

Dietary salt and hypertension: treatment and prevention

Despite the wealth of information on the relation between sodium and blood pressure the place of dietary restriction of salt in the treatment and prevention of essential hypertension remains controversial. There are two distinct questions. Firstly, does dietary salt restriction have anything to offer in the management of patients with established essential hypertension? Secondly, is there a case for reducing the salt intake of the general population in the hope that that would reduce the prevalence of essential hypertension and hence its complications?

Undoubtedly severe short term restriction of dietary salt intake (to around 10 mmol (0.6 g) a day from the normal British intake of around 140-200 mmol (8-12 g) a day) reduces blood pressure in patients with essential hypertension.^{1,2} The dietary measures required to achieve this effect^{3,4} are, however, unacceptable to most patients,¹ and severe restriction of salt has not been shown to be safe.

The effects of moderate restriction of salt (to around 80 mmol (4.7 g) a day) are less clear cut. Several groups have reported that moderate restriction is associated with a fall in blood pressure in patients with essential hypertension.⁵⁻¹⁰ The observed reduction in blood pressure in these studies cannot necessarily be attributed to restriction of salt, however, since it may have been caused by changes in other factors—particularly other constituents of the diet. For example, an increase in potassium intake appears to reduce blood pressure in both hypertensive¹¹ and normotensive¹² people, and in some studies potassium intake was deliberately increased in conjunction with restriction of sodium.⁷⁻¹⁰ Restriction of fats (particularly saturated fats) is also associated with a reduction in blood pressure in hypertensive and normotensive people.¹³ In most cases we do not know whether the dietary measures taken to reduce salt intake also reduced the fat intake, but in one study patients were deliberately asked to cut down fat.⁹ In the one study in which sodium intake was greatly reduced (from 192 to 77 mmol (11.0-4.4 g) a day) without changes in other dietary constituents there was no consistent change in blood pressure, while a reduction in fat intake without reduction in salt intake was associated with a large fall in blood pressure.¹³ Finally, we do not know how far alterations in other dietary constituents such as calcium, alcohol, coffee, and meat, all of which influence the blood pressure,¹⁴⁻¹⁷ may have contributed to the reduction in blood pressure apparently due to restriction of salt.

In an attempt to circumvent these problems MacGregor and his colleagues carried out a double blind, randomised crossover comparison of sodium chloride and placebo in patient who took the same low sodium diet throughout the study.¹⁸ When they reduced their average sodium intake from 162 to 86 mmol (9.4 to 5.0 g) a day their mean supine blood pressure fell by an average of 7.1 mm Hg. Watt *et al.*, however, were unable to reproduce these results using the same study design in general practice.¹⁹ Others have reported that moderate restriction of dietary salt does not reduce the blood pressure in patients with essential hypertension.²⁰⁻²² Of course it may be that dietary restriction of salt may lower the blood pressure in some people and not in others^{23,24}; individual susceptibility may be determined in part by the responsiveness of the renin-angiotensin system²⁵ and in part by genetic factors.²⁶ Furthermore, any relation between daily intake of salt and blood pressure is unlikely to be linear, and there may be a "threshold" above which any change in intake of salt would not be expected to change the blood pressure appreciably. In conclusion, therefore, there is little to commend pure, moderate salt restriction for treating essential hypertension, though more complex dietary manipulation might be beneficial.

The suggestion that restriction of dietary salt should be adopted as a public health measure is based on the circumstantial evidence that intake of salt is causally related to essential hypertension. What is that evidence? Some of the numerous animal models of hypertension are dependent on salt, but their relevance to hypertension in man is dubious.²⁴ Epidemiological studies have shown a positive correlation between average salt intake and blood pressure between different populations.²⁷ Most^{28,29} but not all³⁰ studies, however, have failed to show such a relation within a given population (for critical reviews see references 23 and 24). The reasons for this discrepancy are not clear—the apparent differences between populations might in fact be due to differences in other dietary and social factors, including

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adiposity; on the other hand, the failure to show a correlation within a population might be due to excessive "noise" in the data and the possibility that only certain individuals are susceptible to the effects of dietary salt. Lastly, circumstantial evidence suggests that a reduction in salt intake may be associated with a reduction in the incidence of stroke, but properly controlled prospective studies have not been carried out.³¹

The introduction of a policy of restriction of dietary salt in the population has been likened to the introduction of a new drug, requiring rigorous evaluation.³² Others have argued, however, that restriction of salt is more akin to the removal of a potentially noxious agent,⁴ that it can therefore be advocated on the basis of much less rigorous criteria, and that we should immediately recommend widespread restriction of dietary salt, particularly since a reduction of the mean blood pressure of the population of only 2 or 3 mm Hg could produce a relatively large benefit.³³ This point of view depends heavily on the belief that moderate restriction of salt is completely harmless, but in the absence of evidence to the contrary we cannot ignore the possibility that wide scale moderate restriction of salt could lead to important adverse effects within the population.

The attractions of dietary measures for controlling a disease such as hypertension are obvious, but we believe that there is insufficient evidence to advocate the use of pure restriction of dietary salt in either the treatment or the prevention of essential hypertension. We need carefully controlled, long term, prospective intervention studies in large populations in order to determine the optimal level of dietary intake of salt and to evaluate the importance of other dietary factors, particularly potassium and fats—but most importantly to determine that any proposed measures are safe.

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Chemotherapy of Hodgkin's disease comes of age

Twenty one years ago came the first hint of successful chemotherapy in Hodgkin's disease.¹ A scheme called MOPP (mustine, vincristine, procarbazine, and prednisone) led to a remarkable number of lasting responses in metastatic Hodgkin's disease. The early results have been confirmed by many other studies, and MOPP or variations on the theme now give complete remissions in 60-80% of patients with advanced (stages III and IV) Hodgkin's disease.² Half of those patients will become long term survivors, free of disease, and are probably cured. Alternating combinations of MOPP with other regimens claimed not to be cross resistant (doxorubicin, bleomycin, vinblastine, dacarbazine) may give even better results.³

In the past decade oncologists have concentrated on selecting the best treatment for each individual patient based on the outcome of clinical trials. These trials have also allowed the substantiation of prognostic factors for groups of patients treated either with radiotherapy for limited disease or chemotherapy for more advanced disease. The age of the patient, the number of individual sites of affected lymph nodes, and the size of those lymph nodes—especially the mediastinal mass—all influence the response to radiotherapy in adults treated for Hodgkin's disease. Thus elderly patients with bulky disease particularly in the mediastinum or in multiple sites will do badly.

These factors (in addition to histological picture and stage of disease) are also important for the prediction of long term survival after chemotherapy.⁴ Patients with nodular sclerosis and lymphocyte depleted types do badly, and those with stage IV B do less well than those with stage III. Achievement of a complete remission is a self evident prognostic factor, though very good results may be obtained from salvage treatment for non-responders and those who relapse late either by applying another chemotherapy regimen or by giving local radiotherapy when appropriate.⁵

The treatment of Hodgkin's disease brings in its wake a