

Salt saga continued

Salt has only small importance in hypertension

The idea that salt intake is important in hypertension is now deep rooted among some doctors and some of the public, but the evidence that salt is important is weak. It was Dahl who originally incriminated differences in salt intake as a prime cause of population differences in blood pressure.¹ Although the correlation between salt intake and the prevalence of hypertension that he described was dramatic, the data he used were scanty. Gliberman examined the evidence more critically in a review of 27 published studies and concluded that there was a relation between salt intake and blood pressure, but "since increased dietary salt is usually found with greater acculturation, it cannot be stated whether salt or other cultural changes, or both, are causing the increase in blood pressure."² Her qualification has not been respected by many who have subsequently cited her review as evidence that hypertension in Western society is a disease of salt intake.

The shortcoming of Gliberman's analysis was, however, the quality of the data on which it was based. In only 11 of the 27 reports had urinary sodium excretion been measured and even then not necessarily in those whose blood pressure had been assessed. Now better data are to hand. It is usually foolhardy to claim that a report is definitive on the day that it is published, but the intersalt study published on p 319 is likely to remain the definitive work on cross cultural differences in blood pressure and salt intake. A work can, however, be definitive without being conclusive, and the results of the intersalt study will resolve few of the outstanding issues. The journal also publishes today an intracommunity study from Scotland that suggests that the relation between sodium and blood pressure is weak and that potassium and alcohol are more important (p 329).

No fewer than 52 centres in 32 countries were asked to recruit 200 subjects in the intersalt study. After exclusions data on urinary electrolyte excretion and blood pressure were examined in 10 079 subjects. Subjects from four centres had very low sodium intakes (estimated from 24 hour urinary sodium excretion); those with the lowest sodium intake of all, the Yanomamo Indians of Brazil, excreted only 0.2 mmol sodium in 24 hours compared with about 150 mmol in subjects from a typical Western society. These peoples have previously been reported to have low blood pressures and no increase in blood pressure with age.³ The intersalt study confirmed this. It seems at least possible that salt intakes as low as this may have an important influence on blood pressure, although they are clearly not feasible and possibly

hazardous in most cultures. As data from these cultures weight the results the Intersalt Study Group present separate analyses excluding these four centres. In the remaining 48 centres neither the median blood pressure nor the prevalence of high blood pressure (arbitrarily defined) was related to sodium excretion. On the other hand, the rate of increase of both systolic and diastolic pressure with age was significantly related to sodium excretion. In addition, there was a significant tendency for sodium intake and systolic (but not diastolic) blood pressure to be correlated when individual centres were looked at separately.

This is all a long way from Dahl's straight line relation between salt and blood pressure. Indeed, in tracing the steps in debate from Dahl through Gliberman to the intersalt study the most striking observation seems to be that the more complex the analysis the weaker the relation. The Intersalt Study Group conclude that for a reduction in sodium intake of 100 mmol a day (which is probably as great as can be achieved in a Western society) there would be a reduction in blood pressure of 2.2 mm Hg (systolic) and 0.1 mm Hg (diastolic). These rewards would hardly seem likely to take nutritionists to the barricades (except perhaps the ones already there). This conclusion also assumes that a causal relation lies behind the weak correlations observed and that the manoeuvre carries no adverse consequences. As the potential individual benefit is so low even the smallest harmful effect could negate or reverse the advantage of such a reduction in blood pressure. Folkow and Ely have recently emphasised on physiological grounds that the safety of a reduction in salt intake cannot be assumed: they argue that there are risks, which may be manifest only when a person is stressed, by both very high and very low salt intake.⁴ Even the reversibility of the risk of myocardial infarction by reducing blood pressure has to be taken on trust as most trials of drug treatment have failed to show this.

Nevertheless, these findings may help if evangelical fervour is restrained. The finding of weak correlations may have three explanations. Most simply, they may show that the contribution of sodium intake to blood pressure is minor and is so swamped by other factors that a very large study is needed to show it. A second explanation may be that sodium intake is a powerful determinant of blood pressure but the relation is obscured by insensitive methods. Thus Liu *et al* have emphasised the effect which day to day variability in blood pressure may have in diluting any relation.⁵ For instance, any association between blood pressure and sodium

output would be reduced by half by using only one 24 hour collection; four 24 hour collections would still result in a weakening of a quarter. The analysis of the intersalt study was based on a single measurement with a second measure in a few subjects as a test of reliability. Although the measurement of blood pressure was carefully standardised, single values are clearly only crude estimates of blood pressure over 24 hours. The relation between sodium intake and blood pressure could therefore be much stronger than that observed. I do not believe that this is the case. The striking feature in the current controversy about salt intake and blood pressure has not been merely the difficulty in showing a relation between the two but also the difficulty in showing that salt restriction lowers blood pressure in all but a few subjects with appreciable hypertension.^{6,7} This should be contrasted with the comparative ease of detecting a relation between obesity and blood pressure or between heavy alcohol intake and blood pressure.^{8,9} Likewise, in most cases there has been no difficulty in showing that weight reduction and alcohol restriction lowers blood pressure.^{10,11}

There is a third, and most likely, explanation of the weak associations in the intersalt study: blood pressure probably has many environmental determinants, and few were measured in the study. Dietary constituents are not independent of each other. Thus the salt debate has been paralleled by a debate on the importance of low calcium intake in hypertension.¹² Weak correlations have been shown between calcium intake and blood pressure, which in some analyses disappeared entirely when confounding factors were taken into account.¹² One of the more interesting findings in recent years on the epidemiology of hypertension has been the observation that vegetarians consistently have lower blood pressures than matched controls and that a vegetarian diet will lower blood pressure in omnivores.¹³ It may be relevant that some of the sodium and blood pressure relations in the intersalt study were further weakened when alcohol intake

and body mass index were taken into account. These are the factors that are currently recognised. What of the others that we are not so certain of? Possibly the relations observed may be providing a clue to dietary factors that are more powerful determinants of blood pressure than those perceived at present.

We should guard against giving prescriptive advice based on weak epidemiological relations. Feinstein has pointed to the dangers of such activities: doctors, he warns, should not engage in an "intellectual lobotomy that equates statistical significance with biological, physiological, or quantitative importance."¹⁴ The enormous labour entailed in the intersalt study is of value only if it is seen as a fragment of a large and so far incomplete picture of environmental influences in hypertension.

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Homosexuality

Compatible with full health

AIDS is not a homosexual disease, but in the Western world, and in the United States in particular, male homosexuals have borne the brunt of the epidemic. One consequence has been a vigorous reappraisal of attitudes to homosexuality. Apart from the predictably inflammatory responses of some sections of the press, we have seen both church and government grappling with the issue. The Church of England has reaffirmed that homosexual relationships are inherently immoral whether or not they manifest the virtues of love, mutual caring, and fidelity. The government, in its controversial section 28 of the Local Government Act 1988, has actively discouraged positive attitudes to homosexuality, implying that they encourage homosexual development in young people. What message should doctors be providing at this time?

Doctors have a long and dubious tradition of influencing sexual morality under the guise of medical wisdom. In the past non-procreative sex, such as masturbation, has been proscribed on the grounds of causing serious illness and even insanity. As recently as 1955 the British Medical Association, in its evidence to the Wolfenden committee on homosexuality and prostitution, offered a strong moral condemnation of homosexuality that was in striking contrast to the much more accepting attitude of the committee itself.¹ In general,

doctors have colluded with the process Barbara Wootton described as "the concept of illness expanding at the expense of the concept of moral failure."² It is questionable which label, "sickness" or "sin," does more harm to the well being of homosexuals.

Given that doctors still have considerable influence on public opinion is it reasonable to expect them to maintain a morally neutral position on such matters? Surely we should at least avoid the past errors of obscuring moral values behind pseudomedical science. At the same time there are certain issues on which we should provide an informed opinion to facilitate rational debate. Should homosexuality be regarded as an illness? Is living a homosexual lifestyle bad for your health? Is homosexual development more likely in a society that adopts positive attitudes to this sexual orientation?

Most doctors now accept that there is no rational basis for regarding homosexuality itself as an illness. A homosexual lifestyle is compatible with all the criteria of health except possibly fertility—and voluntary infertility is not regarded as an illness. Illness can be manifested as sexual behaviour, but such behaviour is more likely to be heterosexual than homosexual. On the other hand, those living a homosexual life style in our society are at greater risk of ill health.³ Apart from sexually transmitted disease, this vulnerability is pre-