Blood pressure and sodium balance in hypertensive patients in hospital

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Summary

Ninety-one subjects with uncomplicated essential hypertension were admitted to hospital. They received a diet with a fixed amount of sodium (55 mmol/day), without calorie restriction.

Blood pressure was measured at 2-hr intervals and averaged for each day. In patients who apparently adhered to a low salt diet before admission (group I), BP dropped 5 mmHg during the first 24 hr and then remained stable. In patients who previously ingested more salt (group II) a similar drop in pressure occurred during the first day, but pressure declined another 6 mmHg during the second day, to stabilize thereafter.

Both neurovegetative and sodium-dependent factors appear to contribute to the decrease in BP while the patient is in hospital.

Introduction

The mechanisms involved in the pathogenesis of essential hypertension continue to be elusive (Editorial, 1979). One way of improving our understanding could be to study the background of regressions in blood pressure (BP).

According to common clinical experience, BP tends to fall in many hypertensives as soon as they have entered hospital. This reduction is usually interpreted in terms of mental relaxation or dietary sodium restriction or both. There is no evidence, however, to support such statements. If it is true, as suggested by several epidemiological studies (Dahl, 1961; Isaacson, Modlin and Jackson, 1963; Prior *et al.*, 1968; Freis, 1976), that the BP level is linked to the amount of sodium ingested, one would anticipate the hypotensive response of being in hospital to be related to the change in dietary sodium intake in this period. This hypothesis was tested in the present study.

Patients and methods

Ninety-one subjects (58 men, 33 women) with uncomplicated essential hypertension were admitted to hospital. Mean age was 43 (range 18-68) years

in the men and 46 (range 21-70) years in the women. The diagnosis of essential hypertension was based on routine exclusion of known causes of high BP. All patients had normal values for creatinine clearance (>100 ml/min) and none had associated cardiovascular or metabolic disease. At least 4 weeks before admission, antihypertensive treatment was withdrawn. No patient had received diuretics in the 12 months preceding the study. Salt intake before admission varied over a wide range, although the majority of patients appeared to comply with the routine advice to restrict salt as much as possible. The recommendation to restrict dietary salt intake in these patients was given irrespective of the level of BP. It is the authors' practice to estimate sodium excretion in out-patients, but they refrained from incorporating these data here because of unreliability of such urine collections.

Subjects were admitted to a metabolic ward where sodium intake was maintained at 55 mmol/day. Calorie intake was not restricted. Twenty-four hr urine collections for determination of sodium, potassium and creatinine were started on admission and continued for one week. Creatinine excretion was taken as an index of complete collection. Patients were up and about during the day-time. Blood pressure was recorded in the supine position at 2-hr intervals from 8 a.m. to 12 p.m. The diastolic BP was taken at the 5th phase of the Korotkoff sounds. Mean arterial pressure (MAP) was calculated as diastolic plus one-third of the pulse pressure and averaged for each day. For statistical analysis Student's *t*-test was applied where appropriate. Otherwise Wilcoxon's Rank Sum test was used. Results are expressed as mean $\pm$ s.e. mean.

Results (Fig. 1)

All subjects were in sodium equilibrium within 5 days. According to the patterns of cumulative sodium balance, 2 patient groups could be discerned. Group I consisted of 58 patients in whom there was either net sodium gain (23 patients) or no change in cumulative sodium balance (35 patients) before

urinary sodium excretion matched the new intake. Group II comprised the remaining 33 patients who apparently had a higher sodium intake before admission. All patients in group II lost sodium (on average 125 ± 5 mmol) before attaining the new balance state. Sodium output on the first day of admission was 56 ± 2 mmol/24 hr (range 5–96 mmol/24 hr) in group I, and 134 ± 5 mmol/24 hr (range 102–210 mmol/24 hr) in group II ($P < 0.001$). While in the following days this remained virtually unchanged in group I, it fell to 54 ± 3 mmol/24 hr in group II (Fig. 1). On admission, BP was similar in both groups (MAP 131 ± 2 mmHg in group I v. 130 ± 3 mmHg in group II). During the first 24 hr in hospital, BP fell by 5 mmHg in both groups.

FIG 1. Patterns of urinary sodium excretion (U_{NaV}) and mean arterial pressure (MAP), following admission to hospital, in patients from group I (●) and group II (▲).

Thereafter a further pressure drop was noticed in group II, but not in group I (Fig. 1). By the time BP had stabilized it was 126 ± 2 mmHg in group I and 119 ± 2 mmHg in group II ($P < 0.05$).

Discussion

In this study, the authors attempted to define the contribution of sodium loss to the hypotensive effect of being in hospital. Some doubts could be raised as to the validity of the method of assessing sodium balance. It could be argued, for instance,

that the quantity of sodium excreted during the first day of observation will be less than the daily amount of sodium ingested in the preceding period. However, the renal adaptation to a sudden change in sodium intake usually takes longer than one day (Epstein and Hollenberg, 1976). In the present study, MAP was shown to decrease by an average of 5 mmHg during the first 24 hr after admission. This effect occurred independently of changes in sodium balance and presumably reflects a reduction in neurovegetative pressor activity. A further pressure drop was noticed only in the patients in whom there was net sodium loss after admission (group II). It seems, therefore, that a reduction in body sodium contributed to the fall in BP in this group.

A relationship between salt consumption and the incidence of hypertension is suggested by several epidemiological studies (Dahl, 1961; Isaacson *et al.*, 1963; Prior *et al.*, 1968; Freis, 1976) and the beneficial effects of moderate salt restriction (Parijs *et al.*, 1973; Morgan *et al.*, 1978; Brummelen, Schalekamp and de Graeff, 1978). However, methodological problems do not allow one to draw definite conclusions from these investigations.

Epidemiological studies are hampered by the fact that rigid control of salt intake is not possible and assessing salt habits on the basis of random urinary collections or dietary questionnaires may not be conclusive. Moreover, other studies have failed to show a relationship between sodium excretion and BP (Simpson, 1979). In the present study, BP was similar in group I and II on the first hospital day and a relationship with previous salt intake became apparent only when changes in BP were related to changes in cumulative sodium balance.

Parijs *et al.* (1973), in a cross-over study in 14 patients with mild hypertension, obtained an average reduction in sodium excretion by approximately 50%, i.e. 191.1 ± 61.2 mmol to 92.8 ± 41.8 mmol/24 hr. The decrease in sodium excretion varied across a very wide range; from a reduction by 280 mmol/24 hr to practically no change. The results with respect to BP were also rather erratic. Home BP readings were significantly lower during sodium restriction. In the out-patient clinic, however, the difference in BP readings was not significant.

Morgan *et al.* (1978) studied the effect of sodium restriction in 31 mild hypertensives during an observation period of 2 years. After admission to the trial, subjects were only seen 4 times in that period. Systolic and diastolic BP during upright posture decreased by 10 mmHg; in the recumbent subjects, the decrease was 5 mmHg both systolic and diastolic. Sodium excretion was reported as decreasing from 195 ± 12 mmol to 157 mmol/24 hr. However, in that study, creatinine excretion was

not used as an index of accurate urine collection. Moreover the difference in sodium excretion (38 mmol) between the 2 periods was not based on a paired comparison and this may invalidate the results.

Brummelen *et al.*, (1978) reported a decrease in BP of about 10 mmHg with sodium restriction in 9 patients, but the study does not allow discrimination between the effects of sodium restriction and admission to hospital *per se*. Adaptation to the hospital and frequent BP readings appears to be associated with some reduction of BP. Such an effect was demonstrated in the patients from group I. In contrast to the earlier studies, the present one provides the opportunity to define the additional effect of changes in sodium balance upon BP, showing that a net loss of one gram of salt roughly equals a decrement of one mmHg in MAP. Within limits, this could be taken as a rule of thumb when sodium restriction is considered in treating hypertensive subjects.

After the establishment of sodium equilibrium the authors found they had 2 groups of hypertensives with different grades of severity. It is obvious that, at least in advanced hypertension, the study of the 'hospitalization effect' can provide only part of the answer as to whether a high salt intake increases BP.

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