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Salt and Hypertension – a Dangerous Myth?

The theory that a salt rich diet might be one of the causes of essential hypertension arose in part from Dahl's[1] studies on rats, and in part from a series of epidemiological studies.[2,3,4] These show that hypertension is rare in populations whose diet contains little salt and whose life-style is primitive, and common in populations with salt rich diets and sophisticated or stressful life-styles. Some of the evidence is anomalous, and some of dubious value. The criteria on which the mean daily salt intake of populations has been assessed vary, and many are estimated from inadequate data. Studies carried out comparing groups within a western population who take different and measured amounts of salt in their diets, and controlled trials of the effect of salt restriction or salt supplementation on normotensives drawn from the same populations have failed to support the hypothesis, though salt restriction has long been known to be effective in reducing the blood pressure in hypertensive patients.

Excellent reviews of the evidence have been published by Swales[5] and by Wood,[6] both of whom remain unconvinced of the wisdom of advocating dietary salt restriction for populations.

Despite the lack of firm evidence that salt restriction would be beneficial and not harmful the National Advisory Committee on Nutrition Education in 1983 recommended a reduction of 3 grammes per day in average dietary salt consumption, and much health educational material now includes advice on cutting salt intake. The wisdom of such advice is however questionable. The low prevalence of hypertension in primitive peoples who have lived all their lives on low salt diets may in fact be due to the lethal effects of diarrhoeal diseases on individuals with impaired 'sodium pumps' in the absence of adequate dietary salt. Several groups of workers studying cell membrane ion exchange mechanisms have reported anomalies of these in red, and in white, blood cells from subjects with essential hypertension, and in cells from a proportion of close relatives of cases of hypertension.

If differential mortality rates from cholera, dysentery and salmonellosis had reduced the proportion of individuals with a genetic predisposition to hypertension who survive to reach reproduction age for several generations, one would expect to find a low prevalence of high blood pressure in the resulting populations. It is notable that all the populations which are reported to be free from hypertension, such as the Solomon Islanders, Kenyans,

and Yanamamo Indians, suffer mortality rates in infancy, childhood and early adult life which are high by western standards. As the success of oral rehydration therapy illustrates, the ingestion of suitable amounts of salt and sugar can greatly enhance the body's ability to absorb fluids, maintain electrolytes and hence survive severe diarrhoea. There is evidence from a number of laboratory studies on red or white blood cells from sufferers from essential hypertension, of abnormalities in the movement of sodium and potassium across the cell membrane. All the reports show[7,8,9,10,11] an anomaly, but different workers report the anomaly in different ion transport mechanisms. Some studied the sodium pump which is inhibited by ouabain, others studied the sodium extrusion mechanism which is sensitive to frusemide, and a further group reported a similar anomaly in the sodium/lithium counter transport mechanism. Other workers[12,13,14] have shown that the anomalous sodium transport in hypertensives is mediated through some factor present in their serum. The ion flux in leucocytes from hypertensives became normal after incubation in calf serum, and cells from normotensives incubated in serum from a hypertensive developed the ion transport characteristics of hypertensives. It is possible that this circulating factor alters the membrane's permeability to cations and so affects all the different 'pumps'.

Clearly such a mechanism is unlikely to affect only circulating blood cells. Such a malfunction in the cells of the renal tubules would affect their ability to maintain normal electrolyte concentrations when the sodium intake is unusually low or high. It would impair the ability to conserve salt by actively reabsorbing it from the filtrate and also impair the ability to actively excrete any excess of sodium while conserving water. Subjects with such a defect would thus be at greatly increased risk of death from dehydration if they developed severe diarrhoea, and would also be at increased risk from hypernatraemia if their fluid intake was too low in relation to their dietary sodium. Homeostatic mechanisms work best when operating in the middle of their range, and that which regulates electrolytes is surely no exception to this rule. It is evidently a highly complex mechanism of which the renin-angiotension-aldosterone system forms a small but important part. At high levels of sodium and fluid intake, the production of atrial natriuretic factor in response to expanded blood volume, and the renal kallikrein/kinin/natriuretic hormone system are also important mechanisms for electrolyte regulation. Whether there are supplementary systems to enhance the conservation of salt and water in those with low salt diets is uncertain, but the fact that persons who remain in hot climates for long periods slowly acclimatise, and develop an increased ability to conserve salt suggests that there may be.

Whether or not this is the case, it seems likely that there is an important genetic factor involved in the aetiology of essential hypertension mediated by the production by genetically predisposed individuals of an inhibitor of the sodium transport mechanisms. The finding by Garray *et al.* that the anomaly in ion transfer was present in some normotensives with a family history of hypertension indicates that such impairment precedes the development of hypertension rendering those affected vulnerable to extremes of salt or water intake and to dehydration from diarrhoel disease. Spontaneous hyponatraemia (water intoxication) does occur,[14] and can be fatal.

In view of this, health education material which includes recommendations to restrict salt intake should be revised. During very hot weather, during periods of intense physical exercise, in the course of febrile illness or during attacks of diarrhoea when there may be risk of heat exhaustion, dehydration or hyponatraemia, additional salt taken along with adequate amounts of water can be life saving. Only when the amount of water which can be ingested is severely restricted or the drinking water is excessively saline is there real danger from salt in food. What matters is the maintainance of sufficient fluid throughput to enable the kidneys to excrete the surplus salt in the urine, and a salt intake appropriate to that fluid

throughput. The ratio of water to salt ingested should be such that even a kidney whose ability to conserve or excrete salt has been impaired by a defect in the cell membrane ion transport mechanisms can maintain the body's electrolyte balance and hydration levels. Salt restriction may reduce the incidence of essential hypertension, but if it does this by causing would-be hypertensives to die prematurely from other causes it has no place in preventive medicine!

Whether or not severe salt restriction should be used in the treatment of hypertension is a more difficult question. Salt does appear to sensitise the vasculr tree to angiotension as well as tending to increase fluid retention and expand circulating blood volume. Its restriction can help to reduce arterial blood pressure and so reduce risk of stroke and heart failure. It is for the prescribing doctor to assess whether the risks of these are greater than the risk due to the salt restriction, and possibly to advocate an increase in salt intake during attacks of diarrhoea or periods of hot weather.

Clearly, until more is known about these matters the advice about salt given by health educators should be modified or deleted. Blunderbus recommendations to reduce intake without first ascertaining whether an individual is already consuming below average quantities or has a high fluid intake seem particularly unwise. We must be sure that the advice we give will do no harm, particularly if the benefits are uncertain.

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Tuberculosis and its Challenge to the Public Health

Each generation of Public Health doctors has faced different challenges, and it is appropriate in this Centenary Year that our Journal should contain a paper, 'An old disease ... with some new answers?', by Dr Peter Littlejohns.

The arrival of effective chemotherapy by 1948 has meant that the NHS witnessed during its first 40 years, the control of tuberculosis develop so completely that the latest generation of Public Health doctors finds it difficult to comprehend the magnitude of the problem pre-1948 and the fear that most clinicians had that they themselves might acquire a chronic and virtually untreatable disease. At one time tuberculosis used to be commended to medical students as a pathological model for a chronic disease, and there is value also in regarding tuberculosis as a model of infectious disease control, because it not only illustrates the stages that need to be gone through to achieve success, but as Dr Littlejohns points out, important political and economic factors are involved too.

In the UK, Public Health doctors can be accepted as coming-of-age in 1850 after the Health of Towns Act. Since then each generation has had a different perception of tuberculosis:

Stage 1, before Koch's discoveries, was a time when there was neither cause known nor effective treatment available. There was much morbidity and mortality attributable to 'consumption' but was there truly a specific disease entity? And if so, what underlying factors were involved? Epidemiology is now often defined as 'the study of the distribution of diseases in populations, and the causal factors underlying these distributions' and at this stage it meant that many clinical observations had to be analysed and classified together with crude levels of association with, for example, geography, climate and poverty. It is important to realise in retrospect that much of the early epidemiological work was well done, but was not helpful at the time because it was either not convincing or needed to be associated with casual links that were not yet available.

Stage 2, was the quantum leap forward when the Bacillus was identified and this led rapidly to both (i) the disease entities being more clearly defined, for as well as being able to exclude some non-tuberculous causes of consumption, many other diseases were identified as being associated with the Bacillus that were unexpected, such as meningitis and other secondary organ manifestations, and (ii) treatment in isolation for the control of spread. During this stage, when effective treatments are being searched for, it is important that all the treatment that is offered is safe, i.e. does not make the patient worse, even if it proves to be ineffective. Unfortunately, there is a history of treatments that were both ineffective and harmful, which is now seen to be inexcusable.

Stage 3, is when both the cause is known and effective treatment is available for the affected individual (as well as the control of spread to other persons). It is really in two parts: