Salt and hypertension: a sceptical review of the evidence

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There is plenty of good evidence that sodium has something to do with hypertension. The question that I propose to examine in this paper, is whether a daily intake of 170–200 mmol of sodium is a factor in raising blood pressure. This is the level of intake in various European countries, U.S.A., Australia and New Zealand.

My own ideas on the matter have changed. I always liked Borst’s concept that in hypertension the ‘willingness’ of the kidneys to excrete sodium must be subnormal (Borst & Borst-de Geus, 1963). This type of concept has, of course, been extensively developed by Guyton and his group (Guyton, Coleman, Cowley, Manning, Norman & Ferguson, 1974). So when the opportunity arose, my colleagues and I did a population survey (Simpson, Nye, Bolli, Waal-Manning, Goulding, Phelan, de Hamel, Stewart, Spears, Leek & Stewart, 1978a; Simpson, Waal-Manning, Bolli & Spears, 1978b) with the specific idea of getting evidence relating sodium output to blood pressure within the population, something that we felt had never been done.

However, as we have reported (Simpson, Waal-Manning, Bolli, Phelan & Spears, 1978c), we could find no evidence of a relationship between pressure and sodium output or between pressure and the sodium/potassium ratio or the sodium/creatinine ratio. Our initial reaction was that a single 24 h collection of urine and a blood pressure (albeit measured three times) on a single occasion contained too much error, that there was too much ‘noise’ in the data. And this may well be so. However, obviously an alternative explanation could be that at this sodium intake (giving a 24 h mean excretion of 173 mmol for men, 140 mmol for women) there is no relationship to high blood pressure. It is therefore worthwhile looking at the evidence favouring a link between sodium and blood pressure rather more critically.

We seem, in fact, to have got into a situation where the most slender piece of evidence in favour of such a link is welcomed as further proof of the link, while failure to find such evidence is explained away by one means or another. Francis Bacon (1620) once said of this sort of situation; ‘The human understanding when it has once adopted an opinion ... draws all things else to support and agree with it. And though there be a greater number and weight of instances to be found on the other side, yet these it either neglects and despises, or else by some distinction sets aside and rejects’.

The excess salt theory is in fact a very powerful one, and its protagonists put it forward with tremendous enthusiasm, a little like a religion or a political theory. We could call it ‘antinatriumism’. Now it may be that the antinatriumists are right, that all of us, all over the world, should keep our sodium intake down to such a level that we excrete less than 60 mmol of sodium/day. My own view is that this is not proved and that it is not right to go to the public and launch an anti-salt campaign unless we have better evidence.

The late Dr Lewis Dahl, to whom we owe a major debt for opening up the whole subject, was impatient with anyone with opposing views, quoting (Dahl, 1972) Maeterlinck: ‘at every crossway in the road to the future, each progressive spirit is opposed by a thousand men appointed to guard the past’. The trouble is that progressive spirits sometimes get carried away by their ideas. It may be appropriate in this Scandinavian city of Göteborg to recall that the Norwegian playwright Henrik Ibsen wrote a play (Brand) about a progressive spirit who wanted to lead his people on to new and better things but who would only have succeeded in making them all die in the mountains if they had not been persuaded by two guardians of the past to give up the idea. (Though I am not suggesting that a lower salt intake would lead any of us to death.)

Let us then look at the evidence on which antinatriumism is based.
Animal studies. There is no doubt that a heavy intake of salt, with or without some other manoeuvre, can cause hypertension in animals. However, we have to remember that the doses used are simply enormous. Dahl used an 8% NaCl diet for his rats (Dahl, Heine & Thompson, 1974). A rat eats about 100 g of food per kg body weight per day and therefore the daily NaCl intake would be about 0.8% of its body weight. For a 70 kg man this would represent 560 g of NaCl/day. Even allowing for the smaller turnover of food in a man, 8% NaCl in the dry diet would mean an intake of about 40 g or 680 mmol/day. Vogel (1966) used 2 g of NaCl/kg body weight in dogs, i.e. the equivalent of 140 g/day in man. These are colossal amounts and probably outside the area of relevance. Even so, many rats seem able to withstand such intakes. Aoki, Yamori, Ooshima & Okamoto (1972) found no difference in blood pressures of spontaneously hypertensive rats between those on a low sodium intake and those on a 2.72% NaCl diet; 1% NaCl had to be put in the drinking fluid before a difference in pressure was found. The New Zealand strain of genetically hypertensive rats is equally resistant to salt. This brings up genetic aspects of susceptibility to salt and this point will be taken up again later.

Taste threshold for salt. The notion that hypertensive subjects have less ability to taste salt dates back to a paper by Fallis, Lasagna & Tétreault (1962). This paper is often wrongly quoted; in fact the threshold for noting a difference between distilled water and a solution of salt seems about the same in the hypertensive as in the normotensive groups in taste threshold. Even so, many rats seem able to bring up genetic aspects of susceptibility to salt. A difference between hypertensive and normotensive group, but the hypertensive subjects have less ability to taste salt dates this point will be taken up again later.

relationship of sodium intake to blood pressure within a population. One would think that if there was anything of importance in this matter, then hypertensive subjects would take in and excrete more sodium than normotensive subjects (provided that they had not voluntarily reduced NaCl intake on learning of their hypertension). However, no evidence of this has been obtained.

Ashe & Mosenthal, back in 1937, studied 24 h urinary sodium output in 1000 subjects and stated that 'patients with hypertension do not normally eat more protein or salt than normal persons'. Dahl (1958) published raw data for 24 h urinary sodium excretion in 48 normotensive and 22 hypertensive men, and the mean values can be calculated. Some subjects had both a single value given and a mean value for several collections; in such cases the latter (mean) has been taken as the value for that individual. The mean (±sd) for the normotensive group turned out to be 183 ± 53.1 mmol/day and for the hypertensive group 193 ± 37.8 mmol/day. The difference was not significant by either Student’s t-test or the Mann-Whitney two-sample rank test. Phear (1958) found no difference in sodium output between 20 hypertensive men (mean output 156 mmol/day) and 20 similarly aged (mean 50-56 years) normotensive men (154 mmol/day). Miall (1959) found that hypertensive postmenopausal women (n = 19) excreted slightly less sodium (mean output 123 mmol/day, average of 7 days’ values) than did comparable normotensive women (n = 104, mean output 137 mmol/day, average of 7 days’ values). In the Framingham study, Dawber, Kannel, Kagan, Donabedian, McNamara & Pearson (1967) found no relation between 24 h urinary sodium and blood pressure in 297 men aged 40-49 years. Berglund, Wikstrand, Wallentin & Wilhelmsen (1976) found unconvincing. Dahl & Love (1954, 1957) reported a correlation between self-estimated salt use and blood pressure, but no one since then seems to have confirmed this (Miall, 1959; Swaye, Gifford & Berrettoni, 1972; Meneely, 1974). Dahl (1972) himself later wrote 'our salt habit questionnaire was useful to us but a disservice to others … unless (these methods) give an index of actual salt intake, they have no relevance whatsoever'. Schechter et al. (1973) have claimed that, on a low sodium diet, hypertensive subjects who are given a choice of 0.9% NaCl or water will drink more saline than normotensive subjects, but their subjects were poorly matched for skin colour and also for age: the mean age for the hypertensive group was 42 years and for the controls 22 years.
that sodium output was if anything lower in newly diagnosed hypertensive subjects (175 mmol/day) than in normotensive subjects (198 mmol/day). We find the mean sodium output in patients referred to our clinic (178 mmol/24 h for men, 138 mm/24 h for women) no higher than the mean values in the population of Milton (n = 1206), a small town near Dunedin (173 mmol for men, 140 mmol for women; Simpson et al., 1978a). As mentioned earlier, we were unable to find any correlation between blood pressure and sodium excretion or sodium/potassium or sodium/creatinine ratios in the Milton population (Simpson et al., 1978c).

On the other hand, some investigators claim to have demonstrated the elusive link between sodium and blood pressure within a population. Joossens in Belgium has published interesting epidemiological and historical data linking NaCl intake to stomach cancer and stroke, and has also published results of a population survey demonstrating a link between sodium intake and blood pressure. However, this survey population (Joossens, Willems, Claessens, Claes & Lissen, 1970) was not homogeneous but composed of three subgroups, one of which comprised all the elderly people and they had a particularly high sodium/creatinine ratio in their urine. Most of the conclusions from the study of this very considerable population (n = 2027) seem to be based on analyses which give undue preference to sodium rather than to body weight or even age.

Joossens states, for instance, in a later paper (Joossens, 1973) that he and his colleagues had demonstrated a positive correlation between systolic pressure and 24 h urinary sodium both in 1314 men and in 713 women. Yet it is clear from the earlier paper that the correlation is not significant for women if age is taken into consideration, as it surely must be. In the analysis of a more closely studied group of 19 men and 20 women (Joossens et al., 1970), criticisms can be made that the data for men and women are pooled and that a given individual in summer and the same individual in winter has been counted as two separate subjects.

Morgan, Carney & Wilson (1975) have published data from a population survey indicating that sodium output of a normotensive group (n = 50, mean 111 mmol/day) is lower than that of a group of mild hypertensives (n = 400, mean 148 mmol/day). These two groups are said to be exactly comparable but no data are given on this, not even the male:female ratio. The sodium output of 150 patients referred to the hypertension clinic is also given, mean 185 mmol/day, and is said to be significantly different from that of the normotensive group in the population survey; the hypertensive subjects are described as being 'from the same general population' but in fact they were all male. Virtually all surveys show a higher sodium excretion for men than for women, so this comparison was inappropriate. In a later study (Morgan, Adam, Gillies, Wilson, Morgan & Carney, 1978), the sodium output of a more comparable group of relatively normotensive men and another group of mildly hypertensive men was found to be 191 mmol/day in both cases. And in an earlier paper (Carney, Morgan, Wilson, Matthews & Roberts, 1975) it is stated that 'in Australia many people, hypertensive or not, have a sodium intake of 200 to 400 meq./day'. One's impression is that the population survey reported by Morgan, Carney & Wilson (1975) produced peculiarly low values for sodium excretion in the 50 normotensive subjects.

Schneckloth, Corcoran, Stuart & Moore (1952) found no regular association between output of salt and the occurrence of hypertension in West Indians. Langford, Watson & Douglas (1968) found overnight sodium excretion per hour to be higher in the hypertensive negro girls in a school than in the girls with the lowest blood pressure. But the difference was not significant by the Wilcoxon-White non-parametric test and the authors are more much cautious in their conclusions than are some subsequent authors who quote the paper. Again one would like to see other factors considered, especially age and weight.

Thus there really is no good evidence that within a population the hypertensive individuals habitually take more salt than the normotensive ones. Anti-natriumism has various fall-back positions:

1) 'There is too much noise in the data'. It has to be conceded that there could be some truth in this.

2) 'There is too little variation in salt intake within a population'. This is manifestly not the case: sodium excretion can vary greatly, e.g. from below 70 mmol/day to 300 mmol/day or more.

3) 'There is a genetic variation in susceptibility to salt'. This is true of rats (Dahl et al., 1974) and could be true of man, though it seems strange that this should completely obscure the effect of variation in salt-eating habits.

4) 'Once intake gets above 60 mmol/day, it does not matter how much more is taken'. Freis (1976) puts this forward as a possibility, which it certainly is. This theory is not compatible with the
reductions in blood pressure claimed for more minor reductions in salt intake (Parijs, Joossens, van der Linden, Verstreken & Amery, 1973; Morgan et al., 1978) and it would mean there was little point in, say, halving the average salt intake as this would bring the average down only to about 90–100 mmol/day in many countries.

(5) 'The seeds of hypertension are sown in childhood and adolescence, so that salt intake in later life will not affect the blood pressure'. Again this theory would mean manipulation of salt intake in later life would be unprofitable.

Apart from point (2), none of these explanations is impossible and collectively they demonstrate the resilience and virtual indestructibility of the salt-hypertension hypothesis. Negative data can always be explained away.

Comparisons between populations with different salt-eating habits. These have been extensively reviewed by others (Meneely & Battarbee, 1976; Freis, 1976; Page, 1976) and will not be covered in any detail here. In general they are taken to support the thesis that hypertension is due to a high salt intake. However, even within these studies there are some awkward facts. (1) The life-style of the people who take very little salt (Bushmen, Eskimos and so on) is very different from that of people in the developed countries. (2) Whyte (1958) found in New Guinea that coastal people whose appetite for salt is satisfied did not have higher blood pressure than the people of the highlands who have a very low intake of salt. (3) Malhotra (1970) found that, among railway workers, hypertension was more common in people from South India whereas salt intake was higher in people from North India. (4) Examination of the data from the Solomon Islands provided by Page, Damon & Moellering (1974) indicates a much less clear-cut situation than has been suggested.

This study (Page et al., 1974) of six groups of people in the Solomon Islands is often quoted and Page (1976) has claimed that: (1) the three lowest groups in acculturation rank showed no rise in blood pressure with age whereas age-related changes were found in the three more acculturated groups; (2) 'blood pressure trends were best correlated with salt intake' and 'highest blood pressures were found at all ages and in both sexes among a group (third out of six in rank of acculturation) who boiled their vegetables in sea-water and had by far the highest salt intake of the six groups'. However, the situation does not really seem to be quite so simple. Regarding point (1) above, a perusal of the graph of blood pressure by age in the six groups leaves this reader, at least, singularly unimpressed by the difference between the unacculturated and acculturated. The acculturated groups may have shown a small rise in systolic pressure with age (women only, a point not always mentioned), but then the pressure of two of these groups was very low at ages 20–29 and 30–39 years, as low as in the lowest unacculturated group and lower than the two other unacculturated groups. Is it really worse that blood pressure should be low till the age of 40 and then rise slightly than that it should be at the higher level throughout life? Also two of the unacculturated groups, and only one of the acculturated, had no figures quoted for people over 69. Clearly a rise with age is more likely to be found if the old people are included. Regarding point (2), the group that boiled their vegetables in sea-water (the Lau) certainly had somewhat higher pressures but they evidently lived in rather cramped conditions on artificial islands and the men of this group were 10–19 lb (4.6–9 kg) heavier than the men of the other groups. These other factors should be given consideration equal to that given to sodium.

Neighbouring towns or villages in other countries have also been compared. A fishing village in Newfoundland (Fodor, Abbott & Rusted, 1973) has a higher prevalence of hypertension than a mountain village and a higher estimated salt intake (about 150 mmol/day compared with 122 mmol/day). But both these intakes are fairly low by western standards and the authors hint at many other environmental and sociological differences. They have also stated earlier (Abbott, Perks, Senciall & Rusted, 1971) that dietary factors could not explain the difference in blood pressure. In citing this work in a review article Page (1976) describes the Newfoundland fishing village as having an 'extremely high salt intake', but this does not appear to be correct.

Calabrese & Tuthill (1977) studied two neighbouring high schools in New York, one with 8 mg of sodium/l in drinking water and the other with 107 mg/l, and found higher pressures in the school with higher sodium. The difference in intake is small, about 8 mmol/day (based on an intake of 2 litres of fluid). Other factors such as race, age, body weight, social circumstances etc. need to be fully considered, but they are not properly discussed.

No review of salt intake and hypertension would be complete without a discussion of the situation in Japan, but it is by no means straightforward. On the one hand Hatano (1974) has pointed out that whereas the mean sodium intake in Japan (lowest
about 230 mmol, average perhaps 250–300 mmol/day; Sasaki, 1964) clearly lies well above the corresponding values for western countries, the Japanese people are not in general particularly admitted be a protective factor in this regard. On the other hand, there seems no doubt (Sasaki, 1964; Hatano, 1974) that salt intake, blood pressure and mortality rate from stroke are highest in the north-eastern parts of the main island. The salt intakes in these areas are startlingly high, averaging 350–450 mmol or more per day, though they are reported now to be less than before (Kimura, 1973), and it can be readily conceded that this sort of intake is undesirable. The question remains whether this fairly clear-cut conclusion that a very high sodium intake is bad can, and should, be extrapolated downwards to peoples with intakes of 170–200 mmol/day.

Salt-loading experiments. These have been done by several groups. Gros, Weller & Hoobler (1971) found no effect on blood pressure from the addition of 6 g of NaCl (102 mmol)/day to the diet for 2–9 weeks. Kirkendall, Connor, Abboud, Rastogi, Anderson & Fry (1976) studied eight normotensive subjects on sodium intakes of 10 mmol, 210 mmol and 410 mmol, each diet being given for 4 weeks, and found no change in blood pressure. The duration of these experiments was, of course, as the authors themselves admit, fairly short, so that the negative results do not necessarily prove anything.

Kawasaki, Delea, Bartter & Smith (1978) studied 18 hypertensive subjects on diets containing 109 mmol, 9 mmol and 249 mmol of sodium. Some subjects’ pressures rose markedly when they changed from the 9 mmol to the 249 mmol of sodium diet and the authors classified the group as a whole into two subgroups: salt-sensitive and non-salt-sensitive. The difference between these subgroups lay mainly in that the salt-sensitive subjects responded with a fall in blood pressure on the low sodium diet; there was no significant difference in pressures between their period on 109 mmol of sodium and the period on 249 mmol of sodium. So a very drastic reduction in salt intake would apparently be needed to make use of their ‘salt-sensitivity’. Again, admittedly, this was a short-term experiment.

Calorie restriction and salt restriction as a means of lowering blood pressure. Dahl, Silver & Christie (1958) were convinced that a reducing diet lowers blood pressure because of the concomitant reduction in salt intake. However, Reisin, Abel, Modan, Silverberg, Eliahou & Modan (1978) have shown in a much larger group of patients that loss of weight without salt restriction is effective in lowering pressure in obese individuals. The metabolic effects of weight reduction are, of course, complex and may include some loss of sodium (North, Lascelles & Coates, 1974); and unless special instructions are given (as was done by Reisin et al., 1978) to take salt-rich foods, people who reduce their calorie intake will almost inevitably reduce sodium intake also. So it is not entirely easy to separate these two factors.

The effects of moderate restriction of sodium intake have not been subjected to rigorous trial to any great extent. Some physicians are clearly personally convinced of its value. For instance, Hunt (1977), in a chapter in a major book on hypertension, strongly recommends reduction of sodium intake to below 75 mmol/day and indicates that a large percentage of patients who comply with this will achieve normotension. However, the sodium restriction is part of what he terms ‘nutritional therapy’, which also includes calorie restriction, and he gives insufficient detail of numbers, age, sex, weight and general methodology to enable the results to be properly assessed. No doubt many physicians give somewhat similar advice, though perhaps less enthusiastically and without being certain of the justification for it.

Parijs et al. (1973) found that a deliberate restriction of salt intake for 4 weeks, reducing sodium excretion from 220 to 93 mmol/day, caused a significant fall in home-recorded blood pressure in a group of patients with mild hypertension. What their data show, however, and what they do not discuss, is that the blood pressures recorded at the clinic did not fall significantly on the low salt regimen, and in fact the mean diastolic pressure rose slightly. It is not clear why the home-recorded blood pressures should be considered more significant than the clinic recordings.

Morgan et al. (1975) reported on 12 hypertensive men who reduced their sodium intake from 205 mmol/day to less than 120 mmol/day and who had a significant fall in mean blood pressure compared with a placebo period. Most of them were on a β-adrenoreceptor-blocker or other antihypertensive drug and one could say that the sodium restriction had not necessarily had an independent effect on pressure but had rather made the patients more responsive to the blocking agents. (Though such an effect would, of course, in any case be useful.) In a later study (Morgan et al., 1978), 31 men out of a larger group with mild hypertension were allocated to treatment by
sodium restriction while others had drug treatment or placebo. Sodium intake (as measured by output) fell only from 191 mmol/day to 156 mmol/day but there was a significant fall in blood pressure compared with the placebo-treated group. Some patients evidently hardly lowered their sodium intakes at all and it is a weak point in the evidence or placebo. Sodium intake (as measured by output) there was a significant fall in blood pressure compared with the placebo-treated group. Some patients evidently hardly lowered their sodium intake and fall in blood pressure. No information is given on change in weight. Although interesting, these results from Morgan's group cannot be said to be fully convincing to the sceptic. No doubt further results will be forthcoming and will be awaited with interest. There is no doubt also that similar studies should be done in many countries so that this very important question can be decided once and for all.

Conclusions

1. We certainly should aim to prevent hypertension.
2. We can all agreee we nearly all eat more salt than is necessary, and that addition of salt to baby foods is pointless.
3. Those nations or population groups with exceedingly high sodium intakes (e.g. 250–500 mmol/day) would probably do well to reduce these.
4. However, the data remain unconvincing for countries with a sodium intake (for men) of 170–200 mmol/day.
5. In well-to-do western countries, at least, weight reduction looks much more promising as a preventive measure though in practice it may be difficult to achieve.
6. More studies of the effect on blood pressure of moderate sodium restriction should be done.

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References


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**DISCUSSION**

Langford: We got some lovely quotations from Professor Simpson in his good Scots accent. I would remind him in this context of what Cromwell said to the Scots covenanters, 'Gentlemen, I beseech you, in the bowels of Christ, think it possible you may be mistaken'. I think this is essentially Simpson's message also. Dr Morgan talked about Dahl, who estimated, in his patients, salt intake by means of the dietary history, finding a large percentage of hypertensives among those who took a lot of salt. I read Dahl's paper carefully and I found that those who reported a high salt intake had the same mean blood pressure as those that did not add salt. Therefore I concluded that obtaining from patients a history of adding salt can pick out hypotensives as well as hypertensives. The standard deviation of the blood pressure of that group who said they added salt must have been considerable. I have a second point, reminding you that when we assess salt taste threshold we are doing nothing more than estimating salivary sodium content. The tongue does adapt very rapidly to the ambient concentration of salivary sodium, as Dr Bartoshok of Yale showed years ago. Dr Marino, in collaboration with me, showed recently that there is a nice linear regression of salivary sodium on salt taste threshold. For this reason you can probably get a rough measure of somebody's aldosterone secretion rate by measuring their salivary sodium or their salt taste threshold as well as by putting a probe up
their rectum. Lastly, I would play the little violin that I practise on regularly; Watson and I can find a correlation between sodium/potassium ratio and blood pressure although we fail to find a correlation with sodium, no matter how we fiddle the latter. If we do a stepwise regression we can account for 6% of the variation by the sodium/potassium ratio. As Dr Simpson mentioned, obesity was much more important and we can account for 15% and 18% of the variance by obesity.

Robertson: It is gratifying to find Dr Langford so well informed on Scottish history.

Tobian: One of the things that confounds this whole question in human hypertension is that there are only some people who are susceptible to salt. I think the original Dahl working hypothesis, that genetics plays a role, is still valid. Dahl suggested that only about one person in eight would respond to a high salt intake by becoming hypertensive. Thus, there are going to be a lot of negative sodium/blood pressure correlations, just because seven out of the eight individuals are not going to be affected very much by the usual 170–200 mmol/day intake of sodium. Such individuals might be affected by 800 mmol/day, as we have seen from Dr Luft’s study in Indianapolis. Bearing that reservation in mind, I do think that there is a lot of evidence relating salt to essential hypertension. Dr Morgan mentioned many of these things. There must now be 20 or 25 studies of communities on a life-long low-salt intake, of less than 50 mmol/day. These people do not develop essential hypertension nor do they have a rise in blood pressure with advancing age. When such people move to an environment where they begin to eat more sodium, they begin to develop more hypertension. You cannot explain this original lack of hypertension by the fact that they are lean, because one can find very lean populations that eat a fair amount of salt, and they get their share of hypertension. I do not think these studies can be laughed off; you can perhaps laugh at the first three or four, but it is hard to laugh off 25 such studies. We all know if we just take ordinary people with essential hypertension and put them on a very low sodium intake or given them diuretics, we reduce total body sodium and lower blood pressure. The effect of diuretics can be drowned out by a high salt intake. We also know that a very high salt intake definitely can cause hypertension. In the study in Indianapolis, 800 mmol of sodium chloride/day was seen to raise the blood pressure even in normal individuals. There is a huge amount of animal work to support the human studies showing that high salt diets tend to raise blood pressure and low salt diets to lower it. In the middle ranges of salt intake, correlations may not be so obvious in a population where everyone eats more or less the same amount of salt. The real comparison should be between people who eat 50–60 mmol/day against people who eat 170–200 mmol/day. There is nothing so sacred about a salt intake of 200 mmol/day. Probably every man living two million years ago ate about 40–50 mmol of sodium/day. Early man had a salt intake that was very low. I do not think we need necessarily bring the question of obesity into it either. Certainly obesity affects blood pressure. Salt intake also affects blood pressure. These are probably independent factors and there is no point in using the effect of obesity on blood pressure to deny the effect of salt.

Simpson: First of all, Dr Tobian, I think that the 800 mmol of sodium/day is irrelevant, because that is not what we are talking about. The question really posed is whether any movement of sodium intake over the range 100–200 mmol/day is worthwhile, and I do not know the answer. You say you cannot laugh off 20 or 25 investigations, but there is a sort of cumulative effect of this kind of argument. I think one has to be careful that we are not getting the positive results reported and the negative ones ignored. Many of these studies are being analysed purely in terms of sodium when heaven knows there are a lot of other differences in all these populations, especially when they are living in primitive conditions. I am not convinced that all the other variables have been excluded. Obesity does, I think, have to come into the picture. If you reduce food intake, you also usually reduce sodium intake. The value of the work of Reisin and his group in Israel was that these two things were separated.

Pickering: Mr Chairman, there are really three questions. First, do people with higher pressures in a population eat more salt? That has been answered by Dr Simpson. May I add to the list he presented a paper by Ledingham and his colleagues who made a survey of workers at Oxford and Harwell (Kidney International, 1978, 13, 513). It was shown that the people with the highest blood pressure excreted almost exactly the same amount of sodium per day as those with the lowest pressure. The second question is, can you reduce
arterial pressure by reducing salt intake? There is no doubt about that, you can, but you will have to reduce it an awful long way. This became an issue of great importance in 1946 when Kempner was the first to show that you could reverse the malignant phase of hypertension by a rice/fruit diet. Subsequently a lot of people failed to confirm these findings. I was one of them, and the reason why there were these failures was because patients would not eat the diet. It was shown that the beneficial effect of this diet depended entirely on the sodium content. You have to get the sodium down to something of the order of 5 mmol/day, certainly below 10 mmol/day, if you are going to get any effect. Many studies, including those of the British Medical Research Council and the very careful ones done at Columbia, New York, showed if you added 1 g of salt a day the blood pressure went back to where it started from. So it is an extreme measure and no patient can tolerate it for very long. My patients failed to stick to it while Kempner’s patients succeeded. I went to visit Kempner to see why. I knew that he believed in it but I did not realize that he was a Prussian and also a bully and that he regimented his patients. The first thing he did when we went to the hospital each day was to test the patients’ urines to see if they had any chloride present, and if they had he gave them hell for not sticking to their diets. Volhard was just the same and he used to catechize his patients; they always said when questioned that they had been keeping to their diet. Their colleagues knew that this was untrue, so that there was an expression in Volhard’s clinic ‘lying like a saltless’. This low salt diet is really terrible. Now the third question is, will adding salt put the blood pressure up? Certainly it would; but you do not really get any effect until the intake is above 600 mmol/day. This effect of very high sodium intake was shown in a recent study, already mentioned, from Indianapolis by Luft and his colleagues. Normal subjects were given diets of increasing salt content from 10 to 1500 mmol/day. It was only above 800 mmol/day, however, that any increase in blood pressure was seen. The work of Dahl has already been extensively quoted, and I think that Dr Morgan ought to read Dahl’s paper again, because what he showed was that the people who allegedly had a high salt intake had exactly the same mean blood pressure as those with the low salt intake, but he thought on the family histories that one lot ought to have more hypertension than the other. If that was true they also had more hypotension. Dr Morgan also showed the famous (or infamous) slide of Dahl purporting to show how hypertension in populations is dependent on the amount of salt in the diet. The supposed daily salt intake was proportional to the percentage of hypertensives in a population and nearly all the points lay absolutely on the line; there were just two or three points that were a little out. But how on earth do you measure the percentage of hypertension in the population when you do not know what is the dividing line between ‘hypertension’ and ‘normal blood pressure’? Dahl did not state what that dividing line was, nor did he give age ranges of the subjects studied. How do you estimate the daily salt intake in the United States of America, or in Japan, or in Eskimos? In the population sample we studied at St Mary’s Hospital, London (Clinical Science, 1954, 13, 11), we plotted frequency distribution curves for blood pressure for various age groups. If, for example, in females, we took 150/100 mmHg as the dividing line between normal and ‘hypertension’, some interesting things appeared. If you wanted to get a 50% split between normals and hypertension, you should choose the age range 50–59 years and there it was. You can get this in any population. If you wanted to get no incidence of hypertension, you should choose the age range 20–29 years and you would get none. If you wanted to get 75% of the population who were hypertensive, you could choose the age range 70–79 years. In other words, if you do not know the age of the subjects you are studying and you do not know the arbitrary dividing line separating ‘hypertension’ from normal, the data are quite valueless. The distributions of daily salt excretion in the subjects studied in Framingham showed quite a wide range and, as Dr Simpson said, the mean blood pressures were identical, whatever the salt intake. I think that within the range of salt intake that all of us are accustomed to, and in people with good renal function, the evidence that salt affects blood pressure is non-existent.

Robertson: Dr Morgan says that he would like to reply later in the discussion, so I shall ask Dr Brod to speak next.

Brod: Dr Simpson, I, like the other discussants, admired your literary quotations, but I think that you did not take the quotation of Francis Bacon quite to heart when you said that the excretion of sodium in normotensives and hypertensives is absolutely equal. I would suggest it is equal because in hypertensives the blood pressure is raised. If you lower the blood pressure, for
instance, by vasodilator agents, without affecting in some way the function of the kidney, then immediately the sodium excretion will drop and the patients will retain sodium; they may even become oedematous. I also suggest that there is an explanation for the importance of weight. There is an old paper by Rytand from the 1930s which showed that there was a correlation between the logarithm of the weight of the organism and the logarithm of the glomerular size. I suggest that where the body size exceeds the accepted limits, the kidney is probably too small for the requirements of volume homeostasis. In other words, it is analogous with a kidney with either a clip or renal disease. My third brief comment is on what Sir George Pickering said just now on Kempner’s rice diet. 33 years ago I worked with Homer Smith, and I was involved in the study of the effects of Kempner’s diet. I can confirm that this diet was absolutely unpalatable to practically all patients. Only those patients who were masochists could stand such a diet for more than one week.

Simpson: People are, of course, usually in equilibrium and I think that the first point you make, Professor Brod, is irrelevant. I do not think that when blood pressure changes there will be more than a transient change in sodium excretion. The other point that you made about kidney size does interest me quite a lot and I think probably we should be looking at sodium excretion per unit height. If we do this we would be relating salt to the size of the person and therefore presumably the size of his kidneys. When we do this with our data, the correlation with blood pressure does improve very slightly. I agree that we should, when looking at different populations, examine sodium output per unit height.

Hood: I concur that the critical remarks that have been made, amongst others by Wilhelmsen and Simpson, are entirely true. At the same time, Tobian speaks for individuals and is also correct. For almost exactly the same 24 h urinary sodium output, the handling of sodium is, of course, very different for different individuals. How should we go along with this? We might all subscribe to the idea of Simpson of not adding salt to baby food. I do not take the arguments of Sir George Pickering very much to heart, although I agree that what Kempner did was to give a diet that nobody could eat, apart from those people who had malignant hypertension and then only for a few weeks. You can easily try the effect of sodium intake on blood pressure in a third-generation hypertensive subject. You do not need to be a bully; just let him take care of himself and he will bring down his diastolic pressure by about 25 mmHg of he is originally on a 500 mmol of sodium/day intake and ends up at 100 mmol/day. How should we go about an experiment of this type? I recommend that we take third-generation hypertensive subjects who are at the upper range of blood pressure for their age, randomly allocate them to high and low sodium diets, record the blood pressure by a system which is free from bias, and look for four sets of markers within these individuals to see whether we can pick up those who will eventually respond by decreasing their blood pressure, or at the very least not continuing to increase it. These markers are: the entry of $^{22}$Na into red cells; the whole-body turnover of radioactive sodium; the efflux of labelled noradrenaline from noradrenaline-loaded platelets at different sodium levels in the medium; and plasma volume. All of this will take a long time. In the meantime I agree with the cautious attitude of not transferring entire populations on to a low sodium diet.

Joossens: I have to correct some statements made by Dr Simpson. In our data there was a significant correlation between systolic blood pressure and salt intake, and this was independent of weight, height, pulse rate, urinary creatinine and age for males. For females it was only so when age was omitted as a variable. Males were, however, the largest group: 1314 males as against 713 females. The persons used in this study were all inhabitants of Leuven and the surrounding area and included active and elderly people, all living in their own homes, just as was the case in the Dunedin (Milton) study. Therefore ours was a perfectly homogeneous group. The lower creatinine excretion in the elderly is not surprising by itself; it is one of the reasons for including urinary creatinine as an independent variable. The home readings of blood pressure in the Leuven study were used (not selected) because each person had several blood pressure measurements, the mean of which was used as the blood pressure of that person in a given period. This is, of course, to be preferred to one casual blood pressure reading. It should be noted that the validity, reproducibility and consistency of home readings was tested in a separate experiment with 12 000 home blood pressure readings in 300 persons. The important point of the Leuven study was that a moderate reduction in salt intake from 12 to about 6 g of sodium chloride/day produced a significant
TABLE 2: Salt intake and hypertension

Table 1. North of Belgium: salt excretion in 24 h urine per 1-77 g of creatinine (active population 18–65 years)

Results are from 5535 24 h samples in 2493 persons.

<table>
<thead>
<tr>
<th>Year</th>
<th>No. of persons</th>
<th>No. of samples</th>
<th>Mean NaCl (g/day)</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1966</td>
<td>216</td>
<td>216</td>
<td>15.2</td>
<td>5.3</td>
</tr>
<tr>
<td>1967–69</td>
<td>1538</td>
<td>1538</td>
<td>11.9</td>
<td>4.4</td>
</tr>
<tr>
<td>1970–74</td>
<td>404</td>
<td>3446</td>
<td>11.5</td>
<td>2.9</td>
</tr>
<tr>
<td>1975</td>
<td>65</td>
<td>65</td>
<td>10.3</td>
<td>4.3</td>
</tr>
<tr>
<td>1978</td>
<td>52</td>
<td>52</td>
<td>7.9</td>
<td>3.7</td>
</tr>
<tr>
<td>1978–79</td>
<td>218</td>
<td>218</td>
<td>8.4</td>
<td>3.4</td>
</tr>
</tbody>
</table>

The decrease in blood pressure, independent of and additive to the influence of diuretics.

Table 1 presents data on salt excretion measured in the population from 1966 on. I want to remind you that my interest in salt came from an accidental and unexpected finding in 1964, namely that stroke mortality is strongly related to stomach cancer between countries and that this correlation is much stronger than, for example, that between coronary mortality and lung cancer from the same countries. In 1965 the hypothesis was formulated that the linking factor could be salt intake. We therefore started collecting data on salt excretion from 1966 on. The latest surveys (1978–79), one performed on 218 persons in Ostende by H. Kesteloot and another on 52 people working in a factory in Leuven by A. Amery, showed nearly identical values. Both were about half the value found in 1966. Up to now no data are available on blood pressure distributions in Belgium over the years, but we shall have them in the near future. It is possible, however, to look at stroke and stomach cancer mortality as indicators of blood pressures in the population. From 1968 to 1977 a 34.3% decrease in stroke was observed in Belgium for the average of both sexes in the 45–75+ years group (age-adjusted). There were only two countries in 30 populations examined (western, west-European countries and Japan) with a greater decrease, namely Finland (61.7%) and Japan (37.5%). For stomach cancer the decrease over the same time period was second highest of all 30 examined countries, namely 46.9%. Again Finland was highest with 54.4%. Of course, this is no formal proof of a relation between blood pressure and salt, but it is consistent with it, making it worthwhile going on in this direction. Most people will think that lowering salt intake in the population is difficult. In fact it is not. It should be realized that salt excretion is decreasing, not only in Japan and Belgium where it has been measured over time, but it is also highly probable that this is the case all over the western world. This reduction is probably due to the use of freezers and refrigerators and to a lower intake of bread, potatoes and other cereals, lard, salted meat, fish and vegetables and a higher intake of fresh fruits and fresh vegetables. The resulting decrease is not compensated by the use of salted beans, chips and so on. From the consumption of bread and potatoes alone a salt intake of about 17 g/day can be estimated for Belgium in 1920. Remember that the mean total intake in our country is now about 8 g of sodium chloride/day and that some of the samples from factory workers obtained by A. Amery in 1978 contained less than 2 g of sodium chloride/day. Taste is never a problem in salt reduction, except sometimes in the elderly with totally destroyed taste buds. Of course, it should be done very gradually. Since one never tastes salt as such in the food (it is only tasted when there is less or more salt present than you are used to), it can be lowered gradually and eventually eliminated totally without the subject being conscious of it. This brings me to a point raised by Dr Simpson when he compared taste studies in normotensives and hypertensives. One of those studies was performed in children, with negative results. Taste studies should preferably be performed in adults or, even better, in elderly people. Both taste-bud destruction and high blood pressure are most evident in the elderly.

Robertson: I think that there was one other point that Dr Simpson raised, Dr Joossens, and this was the question whether in the Leuven study there was any weight loss, and whether any of the blood pressure fall could be related to that?

Joossens: There was a weight loss.
**Round Table 2: Salt intake and hypertension**

**Henningsen:** I hope that Dr Simpson will not argue against the information we have obtained in several studies which are still proceeding, and some of which have been presented at this meeting. We believe we have identified part of the population which is salt-sensitive. The first-degree relatives of patients with proved essential hypertension show an abnormal sodium metabolism which is related to their blood pressure. We can also show in these people that if we lower their sodium intake we can lower their blood pressure and postpone the onset of eventual essential hypertension. Coming back to what is called 'noise' in population studies; I agree very much about this. I think that excess weight is optimistic, but I hope that within a year we shall have more definitive answers.

**Simpson:** I should be delighted, Dr Henningsen, if you can convince me about sodium. Frankly, I would rather be arguing on the other side, but the facts as I see them at present require me to be prudently sceptical.

**Henningsen:** The preliminary results are quite optimistic, but I hope that within a year we shall have more definitive answers.

**Beevers:** We have studied 24 h sodium excretion in hypertensives and normotensives in Renfrew, Scotland, and we can confirm that whilst men excrete larger quantities of sodium than women, this trend is no longer seen after correction for urine creatinine excretion. Our hypertensives, however, excreted less, not more, sodium than the normotensive controls (hypertensive men and women 182 mmol/day and 144 mmol/day respectively; normotensive men and women 196 mmol/day and 173 mmol/day). We can confirm that in this area, where hypertension is very common, large salt excretions are found, but within this single society there is little evidence to support the salt hypothesis. However, amongst our hypertensive men, urine sodium was positively correlated with diastolic blood pressure \( r = 0.3229, P < 0.05 \), although this trend was absent amongst the women.

**Robertson:** Can you please tell us what you took as your definition of 'hypertension'?

**Beevers:** 'Hypertension' was a diastolic pressure (fifth phase) of at least 105 mmHg. 'Normals' were required to have a diastolic under 90 mmHg.

**Malliani:** I should like to say something which is perhaps fairly obvious for this Society, but it needs to be emphasized at this point of the discussion. Blood pressure varies continuously and considerably in normal people and also in 'hypertensive' subjects. Probably many complications, such as sudden death, angina, myocardial infarction and stroke are related to these fluctuations. Now it appears to me that these well-known facts are being ignored by the present rather static concept which seems to appreciate only physiological and pathological homeostasis. In the complexity of real life it is quite difficult to pick up only one variable and to infer the dependency of this variable on any single factor. This was apparent, for example, in the slide which was shown earlier in which blood pressures were found to be higher in people who joined the Army. This was attributed by the speaker to a higher sodium intake. It seems to me, however, that other things may change when a man joins the Army and that these things might well put up his blood pressure, independently of sodium intake. Francis Bacon, the philosopher, was mentioned as an adviser against methodological errors. I could perhaps indicate Francis Bacon, the modern painter, as a good interpreter of the complexity of reality.

**Berglund:** I have a short comment on the points made by Dr Simpson and also Dr Beevers about the finding of lower sodium excretion in the hypertensive population than within the normal population. This may very well be because the relationship between blood pressure and salt intake is not linear, but curvilinear, with an increase in the normotensive part of our population and a decrease in hypertensives. If we just compare their mean values obtained in the extremes, we might end up with some false interpretations. Could I ask the panel if they feel that we should be making recommendations to decrease salt intake in the community? In the United States of America a senate committee has agreed to recommend no more than 3 g of sodium chloride as dietary intake per day and in Belgium also firm recommendations have been made regarding a low salt intake. I personally do not think that today we know enough to be able to recommend salt restriction within the total population, but I would like to hear the comment of the panel on this. In people with mild hypertension
it is possible that salt restriction might be a safer way of lowering blood pressure than administering drugs. However, to recommend restriction of salt in a whole population is quite another matter and I think we need a large-scale controlled trial before we are confident enough to advise anything of that sort.

Fodor: In an area in Newfoundland with a high prevalence of hypertension we found almost exactly the same as has been reported here several times; namely, a lower intake of sodium in hypertensives than in normals. However, I can perhaps remind you of a paper by Abramson and his colleagues from Harvard in 1948. They showed that spontaneously hypertensive rats when given a choice between sodium-low and sodium-rich drink, took the sodium-low drink. Maybe similar processes are at work in human hypertensives. On the other hand, when we look at that part of the population in which the normal blood pressure values are distributed, we find the lowest arterial pressures in those people whose sodium intake is below 60 mmol/day. Thus the presence, in a population study, of some people with hypertension, whose kidneys may already have a restricted ability to excrete sodium, may confuse the situation. I do not think that the finding of a decreased sodium intake in hypertensives can be invoked as an argument against the salt hypothesis. In my opinion, a high salt intake is the most likely aetiological culprit in hypertension. In Newfoundland there is also an interesting correlation between cerebral strokes and stomach cancer; both are more frequent than in other parts of North America.

Tuomilehto: I believe we have omitted some very important issues from this discussion so far. As Dr Wilhelmson told us at the start, the main aim of blood pressure control should be to prevent complications amongst people, some of whom may not have very high blood pressures. We are, therefore, really speaking about prevention. We should no longer, when discussing blood pressure, divide people arbitrarily into groups such as 'hypertensives' and 'normotensives'. The use of such arbitrary subdivisions may in some of the discussions here already have obscured the truth. When we make so-called 'cross-sectional' studies we also lose much information. We know that blood pressure increases with age usually and that there are many associated factors. If we make a measurement at one point and try to find correlations, then we are severely limiting our ability to discover something important. In future I think we should follow populations over several years. In such studies we might examine the possibility that different levels of sodium consumption have an effect on the blood pressure and we could examine the way in which this changes over the years. Ideally we should start these surveys in childhood and follow them through into adult life.

Woods: Reference has been made to the interesting association between stroke and cancer in the stomach and it would be of interest to know whether the suggested high intake of sodium, which may be the common factor here, is in the form of sodium nitrite, which is, of course, widely used as a preservative for meats and fish. There is evidence that nitrite in the diet is converted into nitramides and this has been implicated as being a causative factor in carcinoma of the stomach. If the various communities mentioned have a high sodium nitrite intake, this might explain at least partly the relationship between two rather different diseases.

Joossens: It is not widely known that stroke mortality has been coming down in the U.S. for more than 50 years. Fig. 1 shows data from Acheson (1966). Acheson himself did not believe his own data and thought them due to improving classification. However, it has been our experience that better classification always reveals increasing stroke rates and never the contrary. In Fig. 1 one can see that the change from the fifth to the sixth revision produced an increase and not a decrease in recorded stroke mortality. The decrease in stroke mortality before any anti-hypertensive treatment was available has been documented in Baltimore since 1940 by Miller & Kuller (1973). More recently Garraway and colleagues (New England Journal of Medicine, 1979, 300, 449) showed similar results in Rochester, Minnesota, again from 1940 on. So there can be no doubt that stroke was decreasing without antihypertensive drugs. Now the important point is that this behaviour is always paralleled by that of stomach cancer. The decrease of stomach cancer in the U.S.A. was documented since 1930 on, in both sexes and at all age groups. Except for the U.S.A. no early data on stroke mortality are available. In other countries I have only data since about 1950. In data from 12 countries with the best available mortality statistics a decrease in stroke mortality is found for all of them. In fact between 1950 and 1960 the decrease
is somewhat faster than between 1960 and 1973. From 1973 on a new phenomenon becomes visible in certain countries (U.S.A., England and Wales, Finland, Norway and Germany), namely a sudden sharp decline in stroke death rates. Looking at stomach cancer a similar behaviour is noted in the same countries from 1950 to 1973; the only difference is that the sharp decrease, from 1973 on, is not seen. Two facts must be considered. (1) Even rudimentary treatment of hypertension only became possible at the end of the nineteen-forties, with diuretics becoming available around 1958 and β-adrenoreceptor-blockers around 1965. (2) Treatment of hypertension, although available, was used only in selected individuals, not at the population level, at least until very recently. Therefore we cannot easily explain the decreasing stroke rates in this way. The similar behaviour of stroke and stomach cancer may provide a clue to the solution of the problem. Stomach cancer is strongly correlated with stroke within countries and between countries. The quantitative relationship is practically identical under a set of very different conditions (Table 2), making a spurious relationship extremely unlikely.

Although there are many factors influencing separately stroke and cancer mortality in a given population, it is evident that the linking factor between both must be either unique or at least predominant. A multifactorial link cannot provide a similar quantitative relationship as seen in Table 2. The linking factor X must be present in the food, have an influence on blood pressure (the most important factor for stroke mortality), produce a change in the stomach mucosa, decrease over time and be present in high amounts in Japan, Portugal and most east-European countries. From what is known from the epidemiology of stomach cancer factor X must be present in smoked food (Iceland), lard (Netherland), soy sauce or pickled foods (Japan), cereals (U.S.A.) and nearly absent in fresh fruits, fresh vegetables and milk. Salt has all those characteristics. Salt could influence stroke through blood pressure and on the other hand it could destroy the stomach mucosa osmotically (salt is osmotically the strongest factor from all known food constituents), just as it destroys taste buds. Because there are osmoreceptors at the entry of the duodenum, an hypertonic meal stays in the stomach for hours. This could lead to atrophic gastritis, indeed a common feature in Japan. Recent research (see Lancet, 1978, i, 1355) has shown that nitrite concentration in the stomach is inversely related to the acidity of the stomach content. In the absence of HCl, as in atrophic gastritis, high levels of nitrite can combine with...
### TABLE 2. Regression equation between stroke mortality (y) (age adjusted 45–75+ years) and stomach cancer (x) (age adjusted 45–64 years)
(Males + females)/2.

<table>
<thead>
<tr>
<th>Origin of data</th>
<th>Death rates (%)</th>
<th>Regression equation</th>
<th>n</th>
<th>r</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average 12 countries for 21 years (1955–1975)</td>
<td>y = 2.23 + 4.79x</td>
<td>12</td>
<td>0.67</td>
<td>&lt;0.02</td>
<td></td>
</tr>
<tr>
<td>Average of 12 countries per year (1955–1975)</td>
<td>y = 2.24 + 4.74x</td>
<td>21</td>
<td>0.97</td>
<td>&lt;10^-9</td>
<td></td>
</tr>
<tr>
<td>24 countries in 1974</td>
<td>y = 1.87 + 6.53x</td>
<td>24</td>
<td>0.81</td>
<td>&lt;0.00002</td>
<td></td>
</tr>
<tr>
<td>England and Wales (1955–1975)</td>
<td>y = 2.26 + 5.16x</td>
<td>21</td>
<td>0.96</td>
<td>&lt;10^-9</td>
<td></td>
</tr>
<tr>
<td>England and Wales (average 1966–1974) standard regions</td>
<td>y = 1.89 + 6.97x</td>
<td>9</td>
<td>0.92</td>
<td>&lt;0.0005</td>
<td></td>
</tr>
<tr>
<td>Japan (1962–1975)</td>
<td>y = 1.18 + 6.15x</td>
<td>14</td>
<td>0.98</td>
<td>&lt;10^-9</td>
<td></td>
</tr>
<tr>
<td>U.S.A. (1955–1973)</td>
<td>y = 2.18 + 7.65x</td>
<td>19</td>
<td>0.97</td>
<td>&lt;10^-9</td>
<td></td>
</tr>
<tr>
<td>The Netherlands (1955–1977)</td>
<td>y = 1.40 + 5.01x</td>
<td>23</td>
<td>0.96</td>
<td>&lt;10^-9</td>
<td></td>
</tr>
<tr>
<td>Belgium (1968–1976)</td>
<td>y = 2.33 + 5.64x</td>
<td>9</td>
<td>0.84</td>
<td>&lt;0.005</td>
<td></td>
</tr>
<tr>
<td>Sweden (1951–1976)</td>
<td>y = 1.14 + 6.68x</td>
<td>26</td>
<td>0.97</td>
<td>&lt;10^-9</td>
<td></td>
</tr>
<tr>
<td>Norway (1960–1973)</td>
<td>y = 2.61 + 3.48x</td>
<td>14</td>
<td>0.94</td>
<td>&lt;10^-4</td>
<td></td>
</tr>
<tr>
<td>Denmark (1951–1976)</td>
<td>y = 1.40 + 6.22x</td>
<td>26</td>
<td>0.94</td>
<td>&lt;10^-9</td>
<td></td>
</tr>
<tr>
<td>Italy (1958–1974)</td>
<td>y = 2.07 + 4.26x</td>
<td>17</td>
<td>0.96</td>
<td>&lt;10^-8</td>
<td></td>
</tr>
<tr>
<td>W. Germany (1956–1971)</td>
<td>y = 2.68 + 4.49x</td>
<td>16</td>
<td>0.98</td>
<td>&lt;10^-9</td>
<td></td>
</tr>
<tr>
<td>Canada (1955–1974)</td>
<td>y = 1.31 + 7.54x</td>
<td>20</td>
<td>0.95</td>
<td>&lt;10^-8</td>
<td></td>
</tr>
</tbody>
</table>

**Fig. 2.** Between-countries relationship between death rates (%) for stroke and stomach cancer in 1974.

Food amines to nitrosamides and this could lead to stomach cancer. I do not think nitrate intake is important since the amount of Na linked to it is too small to influence blood pressure. In Fig. 2 the relation between stomach cancer and stroke is shown for 24 countries in 1974. Japan, Bulgaria and Portugal have documented high salt intakes (18–30 g/day). The U.S.A. with 6–10 g/day and Belgium with 11 g/day also fit into the picture. New Zealand with 10−6 g of sodium chloride/1.77 g of creatinine in 1975 (mentioned by Dr Simpson), stroke rates of 3.5% and stomach cancer of 0.16% is near to the calculated regression line. Practically all the evidence accumulated since 1965, when the salt–stroke–stomach cancer hypothesis was first presented, has been consistent with it. A few conclusions can be drawn for the stroke–stomach cancer relationship. First, because of the identical linear relation between countries up to 1973 it is not possible to ascribe the fall in stroke to treatment of hypertension (see New England Journal of Medicine, 1979, 300, 1396). Treating hypertension cannot cure stomach cancer. Secondly, in a few countries (mentioned above) a marked deviation from linearity is seen since 1973. This is the first available evidence pointing to the success of treating hypertension at the population level.

Thirdly, the possible linking factor between stroke and stomach cancer must be studied more intensively. Therefore epidemiological studies on salt excretion should be performed in as many countries as possible and be followed over time. In certain countries like England and Wales where documented differences exist between, for example, north and south in terms of stroke and stomach cancer mortality, regional salt excretion should be studied.

**Maxwell:** I would like to criticize some of the points made about weight and blood pressure. I really think that all we can say is that there does appear to be some association between body habitus and blood pressure. There also seems to be
some lowering of blood pressure concomitant with weight reduction, but the fall in pressure is very slight. Even Dr Tyroler at Evans County showed only a few mmHg reduction of blood pressure in this way. I do not think that anyone has followed these patients into realimentation. Sodium has to be considered in this context; for example, I do not think that the article mentioned by Reisin and his colleagues from Israel fully excluded the possibility of sodium changes. We have organized a very large obesity clinic at the University of California in Los Angeles for the purpose of studying the relationship of blood pressure to body habitus. We have been particularly interested in what happens during rapid weight reduction. We now have several hundred patients, some of whom have lost up to 150 lb. These people show a marked early diuresis during the first week of weight reduction on a very low calorie diet. Some 80% of the blood pressure fall during the active phase of weight loss occurs during this first week. Furthermore, obese people eat a lot more salt than non-obese people, just as they eat a lot more sugar and a lot more calories and a lot more of everything else. I do not think that there is very good evidence that weight reduction, long-term, particularly when patients are followed into realimentation, when they again eat more salt, actually reduces blood pressure. I suspect that the blood pressure reduction during weight loss may well be associated with a lowering of sodium intake.

Weber: I really do not believe that there is a causal connection between stomach cancer and stroke. I do not think that these diseases share the same mechanism. If those splendid correlations shown by Dr Joossens have any meaning, then we should, when we see a lot of hypertensive patients, see a lot of individuals with cancer of the stomach. Conversely, when we see a lot of patients with cancer of the stomach we should also see a lot of patients with hypertension. In my experience neither of these circumstances is true.

Joossens: I agree with that. I have looked at blood pressure in patients with stomach cancer and found it to be lower than in patients with lung cancer. But you must not forget that at that stage the patient with stomach cancer is necessarily taking a low-sodium diet, because he cannot eat.

Unidentified voice: Is it not possible that the stroke that the patients with the stomach cancer have, is not at all the same as the stroke that people with hypertension get? The poor man with stomach cancer has just got a secondary in his head.

Robertson: I shall now ask the three panelists to reply to the various points that have been made in the discussion. I shall ask them to speak in the sequence in which they performed initially. Dr Wilhelmsen, therefore, will speak first. Perhaps I might also ask you to say, Dr Wilhelmsen, in reply to the question posed by your colleague, Dr Berglund, whether you want any legislative recommendation made by this Society?

Wilhelmsen: Mr Chairman, I should like perhaps just to repeat a few things which I believe are important with respect to blood pressure in the community. We need to do two things: to prevent hypertension occurring; and, if it has occurred, to prevent it increasing. I think nearly all of us are agreed that the distribution of blood pressures in the community is continuous. The important thing here is that there are really, so far as we know at present, very few measures which we might implement and therefore, to me, salt restriction is important. It is almost the only thing we can do apart from administering drugs. Drug therapy is not a realistic proposition in the vast majority of those people who have only slight blood pressure elevation. I might also add that we should consider the history of salt intake over the whole life of these people who have blood pressure at the upper end of the distribution curve. We have discussed today several studies of the ‘cross-sectional’ type. However, in epidemiology we always like to have prospective studies of any risk factors. Ideally, therefore, what we should like to do would be to identify young people, perhaps as early as 5 or 10 years of age, and follow them, looking at their salt intake, over several years and see what happens to their blood pressure. I think that if anyone could perform such a study it would provide a great deal of very valuable information. But epidemiology is much more complicated than merely looking at populations and correlating data from various studies, nearly all of which have a lot of what has here been called ‘noise’ in them. I think that many of the things that have been said here today will be shown to have been rather inaccurate in 5 or 10 years’ time. If, therefore, we make very firm recommendations, the public will come back to us and say that they will take no notice of what we recommend since they do not believe in us. We have already seen this happen for certain other proposals and I think, therefore, that we should be
careful to obtain very firm data and only advise the public when we are sure of our facts. I personally think that Dr Hood made some very interesting comments and studies like these are exactly what we shall have to do.

Robertson: Dr Morgan, you have been storing several ripostes for the end.

Morgan: Several points were directed to me during the course of this discussion. However, I will try to be brief. It is true that in the report by Dahl, the mean diastolic blood pressure did not rise. However, the number of people who showed a diastolic greater than 90 mmHg did increase. If the Framingham study is analysed properly, the data reveal a correlation between salt intake and blood pressure. Most people have agreed that a sodium intake of 800 mmol/day does cause hypertension. If salt intake and blood pressure were continuously correlated surely this means that as salt intake increases, so also does the blood pressure increase. I should be surprised if there was a sudden cut-off point at a sodium intake above 400 or say 800 mmol/day. In a population with a mean sodium intake of 200 mmol/day, many individuals must have a salt intake of greater than 400 mmol/day, which is getting into the range where even the sceptics agree that restriction of salt intake is important. The Kempner diet has been mentioned and always is raised as a red herring. No-one, I think, is here advocating such a diet, but a reduction in the excess of the salt that we eat, preferably achieved gradually so that people can adjust and get down to tolerable intakes (say 20–40 mmol/day), is realistic. As I mentioned in my talk, there is no requirement, for my hypothesis to be viable, that hypertensive patients should have a higher mean salt excretion than normotensive subjects in the same population. In fact the data mentioned stating that salt excretion of the hypertensive group is less than that of the normotensive group, are what I would predict. As salt intake increases, the capacity to maintain salt balance by the usual control mechanisms is exceeded. This stimulation of the humoral signals would inhibit salt appetite and people with hypertension should have a salt intake less than that of the normotensive population. The hypothesis that I put forward is that some people do not handle salt as well as others. It is these people who will have blood pressures in the range that we now call ‘hypertension’. Accordingly I do not expect that hypertensive patients should have a higher salt intake than normotensives in the same population sample. It is failure to excrete salt by the usual means that causes more and more people to develop hypertension as salt intake goes up. Dr Simpson’s study, of the town of Milton in New Zealand, is an excellent one which did not show in a population a correlation of salt intake and blood pressure. However, the mean sodium intake of the population was around 170 mmol/day and there was an incidence of hypertension of about 15%. This fits right along the line of the correlation that has been shown in population studies around the world. To summarize the evidence: (1) salt intake of communities correlates with the incidence of hypertension in that community; (2) hypertension can be caused by salt when the intake is very high or when excretory capacity of the kidneys is grossly disturbed; (3) anomalies inside a community are seen between salt intake and blood pressures (the inter-relationship is complex); (4) reduction of salt intake in an individual can reduce blood pressure. All of these points have been made, supported or not contradicted by the opposition at this debate. Two problems only remain: (a) how does the high salt intake cause an increased peripheral resistance and high blood pressure?; (b) how are we going to reduce salt intake in the community at large? I suggest that those people who do not accept my propositions and who eat a lot of salt should have their blood pressures checked.

Robertson: Thank you very much, Dr Morgan. I think Sir George Pickering is a little worried about the Framingham reference.

Morgan: If statistics are done on the data it can be shown that there is a difference in blood pressure related to salt intake. The diastolic blood pressure of people with a sodium intake of less than 8.5 g/day was 81 mmHg, while the mean blood pressure of people with a higher salt intake was 89 mmHg. With the number of persons involved, the difference was highly significant. There was no continuous correlation when different levels of salt intake were compared.

Pickering: I should like to comment on that. If you use an artificial cut-off point you can get support for any hypothesis you like and there is no reason why you should use 90 mmHg as a cut-off point rather than 80 or 100 mmHg, is there?

Morgan: I have not put forward a hypothesis that there is a continuous correlation at all levels
between salt intake and blood pressure, and I believe that the prevalence of any particular blood pressure is the only way to analyse the data. We should look at the distribution of blood pressure, not the mean pressure. Many people do not get an increase in pressure with increased salt intake but some do have a rise into the range that causes morbidity and mortality. This number goes up as the salt intake of the community rises. However, it may only be when 20% of the population has a raised pressure that we shall see a significant increase in the mean. Many young members of the society can excrete a large amount of salt without a rise in blood pressure. In the Framingham study there was a higher mean arterial blood pressure with higher salt intake.

Pickering: In fact, Mr Chairman, if you look at mortality and arterial pressure, the relation is continuous from the lowest to the highest levels recorded.

Robertson: I now ask Dr Simpson to reply to his critics.

Simpson: Dr Berglund suggests that one should be looking for a curvilinear relationship rather than a linear one. We tried this but found nothing of note. Regarding a possible recommendation on dietary salt intake, I feel that it would be distinctly premature to do this now. However, it does highlight the confusion in this area and we all ought to get going and obtain more evidence. It really is rather odd that we spend so much time studying drugs and their effects while most of us have neglected this matter of salt intake. Dr Maxwell challenged the data of Reisin and his colleagues. I know that the data can be challenged; for example, in that paper there are no details of sodium intake before the trial started. Despite that, it seems to me that the conclusions stand. I do not think that the matter of acute reduction of weight is relevant. In the average person it is a matter of reducing weight by 5–10 kg and this can usually be done without a large sudden loss of weight. Certainly in the acute phase of massive weight reduction sodium excretion and fluid loss are complicating factors. I do feel, finally, that Dr Wilhelmsen sounded at one stage of his talk as if he was a prosecuting counsel in a murder case where the murder itself was so horrible that the prisoner should be found guilty regardless of doubts about the evidence. I agree that when we are considering hypertension the crime is terrible; the toll from heart disease and cerebrovascular accident is appalling. However, on the evidence as I see it at present, I am not sure that sodium intake is, even partly, guilty.

Robertson: I think it is quite apparent that we cannot achieve a consensus on this issue today. While I, personally, find attractive the concept of the sodium intake being important in determining the blood pressure values we find in our patients or in the population at large, at the moment I am perhaps nearer to Simpson than to any of the other speakers. In my view, proof either of the pathogenic effect of a high sodium intake, or of the therapeutic value of restricting dietary sodium, remains wanting. At the same time I should be very ready to be convinced if and when clear evidence is obtained. I agree that the data produced by Professor Joossens and his group in Leuven and Dr Morgan and his colleagues in Australia seem the most compelling so far. I think we can take some reassurance from the thought that if the therapeutic effects of quite modest dietary sodium restriction are as clear-cut as they appear to be from the reports of these workers, it should not be too difficult for others to confirm or refute the findings. The critical examination of any concept can surely only be profitable. If the basic hypothesis is sound, then it will be strengthened by additional evidence; alternatively, we may discover some aspect which may require the original hypothesis to be modified in some important way in order to remain viable; or, thirdly, the evidence may turn out to be incompatible with the hypothesis, and it will be abandoned. In my opinion, we do not at present have sufficient evidence to make firm recommendations to the general public. For such advice to carry conviction, we require first to convince ourselves. For the same reasons, and in this I think I am endorsing what several speakers in the discussion said, proposals about legislation are premature. Perhaps when this Society meets in George Orwell’s fateful year of 1984, this may be one of the topics about which we shall have clearer insight than we have at the moment.

In conclusion, I should like to thank the three principal speakers for tackling the issue so positively and for putting forward their views with such clear advocacy. The extensive discussion from the floor has emphasized how much interest there is in this matter. The arguments have been replete with learned and relevant literary, artistic and historical allusions. I thank all who have participated for their valuable contributions.