Dietary sodium restriction for mild hypertension in general practice

G C M WATT, CATHERINE EDWARDS, J T HART, MARY HART, PAM WALTON, C J W FOY

Abstract

Eighteen patients with stable mild hypertension (mean blood pressure 144/93 mm Hg) restricted their sodium intake for eight weeks while taking part in a double blind randomised crossover trial of slow sodium and placebo tablets. Mean 24 hour urinary sodium excretion was 143 mmol(mEq) during the period on slow sodium and 87 mmol during the period on placebo. Five patients were unable to reduce their sodium intake below 120 mmol, but the others had a mean 24 hour urinary sodium excretion of 59 mmol during the period on placebo. There was no significant difference in blood pressure between the slow sodium and placebo treatment periods, although the study had a power of 98% to detect a difference of 5 mm Hg in mean arterial pressure between the two periods.

Moderate dietary sodium restriction does not lower blood pressure in patients with this degree of hypertension.

Introduction

Recent studies suggest that moderate dietary sodium restriction is effective in lowering blood pressure in hypertensive patients.

These studies were based on patients referred to hospital and managed subsequently as inpatients or outpatients. We set out to establish whether dietary sodium restriction is also feasible and effective in the treatment of mild hypertension in general practice.

Patients and methods

Glyncoeur was a relatively isolated former mining village in the South Wales valleys. About 1500 residents are registered with one general practitioner, along with about 600 other patients in neighbouring villages. Case finding for high blood pressure began in 1968-9, when 98% of adults aged 20-64 had their blood pressure measured. Subsequently ascertainment, treatment, and follow up have been maintained more or less completely.

Until mid-1981 the policy was to treat patients with hypertensive drugs when the mean of three consecutive readings exceeded 105 mm Hg diastolic (phase V: reduced to 99 mm Hg in 1981) or 179
mm Hg systolic, or both. People with pressures in the diastolic range 90-104 mm Hg were reviewed yearly and treated only if warranted clinically. Known hypertensive patients had diastolic pressures of 105 mm Hg or over have been treated, as have 11 patients with pressures below this level. At any time about 90%, of the adult population have had their blood pressure measured at least once during the period of this survey. Virtually all measurements were taken by two practice nurses using a random zero sphygmomanometer and all the data were recorded on computer file at the MRC Epidemiology and Medical Care Unit.

Patients were considered eligible for the study if their diastolic pressures were in the second or fourth quartile of the six most recent blood pressure measurements at the health centre. Of the 51 patients who met this condition, one had recently died and another had moved away. Ten other patients were excluded because we hoped to include them in family studies of dietary sodium restriction. Twenty three of the remaining patients responded to an invitation to attend, with their spouses, a meeting in the local school hall, at which the risks and benefits of treating mild hypertension were explained. We also introduced the possible role of dietary sodium restriction as an alternative to drug treatment and described the design of a proposed trial of dietary sodium restriction. Twenty patients agreed to take part in the trial. This number was sufficient for the statistical requirements of the study, and no effort was made to recruit subjects from the 16 people who had not attended the meeting.

During the trial, all patients attended the health centre twice for baseline measurements of blood pressure, height, and weight. Two 24 hour urine specimens were collected for estimation of sodium and potassium intake and a blood sample taken for estimation of plasma renin activity. Dietary advice was also given at that stage.

The study consisted of a randomised double blind controlled trial of slow sodium and placebo tablets in patients who all restricted their sodium intake for eight weeks. Patients were allocated at random to four weeks of slow sodium tablets (Ciba, 10 mmol(mEq) sodium 105 mmol(mEq) sodium powder) taken after each meal, or the placebo (tablets) followed by four weeks of slow sodium placebo tablets (Ciba), or vice versa, with a fixed order of eight tablets a day. The study design was explained carefully to each patient, but they did not know the number of tablets or the day of administration. Patients were asked not to add salt to their food, to use as little salt as possible in cooking, and to avoid salty foods. Each person was given a shopping guide, a list of local recipes adapted for low sodium cooking, and a wall poster indicating for each meal of the day which foods should be avoided, taken in moderation, or eaten freely. Low sodium bread baked with half a usual salt content was ordered from a local baker and salt free butter was imported from a nearby town. Both items were sold to patients at cost price. Low sodium baking powder and grany browning were provided free of charge. No attempt was made to influence potassium or energy intake. Before the trial each person kept a simple seven day food diary, which provided a basis for individual dietary advice. Halfway through the study there was a meeting of all participants to discuss their experience of sodium restriction.

Blood pressure and weight were measured at weekly afternoon clinics at the health centre. Each patient was seen on the same day of the week at the same time of day. All blood pressure measurements were taken by two experienced MRC fieldworkers, who took the same number of readings from each patient during each period of the trial. Blood pressure was measured in the left arm using a random zero sphygmomonanometer with the patient seated for five minutes beforehand. Large cuffs were used for obese arms and diastolic pressures read at phase V. Patients started a 24 hour urine collection with the second void on the day of their attendance at the clinic, and completed it with the first void the next day.

Blood samples were taken for estimation of plasma renin activity at the end of the third clinic attendance in each period. Blood was taken without stasis with the patient supine for three minutes beforehand. Clinically. From 1968, 91 patients with diastolic pressures of 105 mm Hg or over had taken part in the study, as have 11 patients with pressures below this level. At any time about 90%, of the adult population have had their blood pressure measured at least once during the period of this survey. Virtually all measurements were taken by two practice nurses using a random zero sphygmomanometer and all the data were recorded on computer file at the MRC Epidemiology and Medical Care Unit.

Patients were considered eligible for the study if their diastolic pressures were in the second or fourth quartile of the six most recent blood pressure measurements at the health centre. Of the 51 patients who met this condition, one had recently died and another had moved away. Ten other patients were excluded because we hoped to include them in family studies of dietary sodium restriction. Twenty three of the remaining patients responded to an invitation to attend, with their spouses, a meeting in the local school hall, at which the risks and benefits of treating mild hypertension were explained. We also introduced the possible role of dietary sodium restriction as an alternative to drug treatment and described the design of a proposed trial of dietary sodium restriction. Twenty patients agreed to take part in the trial. This number was sufficient for the statistical requirements of the study, and no effort was made to recruit subjects from the 16 people who had not attended the meeting.

During the trial, all patients attended the health centre twice for baseline measurements of blood pressure, height, and weight. Two 24 hour urine specimens were collected for estimation of sodium and potassium intake and a blood sample taken for estimation of plasma renin activity. Dietary advice was also given at that stage.

The study consisted of a randomised double blind controlled trial of slow sodium and placebo tablets in patients who all restricted their sodium intake for eight weeks. Patients were allocated at random to four weeks of slow sodium tablets (Ciba, 10 mmol(mEq) sodium 105 mmol(mEq) sodium powder) taken after each meal, or the placebo (tablets) followed by four weeks of slow sodium placebo tablets (Ciba), or vice versa, with a fixed order of eight tablets a day. The study design was explained carefully to each patient, but they did not know the number of tablets or the day of administration. Patients were asked not to add salt to their food, to use as little salt as possible in cooking, and to avoid salty foods. Each person was given a shopping guide, a list of local recipes adapted for low sodium cooking, and a wall poster indicating for each meal of the day which foods should be avoided, taken in moderation, or eaten freely. Low sodium bread baked with half a usual salt content was ordered from a local baker and salt free butter was imported from a nearby town. Both items were sold to patients at cost price. Low sodium baking powder and grany browning were provided free of charge. No attempt was made to influence potassium or energy intake. Before the trial each person kept a simple seven day food diary, which provided a basis for individual dietary advice. Halfway through the study there was a meeting of all participants to discuss their experience of sodium restriction.

Blood pressure and weight were measured at weekly afternoon clinics at the health centre. Each patient was seen on the same day of the week at the same time of day. All blood pressure measurements were taken by two experienced MRC fieldworkers, who took the same number of readings from each patient during each period of the trial. Blood pressure was measured in the left arm using a random zero sphygmomonanometer with the patient seated for five minutes beforehand. Large cuffs were used for obese arms and diastolic pressures read at phase V. Patients started a 24 hour urine collection with the second void on the day of their attendance at the clinic, and completed it with the first void the next day.

Blood samples were taken for estimation of plasma renin activity at the end of the third clinic attendance in each period. Blood was taken without stasis with the patient supine for three minutes beforehand. Clinically. From 1968, 91 patients with diastolic pressures of 105 mm Hg or over had taken part in the study, as have 11 patients with pressures below this level. At any time about 90%, of the adult population have had their blood pressure measured at least once during the period of this survey. Virtually all measurements were taken by two practice nurses using a random zero sphygmomonanometer with the patient seated for five minutes beforehand. Large cuffs were used for obese arms and diastolic pressures read at phase V. Patients started a 24 hour urine collection with the second void on the day of their attendance at the clinic, and completed it with the first void the next day.

Blood pressure and weight were measured at weekly afternoon clinics at the health centre. Each patient was seen on the same day of the week at the same time of day. All blood pressure measurements were taken by two experienced MRC fieldworkers, who took the same number of readings from each patient during each period of the trial. Blood pressure was measured in the left arm using a random zero sphygmomonanometer with the patient seated for five minutes beforehand. Large cuffs were used for obese arms and diastolic pressures read at phase V. Patients started a 24 hour urine collection with the second void on the day of their attendance at the clinic, and completed it with the first void the next day.

Blood samples were taken for estimation of plasma renin activity at the end of the third clinic attendance in each period. Blood was taken without stasis with the patient supine for three minutes beforehand. Clinically. From 1968, 91 patients with diastolic pressures of 105 mm Hg or over had taken part in the study, as have 11 patients with pressures below this level. At any time about 90%, of the adult population have had their blood pressure measured at least once during the period of this survey. Virtually all measurements were taken by two practice nurses using a random zero sphygmomonanometer with the patient seated for five minutes beforehand. Large cuffs were used for obese arms and diastolic pressures read at phase V. Patients started a 24 hour urine collection with the second void on the day of their attendance at the clinic, and completed it with the first void the next day.

Blood samples were taken for estimation of plasma renin activity at the end of the third clinic attendance in each period. Blood was taken without stasis with the patient supine for three minutes beforehand. Clinically. From 1968, 91 patients with diastolic pressures of 105 mm Hg or over had taken part in the study, as have 11 patients with pressures below this level. At any time about 90%, of the adult population have had their blood pressure measured at least once during the period of this survey. Virtually all measurements were taken by two practice nurses using a random zero sphygmomonanometer with the patient seated for five minutes beforehand. Large cuffs were used for obese arms and diastolic pressures read at phase V. Patients started a 24 hour urine collection with the second void on the day of their attendance at the clinic, and completed it with the first void the next day.
in general practice. For example, Kawasaki et al.\(^*18\) and Van Brummen et al.\(^*19\) admitted their patients to hospital where they could administer low sodium diets of 9 and 50 mmol a day, respectively. These studies provide no guide to the effect and feasibility of achieving these levels of intake in free living subjects.

Pariss et al.\(^*20\) studied patients with an initial mean diastolic pressure of 115 mm Hg, and though blood pressure fell by 7.7/4.4 mm Hg when sodium intake was reduced from 191 to 93 mmol daily, this finding may not apply to patients with mild hypertension.

Morgan et al.\(^*10\) reported a reduction in pressure in patients who reduced their sodium intake from 195 mmol to 157 mmol a day, but the evidence for this reduction in sodium intake was based on an unequal number of 24 hour urine collections from each patient, and one third of the patients provided no initial urine specimen.

MacGregor et al.\(^*21\) admitted patients to their study if diastolic pressure lay in the range 90-109 mm Hg after two months of observation. As mean diastolic pressure was 98 mm Hg, probably many of their patients had pressures high enough at the outset to justify drug treatment. Their data were also based on supine recordings. General practitioners do not as a rule measure blood pressure in this position, and it is not clear how these pressures relate to observations made in general practice. It cannot be assumed that supine pressures are comparable with sitting pressures, as standing pressures were on average 9 mm Hg higher than supine pressures throughout this study.

In view of this uncertainty we set out to study the feasibility

![FIG 1—Individual changes in 24 hour urinary sodium excretion (n = 18). (1 mmol sodium/24 h = 1 mEq sodium/24 h.)](image)

<table>
<thead>
<tr>
<th>Table I—Randomised controlled trial of dietary sodium restriction: effects of changes in sodium intake. Values are means</th>
</tr>
</thead>
<tbody>
<tr>
<td>All 18 subjects</td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>Placebo</td>
</tr>
<tr>
<td>Systolic pressure (mm Hg)</td>
</tr>
<tr>
<td>Diastolic pressure (mm Hg)</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
</tr>
<tr>
<td>Weight (kg)</td>
</tr>
<tr>
<td>24 h Urinary sodium excretion (mmol)</td>
</tr>
<tr>
<td>24 h Urinary potassium excretion (mmol)</td>
</tr>
<tr>
<td>Plasma renin activity (ng/l/h)</td>
</tr>
</tbody>
</table>

\(^*\) p < 0.001; \(^\dagger\) p < 0.05; all other differences not significant.

Conversion: Si to traditional units—Sodium: 1 mmol = 1 mEq. Potassium: 1 mmol = 1 mEq.

Discussion

Current clinical practice is to begin hypotensive medication when diastolic pressures are sustained at 100 mm Hg or over.\(^*\)

The value of drug treatment in patients with pressures in the range 90-99 mm Hg is not yet established,\(^*\) and evidence is awaited from the MRC Mild Hypertension Trial.\(^*\) With increasing awareness of adverse reactions to common hypotensive drugs\(^*14–16\) there is doubt about drug treatment as the first line measure in managing mild hypertension and great interest in methods of lowering blood pressure without drugs.\(^*\)

Extreme dietary sodium restriction down to 10 mmol a day does reduce blood pressure in severe hypertension,\(^*14–17\) but this monotonous and unpalatable diet has no place in the management of large numbers of free living, healthy people. The key question is whether lesser degrees of sodium restriction are also effective in lowering pressure. Reviewing four controlled clinical trials of dietary sodium restriction,\(^*18–21\) Amery et al.\(^*\) concluded that blood pressure would fall by 11 mm Hg systolic and 10 mm Hg diastolic if sodium intake could be reduced from 200 mmol to 100 mmol daily. This opinion was supported by MacGregor et al.\(^*\) who found that mean arterial pressure fell by 7.1 mm Hg when sodium intake was changed from 162 to 86 mmol daily. There are several problems, however, in relating the results of the studies to the management of mild hypertension.

![FIG 2—Individual changes in mean blood pressure (diastolic pressure plus one third of pulse pressure) (n = 18).](image)
and effectiveness of moderate sodium restriction in the treatment of patients with mild hypertension in general practice.

We used the study design proposed by Paris et al26 in 1973 and first implemented by MacGregor et al,24 in which all patients restrict their sodium intake while taking either slow sodium or placebo tablets. The principal advantage of this design is that treatment and control groups are managed in an identical way, and the results can therefore be interpreted solely on the basis of changes in sodium intake.

Patients were eligible for the study if diastolic pressure lay in the range 90-104 mm Hg, taken as the mean of six casual measurements. This number of readings having been chosen in an attempt to exclude patients with only transiently raised pressure. The 18 patients who completed the study had an initial mean diastolic pressure of 92.9 mm Hg, compared with 93.3 mm Hg in the 33 patients who were also eligible but did not take part. We consider therefore that the patients in this study were representative of patients with stable mild hypertension, with the reservation that the practice policy of treating all diastolic pressures at or above 105 mm Hg (mean of three readings) over more than 10 years will have systematically culled those with casual pressures at the upper end of the diastolic range 90-104 mm Hg.

The relatively low mean pressure of these patients may also be explained by two other factors. Firstly, the distribution of pressure in the range 90-104 mm Hg is weighted towards lower pressures. Secondly, we calculate mean diastolic pressures may not account the tendency of blood pressure to fall with repeated measurement.21 Based on only the three highest consecutive pressures of the six used to classify patients for this study, mean diastolic pressure was 96.9 mm Hg.

The study achieved its main aim of comparing blood pressure at two levels of sodium intake in patients with mild hypertension. Not all patients were willing or able to restrict their sodium intake, and some patients were non-compliant with the tablets. Two patients (fig 1) actually had higher sodium excretion during the period on placebo and were thought not to have taken any of the tablets despite continuing to attend the weekly clinics. In general these results show that substantial reductions in sodium intake are feasible in general practice with willing patients given appropriate advice and support. Studies in which all subjects achieve low sodium intakes are probably not representative of the general population.

Compared with baseline measurements blood pressure fell in all patients during the study, irrespective of whether they were taking slow sodium or placebo tablets. The fall of 10 mm Hg in diastolic pressure is most likely explained by diminution of the alerting response and a small reduction in body weight.

There was no significant difference in mean arterial pressure between the periods on slow sodium and placebo tablets, despite a difference of 56 mmol in 24 hour urinary sodium excretion and a twofold difference in plasma renin activity. In contrast, in a virtually identical study MacGregor et al24 observed a fall of 7.1 mm Hg in mean arterial pressure between the fourth week of slow sodium and the fourth week of placebo, in association with a fall of 76 mmol in 24 hour urinary sodium excretion and a similar difference in plasma renin activity. Making this comparison to the 13 patients who were compliant with their sodium tablets unsatisfactorily in our study, we found no significant difference in mean arterial pressure despite a fall of 57 mmol in sodium excretion: the 95th confidence interval extended from a rise of 3.0 mm Hg to a fall of 5.4 mm Hg.

The difference in urinary sodium excretion during the periods on slow sodium in the two studies (162 v 142 mmol) was insufficient to account for the difference in blood pressure findings. There were more men and a greater racial mix in the study by MacGregor et al24 and these differences may be important. The studies were not large enough to investigate differences between men and women and between blacks and whites.

Arguably our study did not last long enough to detect an effect of sodium restriction on patients with only mildly raised pressures. For example, Morgan et al26 found that blood pressure fell only marginally during the first six months of their study, and the maximum fall did not occur until 18 months. Nevertheless, the evidence that the fall in blood pressure in their study was accompanied by a reduction in sodium intake was unsatisfactory (see above). Most studies reporting an effect of sodium restriction on blood pressure have observed this effect within 28 days, and usually much sooner.

The major difference between these two studies which might explain their different results is the level of initial blood pressure in the two groups of patients. Together the results of the two studies are consistent with the suggestion that the effect of sodium restriction on blood pressure is related directly to the level of the initial blood pressure, in agreement with uncontrolled observations.23

Conclusion

Our study provides no evidence that moderate dietary sodium restriction lowers the blood pressure in patients with mild hypertension in the general population. Evidence from other studies is consistent with a hypotensive effect in patients with higher pressures, who would normally be treated with drugs. Sodium restriction may reduce drug requirements,24 but it is not clear whether the effect of a low sodium diet alone is large enough to provide adequate control of blood pressure in these patients. Even in the most convincing study of the effect of sodium restriction26 blood pressure stayed the same, or rose, during the period of low sodium diet in one third of the patients. Larger studies are required to determine the effect of sodium restriction on patients with different levels of blood pressure, and to identify factors which may indicate susceptibility to sodium restriction.

This work was supported by the British Heart Foundation. We thank Dr Robin Elsdon-Dew (Ciba, Horsham) for supplies of slow sodium and placebo tablets; Dr David Gordon, of St Mary’s Hospital Medical School, London, for estimations of plasma renin activity; Dr Tony Ames and Mr Tony Thomas, of Neath General Hospital, West Glamorgan, for urine analyses; and Dr Tom Meade for comments on an earlier draft of this paper. Catherine Edwards is supported by grant 81 37 from the British Heart Foundation.

Requests for reprints should be sent to Dr G C M Watt.

References

Method of healing diabetic forefoot ulcers

J P POLLARD, L P LE QUESNE

Abstract

Six diabetic patients with neuropathic ulceration of the sole of the foot (seven feet, eight ulcers) were treated by the application of a below knee walking plaster with a rubber rocker. All the ulcers healed with this treatment, greatly reducing the usual period of hospital inpatient stay. After healing, study of the forces acting on the sole of the foot showed that these ulcers occur at the site of maximal horizontal shear force and confirmed that they occur at the site of maximal vertical force.

This treatment is highly effective for neuropathic ulcers of the sole not affecting bone or complicated by deep sepsis. There may be a high rate of recurrence, however, reflecting inadequacy of methods of protecting damaged neuropathic feet.

Introduction

It has long been thought that mechanical factors play a part in diabetic neuropathic ulcers of the sole. A recent study showed clearly that these ulcers occur at the site of maximum pressure (vertical force), and although there are certainly other factors in their formation, the site of the ulcer is clearly determined by mechanical factors.

We have shown that both the vertical and shearing (longitudinal and transverse) components of force acting on the foot during walking are greatly modified by the type of footwear, and in particular that a below knee plaster cast with a rubber rocker greatly reduces all three components of force. This suggested that it might be possible to heal diabetic neuropathic ulcers in such a cast, and we therefore report the results of this method of treatment. We also give the results of measuring the components of force after the ulcers had been healed.

Method

TREATMENT OF ULCERS

For treatment in a plaster cast the ulcer had to be moderately clean with healthy granulation tissue and without excessive discharge. We achieved this by bed rest, paring the overhanging edges of the ulcer, and on occasions antibiotics for a short period. Deep sepsis or osteomyelitis of the metatarsal head rendered the ulcer unsuitable for treatment by this method.

A conventional below knee plaster cast with a rubber rocker for weight bearing was then applied. On account of the sensory peripheral neuropathy great care was taken to apply adequate padding, and an extra layer of self adhesive foam rubber was applied around the malleoli. The forward projection of the cast was restricted to the level of the mid-shaft of the proximal phalanges of the toes, so that by dorsiflexion of the toes the dressing could be withdrawn, the ulcer inspected, and a new dressing inserted. Redressing was usually required daily, the dressing used being non-adhesive, presterilised Melolin (Smith and Nephew Ltd). The plaster was retained for two weeks after healing to allow some consolidation to occur.

In practice the plaster casts required changing after three or four weeks because of softening or soiling from discharge, or to allay anxiety of the state of the neuropathic foot in a plaster cast; however, no troublesome pressure problems were encountered.

ANALYSIS OF FORCES

Once the ulcer was healed it was possible to analyse the forces under the foot, as described. The three components of force (vertical, longitudinal shear, transverse shear) were analysed separately in different footwear using transducers attached to the sole of the foot with double sided adhesive tape. Transducers were located under the hallux, under the first, second and third, and fourth and fifth metatarsal heads, and under the midpoint of the heel.

Recordings were made with the patients walking barefoot, in a pair of conventional leather shoes, in the same shoes with a single 6 mm layer of low density Plastazote as an insole, in a pair of extra depth surgical shoes (Drushoe) designed to accommodate two 6 mm layers of Plastazote (one of medium density, one of low density), and in a below knee plaster cast.

PATIENTS

Seven ulcerated feet in six diabetic patients (age range 27-75 years; mean 47) were treated by the methods described. All patients had severe peripheral neuropathy affecting the legs with absent or severely impaired sensation to light touch and pain. In all except one patient both peripheral pulses were palpable at the ankle; in the oldest patient