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Double-blind randomised trial of modest salt restriction in older people

Francesco P Cappuccio, Nirmala D Markandu, Christine Carney, Giuseppe A Sagnella, Graham A MacGregor

Summary

Background Stroke is directly related to blood pressure and treatment trials in older hypertensive individuals show a reduction in strokes. However, the majority of strokes occur in normotensive individuals in whom no attempt is made to lower blood pressure. We compared the effects of modest salt restriction on blood pressure in older hypertensive and normotensive people.

Methods 47 untreated elderly people (24 men, age range 60–78 years; blood-pressure range 123–205 mm Hg systolic and 64–112 mm Hg diastolic) completed a 2-month double-blind randomised placebo-controlled crossover study of modest salt restriction with slow sodium and placebo to give a salt intake of either 10 g (equivalent to the normal amount for the UK population) or 5 g.

Findings On the normal salt intake for the UK population, supine blood pressure was 163/90 (SD 21/10) mm Hg with urinary sodium excretion of 177 (49) mmol/day. With modest sodium restriction, blood pressure fell to 156/87 (22/9) mm Hg ($p<0·001/0·004$) with a urinary sodium excretion of 94 (50) mmol/day. A reduction in sodium intake of 83 mmol/day was associated with a reduction of 7·2/3·2 mm Hg. There was no significant difference in the blood-pressure fall between 18 normotensive and 29 hypertensive participants (8·2/3·9 *vs* 6·6/2·7 mm Hg).

Interpretation A modest reduction in salt intake leads to a fall in blood pressure in both normotensive and hypertensive older people similar to that in outcome trials of thiazide-based treatment. Since the majority of strokes in older people occur below the current definition of hypertension, our results have important implications for the prevention of stroke.

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Blood Pressure Unit, Department of Medicine, St George's Hospital Medical School, London SW17 0RE, UK (F P Cappuccio MRCP, N D Markandu RGN, C Carney RGN, G A Sagnella PhD, Prof G A MacGregor FRCP)

Correspondence to: Dr F P Cappuccio

Introduction

Increasing blood pressure throughout its range is the major risk factor for the development of stroke. The absolute risk of a stroke is related not only to the height of blood pressure, but also to age.[1] Treatment trials, mainly diuretic based, in older people with high blood pressure, including isolated systolic hypertension, have shown a reduction in strokes, both fatal and non-fatal, with almost complete reversal of the calculated epidemiological risks.[2,3]

Reductions in blood pressure in older hypertensive individuals similar to those in the treatment trials can be achieved by non-pharmacological intervention. For instance, a moderate reduction in salt intake alone,[4,5] or combined with an increase in potassium intake,[6] or with a reduction in bodyweight,[5,7] lowers blood pressure in older hypertensive patients. However, no studies have been carried out in older people with blood pressure in the normal range, which is surprising given that the majority of strokes in older people occur at blood pressures in the upper range of normal where, at present, drug treatment is not considered and no attempt is made at prevention.

We thus decided to carry out a double-blind randomised trial of the effect on blood pressure of a modest reduction—5 g per day—in salt intake from the

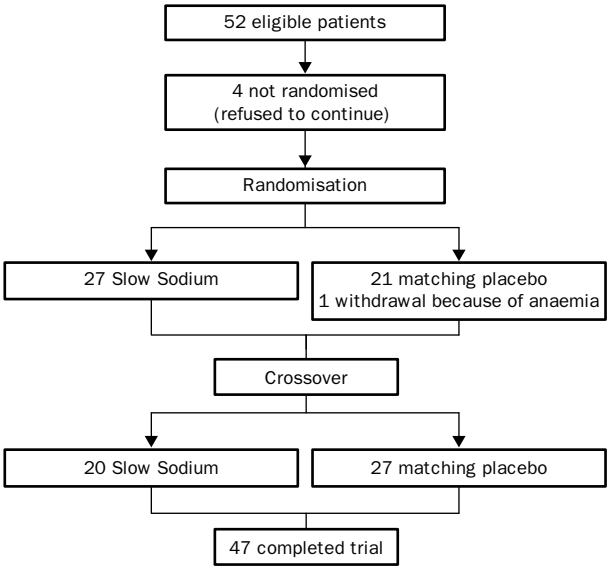

Figure 1: **Trial profile**

normal UK levels (10 g salt per day) in a group of older people with a broad range of blood pressures, and to compare the effect on blood pressure in both hypertensive and normotensive individuals.

Patients and methods

People were recruited from either general-practitioner referrals or volunteers, usually spouses or friends of the patients, aged 60 years or more, with no previous treatment for high blood pressure or withdrawal of treatment for at least 4 or 8 weeks if diuretics, and informed written consent. The study was approved by the St George's Hospital Ethics Committee. Exclusion criteria were systolic blood pressure greater than 210 mm Hg or diastolic pressure greater than 115 mm Hg, secondary or accelerated hypertension, recent evidence of ischaemic heart disease or congestive heart failure, or cerebrovascular disease. 52 participants entered the study, four withdrew during the run-in before randomisation, and one after 1 month of the crossover phase due to anaemia (figure 1). 47 participants completed the study. The mean age was 66·8 years (SD 5·3; range 60–78). There were 24 men, 23 women; 42 were white, three South Asian, and two black. There were five couples (husband and wife).

All participants underwent a period of familiarisation in the Blood Pressure Unit with blood-pressure measurements on no treatment for at least 1 month. Their usual salt intakes were then measured, after which they were told how to reduce their salt intake to about 5 g (80 mmoles sodium) daily. Participants achieved this reduction by not adding salt at the table or in cooking, and by avoiding foods that contain large amounts of salt. Salt-free bread was provided for those who had no easy access to it, but no other foods were given. Written instructions and recipes were given to all participants, who were instructed by specially trained nurses. In appropriate cases, the spouse or whoever cooked in the household was also seen. At every visit the nurses reiterated these instructions. After 2 weeks of reduced sodium intake, further measurements were made, after which participants entered a double-blind, randomised, crossover study. They were allocated in random order (by means of random-generated numbers handled by one of us not involved in the clinical assessments) to take 12 Slow Sodium tablets daily (10 mmoles sodium per tablet) or 12 Slow Sodium matching placebo tablets daily. After 1 month, measurements were repeated and participants crossed over to the opposite treatment for a further month. Neither nurses nor participants were aware of the treatment allocation at the time of the study. The amount of Slow Sodium administered was calculated to give a salt intake equivalent to the average sodium intake for the UK. 27 individuals were given Slow Sodium first, and 20 placebo first. We detected no significant order-of-treatment effect.

Measurements were done at entry to the study, 2 weeks after the salt restriction, and at the end of each month of the double-blind, crossover phase, and consisted of blood pressure, pulse, weight, and two consecutive 24 h urine collections. Blood was also taken for measurements of routine biochemistry and plasma renin activity. Participants were seen on the same day of the week, at the same time of day, by the same nurse, in the same room; blood pressure was measured in the same arm by nurses with semi-automatic ultrasound sphygmomanometers (Arteriosonde) with attached recorders. Supine and standing blood pressures were the mean of five readings taken with 1–2 min intervals. Bodyweight was measured by automatic scales (Seca) during which participants wore light indoor clothing and no shoes. Participants were carefully instructed—orally and by printed material—on how to collect urine, and the mean of two consecutive 24 h urine collections was taken as sodium excretion at that time. All blood was taken without stasis after participants had been sitting upright for 5–10 min between 0900 and 1200 h.

Results are expressed as the mean with SD, SE, or 95% CI, as appropriate. We used paired Student's t tests to compare differences between the variables during the two phases in the crossover part of the study. A subgroup analysis between hypertensive and normotensive participants was planned a priori with unpaired comparison of mean changes in blood pressure. We used correlation analysis (after log-transformation of non-normally distributed variables) to study associations between blood-pressure response to changes in sodium intake and other variables. By means of sample-size calculations, we estimated that a minimum of 44 participants (allowing for subgroup analysis and 10% drop-out rate) were needed to detect a change in blood pressure of 10/5 mm Hg with $\beta=0{\cdot}90$ and $\alpha=0{\cdot}05$ between the two sodium intakes. Given the conservative estimate of the SD of difference used in the sample-size calculation, we actually achieved a greater power in our study.

Results

Run-in phase

Table 1 shows the baseline characteristics of the participants at the end of the run-in period, and after

Variable	Run-in on habitual diet week 4	Run-in on reduced sodium diet week 2	Randomised double-blind crossover		Difference (95% CI)	p for difference
			Reduced sodium intake week 4	Normal sodium intake week 4		
Supine blood pressure (mm Hg)						
Systolic	161·7 (19·9)	154·9 (20·4)	155·9 (21·6)	163·2 (20·6)	7·2 (3·3, 11·1)	<0·001
Diastolic	89·7 (9·7)	87·1 (9·5)	86·9 (8·8)	90·1 (10·5)	3·2 (1·1, 5·3)	0·004
Standing blood pressure (mm Hg)						
Systolic	155·5 (19·6)	148·0 (20·2)	151·1 (21·2)	155·0 (21·5)	3·9 (0·1, 7·7)	0·05
Diastolic	93·4 (11·5)	91·3 (10·2)	91·6 (8·7)	93·7 (10·5)	2·1 (0, 4·2)	0·05
Urinary excretion (mmol/24 h)						
Sodium	127 (38)	72 (40)	94 (50)	177 (49)	83 (66, 100)	<0·001
Potassium	63 (17)	66 (20)	66 (18)	65 (18)	–0·9 (–5, 3)	0·65

Results are mean (SD).

Table 1: **Blood pressure and urinary sodium and potassium excretion in 47 participants throughout study**

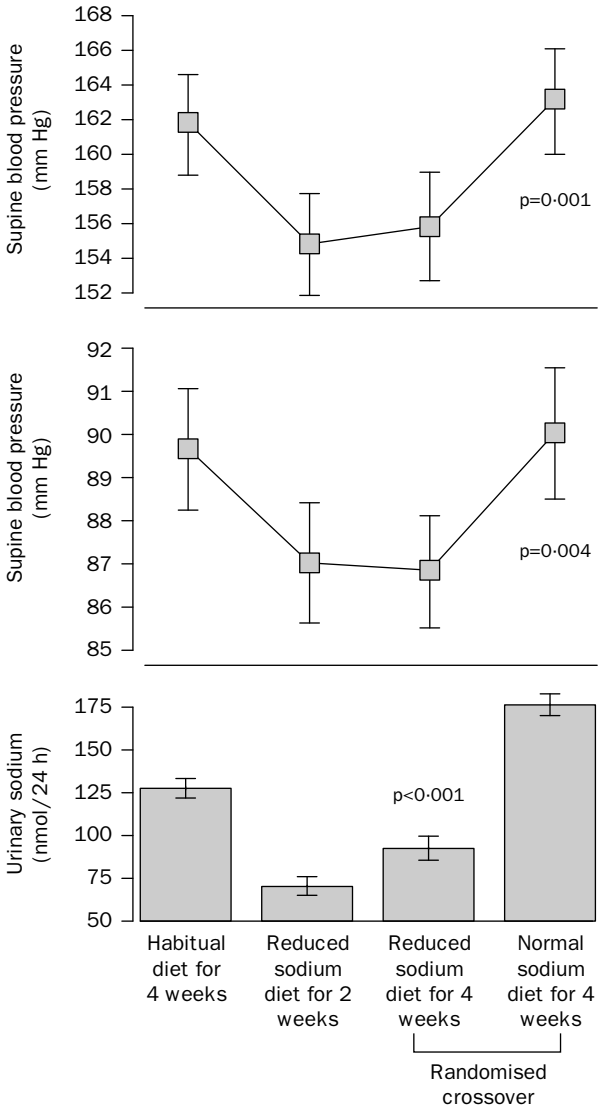

Figure 2: **Blood pressure and urinary sodium excretion at end of each dietary period in 47 participants**
Statistical comparison by paired *t* test in crossover phase. Results expressed as mean and SE. SBP=systolic blood pressure; DBP=diastolic blood pressure.

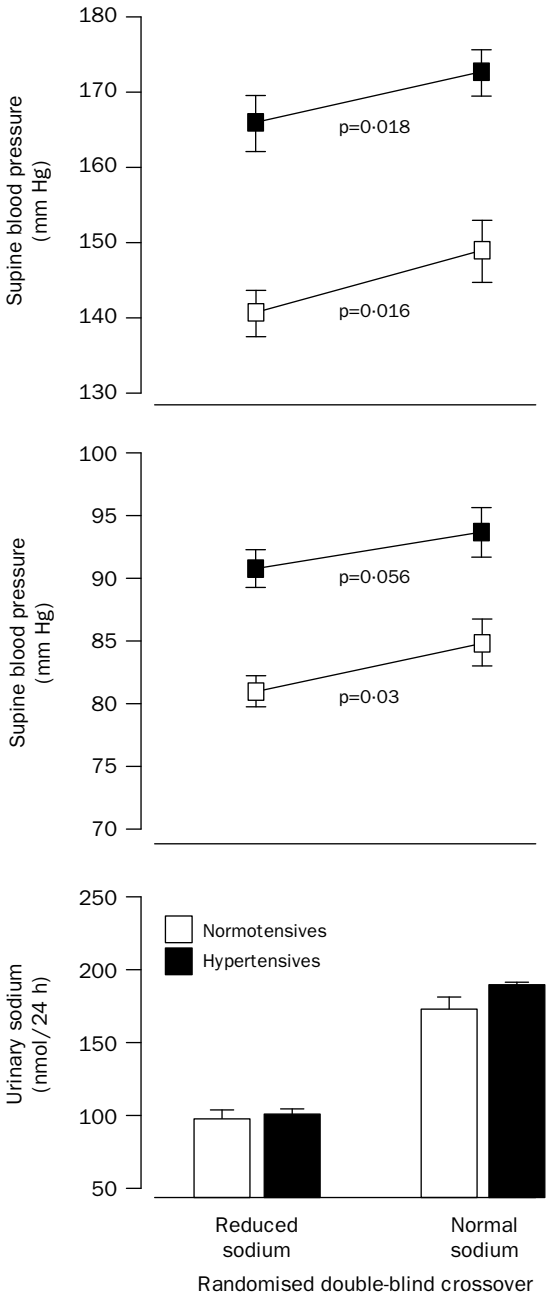

Figure 3: **Blood pressure and urinary sodium excretion after 4 weeks during each phase of crossover study in normotensive (n=18) and hypertensive (n=29) participants**
Statistical comparison by paired *t* test. Results expressed as mean and SE.

2 weeks on a reduced sodium intake. After the 4-week run-in phase, supine systolic blood pressure ranged from 123 to 205 mm Hg, and diastolic pressure ranged between 64 and 112 mm Hg. After 2 weeks of sodium restriction, average blood pressure fell with a reduction in urinary sodium that ranged from 59 to 239 mmol/24 h (figure 2).

Randomised double-blind crossover phase

After 1 month of Slow Sodium tablets (normal sodium intake) the supine blood pressure was 163/90 (SD 21/10) mm Hg (urinary sodium excretion 177 [49] mmol/24 h). Blood pressure fell after 1 month of Slow Sodium placebo tablets (reduced sodium intake) to 156/87 (22/9) mm Hg (p=0·001 for systolic, p=0·004 for diastolic) with a urinary sodium excretion of 94 (50) mmol/24 h (table 1). In the group as a whole, there was thus a fall in blood pressure of 7·3/3·2 mm Hg with an average reduction in sodium intake of 83 mmol/24 h (figure 2). Of the 47 participants, five did not comply with either their diet or taking of Slow Sodium tablets since their 24 h urinary sodium excretion was not lower on the reduced sodium intake. After exclusion of these participants from analysis, the blood-pressure reduction during the period of reduced sodium intake was similar to that for the entire population (7·5/3·6 mm Hg). We also recorded smaller but significant reductions for standing blood pressure in the group as a whole (table 1).

Normotensive and hypertensive people

There was a significant fall in supine blood pressure after reduction of sodium intake both in the 18 normotensive individuals and in the 29 hypertensive participants (table 2). However, there was no significant difference in the fall in blood pressure between these two groups (p=0·70 for

Variable	Normotensive people (n=18)				Hypertensive people (n=29)			
	Randomised double-blind crossover		Difference (95% CI)	p for difference	Randomised double-blind crossover		Difference (95% CI)	p for difference
	Reduced sodium intake week 4	Normal sodium intake week 4			Reduced sodium intake week 4	Normal sodium intake week 4		
Supine blood pressure (mm Hg)								
Systolic	140·4 (13·4)	148·5 (18·0)	8·2 (2·3, 14·0)	0·016	165·6 (20·1)	172·2 (16·6)	6·6 (1·5, 11·8)	0·018
Diastolic	80·8 (5·4)	84·7 (7·9)	3·9 (0·7, 7·2)	0·03	90·7 (8·3)	93·5 (10·6)	2·7 (0·05, 5·5)	0·05
Standing blood pressure (mm Hg)								
Systolic	138·3 (16·7)	142·9 (20·5)	4·5 (–0·5, 9·5)	0·09	159·1 (19·9)	162·6 (18·7)	3·5 (–1·9, 8·9)	0·21
Diastolic	85·7 (6·5)	88·0 (9·6)	2·3 (–1·5, 6·2)	0·25	95·2 (7·9)	97·2 (9·7)	2·0 (–0·5, 4·4)	0·12
Urinary excretion (mmol/24 h)								
Sodium	91 (54)	167 (54)	76 (50, 102)	<0·001	95 (48)	182 (46)	87 (64, 110)	<0·001
Potassium	69 (18)	65 (22)	–3·7 (–10·9, 3·5)	0·32	65 (17)	64 (18)	–0·7 (–4·3, 5·7)	0·77

Results are mean (SD).

Table 2: **Blood pressure and urinary sodium and potassium excretion in normotensive and hypertensive participants**

systolic and p=0·59 for diastolic; table 2 and figure 3). Standing blood pressures showed similar trends, though they were not significant (table 2).

Other variables

After 4 weeks of a normal salt intake compared with 4 weeks of reduced salt intake, there were no significant changes in plasma sodium (141 [2] to 141 [3] mmol/L), plasma creatinine (88 [17] to 89 [16] μmol/L), fasting glucose (4·9 [0·7] to 4·9 [0·6] mmol/L), serum cholesterol (5·9 [1·1] to 6·0 [1·0] mmol/L, or serum triglycerides (1·4 [0·7] to 1·5 [0·6] mmol/L. There were significant increases in plasma potassium and in plasma renin activity on the lower salt intake (4·1 [0·3] to 4·2 [0·4] mmol/L, p=0·011; 1·22 [0·98] to 1·56 [1·40] ng mL^{-1} h^{-1}, p=0·004; respectively). There was no significant change in bodyweight or pulse rate. There were similar changes in plasma renin activity in normotensive and hypertensive participants (1·16 [0·90] to 1·47 [0·85] ng mL^{-1} h^{-1}, p=0·024; and 1·27 [1·06] to 1·62 [1·67] ng mL^{-1} h^{-1}, p=0·04; respectively).

Correlations between blood-pressure response and other variables

The reduction in supine blood pressure during lower sodium intake was not significantly associated with age (r=0·20 for systolic and –0·15 for diastolic), sex, urinary sodium excretion (r=0·04 for systolic and 0·01 for diastolic), or plasma renin activity (r=–0·22 for systolic and –0·14 for diastolic); nor was it associated with changes in urinary sodium (r=0·04 for systolic and 0·12 for diastolic) or plasma renin activity (r=–0·12 for systolic and –0·11 for diastolic) during changes in sodium intake (all r values with p values greater than 0·1).

Discussion

Our results show that in untreated older people with a broad range of blood pressures, a modest reduction in sodium intake of 80 mmol/24 h (about 5 g salt) over 1 month produced a substantial reduction in both systolic and diastolic blood pressure of 7·2/3·2 mm Hg. This reduction took place in participants in both supine and standing positions, with no evidence of orthostatic hypotension, postural symptoms, or metabolic side-effects. More importantly, our study clearly shows that similar falls in blood pressure occur in normotensive older people (fall in blood pressure 8·3/2·9 mm Hg).

These findings differ from those in younger people, in whom both baseline blood-pressure measurements and the reactivity of the renin-angiotensin system are strong predictors of the response to salt-intake reduction.[8] This difference between younger and older people is likely to result from suppression of the renin-angiotensin system with increasing age, since we found no difference in the rise in plasma renin activity with the reduction in salt intake in the older normotensive or hypertensive people.

The fall in blood pressure in our study is similar to that in the controlled-outcome trials of drug therapy in older hypertensive people with the use of thiazide diuretics.[2,3] A quantitative overview of these trials has estimated a 36% reduction in the 5-year incidence of stroke with this blood-pressure fall, and that the short-term benefits are much greater in older than in younger people.[2] To prevent one vascular event (particularly stroke) over a given period of time, four times more younger people need to be treated than those over the age of 60.[1] Despite the fact that drug therapy is effective and beneficial, it is currently only considered for those with sustained hypertension—because no outcome trials are available in people with lower blood pressure. However, since the increased risk of development of stroke is present throughout the blood-pressure range, this high-risk strategy ignores the majority of older people, in whom the greater number of strokes occur. A much more comprehensive population approach is necessary to yield the greatest benefits.

Swales[9] has highlighted the need for a population approach despite the absence of long-term trials with hard endpoints, since "it is legitimate to argue that reducing the blood pressure even of patients conventionally regarded as 'normotensive' would result in reduction of risk [for strokes]". Moreover, these blood-pressure changes in the population could possibly be achieved by effective and sustained reform of diet and lifestyle.[10] In a reanalysis of all evidence relating salt intake to blood pressure, Law and colleagues[11] estimated that a reduction in sodium intake of 50 mmol/24 h (about 3 g salt) in older people would lower the population's systolic blood pressure by an average of 5 mm Hg. Our findings fit almost exactly with Law's predictions in this older population. Law also predicted that this lowering would lead to a 26% reduction in strokes. In the UK alone this reduction would translate into 40 000 fewer deaths from stroke every year.

However, there are two questions that need to be answered. Can such a reduction in sodium intake be sustained over a long period, and is the fall in blood pressure long lasting? As yet, we do not have follow-up data on these older people, but we have shown in a previous trial of similar design that even after 1 year of follow-up, the sodium intake of the participants remained

at around 50 mmol/24 h and the blood pressure was unchanged.[12] We have no reason to believe that the same would not apply to an older age group. Law's study, and others,[13] clearly show that a modest reduction in sodium intake is feasible and can be achieved over a long period, provided salt is not added to food or in cooking, and highly salted processed foods are avoided. The availability of salt-free bread allows greater reductions in salt intake.[14] Some argue that a reduction in salt intake makes the diet unpalatable. But there is now good evidence that when individuals choose to reduce their salt intake their taste preferences change,[15–17] and increased sensitivity of salt-taste receptors with a lower sodium concentration gives the same salty taste. Food with less salt becomes preferred over more salty food, and this ensures long-term compliance.

An increasing problem as we adopt a more hectic lifestyle is that we eat more processed food and do not cook from natural ingredients. 70–80% of our salt intake now comes from salt hidden in processed food and bread. Clearly, we need the cooperation of the food-processing industry to reduce the very high concentrations of salt in processed foods often equivalent to sea water.[18] The immediate benefits to the UK population of a modest reduction in salt intake, particularly in older people as shown in our study, would far outweigh the commercial reasons for the addition of salt to food.[19] Long term, this modest reduction is also likely to help prevent hypertension and its vascular complications, as well as bone demineralisation (osteoporosis)[20] and its complications in older people, both likely consequences of a life-time of high intakes of salt.

Our results suggest that a modest reduction in salt intake in all older people, irrespective of blood-pressure status, will cause a fall in blood pressure similar to that seen in thiazide-based outcome studies. We believe that this fall in blood pressure, as in the outcome studies,[2,3] will result in a major reduction in strokes—not only in older hypertensive patients, but also in older normotensive people.

Contributors

F P Cappuccio was responsible for the conception of the study, its design, ethical approval, funding, direct supervision, recruitment, statistical analysis, interpretation, and the writing of the paper. N D Markandu and C Carney were responsible for recruitment, day-to-day contact with participants, dietary counselling, measurements, and data handling. G A Sagnella was responsible for randomisation, data handling, biochemical measurements, statistical analysis, and interpretation. G A MacGregor was responsible for the conception of the study, interpretation, and the writing of the paper.

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