium ratio, although the degree of interdependence (r^2) was only 15%. Dividing the 28 subjects into groups with different renal sodium and potassium excretions (tables IV and V) showed no significant differences in blood pressure between these groups.

The subjects who increased their sodium intake showed a rapid weight increase of 1·5% ($p < 0·01$) after the start of the experimental period. This weight was lost promptly after the experimental period. Increasing the potassium intake caused no consistent weight change.

Only six of the 28 individuals had any family history of hypertension. This was too small a sample from which to obtain a correlation coefficient between blood pressure and renal electrolyte excretion. There was no evidence that these six individuals were different from the rest of the subjects in any way.

Discussion

Our results suggest that increasing the sodium intake of normal individuals to amounts similar to those eaten in Japan[4] has no effect on blood pressure in the short term. Murray *et al* showed increases in the blood pressures of their volunteers only when very large quantities of sodium were administered[22]—amounts far greater than those consumed by any human society. The response of the blood pressure to changes in sodium intake appears to be rapid, occurring within seven days in one report[18] and within three days in another.[22]

The correlation between blood pressure and renal sodium excretion reported here was small and not statistically significant. Pietinen *et al* showed a good correlation between blood pressure and renal sodium excretion but only in those individuals with a family history of hypertension.[19] Too few of our volunteers had family histories of hypertension for us to confirm this observation. Kawasaki *et al*, studying a group of hypertensive patients, showed that the blood pressures of some responded well to changes in sodium intake while others were apparently insensitive.[18] Nevertheless, recent surveys of unselected populations have failed to find any relation between blood pressure and sodium intake.[9 10]

Increasing the potassium intake in two stages had no effect on the blood pressures of our volunteers. Mickelsen *et al* achieved an increase in renal potassium excretion similar to that seen in our first stage using the same technique.[23] They measured blood pressures only twice but thought that there had been no change. Gros *et al* gave potassium supplements to patients with essential hypertension but failed to decrease their blood pressures.[24]

Before the advent of reliable drug treatment special salt-free diets were used to lower blood pressures in hypertensive patients,[25] although some contemporary workers reported effects which were too small to be clinically useful.[26] More recently Parijs *et al* showed that halving the salt intake of a group of hypertensive patients for several weeks resulted in only small reductions (about 6%) in blood pressure.[27] The suggestion made by Freis that "reduction of salt in the diet to below 2 g (34 mmol) per day would result in the prevention of essential hypertension and its disappearance as a major public health problem"[2] appears optimistic. The blood pressures of the Japanese,[28 29] whose sodium intake is chronically high, are not much higher than those measured in a London survey[30] or in the USA,[29] despite the prevalence of hypertension in Japan.[1] On the other hand, blood pressures are generally high in Finland while the renal sodium excretion is lower than that in Japan.[5] We consider that genetic[18 19] and dietary factors[31–33] have at least as large a part to play in essential hypertension as sodium or potassium. The relatively small reductions in sodium intake and increases in potassium intake that might be achieved through propaganda and changes in food processing are unlikely to lower the mean blood pressure in Western countries. Such manoeuvres may be useful to people who are genetically susceptible to salt-induced hypertension or whose salt intake is unusually high.

TABLE III—*Correlation coefficients (r) between control values of systolic and diastolic pressures and renal electrolyte excretion from tables I and II*

	Blood pressure	
	Systolic	Diastolic
Sodium excretion ..	−0·252	0·160
Potassium excretion ..	0·204	0·252
Sodium:potassium ..	−0·390	−0·148
	(p<0·05)	

TABLE IV—*Comparison between individuals with different renal sodium excretions. Control values for renal electrolyte excretion and blood pressure are given. Values are means ± SE*

	Na^+ excretion (mmol/day)	K^+ excretion (mmol/day)	Blood pressure (mm Hg)		Age
			Systolic	Diastolic	
High Na ..	258·4±32·5	56·3±12·1	116·2±3·0	81·3±1·8	25·2±4·4
(n=6)	p<0·01				
Medium Na	150·8±4·5	58·5±4·0	116·3±1·8	78·5±0·9	22·7±1·1
(n=17)	p<0·001				
Low Na ..	97·2±9·8	50·7±6·3	120·4±2·9	81·6±0·9	24·2±3·2
(n=5)					

TABLE V—*Comparisons between individuals with different renal potassium excretions. Control values for renal electrolyte excretion and blood pressure are given. Values are means ± SE*

	Na^+ excretion (mmol/day)	K^+ excretion (mmol/day)	Blood pressure (mm Hg)		Age
			Systolic	Diastolic	
High K^+ ..	176·2±35·9	79·1±4·0	118·6±2·9	80·1±2·1	26·6±3·5
(n=8)		p<0·001			
Medium K^+	156·6±12·8	51·8±1·9	116·8±1·8	79·5±0·8	22·7±1·2
(n=16)		p<0·001			
Low K^+ ..	170·8±18·1	29·5±2·7	112·0±1·6	78·2±1·0	20·5±0·5
(n=4)					

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"Benign" monoclonal IgE gammopathy

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Summary and conclusions

So far IgE monoclonal paraproteins have been found only in patients with malignant diseases, though there are benign monoclonal paraproteins of other immunoglobulin classes. A patient with osteoporosis first seen in Paris in 1965 was found to have a paraprotein type λ. In 1977 immunoelectrophoresis identified this as IgE λ paraprotein, and immunodiffusion studies showed precipitin bands identical with those in patients with IgE myeloma.

This patient seemed to have a benign monoclonal IgE gammopathy which had existed for 14 years. Though the possibility of transition into multiple myeloma cannot be excluded, this case suggests that a monoclonal expansion of IgE lymphocytes need not produce malignant change.

Introduction

"Benign" monoclonal paraproteins can be found in about 1% of the population over 25 years of age. With increasing age the incidence of paraprotein rises to more than 2·5% in individuals over 70. Thus benign paraproteinaemia of the IgG, IgA, IgM class or of light-chain type is relatively common and only rarely followed by multiple myeloma or Waldenström's macroglobulinaemia. For IgE, however, the immunoglobulin class with normally only trace serum concentrations, no benign monoclonal IgE gammopathy has been reported so far, although 13 cases of IgE myeloma[1-4] and two patients with lymphoproliferative disorders[5 6] and IgE paraproteinaemia have been described up to now.

Case report

A 71-year old woman was referred in 1977 for evaluation of occasional discomfort in her lumbar spine. She had suffered from malnutrition and anaemia from 1939 to 1946. In 1963 she had complained of pain in the thoracolumbar region. Calcium injections were given, but no radiographs taken until December 1965, when severe pain and almost complete immobilisation led to her admission to hospital in Paris, where osteoporosis with collapse of several vertebrae was found. Haemoglobin was 8·2 g/dl, leucocyte count $6·2 \times 10^9$/l with 35% lymphocytes (half atypical). The erythrocyte sedimentation rate was 40 mm in 1 h, while serum calcium, phosphate, and serum protein concentrations were normal. Serum electrophoresis disclosed a minimal spike in the gamma region. Proteinuria was 250 mg/24 h with no Bence Jones protein. Calcium excretion was 60 mg/24 h on an unrestricted diet and 150 mg/24 h after intravenous calcium. In January 1966 immunoelectrophoresis (Professor M Seligmann) showed a paraprotein type λ, which was not detected in unconcen-

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