Salt restriction for borderline hypertension

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Dietary sodium overload was suggested as a cause of hypertension almost as soon as the latter was recognized as a problem.1 At about 150–200 mmol of sodium (9–12 g NaCl), the daily sodium load in an average British diet is 15–20 times the minimum physiological requirement.2 Human taste for salt seems to be unrelated to need.3

In 1948 reduction of dietary sodium below 5 mmol (0.3 g NaCl) daily was shown to be effective in controlling most cases of severe malignant hypertension by Kempen4 with his rice and fruit diet, and this was confirmed by a Medical Research Council (MRC) trial.5 Systolic pressures fell by about 50 mm Hg, and diastolic pressures by about 25 mm Hg. However, above this 5 mmol threshold control was lost in most cases, and the diet was so vile that even Kempen's style of patient management failed to ensure compliance, though the penalty in most cases was early death. When effective oral diuretics arrived in the 1950s, salt restriction disappeared from clinical medicine in Britain and North America, though it persisted as a (generally unverified) exhortation in continental Europe.

In 1954 Dahl bred a strain of rats that became hypertensive when they ate a lot of salt, and another strain that could eat as much salt as they liked without becoming hypertensive.4 He suggested there might be a similar genetic difference in humans. At the same time it was becoming obvious that arterial pressure was continuously and unimodally distributed through the whole population, which was difficult to reconcile with Dahl's idea in this simple form.

During the 1950s and 1960s a mass of generally consistent evidence came from all over the world, showing that where people ate less than about 30 mmol of salt a day (1.7 g NaCl), for example in tribal Africa, some of the Polynesian islands, and among unassimilated Indians in South America, there was no such thing as hypertension, though these same people quickly developed the same incidence of hypertension as the rest of the world as soon as they adopted a high-sodium diet. On the other hand, where people ate exceptionally large amounts of salt, for example in north-eastern Japan and in Portugal, there was a high prevalence of hypertension and stroke.

Finally, in the 1970s, German work showing consistent differences between hypertensives and normotensives in ion transport across erythrocyte membranes was rediscovered, rapidly extended and continues to advance with bewildering speed.7 We now know of systematic differences in ion transport across all cell membranes, not just red cells, and an increasing variety of different ion transport systems, each with its own potential defects, which mostly have a different distribution in normotensive and hypertensive groups. This means that genetic susceptibility to hypertension could be multifactorial, and could also depend on defects in cell-membrane ion transport of sodium and potassium, thus reconciling Dahl's hypothesis with the facts of epidemiology. Cooper and his group8 in Chicago have now shown consistent differences in cell-membrane ion transport between normotensive offspring of hypertensive parents, and normotensive offspring of normotensive parents; in other words, cell-membrane differences are probable precursors of clinical hypertension. There is no longer room for doubt that sodium handling must in some way play a causal role in primary hypertension.

An early conclusion from all this was to resume sodium restriction as a method of prevention and treatment for hypertension. We are just beginning to understand the often serious impairments we impose on our patients when we start them on a lifetime of medication. It is salutary to recall that only after more than 20 years experience of treatment of many millions of patients did we discover, through the current MRC general practice trial of treatment of mild hypertension, that about one man in five put on thiazide diuretics becomes impotent.9 Another British general practice study showed that of 75 hypertensive patients on treatment, none of whom were thought by their family doctors to suffer any significant side-effects, 10 per cent complained of impairments when questioned personally, and 74 per cent were thought by their close friends or relatives to be impaired, about one-third of them severely so.10 At the same time, pressure to include more and more millions of people in the definition of hypertension requiring drug intervention, with authoritative calls for mandatory treatment for all from a diastolic thresh-

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old of 90 mm Hg,\textsuperscript{11,12} pushes us towards continuous, lifelong, antihypertensive medication for 15–20 per cent of the whole adult population. To find some simple change in behaviour as an alternative to drug treatment in mild hypertension (diastolic blood pressure between 90 and 99 mm Hg) might rescue us from this dilemma. The desire for such a miracle, and the compelling case for a causal role of sodium from laboratory and comparative epidemiological studies, have joined to induce a state of uncritical credulity regarding the effectiveness of relatively small reductions in dietary sodium in treatment of mild hypertension.

Does moderate salt restriction reduce pressure in hypertension?

We already know that drastic reduction of dietary sodium is effective in decreasing hypertension, and so the relative inefficacy of moderate restriction found in the MRC 1950 trial may, for our present purposes, be misleading. A reduction of 5 or 10 mm Hg in the severe and malignant hypertension of Kempner’s patients was irrelevant to their care, but it would be highly relevant to the care of hypertensives in the diastolic range 90–104 mm Hg. In 1978 Morgan\textsuperscript{13} in Australia published a controlled trial of moderate sodium restriction in moderate hypertension, aiming to reduce daily sodium load from the 195 mmol his patients were taking at the outset of the trial to a target level of 70–100 mmol. Although his treated group only managed to reduce their average sodium intake to 157 mmol, their group mean diastolic pressure fell by 7 mm Hg compared with the controls. This was good news for physicians everywhere. You tell your patients to do something difficult and uncomfortable that will be good for their health; they do not do it, but nevertheless they painlessly obtain the health benefits you promised them; everyone is satisfied. Of course, one nagging doubt remains: how did it happen? Well, one explanation might be that the salt restricters were examined more often than the controls, got more used to the measurement procedure, and so their blood pressures fell more. In the Australian mild hypertension trial the blood pressure of nearly half the placebo-treated controls fell to normotensive levels after two years,\textsuperscript{14} and all but the most rigorously designed studies can give spurious positive results because of this generally favourable trend in the first year or two.

However, there is better evidence than this that short-term reduction of dietary sodium below about 85 mmol (5 g NaCl) a day does reduce blood pressure, at least in moderate hypertensives, by about the same amount as some antihypertensive drugs used alone. McGregor and his group\textsuperscript{15} at Charing Cross Hospital showed a small but worthwhile drop in pressure in most, though not all, of 18 moderate hypertensives who restricted their dietary sodium from a group mean of 162 to 86 mmol daily over four weeks. Their patients had been referred to hospital for hypertension at levels that have not been published, and after a two-month run-in, but before dietary restriction, they had diastolic pressures at 90–109 mm Hg, with a group mean of 98 mm Hg.

In Glyncorrwg we set out to replicate this well-designed study on a free-living and unreferred population identified by semicontinuous screening since 1968.\textsuperscript{16} Our treatment policy until 1981 was to start medication only after three readings with a mean diastolic pressure of 105 mm Hg or over. For this trial we were looking for patients with diastolic pressures in the range 90–104 mm Hg, taken from the mean of the last six casual readings. Of 49 patients meeting this criterion, 20 volunteered for the study, 18 completed it, and 13 complied fully with our programme of salt restriction and reduced their daily sodium load from a group mean of 149 mmol before the trial to 59 mmol during the trial. We used McGregor’s double-blind crossover design; all the patients restricted their dietary sodium throughout eight weeks, but half of them took 80 mmol of slow sodium daily and the other half took identical dummy tablets, the two groups crossing over at the fourth week. Comparing all 18 who completed the trial, in the fourth and final weeks there was no difference in blood pressure between the two groups, one excreting a mean 87 mmol, the other 143 mmol of sodium daily. The result was the same for the 13 successful salt restricters, taking 59 mmol sodium daily against 139 mmol for controls.\textsuperscript{17} It was a disappointing result, and not at all what we expected. Other trials of salt restriction in mild and borderline hypertension have confirmed our results.\textsuperscript{18,19}

Our study has been attacked by McGregor,\textsuperscript{20} mainly on the grounds that our subjects were not ‘real hypertensives’. We do not deny that in our subjects the blood pressures were substantially lower than those in the subjects of his study, but in my opinion there is not necessarily a conflict between the studies; the results of nearly all rigorous studies of salt restriction in hypertension are consistent with the view that the fall in blood pressure that occurs with reduction in dietary sodium below about 85 mmol (5 g NaCl) daily is proportional to the original pressure.\textsuperscript{21} High blood pressure, which needs drugs anyway, responds in a small but clinically significant way to salt restriction; mild and borderline hypertension show no useful response, certainly nothing commensurate with the work involved in attaining it. We have therefore concluded that sodium restriction is not a substitute for drug treatment in this group, which should be managed by careful follow-up (about 10 per cent of untreated mild hypertensives in the Australian trial showed a rise of 10 mm Hg or more after two years follow-up)\textsuperscript{14} and by action on other risk factors, particularly smoking.

Is salt restriction a useful adjunct to drug treatment in those who really need this, in the diastolic range above 100 mm Hg? Beard’s group in Australia\textsuperscript{22} has shown that reduction of dietary sodium, to 37 mmol from a usual level of 161 mmol, allows medication to be halved.
without any loss of control. This extremely rigorous diet was maintained for 12 weeks; salt restricters felt less depressed than controls, and 67 per cent of them said they would stick to the diet indefinitely. Several other studies have shown similar results, but it is difficult to exclude an overspill effect from compliance with diet to compliance with antihypertensive tablet taking.

Is salt restriction feasible?

Much depends on what one thinks about the taste of a low-sodium diet. In this matter there is no substitute for experience. All our research team and their families maintained a low-sodium diet for at least a week before asking our volunteers to do so. All of us found it difficult, most of us very difficult. It may get better as time goes on, but after eight weeks of sodium restriction only two of our subjects said that they actually preferred the low-sodium diet to normal food; in both cases, analysis of 24-hour urine samples showed that they were not complying with the diet. Though there are honourable exceptions, I find that most of the enthusiasts for sodium restriction have not subjected themselves both to the experience of salt restriction and to verification by analysis of 24-hour urine collections.

Some experienced workers in this field claim that 'elimination of salt at the table and simple modifications in cooking procedures will enable the patient to adhere easily to an intake of 90 mmol of sodium'. This optimism is not shared by workers in the successful North Karelia project, who reduced sodium load by 25 per cent from a high initial mean of 210 mmol on a community scale and found this task anything but easy. Nor was this our experience in Glyncorrwyg; despite provision of sodium-reduced bread, personal nutritional advice, many foods specially prepared by the cook on our team, and a great deal of group and personal support and education, the diet evidently made heavy demands on our subjects.

I rarely attempt serious salt restriction with my hypertensive patients, because I think interference with normal eating behaviour is, for most people, a side-effect of treatment just as important as the minor but lifelong side-effects of most if not all antihypertensive drugs. If we had an antihypertensive drug that made many foods taste like blotting paper, we would regard that as an important adverse effect. It is true that sodium restriction to 85 mmol (5 g NaCl) daily is probably tolerable to most people if they can be motivated; but most of us do not expect to tolerate our food, we expect to enjoy it. Salt restriction is simply one of many options, whose advantages and disadvantages must be balanced in each individual case, just as we do with drugs. Analysis of 24-hour urinary sodium, not just once but many times, is essential. In practice, the main indication is the patient who does not respond in the usual way to antihypertensive drugs. Such people need a careful dietary history and a serious trial of sodium restriction.

General dietary change

Is there then no place for dietary modification in the treatment or prevention of hypertension and its cardiovascular and cerebrovascular complications? There is now massive evidence that vascular health is improved and maintained on a diet containing less meat, less fat and more fruit and vegetables than the diet that most people, particularly working-class people, eat today, and that all of these changes have a small but significant blood-pressure-lowering effect. There is also every reason to think that any reduction in dietary sodium load is likely to be beneficial rather than harmful, that processed foods generally contain far more salt than is necessary, and that we should adopt a cautious and not too radical national food policy as we did during the Second World War, when for the first time in our history all schoolchildren got enough to eat, and the composition of bread was modified to take scientific knowledge into account. It will be difficult to secure such a policy against the opposition of the tasty rubbish industry, and the task is not made easier by anticipating the results of properly conducted trials; we simply do not know what the trials will show, as our customary levels of sodium intake are much lower than those in the United States and Australia. Nor is clinical medicine made easier by adding to the list of unverified exhortations that we shower on our patients. As I read it, there is a good case for a gradual reduction in salt consumption of about 20-25 per cent across the whole population, mainly by changes in manufactured prepared foods. There is also a case for occasional sodium restriction in moderate or severe hypertension that is refractory to standard drugs. There is no case at all for routine sodium restriction in borderline hypertension to the 85 mmol (5 g) levels currently in vogue.

References


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Determination of fetal sex

Fetal sex was determined ultrasonically by visualization of the external genitalia with 99 per cent accuracy in 69 per cent of 855 consecutive fetuses of 15 or more weeks' gestational age. Definitive visualization of the external genitalia was achieved in more than 90 per cent of all cases after the fifth month. We found that 62 per cent of male fetuses have testicular descent into the scrotum between 28 and 30 weeks and 93 per cent after 32 weeks. The wide span of gestational age at the time of testicular descent limits use of this feature as an indicator of fetal maturity.


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