LOW SODIUM/HIGH POTASSIUM DIET FOR PREVENTION OF HYPERTENSION: PROBABLE MECHANISMS OF ACTION

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Summary 20 normotensive subjects (10 with a family history of hypertension) were investigated as to whether moderate salt restriction and/or a high potassium intake had a beneficial effect on blood pressure regulation and prevention of hypertension. In all subjects a moderate reduction of salt intake from 200 to 50 mmol/day over 2 weeks reduced the rise in blood pressure induced by various doses of noradrenaline (0·1, 0·2, and 0·4 μg/kg/min). Furthermore, of 20 subjects 12 (8 with a family history of hypertension) responded to salt restriction with a fall in systolic or diastolic blood pressure of at least 5 mm Hg. There were no significant differences in plasma renin, aldosterone, vasopressin, and catecholamine levels between responders (salt-sensitive subjects) and non-responders, but salt-sensitive subjects had a mean baseline diastolic blood pressure which was higher than that of salt-insensitive subjects by 13 mm Hg (77·3±3·26 vs. 64·6±2·06, $p<0·001$). A high potassium intake reduced diastolic blood pressure by at least 5 mm Hg in 10 out of 20 subjects; of the 10 7 had a family history of

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hypertension and 9 responded to salt restriction. A high potassium intake also improved compliance with a low salt regimen, promoted sodium loss, prevented the rise in plasma catecholamines induced by a low salt diet, and increased the sensitivity of the baroreceptor reflex. These four effects occurred in the group as a whole and were probably the means by which a high potassium intake reduced blood pressure. In all subjects 2 weeks of a combined low sodium/high potassium intake reduced blood pressure rises induced by mental stress or noradrenaline infusion by 10 mm Hg. The results of this study suggest that moderate salt restriction combined with a high potassium intake helps to prevent hypertension, that salt-sensitive subjects exist, and that these individuals would profit most.

Introduction

TO prove whether man's usual high sodium/low potassium diet may be a cause of essential hypertension[1] and whether a low sodium/high potassium diet prevents hypertension, a study of the therapeutic effect of a low sodium/high potassium diet in already established hypertension probably does not suffice. Such studies[2-7] have given equivocal results. Since the hypertensive process affects the ability of the kidney to excrete sodium,[8,9] studies of the role of sodium in the development of hypertension would have to be conducted in normotensive subjects likely to become hypertensive in later life, and over the time hypertension takes to develop. We have investigated the effects of salt restriction and/or high potassium intake in normotensive subjects with and without a familial predisposition for hypertension. Because changes of blood pressure after short-term intervention are likely to be small or absent, we have investigated the effects of dietary change not only on blood pressure but also on those mechanisms said to contribute to the pathogenesis of hypertension. These are: (a) mental stress and sympathetic nervous system activity,[10-12] and (b) baroreceptor dysfunction.[13,14] This work was presented at the 7th Meeting of the International Society of Hypertension (New Orleans, 1980) and a short account of part of this work has been published.[15]

Material and Methods

20 normotensive medical students, all male (aged 21–25 years), were studied. 10 of them had a family history of established hypertension in parents or grandparents. The rest had parents and grandparents who were known to be normotensive.

The order in which subjects were given test diets was randomised. The four diets were: the "usual" (high sodium/low potassium) diet containing; 200 mmol of salt and 80 mmol of potassium per day; a high potassium diet containing 200 mmol salt and 200 mmol potassium; a low sodium diet containing 50 mmol salt and 80 mmol potassium; or a low sodium/high potassium diet containing 50 mmol salt and 200 mmol potassium. Each type of diet was taken for 2 weeks. The caloric intake was not changed.

The diets were provided by the dietetic department of the hospital, the amount of sodium and potassium in the usual diet being that usually consumed in Western Europe; the moderate salt restriction was achieved by eliminating food containing added salt and using no salt for cooking or baking except in bread. A high potassium intake was achieved by the inclusion of large amounts of vegetables and fruit; by the use of a commercial salt substitute containing a mixture of potassium salts, mainly potassium chloride ('Sina-Salz', Nordmark-Werke, Hamburg, Federal Republic of Germany); and by taking daily a tablet of 'Kalinor' (Nordmark-Werke) containing 40 mmol of potassium.

After a 24 h urine specimen had been collected, a needle was placed in a forearm vein of the fasting subject at 0800h. After 90 min of supine bed rest heparinised blood was taken for determination of plasma renin, aldosterone, vasopressin, noradrenaline, and adrenaline. During this time pulse rate and systolic, diastolic, and mean blood pressures were recorded every minute by the use of a 'Dinamap' model 849 with recorder 950 (Applied Medical Research Corp., Tampa, Florida, U.S.A.). The subjects then had to do a standardised calculation stress for 3 min while blood pressure and pulse continued to be monitored. Immediately after the mental stress test blood was collected for plasma catecholamines and vasopressin determination. In the afternoon of the same day, after 30 min supine bed rest, graded doses of noradrenaline (0·1 μg/kg/min, 0·2 μg, and 0·4 μg) were infused each over 5 min, while blood pressure and pulse rate were recorded every minute.

The methods of hormone determinations were: plasma renin by the method of Boyd et al.;[16] aldosterone by the method of Ito et al.;[17] vasopressin by the method of Robertson et al.;[18] and catecholamines by the method of Hörtnagl et al.[19] Blood pressures and pulse rates used for comparison of the effects of the different diets were the means of values recorded during the morning and afternoon resting periods. Values used to indicate the effect of mental stress were the highest pulse rate and the mean of the two highest blood pressure values recorded after the stress test. Values used for the evaluation of pressor response to noradrenaline were the differences between the mean values of blood pressure and pulse rates of the resting period and those of each 5 min period of noradrenaline infusion. The "steady-state" baroreceptor reflex properties were assessed by a simplification of the method of Korner et al.[14] In 5 subjects sodium space was also measured by the use of sodium-24, when they were on high sodium/low potassium and low sodium/high potassium diets. Since each subject was his own control, paired *t*-tests were used for statistical comparison of all results, except where not normally distributed. These were evaluated by the Wilcoxon matched pairs signed rank test.

Results

Moderate salt restriction or a high potassium intake alone resulted in the loss of about 1 kg body weight associated with small but significant increases of serum creatinine and uric acid (table I). In the 5 subjects in whom sodium space was measured, low sodium/high potassium diets reduced the space from 25·75±1·79 (±SEM) to 21·83±1·24 litres ($p<0·005$). None of the diets produced major changes in serum potassium, whereas serum sodium rose after the low sodium/high potassium diet. Plasma renin and aldosterone showed the expected changes, the greatest stimulation of the renin-aldosterone system occurring after the low sodium/high potassium intake, during which urinary sodium excretion was lower and urinary potassium higher than when sodium intake was reduced and potassium intake was increased separately. There were no major changes in plasma adrenaline or plasma vasopressin. Plasma noradrenaline rose with the low sodium diet; this increase did not occur when a combined low sodium/high potassium diet was taken, although urinary sodium excretion was even lower on the combined diet. Calculation stress caused equal maximum rises in blood pressure, whether the subject was on the usual diet, the low sodium diet, or the high potassium diet, but heart rates were significantly higher (by about 10 beats/min with the low sodium and high potassium diets.) The combined low sodium/high potassium diet reduced the rise in blood pressure during mental stress.

A tracing of blood pressure and pulse rate recordings obtained during noradrenaline infusion is shown in fig. 1. Fig. 2 shows that, compared with the usual diet, a high potassium diet resulted in greater falls in heart rate associated with blood pressure rises during noradrenaline infusion; a low sodium diet led to smaller blood pressure rises during

TABLE I—EFFECTS OF SODIUM RESTRICTION AND/OR HIGH POTASSIUM INTAKE IN NORMOTENSIVE SUBJECTS ON BODY WEIGHT, ELECTROLYTES, PRESSOR HORMONES AND RESPONSE TO MENTAL STRESS (MEAN±SEM)

	Usual diet	Low sodium diet	High potassium diet	Low sodium/high potassium diet
Change in body weight (kg)	75·8±2·66	−1·02±0·07†	−0·89±0·13*	−1·20±0·10 ‡
Systolic blood pressure (mm Hg)	125·0±2·39	122·3±2·32	123·3±2·54	122·7±1·81
Diastolic blood pressure (mm Hg)	73·1±2·17	70·1±1·86	68·6±1·98	69·6±1·67
Heart rate (beats/min)	62·2±2·44	67·4±3·29	62·4±2·06	71·0±6·88
Serum sodium (mmol/l)	141·2±1·25	144·3±1·29	142·6±0·90	149·1±0·49‡
Serum potassium (mmol/l)	4·69±0·13	4·64±0·09	4·51±0·10	4·75±0·20
Serum uric acid (mg/dl)	5·74±0·28	6·92±0·28‡	5·76±0·27	7·22±0·63‡
Serum creatinine (mg/dl)	1·07±0·02	1·13±0·03†	1·13±0·03†	1·28±0·04‡
Plasma renin (pg/ml/h)	328·3±32·02	602·8±72·20‡	274·8±46·10	1439·4±413·67‡
Plasma aldosterone (ng/100ml)	5·3±1·34	15·9±1·79‡	11·1±1·58†	43·1±9·75‡
Plasma noradrenaline (ng/ml)				
Basal	0·353±0·075	0·605±0·177‡	0·440±0·101	0·418±0·056
After mental stress	0·413±0·108	0·590±0·169	0·401±0·078	0·448±0·031
Plasma adrenaline (ng/ml)				
Basal	0·053±0·009	0·062±0·017	0·062±0·011	0·048±0·009
After mental stress	0·087±0·009	0·071±0·019	0·066±0·015	0·076±0·009
Plasma vasopressin (pg/ml)				
Basal	10·1±0·76	11·9±1·49	10·5±0·88	Not done
After mental stress	11·8±0·90	13·4±1·41	11·9±1·00	Not done
24 h urinary sodium (mmol/day)	210·5±23·6	40·3±7·5‡	155·1±20·9	28·4±5·3‡
24 h urinary potassium (mmol/day)	71·4±5·8	65·4±5·6	115·5±11·5†	172·4±4·3‡
Response to mental stress				
Heart rate at 1 min (beats/min)	83·50±5·23	93·60±5·98†	88·30±5·25*	100·00±9·81‡
Systolic blood pressure at 2 min (mm/Hg)	134·50±4·92	134·50±4·27	134·30±3·70	124·40±4·25‡

*p<0·05; †p<0·01; ‡p<0·001.

each step of noradrenaline infusion; and the low sodium/high potassium diet dampened the pressor response to exogenous noradrenaline so much that 0·8 μg noradrenaline/kg/min was required to achieve the rises in blood pressure induced by 0·4 μg/kg/min noradrenaline when the subject was on the high sodium/low potassium diet.

Fig. 3 shows that baroreceptor sensitivity (ratio of decrease in pulse rate to increase in mean arterial pressure during infusion of 0·2 μg noradrenaline/kg/min) rose from 1·23±0·257 (mean±SEM) to 3·33±1·025 when the potassium intake of 80 mmol/day was raised to 200 mmol/day (p<0·01, Wilcoxon matched pairs signed rank test). In contrast, baroreceptor sensitivity remained unchanged when sodium intake was altered. A linear regression was calculated from the logarithms of changes in blood pressure as related to changes of pulse rate during noradrenaline infusions. The linear regression equation derived from all subjects for the combined low potassium (high and low sodium) diet was Δ pulse rate (beat/min)= 15·8−20·6 log (Δ mean blood pressure + 10 mm Hg), $r = -0·51$, n=100, p<0·001; for the combined high potassium (high and low sodium) diet it was Δ pulse rate = 19·1 − 25·9 log (Δ mean blood pressure + 10),

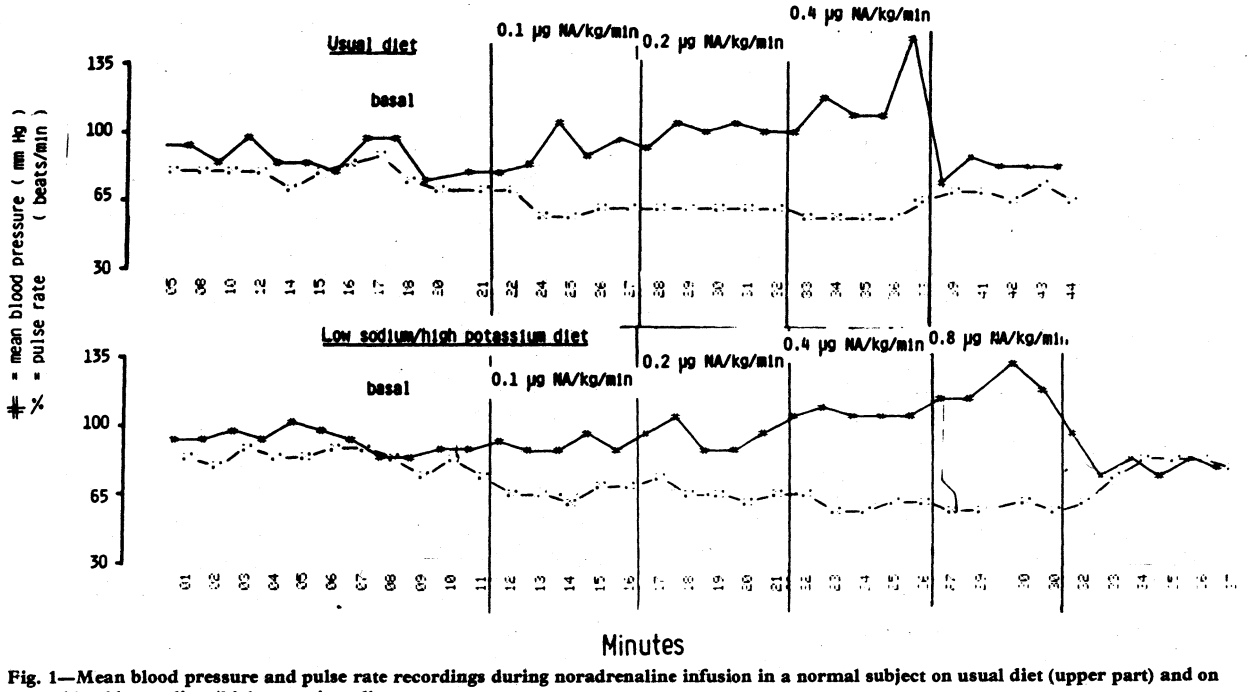

Fig. 1—Mean blood pressure and pulse rate recordings during noradrenaline infusion in a normal subject on usual diet (upper part) and on combined low sodium/high potassium diet.

At each step of infusion blood pressure rises are smaller and pulse rate responses are more pronounced during the low sodium/high potassium diet.

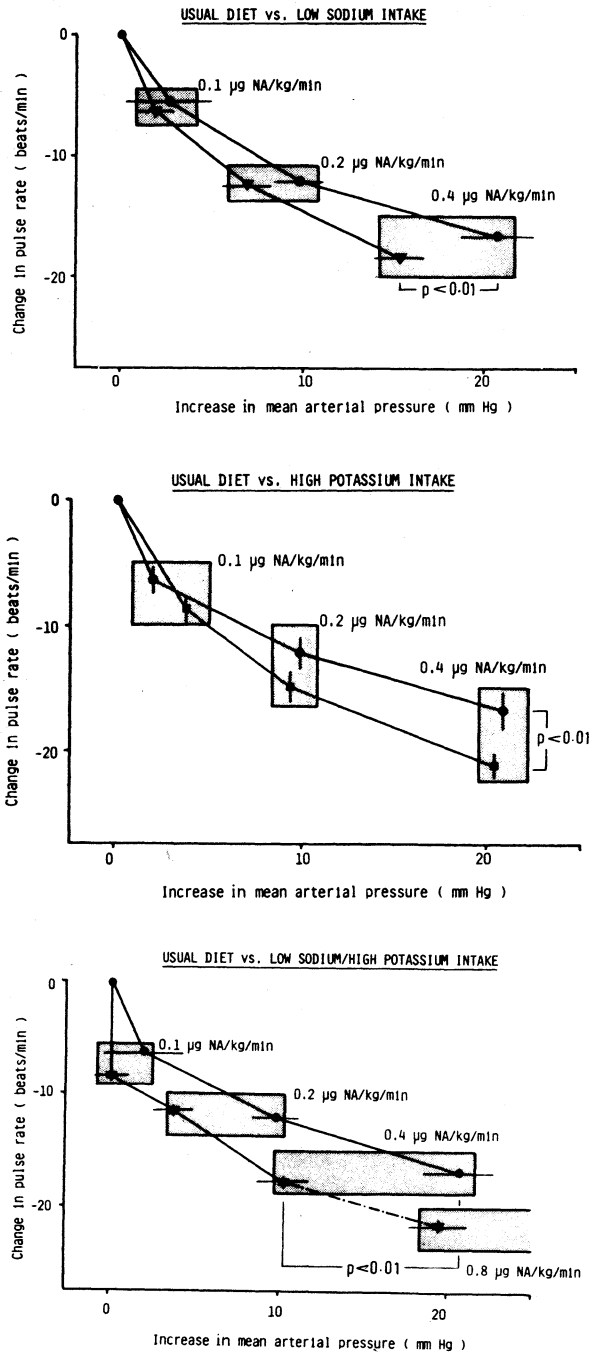

Fig. 2—Mean changes of mean blood pressure and pulse rates in 20 normal subjects switched from their usual "high sodium/low potassium" diet (●) to a salt restricted diet (▼, top), a high potassium diet (■, middle), or a combined low sodium/high potassium diet (★, bottom).

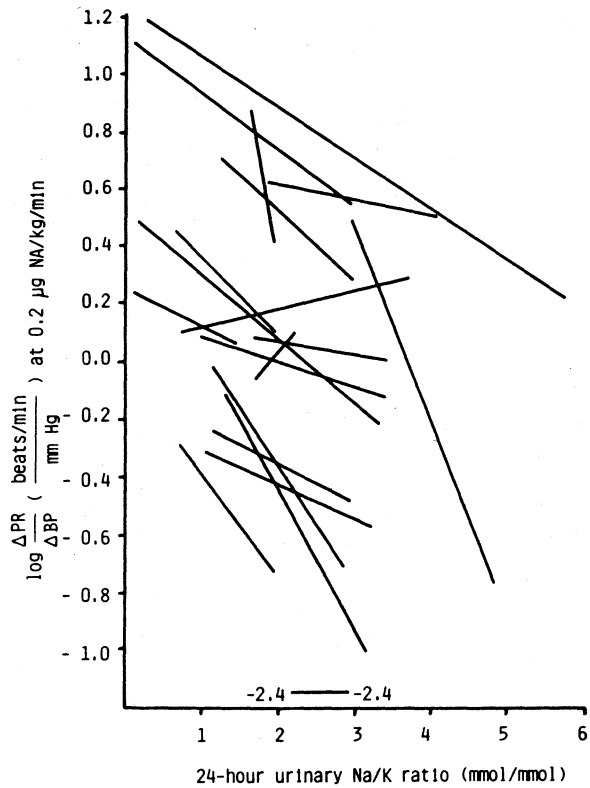

Fig. 3—Baroreceptor sensitivity (ratio of decrease in pulse rate to increase in mean arterial pressure on 0·2 μg noradrenaline/-kg/min) in relation to 24 h urinary sodium/potassium ratio.

Each line shows the change in value for a subject changing from his usual diet to a high potassium diet ($p<0·01$, Wilcoxon matched pairs signed rank test).

$r = -0·63$, $p<0·001$. The slope of baroreceptor sensitivity in subjects on a high potassium intake was significantly different from that when subjects were on their usual ("low") potassium intake ($p<0·01$).

Although basal blood pressure did not change significantly during either the low sodium or high potassium diet when calculated for the whole group (table I), after salt restriction it fell by between 5 and 11 mm Hg in 12 out of 20 subjects (8 out of 10 with, and only 4 out of 10 without, a family history of hypertension); after an increase of potassium intake it also fell by at least 5 mm Hg in 10 out of 20 subjects (7 of the 10 had a family history of hypertension and 9 were salt-responsive). Blood pressure did not change (±4 mm Hg) in the others. Subjects were classed as responders and non-responders according to whether there was a fall in systolic or diastolic blood pressure after sodium restriction or after an increase of potassium intake (table II). There were no significant differences in urinary electrolyte excretion or pressor hormones between the two groups, but subjects in whom blood pressure fell after a sodium restricted diet or a high potassium diet had started with higher ($p<0·001$) baseline diastolic blood pressures than had the non-responders (difference of 12·7 mm Hg between salt responders and non-responders, and 6·7 mm Hg between potassium responders and non-responders).

Discussion

The aim of our study was to show whether a moderate reduction in sodium intake from the average of 200 mmol per day consumed in industrialised nations to 50 mmol per day affected blood pressure and its regulation, and whether sodium replacement by potassium produced additional effects. In that and in other respects our study is different from the study of Parfrey et al.[20] who considered the usual sodium intake "low" and raised it. 50 mmol sodium/day was chosen as our lower limit not only because it would be practicable but also because this amount seems to be the

TABLE II—COMPARISON OF BLOOD PRESSURE AND URINARY ELECTROLYTE EXCRETION IN NORMOTENSIVE SUBJECTS IN RELATION TO A FALL OF BASAL BLOOD PRESSURE AFTER MODIFICATION OF DIET (MEAN±SEM)

	Diet	p between responders in effect of different diets	Responders	p between responders and non-responders in effect of specific diet	Non-responders	p between non-responders in effect of different diet
Systolic blood pressure	High Na Low Na	p<0·001	126·3±3·31 121·2±3·26		122·4±2·87 124·6±2·64	NS
Diastolic blood pressure	High Na Low Na	p<0·001	77·3±3·26 72·7±2·21	p<0·001 p<0·001	64·6±2·06 65·6±1·83	NS
24-h urinary sodium	High Na Low Na		215·6±23·0 42·2±12·3		200·2±58·3 52·0±17·6	
Systolic blood pressure	Low K High K		127·5±3·16 122·5±3·40	p<0·001	118·5±2·53 123·8±3·88	
Diastolic blood pressure	Low K High K	p<0·001	74·0±3·53 65·6±3·15	p<0·01	67·3±3·08 69·7±2·90	NS
24-h urinary potassium	Low K High K		80·8±8·58 117·8±20·5		60·0±8·51 97·3±15·4	

maximum amount to which the feedback loops controlling sodium balance were originally aligned.[21] Hence in our study moderate salt restriction or increase in potassium intake led to a significant decrease of body weight and concomitant increases in serum creatinine and uric acid in all subjects. We therefore infer that when on their usual high sodium/low potassium diet these subjects live with a larger extracellular fluid volume (we estimate by about 1 litre) than when on a low salt diet containing at least five times the minimum salt requirement. This inference is confirmed by the contraction in sodium space after the low sodium/high potassium diet in 5 of the subject—the contraction of 3·91±0·34 litres (±SEM, p<0·005) is even greater than that expected from the change in body weight.

In all subjects sodium restriction reduced the pressor response to exogenous noradrenaline. This effect was independent of baseline blood pressure (basal pressure when on the usual diet) and of a family history of hypertension (of the 13 subjects whose pressor response was much impaired 7 had a family history of hypertension). This reduction in pressor response to noradrenaline, which has also been observed after severe sodium restriction,[23] may be partly due to the rise in plasma noradrenaline, observed in the present study and by others.[24] However, this cannot be the sole explanation, since the reduced pressor response was also observed after the combined low sodium/high potassium diet, after which there was no increase of plasma noradrenaline. Although changes in blood pressure after mental stress were not altered by sodium restriction or by a high potassium intake, there was evidence of the altered haemodynamics associated with sodium restriction[25]—namely, the higher pulse rates needed to produce a specific blood pressure increment; this change must be a consequence of either a reduced cardiac pre-load and/or afterload. All these effects were seen in the entire group.

Since our findings suggest that there are probably four ways whereby a potassium intake of 80 to 200 mmol/day improves blood pressure regulation, such a diet may be of value in the prevention of hypertension in man. Like sodium restriction, a high potassium intake alone produced weight loss and increased serum creatinine; these findings indicate a reduction in extracellular fluid volume, presumably due to a direct saluretic effect of potassium on the kidney.[26] But, unlike sodium restriction, a high potassium intake improves baroreceptor function. This improvement is indicated by the increase of the ratio Δpulse rate/Δblood pressure from 1·23±0·257 (mean±SEM) to 3·33±1·025 at 0·2 μg NA/kg/min (p<0·01); and secondly, by the significantly steeper slope of baroreceptor sensitivity after transformation of pulse rate and blood pressure changes into a linear regression. Although in animal work baroreceptors are sensitive to volume changes[27] or to changes in extracellular sodium and potassium concentration,[28,29] we were surprised when the different diets containing physiological amounts of sodium and potassium affected carotid sinus reflex after only 2 weeks. An increase in potassium intake also prevents the increase in plasma noradrenaline which usually occurs after sodium restriction (table I)—a phenomenon for which we do not have a ready explanation and which will require further investigation.

The fourth way by which a high potassium intake might help to prevent hypertension is probably by improving compliance with a low salt diet. Food usually consumed with salt is replaced by food, such as fruit, which is not generally taken with salt; also, the subjects found that the mixture of potassium salts supplied as salt substitute was very acceptable.

When subjects on the low sodium or high potassium diet were classed as responders or non-responders,[22] the only criterion of response being a fall of basal blood pressure of at least 5 mm Hg, the only significant difference between the two groups was a higher baseline diastolic blood pressure among the responders (table II). There were no significant differences in urinary electrolyte excretion or in levels of pressor hormones. Although severe sodium restriction may induce a response in more subjects, those who responded to our moderate sodium restriction or to an increase in potassium intake probably form the group most susceptible to hypertension since they were more likely to have a family history of hypertension, and to have higher baseline diastolic blood pressures (the latter probably as a result of their salt sensitivity).

In some parts of the industrialised world potassium intake may be half that in Europe.[1] In these places an increase of potassium intake may be even more useful in improving blood-pressure regulation.

Of the many feed-back loops regulating blood pressure, at

least four are influenced beneficially by moderate salt restriction and high potassium intake: (1) the renin-angiotensin system and (2) the aldosterone system which are brought back into their regulatory range where they can help to control sodium balance;[21] (3) the sympathetic nervous system (said to be overactive in hypertensive subjects, especially those with borderline hypertension), which would raise blood pressure to a lesser extent than would be reached after the usual high sodium/low potassium diet; and (4) the baroreceptor reflex which becomes more sensitive. Only very simple preventive measures can be put into practice on a large scale. Since compliance with sodium restriction alone is poor, and since additional benefits may be expected, replacement of sodium by potassium deserves a long-term trial for preventing hypertension. Further studies will be required to confirm whether there indeed exists a salt-sensitive normotensive group who would benefit more from the regimen than would the population as a whole.

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