IMPORTANCE OF WEIGHT LOSS AND SODIUM RESTRICTION IN THE TREATMENT OF MILD AND MODERATE ESSENTIAL HYPERTENSION

J-M. Krzesinski, M. Janssens, F. Vanderspeeten, G. Rorive*

SUMMARY

The effectiveness of non pharmacological treatment of chronic arterial hypertension has been evaluated in 116 treated or not primary hypertensive out-patients. Those receiving diuretics were however excluded. This population was requested to follow for 3 months a modification of their usual diet characterized by salt restriction combined with energy intake restriction when the patients were above their ideal body weight. Only 62 patients (53%) completed the study. However, this approach was effective by decreasing blood pressure and total cholesterol. The weight loss appears the most effective way to decrease blood pressure in untreated patients, but needs 3 months to be significant. In drug-treated patients, the sodium restriction significantly influences the blood pressure level, already after 1 month. However, salt sensitivity has been noted, especially in the most severe forms of the hypertensive disease. The combination of both modifications (weight loss and sodium restriction) does not appear to be more effective than each separate dietary measure .

Acta Clin Belg. 48, 4: 234-45.

INTRODUCTION

Non-pharmacological treatment may be of interest for the hypertensive patient, mainly in the mild to moderate forms of the disease (1, 2).

If the efficiency of weight reduction in obese people has been well established for the treatment of hypertension, the usefulness of adding sodium restriction in these obese hypertensive patients is more controversial (3-5). Moreover, the feasibility of such an approach in clinical practice has been challenged (6-9) as it requires highly motivated individuals and a high level of intervention (10).

The aim of the present study was to assess first the short-term feasability of a dietary approach of mild and moderate hypertension in consecutive but not unselected out-patients with essential hypertension. Secondly using this population, the respective efficiency of sodium restriction and weight loss was compared in untreated as well as drug-treated patients. Thirdly, from these data, it was attempted to identify the predictors of blood pressure (BP) response to sodium restriction or overweight correction.

METHODS

The first 116 uncomplicated essential hypertensive patients, not treated by diuretics, with a BP level over 140/90 mmHg in the supine position after 3 visits, during a period of 3 months of follow-up were included in the study with the

*Nephrology-Hypertension Unit
C.H.U. Sart-Tilman (Bât. B35)
4000 SART TILMAN/LIEGE I - BELGIUM
Reprints : J.M. Krzesinski

TABLE 1: CHARACTERISTICS OF 116 ESSENTIAL HYPERTENSIVE PATIENTS

Age (Years)	44 ± 18
Sex ratio (M/F)	56 / 60
Hypertensive heredity: negative	52
1 parent	54
2 parents	10
Duration of hypertension (years)	2 ± 0.5 (3m-7y)
Left ventricular hypertrophy (% of the total)	positive in 19%
Antihypertensive drugs 0	58
1	29*
2 or more	29**
Systolic Blood Pressure (mmHg)	163 ± 18
Diastolic Blood Pressure (mmHg)	101 ± 10
Heart Rate (beats/min)	77 ± 11
Body Weight (kg)	82 ± 15
Body Mass Index (kg/m²)	29 ± 5
% of obeses	59%
Urinary Sodium (mmol/24 H)	164 ± 80
Urinary Potassium (mmol/24 H)	79 ± 40
Plasma Renin Activity (ng/ml/h)	2 ± 1.6
Total Cholesterol (mmol/l)	6.3 ± 2

Description of the drugs :
*** : 10 received a ß-blocker, 9 a calcium entry blocker and 10 a converting enzyme inhibitor.**
**** : 17 and 12 patients received a combination of 2 or 3 of these drugs, respectively.**

approval of the Ethics Committee of our hospital. Their characteristics are summarized in Table 1.

A family history of hypertension was considered positive when a relative younger than 65 years either died from cardiovascular disease related to high BP, or was being treated for this disease, or had a diastolic BP higher than 90 mmHg measured on 2 occasions.

Some patients had never been treated by antihypertensive drugs or had stopped their drugs several months before the study. People already treated were receiving the same treatment for at least 2 months. Those receiving diuretics were excluded. During the study, no modification of the antihypertensive treatment was allowed. For this reason, the duration of the study has been decided not to be longer than 3 months.

Obesity was defined by the body mass index (BMI) equal or above 27.2 for men and 26.9 kg/m² for women according to Burton et al. (11).

Echocardiography was performed for each subject before starting the study for detection of left ventricular hypertrophy according to Devereux et al. (12) and evaluation of cardiac function. Only people with normal cardiac function were selected.

Twenty-four hour urine collections were performed to measure creatinine clearance. Only people with a normal renal function (creatinine clearance above 80 ml/min./1.73 m²) and without proteinuria were recruited.

PROTOCOL OF THE STUDY

All patients who remained hypertensive after 3 visits for at least 3 months prior to the study and showing no secondary causes of high blood pressure were enrolled to start the diet. The blood

pressure at the third visit was considered as the baseline blood pressure. These people were then examined after one and three months of such a diet, in the morning, after an overnight fast. At each occasion, they were weighted and blood pressure was recorded in supine position by the same observer, after 10, 15 and 20 minutes with a random zero sphygmomanometer. The mean of the 3 BP measurements was taken as the arterial pressure at the different steps of the study.

At each visit, subjects were asked to provide 24 h urine collection for sodium (Na) and potassium (K) estimations. The creatinine was also measured to assess the validity of this collection. Outliers were identified on the basis of previously reported criteria (13) and also of variation of urinary creatinine excretion of more than 20% between the baseline value and those noted after the first or the third month. This validity was also assessed by measuring the urinary volume at follow-up examination. When this volume was different by more than 50 percent from the preceding collected 24 h urine samples, the patient was excluded from the statistical analysis.

At the end of the visit, and after 30 min. rest, a blood sample was drawn for plasma renin activity (PRA) (a possible salt sensitivity criteria) and serum total cholesterol (another cardiovascular risk factor) measurements.

The patients were also questioned by a dietician at the start of the study about their nutritional habits characterized by the dietary recall technic (food frequency) and received a personalized and modified food intake. At each visit, adherence to the diet was checked by interview and patients who had difficulty with the diet received additional instructions. This modified diet included a decreased by half of sodium intake from food (based on the patient's usual sodium intake which was determined through interview) and suppression of any addition of salt.

As proposed by Russel et al. (14), a decrease by 1/3 of total caloric intake permitted for calculated ideal body weight for the height, sex and age was also advocated in obese people (Table 2). A more balanced diet was also proposed. Tobacco and alcohol use were asked to be reduced or, if possible, stopped.

TABLE 2: CHARACTERISTICS OF THE DIETARY RECALL AND THE PROPOSED DIET IN THE HYPERTENSIVE POPULATION.

	Dietary recall	Proposed diet
k cal}	2798 ± 940	1/3 decrease of the ideal
kJ }Energy per day	[1345-7212]	energy intake for the ideal
	11445 ± 3930	weight
Protids (%)	16	15
Lipids (%)	42	30
Carbohydrates (%)	42	55
Sodium intake from food (mmol/24h)	135 ± 114 [26-287]	1/2 decrease
Sodium added	in all but 1	0
Potassium intake (mmol/24h)	109 ± 50	unchanged
Calcium intake (mmol/24h)	23 ± 20	unchanged
Polyunsaturated/saturated fatty acids	0.27 ± 0.1	doubled
Dietary fibers (g/24h)	22 ± 10	unchanged

[ ] = range

About salt sensitivity criteria, no consistent definition has been advanced in the literature that has been universally accepted, as summarized by Sullivan (15) or Wedler et al. (16). Diverse protocols have been employed. In our study, designed for giving rapid routine usable advice for the possible presence of salt sensitivity, this definition was based on an arbitrary change in mean blood pressure when salt intake was decreased. A decrease of mean BP greater than 5% (corresponding to a decrease of at least 5 mmHg for patients with BP over 140/90 mmHg) characterized the salt sensitive patients when 24 h urinary sodium excretion decreased by more than 85 mmol between the first and third visits of the study. When such a variation of urinary sodium was also obtained but with a mean BP variation below 5 %, the patients were considered salt resistant.

Statistical analysis

All values reported are mean ± SD.

One-way analysis of variance (ANOVA test) was used with BONFERRONI correction to compare differences between treated and not treated groups. Categorical variables were compared using Chi square test. Comparison of the change of blood pressure, weight, heart rate or biochemical values within groups according to time were made with use of ANOVA with repeated measures. The relation between different variables was tested by univariate linear regression analysis. For selecting variables with the greatest influence on blood pressure response, multiple regression analysis was used.

Significance was accepted at the 0.05 level of probability.

RESULTS

1. In the whole population, a significant correlation was found, at the start of the study, between age and systolic BP ($r=0.29$; $p<0.002$) or total cholesterol ($r=0.43$; $p<0.001$). On the contrary, no significant correlation was noted between BP and urinary sodium excretion ($r=0.09$; NS) or weight ($r=0.11$; NS). An excellent positive correlation was observed between the amount of salt intake estimated either by the dietary recall technic or by the 24 h urinary sodium excretion measurement ($r=0.88$; $p<0.0001$).

2. Only 62 patients (from the initial 116 patients enrolled in this diet intervention trial) completed the 3 months of observation (53% of the total population). The reasons for such a drop-out are multiple: need for a modification of the treatment during the study (23%), poor compliance to the medical appointments (44%), failure to provide a 24 h sample or incomplete 24 h urine collection (33%).

In spite of such a large drop-out, the characteristics of these 62 patients at the start of the study were very similar to those of the drop-outs after 1 month (n=32) or 3 months (n=22) (Table 3). Thus for the interpretation of the results during the diet intervention study, only these 62 patients have been considered. It is worth noting that within this population, 36 patients suffered from obesity. Sixty-four percent of these obeses (23/36) needed antihypertensive drug treatment whereas only 35 % of the non obese patients (9/26) belonged to this drug-treated group ($p<0.05$).

3. During this study, there was a decrease in systolic and diastolic BP ($p<0.01$), heart rate ($p<0.05$), body weight ($p<0.01$), urinary Na excretion ($p<0.001$) and total cholesterol ($p<0.01$). The PRA was stimulated ($p<0.001$). The K excretion did not change (Table 4).

4. The 62 patients were divided into 2 groups according to antihypertensive treatment (30 without and 32 with antihypertensive drugs). No difference was noted between these 2 groups according to the presence of hypertensive heredity or left ventricular hypertrophy. Sixty percent (18/30) had a hypertensive heredity in the untreated group and 59% (19/32) in the drug-

TABLE 3: COMPARISON OF THE INITIAL CHARACTERISTICS OF THE 62 PATIENTS WHO COMPLETED THE STUDY AND THOSE FROM PATIENTS DISCARDED FROM THE STUDY AFTER 1 (n=32) AND 3 (n=22) MONTHS.

	Completing the study (n=62)	Drop-out after 1 visit (n=32)	Drop-out after 2 visits (n=22)
Age (yrs)	44 ± 15	44 ± 12	44 ± 17
BMI (kg/m²)	28.7± 5	30 ± 4	27.3± 2.6
Weight (kg)	82 ± 17	85 ± 10	78 ± 15
SBP (mmHg)	164 ± 18	161 ± 20	166 ± 18
DBP (mmHg)	101 ± 10	102 ± 10	100 ± 11
Heart rate (bts/min.)	77 ± 8	74 ± 10	82 ± 5
Urin. Na (mmol/24h)	172 ± 72	160 ± 50	150 ± 45
Urin. K (mmol/24h)	75 ± 25	81 ± 20	83 ± 15
PRA (ng/ml/h)	1.9 ± 1.6	1.8 ± 0.5	2.2 ± 0.7
Total cholesterol (mmol/l)	6.3 ± 1.6	6.2 ± 1.5	6.6 ± 0.8
Sex ratio (M/F)	32/30	14/18	10/12
Hypertensive heredity -	25	19	8
+	32	12	10
++	5	1	4
Untreated patients (n)	30	15	12
Left ventricular hypertrophy (%)	23	10	23

TABLE 4: EVOLUTION OF THE CLINICAL AND BIOLOGICAL PARAMETERS IN 62 HYPERTENSIVE PATIENTS AFTER 1 AND 3 MONTHS OF THE DIET

	t = 0	t = 1 month	t = 3 months
SBP (mmHg)	164 ± 18	155 ± 20 (-6.5±9)	151 ± 20[c] (-7.1±10)
DBP (mmHg)	101 ± 10	98 ± 10 (-3.4±9)	94 ± 8[c] (-7±11)
H.R. (beats/min)	77 ± 7	74 ± 8 (-4±12)	72 ± 8[a] (-6±14)
WEIGHT (Kg)	82 ± 17	80.4 ± 20 (-2.8±4)	77 ± 15[c] (-5±6)
URIN. Na (mmol/24 h)	172 ± 72	137 ± 35 (-24±34)	105 ± 42[c] (-31±37)
URIN. K (mmol/24 h)	75 ± 25	81 ± 35 (+8±42)	70 ± 28 (-2.8±43)
PRA (ng/ml x h)	1.9 ± 2	2.8 ± 2 (+48±98)	3.4 ± 2.1[c] (+73±97)
TOTAL CHOLESTEROL (mmol/l)	6.3 ± 2	5.9 ± 2.4 (-3.9±9)	5.8 ± 1.4[b] (-5.1±12)

a : $p<0.05$
b : $p<0.01$
c : $p<0.001$
(+ or -): ▲ in % ± 1 SD

treated group (NS). Left ventricular hypertrophy was present in 28% of the group with antihypertensive treatment and in 17% of the untreated group (NS).

4.a. *In the untreated patients* (n=30), the BP was significantly decreased in both groups divided according to family history of hypertension during the 3 months of the study ($p<0.05$): in the people without hypertensive heredity, from 160 ± 10 to 149 ± 11 mmHg for the systolic BP and for the diastolic one from 97 ± 5 to 93 ± 5 mmHg; in the people with such a positive hypertensive heredity the systolic and diastolic BP were significantly decreased during the diet period from 165 ± 14 to 152 ± 12 and from 101 ± 6 to 93 ± 5 mmHg, respectively.

In the untreated patients, the systolic BP decrease after 1 month was significantly correlated, at the 5% level, to their initial value (r=0.50), to the sodium intake at the start (r=0.38), and to weight reduction (r=0.40). After 3 months, the systolic BP decrease remained correlated to their baseline value (r=0.56), and to weight loss (r=0.53). The diastolic BP decrease was correlated, after 1 and 3 months, to their initial value (r=0.63 and 0.62), and to weight loss (r=0.36 and 0.49, respectively). However, there was no significant relationship between BP decrease and initial weight.

When the sodium restriction was absent (measured by a decrease in 24 h sodium excretion less than 10 mmol between the start and the end of the study), a weight loss of more than 4 kg (n=9) was associated with a systolic BP decrease of 12± 2% and a diastolic BP fall of 13±3%. For 7 patients characterized by a weight change of less than 1 kg during the study, but a large decrease in salt intake (more than 85 mmol/24 h), the systolic and diastolic decreases were 10 ± 2 and 7 ± 2%, respectively. Combination of weight loss of more than 4 kg and salt restriction of more than 85 mmol/24 h (n=7) did not induce significantly greater BP response than separate reductions in weight or sodium intake: 13± 3% and 10 ± 2%, respectively.

4.b. *In the drug-treated group* (n=32) (16 receiving 1 drug), the BP decrease induced by the diet was not affected by a family history of hypertension: in people with positive heredity, the BP was lowered from 163 ± 11 to 150 ± 10 and from 103 ± 4 to 94 ± 5 mmHg during the diet, and in people without such heredity from 166 ± 12 to 153 ± 9 and from 100 ± 5 to 93 ± 6 mmHg.

In the treated group, the systolic BP decrease was significantly correlated to the baseline BP level (r=0.57 and 0.68, after 1 and 3 months, respectively), to the initial sodium intake (r=0.56 and 0.55), and to sodium restriction (r=0.43 and 0.42, after 1 and 3 months, respectively). For the diastolic BP response, significant correlations were noted between this parameter and either baseline diastolic BP (r=0.38), or salt intake (r=0.62) and its change (r=0.41 and 0.48, after 1 and 3 months, respectively). In this group receiving antihypertensive drugs, there was no correlation between blood pressure changes and initial weight or weight changes induced by the diet.

For these patients, a reduction of weight of more than 4 kg (n=9) without change in salt intake decreased by 9 ± 1% the systolic and by 6 ± 1% the diastolic BP. For a sodium reduction of more than 85 mmol/24 h with a weight loss less than 1 kg (n=8), a decrease of 14 ± 3% and 12 ± 2% was noted for systolic and diastolic BP, respectively. Again, combination of weight loss and sodium intake restriction (n=7) did not give greater BP response: - 11 ± 2% and - 13 ± 2% for systolic and diastolic BP, respectively.

5. The Stepwise regression studying the respective effect of salt and weight reductions confirms a major effect of weight loss on BP in the untreated groups, which needs 3 months to reach the significant level ($p<0.01$). Salt restriction plays the greatest role on BP reduction in the drug-treated patients, and this influence is already significant after 1 month (Table 5).

TABLE 5: RESPECTIVE INFLUENCE OF WEIGHT LOSS AND SODIUM RESTRICTION ON THE BLOOD PRESSURE RESPONSE IN 62 ESSENTIAL HYPERTENSIVE PATIENTS (Equation and statistical significance given by multiple regression)

	ANTIHYPERTENSIVE TREATMENT	
	ABSENCE (n=30)	PRESENCE (n=32)
▲ SBP (0 - 1 m)	-6.65 +2.34 ▲W + 0.05 ▲UNa	-2.96 + 0.07 ▲W + 0.21 ▲UNa[b]
▲ SBP (0 - 3 m)	0.03 +2.45 ▲W[b] + 0.06 ▲UNa	-4.03 - 0.03 ▲W + 0.22 ▲UNa[b]
▲ DBP (0 - 1 m)	-1.15 +1.22 ▲W + 0.03 ▲UNa	0.7 + 0.21 ▲W + 0.11 ▲UNa[a]
▲ DBP (0 - 3 m)	0.04 +1.48 ▲W[b] + 0.02 ▲UNa	-2.81 - 0.01 ▲W + 0.13 ▲UNa[b]

(0 - 1 m) or (0 - 3 m) : study between baseline and 1 or 3 months.
▲W : variation in weight ▲UNa : variation in 24 h urinary Na excretion
a : $p < 0,05$ and b : $p < 0,01$

TABLE 6: PREDICTIVE FACTORS FOR SALT SENSITIVITY IN 62 ESSENTIAL HYPERTENSIVE PATIENTS ACCORDING TO THE FIRST VISIT PARAMETERS.

	Salt Sensitive	Salt Resistant	p
n	19	10	
Age (years)	44 ± 8	42 ± 6	NS
Sex ratio (M/F)	9/10	6/4	NS
BMI (kg/m²)	30 ± 2	29.4 ± 1	NS
Energy intake Kcal (dietary recall)	2731 ± 360	2790 ± 310	NS
Weight (kg)	85.8 ± 8.2	85.7 ± 4.5	NS
SBP (mmHg)	168 ± 8	153 ± 9	< 0.05
DBP (mmHg)	105 ± 4	100 ± 6	NS
Urinary Na (mmol/24 h)	210 ± 28	179 ± 15	< 0.05
Hypertensive heredity 0 + ++	 9 9 1	 4 6 0	NS
Left ventricular hypertrophy − +	 13 6	 10 0	<0.05
Antihypertensive drug treatment 0 1 >1	 9 4 6	 6 2 2	NS

6. In the search for patients' baseline characteristics about sodium sensitivity, 29 patients presented a decrease of more than 85 mmol/ 24 h urinary Na excretion between the first and third visits of the study. Nineteen were considered as salt sensitive according to our definition (see methods) and 10 salt resistant. The table 6 summarizes the characteristics of both groups at the start of the study. These groups do not differ for sex ratio, age, BMI, energy intake, distribution of protids, lipids and carbohydrates in their diet, heart rate, family history of hypertension or treatment by antihypertensive drugs, or PRA. However, the salt sensitive group presented at the start of the study a greater systolic BP level, a more severe form of hypertension as illustrated by higher frequency of left ventricular hypertrophy, and a larger amount of salt intake prior to the study.

DISCUSSION

There is no doubt that non-drug treatment of hypertension is attractive, but the limit of its widespread use is the difficulty in convincing pauci-symptomatic mild hypertensive patients to change their diet and life-style (for instance increasing physical activity, decreasing tobacco use).

Our study takes place in this debate in general practice. In spite of the absence of a control group, our open approach has been very effective in lowering BP, but a very large number of exclusion or drop-out (almost 50%) has been noted. This phenomenon has already been noted after a short period (3 months) of follow-up in spite of many efforts to encourage all patients to comply with the protocol. Twenty-three percent of the drop-outs could be explained by need for medical treatment modification: for uncontrolled high BP (4%), for side-effects (5%), or intercurrent illness (14%) such as infectious diseases, gastrointestinal disorders, cardiovascular events .... Seventy-seven percent could not be accepted in our analysis due to either incomplete 24 h urinary collection (33%) according to the criteria or poor adherence to the appointments (44%) due mainly to insufficient dietary compliance or interest for this more physiological therapeutic approach. This remains an important problem already encountered in other studies (8). For Kumanyika (10), salt restriction would require intensive individualized counseling programs. Another cause for this large number of drop-outs could come from our choice of imposing a diet which greatly tends to modify the life-style and dietary habits of our patients. For such a large modification, much more time is probably needed for adherence. However, the adhesion to this non pharmacological approach could also be decreased by the variety of opinions about what advice should be offered to hypertensive people. One of the most debated points in this therapeutic approach is the real importance and effect of dietary sodium restriction. An association between sodium excess in the diet and elevated arterial pressure has been proposed by several epidemiological, experimental and clinical observations (for review, 17) but arguments for effectiveness of sodium restriction for treating high BP are disputed (6). This controversy is particularly acute in hypertensive obeses. In this particular form of hypertension, with multiple interactive pathogenic factors (18), the usefulness of sodium restriction in parallel with decreasing body weight is largely debated (3, 5, 7). For Elihaou et al. (19), sodium restriction is not useful for treating such patients. On the other hand, for Gillum et al. (5), low salt diet increases the hypotensive effect of weight loss. Our non-pharmacological approach (salt restriction with, in obese patients, weight reduction) has been effective to reduce BP (table 4). This therapeutic response, already effective after 1 month, has not been different according to hypertensive heredity, sex, age, or weight excess.

In our study, however, most of the hypertensive patients are obese (59%), a rather classical observation in our industrialized countries (13).

Although a correlation has been observed between age and the baseline systolic pressure level, no relationship has been noted between BP and urinary sodium excretion, in agreement with Staessen et al (20). Surprisingly, moreover, no correlation has been found between weight and BP at the start of the study in the whole population (n= 116) as well as in the 62 patients who completed the study. This discrepant observation as well as that of some authors (13, 20) could be explained by a large proportion of obese people in the group receiving antihypertensive drugs.

The systolic and diastolic BP responses during diet are dependent of the baseline BP level. The effectiveness of body weight reduction has been confirmed as the main factor explaining the BP decrease in patients free of antihypertensive drugs. However, this effect of weight loss needs 3 months to become significant, and is independent of the initial weight.

In the drug-treated group (receiving either a beta-blocker, a converting enzyme inhibitor or a calcium entry blocker alone or in combination) the BP lowering effect is correlated to the amount of salt consumption at the start of the study and the quantitative importance of sodium restriction. Moreover, the latter influences the BP level more rapidly as it shows its favourable lowering effect already after 1 month. Although this treated group is characterized by a large proportion of obese hypertensive people (23/32), the initial body weight and the amount of weight loss have less influence on the BP in this group than sodium restriction. One important observation we have done is that weight and salt reductions together do not give a better antihypertensive effect than each reduction separately.

Looking for salt sensitivity criteria, we have found it in 2/3 of the patients who presented a good compliance to salt restriction. These salt sensitive people are characterized at the start of the study by a more severe form of high BP (higher level of systolic BP and/or presence of left ventricular hypertrophy) and a larger baseline amount of salt intake. Grobbee and Hofman (21) also observed, from a review of 13 randomised trials, that salt restriction effect increased in those with higher BP as in aging subjects. However, in our study the patients' age does not play a great role besides other parameters, also considered as important factors of salt sensitivity, (PRA, BMI, hypertensive heredity) (22-25), probably because this effect of age has been blunted by the severity of hypertension and the pharmacological treatment of the latter. Indeed, many of our patients with the most severe forms of hypertension are young and belong to the drug-treated group. Concerning obesity as a marker of salt sensitivity, Rocchini et al. (23) showed that in untreated obese adolescents salt sensitivity existed but, after weight loss, this characterictic disappeared. This could perhaps be explained by a decrease in hyperinsulinemia frequently obser-ved in obese people (26). In our study, however, the majority of obese patients belonged to the drug-treated group as before mentioned. This could have modified salt sensitivity. Moreover obese people were asked in our protocol to follow, in parallel, sodium and caloric restrictions, the latter being able to mask the former effect on blood pressure.

Beside the existence of salt sensitivity in the most severe form of hypertension and in largely salt consuming patients, the problem of salt intake in people with left ventricular hypertrophy is very important. Indeed, a positive relationship between salt intake and echocardiographic evidence of left ventricular hypertrophy (27) or exercise left ventricular dysfunction (28) has been demonstrated. In these complicated forms of hypertension, some other arguments for limiting sodium intake come from the observations made by Tobian and Hanlon (29) in rats. In human, this negative effect linked to sodium intake has been observed in epidemiological studies on stroke (30).

Finally, in parallel with the BP decrease during the diet, we have noted a decrease of total

cholesterol in both groups of hypertensives, strengthening the beneficial effect of the diet on the reversibility of the cardiovascular risk of these patients as recently pointed out by Berglund et al. (31)

CONCLUSIONS

The non pharmacological approach used alone or in combination with antihypertensive drugs is effective in the treatment of essential hypertension by decreasing high BP and total cholesterol. However, it requires many efforts from both the patients and the medical staff. The BP response is related to the baseline BP level.

The salt restriction remains very efficient for the most severe forms of hypertensive disease, with or without obesity, and in the drug treated group. This effect is already observed after 1 month.

The effect of body weight reduction on the BP decrease is more effective in untreated patients, positively related to the amount of weight reduction. A significant effect of weight loss on the BP is observed before reaching the ideal weight, but needs at least 3 months of dietary compliance to be significant.

The favourable effect of diet in the treatment of hypertensive people is independent of sex, age, hypertensive heredity, obesity or antihypertensive treatment, but combination of weight loss and sodium restriction does not produce a greater response than each measure separately.

SAMENVATTING

De doeltreffendheid van niet-farmacologische behandeling van chronische arteriële hypertensie werd bestudeerd bij 116 behandelde of niet, primaire hypertensieve ambulante patiënten. Deze die op diuretica stonden werden niet opgenomen in de studie. De patiënten werden gevraagd gedurende 3 maand hun dieet te wijzigen, namelijk zoutbeperking toe te passen gecombineerd met verminderde calorie-inname indien het ideale lichaamsgewicht werd overschreden. Slechts 62 (53 %) van de patiënten bleven tot het einde in de studie. Een verlaging van de bloeddruk en van het totaal cholesterol werd gezien. Gewichtsverlies lijkt het meest effectieve middel om bij onbehandelde patiënten de bloeddruk te doen dalen maar het duurt 3 maand vooraleer dit significant is. Bij patiënten behandeld met geneesmiddelen geeft sodiumrestrictie reeds na 1 maand een significant effect op de bloeddruk. Zoutgevoeligheid werd gezien, vooral bij meer ernstige vormen van hypertensie. De combinatie van beide interventies (gewichtsverlies en zoutrestrictie) leek niet meer effectief dan elk van deze maatregelen afzonderlijk.

RESUME

L'utilité d'une approche thérapeutique non médicamenteuse dans l'hypertension artérielle a été évaluée chez 116 patients avec hypertension de type essentiel, traités ou non par médicaments antihypertenseurs (sauf les diurétiques). Cette population a été soumise pendant 3 mois à un régime comprenant une restriction sodée chez tous, associée à une alimentation hypocalorique chez les sujets présentant parallèlement un excès pondéral. Seulement 62 patients (53%) ont terminé l'étude. Ces derniers ont vu leur pression artérielle et leur taux de cholestérol total s'abaisser significativement. La réponse de la pression artérielle à la diététique a été proportionnelle au niveau de pression artérielle de départ. Dans le groupe non traité pharmacologiquement, la perte de poids a constitué la mesure la plus efficace, mais nécessitant trois mois pour devenir significative. Par contre, chez les sujets traités par médicaments antihypertenseurs et/ou présentant une hypertrophie ventriculaire gauche, c'est la restriction sodée qui a été la plus utile avec un effet significatif déjà après 1 mois. L'association des deux mesures (perte de poids et restriction sodée) n'a pas été plus efficace que chaque approche considérée séparément.

REFERENCES

1. Amery A, Bulpitt C, Fagard R, Staessen J. Does diet matter in hypertension? *Eur Heart J.* 1980;1:

299-308.
2. Elmer PJ, Grimm JrRH, Flack J, Laing B. Dietary sodium reduction for hypertension prevention and treatment. *Hypertension.* 1991; 17 (suppl. 1): I-182 - I-189.
3. Reisin E, Abel R, Modan M, Silverberg D, Eliahou H, Modan B. Effect of weight loss without salt restriction on the reduction of blood pressure in overweight hypertensive patients. *N Engl J Med.* 1978; 298: 1-6.
4. Tuck M, Sowers J, Dornfeld L, Kledzid G, Maxwell M. The effect of weight reduction on blood pressure, plasma renin activity and plasma aldosterone levels in obese patients. *N Engl J Med.* 1981; 304: 930-3.
5. Gillum R, Prineas R, Jeffery R et al. Non pharmacological therapy of hypertension: The independent effects of weight reduction and sodium restriction in overweight borderline hypertensive patients. *Am Heart J.* 1983; 105: 128-33.
6. Watt G, Edwards C, Hart J, Hart M, Walton P, Foy C. Dietary sodium restriction for mild hypertension in general practice. *Br Med J.* 1983; 286: 432-6.
7. Fagerberg B, Andersson O, Isaksson B, Bjorntorp B. Blood pressure control during weight reduction in obese hypertensive men: separate effects of sodium and energy restriction. *Br Med J.* 1984; 288: 11-4.
8. Reisin E. Weight reduction as a therapeutic modality in hypertension. *In* Hypertension, pathophysiology, diagnosis and management edited by Laragh & Brenner, New York, Raven Press, 1990; Chapter 128: 2025-35.
9. Mac Gregor G, Sagnella G, Markandu N, Singer D, Capuccio F. Double-blind study of three sodium intakes and long-term effects of sodium restriction in essential hypertension. *Lancet.* 1989; 2: 1244-47.
10. Kumanyika S. Behavioral aspects of intervention strategies to reduce dietary sodium. *Hypertension* 1991; 17 (suppl. 1): I-190 -I-195.
11. Burton B, Foster W, Hirsch J, Van Itallie T. Report of conference proceedings, Health implications of obesity: an NIH consensus development conference. *Int J Obes.* 1985; 9: 155- 69.
12. Devereux R, Pickering T ,Harshfield G et al. Left ventricular hypertrophy in patients with hypertension: importance of blood pressure response to regularly recurring stress. *Circulation* 1983; 68: 470-6.
13. Staessen J, Bulpitt C J, Fagard R, Joossens J V, Lijnen P, Amery A. Salt intake and blood pressure in the general population: a controlled intervention trial in two towns. *Am J Hypertension.* 1988; 6: 965- 73.
14. Russel R, Mc Gandy R, Jelliffe D. Reference weights: practical considerations. *Am J Med.* 1984; 76: 767- 9.
15. Sullivan J M. Salt sensitivity: definition, conception, methodology, and long-term issues. *Hypertension.* 1991; 17 (suppl. 1): I-61-I-68.
16. Wedler B, Wiersbitzki M , Gruska S, Wolf E, Luft F. Definitions and characteristics of salt-sensitivity and resistance of blood pressure; Should the diagnosis depend on diastolic blood pressure? *Clin Exp Hypertens Theory and Practice.* 1992; A14: 1037-49.
17. Porter G. Sodium and blood pressure. Chronology of the sodium hypothesis and hypertension. *Ann Intern Med* 1983; 98 (part 2): 72-3.
18. Dornfeld L, Maxwell M, Waks A, Tuck M. Mechanisms of hypertension in obesity. *Kidney Int.* 1987; 32 (suppl. 22): S-254 - S-258.
19. Eliahou H, Erdberg A, Blau A. Energy restriction or salt restriction in the treatment of overweight hypertension. Which one? A point of view. *Clin Exp Hypertens - Theory and Practice.* 1990; A 12: 795-802.
20. Staessen J, Fagard R, Lijnen P, Amery A, Bulpitt C, Joossens J. Salt and blood pressure in Belgium. *J Epidemiol Community Health.* 1981; 35: 256- 61.
21. Grobbee D, Hofman A. Does sodium restriction lower blood pressure? *Br Med J.* 1986; 293: 27-9.
22. Koolen M, Van Brummelen P. Sodium sensitivity in essential hypertension: role of the renin-angiotensin-aldosterone system and predictive values of an intravenous Furosemide test. *J Hypertens.* 1984; 2: 55-9.
23. Rocchini A, Key J, Bondie D et al.The effect of weight loss on the sensitivity of blood pressure to sodium in obese adolescents. *N Engl J Med.* 1989; 321 : 580-5.
24. Kawasaki T, Delea C, Barter F, Smith H. The effect of high-sodium and low-sodium intakes on blood pressure and other related variables in human subjects with idiopathic hypertension. *Am J Med.* 1978; 64: 193-8.
25. Luft FC, Weinberger MH, Grim CE. Sodium sensitivity and resistance in normotensive humans. *Am J Med.* 1982, 7: 726-35.

26. DeFronzo R, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care.* 1991, 14: 173-94.
27. du Cailar G, Ribstein J, Grolleau R, Mimram A. Influence of sodium intake on left ventricular structure in untreated essential hypertensives. *J Hypertens.* 1989; 7(suppl. 6): S-258 - S-259.
28. Blake J, Devereux R, Borer J, Szulc M, Pappas T, Laragh J. Relation of obesity, high sodium intake, and eccentric left ventricular hypertrophy to left ventricular exercise dysfunction in essential hypertension. *Am J Med.* 1990; 88: 477-85.
29. Tobian L, Hanlon S. High sodium chloride diets injure arteries and raise mortality without changing blood pressure. *Hypertension.* 1990; 15: 900-3.
30. Perry I G, Beevers D G. Salt intake and stroke: a possible direct effect. *J Hum Hypertension* 1992; 6: 23- 5.
31. Berglund A, Andersson O, Berglynd G, Fagerberg B. Antihypertensive effect of diet compared with drug treatment in obese men with mild hypertension. *Br Med J.* 1989; 299: 480- 5.