Lack of Impact of a Comprehensive Intervention on Hypertension in the Primary Care Setting

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BACKGROUND
The implementation of lifestyle modifications, home blood pressure (BP) measurement, and optimization of antihypertensive drug therapy have been shown to improve BP control in tightly controlled research settings. Our objective was to determine the effect of these interventions in a primary care setting, with the family practitioners and nurses serving as the interventionists.

METHODS
Two hundred twenty hypertensive patients were recruited from 2 health centers that operated in the same building and covered similar populations, with the health centers randomized to function as intervention or control sites. Participants in the intervention group received repeated individual and group counseling from the centrally trained staff of the health center on healthy lifestyles. In addition, their antihypertensive drug therapy was guided by home BP measurements performed at 3-month intervals instead of by conventional office measurements.

RESULTS
After 12 months of follow-up, the between-group differences in the changes of lifestyle variables (body mass index, physical activity, dietary recalls, and urinary sodium/potassium) were nonsignificant. Antihypertensive treatment intensity increased in both groups, but the between-group difference was nonsignificant ($P = 0.63$). Office systolic/diastolic BP decreased significantly in the intervention (8/6 mm Hg; $P < 0.001$) and control (11/7 mm Hg; $P < 0.001$) groups, but the between-group differences were nonsignificant ($P = 0.25/0.16$).

CONCLUSIONS
Our intervention did not improve BP control as suggested by many prior studies performed in controlled academic settings. This result could be attributed to a lack of motivation and incentives among the staff or because the population was relatively unselected. Greater attention to education and financial incentives might be required in typical primary care settings to obtain better results.

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Despite the high prevalence of hypertension, its control rates (blood pressure (BP) < 140/90 mm Hg among treated hypertensives) are poor, even in the developed world.[1] The reason for inadequate hypertension control is somewhat unclear because the prevalence of true resistant hypertension among adults with hypertension is estimated to be 9%.[2]

Modern physicians have a multitude of tools at their disposal to increase hypertension control. A plethora of evidence has been provided recently confirming the significant effects of a multicomponent lifestyle intervention on BP.[3–6] In addition, home BP measurement and combination therapy with drugs tailored on an individual basis have been shown to result in improved adherence to treatment and control of hypertension.[7–10] Why are the results of these trials not being translated into everyday clinical practice? One reason could be that motivated research physicians working in tightly controlled academic settings have performed the previously cited interventions. This setting, however, does not reflect reality because the majority of patients with hypertension are treated in the primary care setting by family practitioners and nurses.

The objective of our study was to determine the feasibility of a comprehensive intervention on hypertension control in a primary care setting through optimization of antihypertensive drug therapy, introduction of home BP monitoring, and lifestyle guidance, with family practitioners and nurses serving as the interventionists.

METHODS

Participants

The target population consisted of hypertensive adults aged 35–74 years with (i) an untreated systolic office BP ≥160 mm Hg and/or a diastolic office BP ≥100 mm Hg or (ii) antihypertensive treatment and controlled or uncontrolled hypertension. The screening office BP was the mean of 2

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measurements. Participants who were currently participating in a drug trial or had severe psychiatric or neurologic illnesses, heart failure (ejection fraction <40% or previous hospitalization for heart failure), hemodynamically significant valvular disease, unstable coronary heart disease, or chronic kidney disease (proteinuria >1 g/L or a serum creatinine concentration >1.81 mg/dl) were excluded. Each participant provided written consent, and the local ethics committee approved the study protocol.

Study conduct

Two health centers (public primary care units) operating independently in the same building in the city of Turku, Finland, were randomized to function as an intervention and a control site (2-cluster design). The health centers are municipality-funded primary care providers, which provide the most day-to-day medical services in Finland. The two health centers employed approximately 10 physicians and 5 nurses each. The populations covered by these health centers had a similar age distribution and socioeconomic structure, and the working habits of the respective staffs resembled each other closely.

The staff nurses and physicians of the 2 health centers recruited the participants from patients who presented at the health center with previous or newly diagnosed hypertension. The patients were given information about the study, and written consent was obtained. The patients were then invited to the Research Center of the Finnish Social Insurance Institution in Turku, Finland, for baseline examinations. During 2 consecutive days, baseline data were collected, and the patients' eligibility for the study was determined. Follow-up data were collected with similar methods 12 months later.

The patients and the staff of the control health center did not receive any intervention. No contact between the control group and the study organization occurred between the baseline examinations and the follow-up examinations at 12 months, and hypertension treatment continued according to conventional practice.

Behavioral interventions

Before the start of the study, the physicians and nurses of the intervention health center attended three 90-minute lectures and three 30-minute group sessions held by the study investigators, which dealt with healthy lifestyles, nutrition guidance, and optimal pharmaceutical treatment of hypertension. The staff also received written instructions on the same topics.[8] The training sessions occurred during regular working hours, and no clinical activities were scheduled at the intervention health center during the sessions.

After baseline data collection, patients in the intervention cohort, who did not receive any direct education or treatment from the study investigators, received lifestyle guidance from an investigator-educated nurse during two 30-minute individual counseling sessions held at 4-week intervals and at a 60-minute group session of 10–12 participants held 4 weeks later. In addition, written instructions were distributed to the participants. The participants were instructed to avoid added salt, use low-salt food ingredients, increase intake of fruits, vegetables, and berries, favor unsaturated fat over saturated fat, use low-fat dairy products, eat fish for 1–2 meals per week, exercise at least 3 hours per week, lose weight if necessary, and use no more than moderate amounts of alcohol. The lifestyle goals for the intervention group were as follows: (i) body mass index <25 kg/m^2 or a weight loss of >5% among the obese (body mass index ≥30 kg/m^2); (ii) >180 minutes per week of moderate-intensity physical activity; (iii) daily intake of <2 grams of sodium; (iv) daily intake of >3/3.5 grams of dietary potassium for women/men; (v) smoking cessation; (vi) <10% of daily energy intake from saturated fatty acids; (vii) >1% of daily energy intake from omega-3 fatty acids; and (viii) daily intake of ≤3 drinks of alcohol for men and ≤2 drinks for women. During follow-up, the intervention group patients completed a lifestyle questionnaire on exercise, nutrition, alcohol use, and smoking at 0, 3, 6, 9, and 12 months from the beginning of the study to observe whether the lifestyle goals were being met.

Implementation of home BP measurement and pharmaceutical intervention

In addition to a comprehensive lifestyle intervention, the participants' antihypertensive treatment was guided by systematic home BP measurements instead of conventional office measurements. In the control group, the target BP was an office BP <140/85 mm Hg, as recommended by the Finnish Hypertension guidelines.[11] In the intervention group, the target was a home BP <135/83 mm Hg, derived from the cross-sectional data obtained in the Pressioni Arteriose Monitorate e Loro Associazioni study.[12,13] The participants self-measured their BP at 0, 3, 6, 9, and 12 months from the beginning of the study and additionally 1 month after any changes in antihypertensive medication. The BP readings were mailed to the treating physician, and the participant was contacted by telephone. The results of the lifestyle questionnaire were examined at the same time, and lifestyle guidance was given. A face-to-face appointment was scheduled, if deemed necessary. If home BP was greater than the target pressure, the drug therapy was intensified. Physicians had free choice of which medications to use, but they had been educated on rational drug choices and combinations.[8]

Aim and outcomes

The specific aim of the study was to test the effects of a comprehensive intervention on hypertension compared with conventional practice. The intervention was delivered by primary care staff, which had been trained to deliver the intervention. The primary outcomes were the changes in office systolic and diastolic BP from baseline to 12 months. The secondary outcomes were the changes in lifestyle, nutrition, and left ventricle size.

Measurements

The staff of the Research Center of the Finnish Social Insurance Institution, who were masked to randomization

assignment, collected baseline and 12-month follow-up measurements.

Nurses measured office BP with a mercury sphygmomanometer after a 10-minute rest from the right arm with an appropriately sized cuff. Three BP measurements separated by 1 minute were obtained. Office BP was the mean of all available measurements. Home BP was measured in the intervention group using validated monitors (Omron HEM-722C, Omron Healthcare Inc., Kyoto, Japan) and similar preparations as for office BP measurement.[14] Home BP measurement was measured twice in the morning and evening on 4 consecutive days for a total of 16 measurements. The mean of all measurements was used as home BP. Ambulatory BP was recorded over 24 hours at baseline and 12 months with a validated Diasys Integra device (Novacor SA, Rueil-Malmaison, France).[15] Ambulatory BP was measured every 15 minutes between 6:00 AM and 11:00 PM and every 30 minutes between 11:00 PM and 6:00 AM. The average of all measurements was used as ambulatory BP.

Intake of nutrients and food groups was assessed from 7-day dietary recalls. The questionnaires were analyzed with the validated food intake and nutrient calculation software Nutrica (Social Insurance Institution, Turku, Finland).[16] Biomarkers of dietary intake were 8-hour overnight urinary excretion of sodium and potassium.[17] Participants filled out questions on alcohol consumption during the previous week (in units of wine, beer, spirits, and liqueur). A total alcohol consumption variable was calculated by adding the amount of each of the various beverages, similar to definitions by Klatsky.[18] Leisure time physical activity was assessed with a questionnaire that included questions about the average frequency, intensity, and duration of exercise. Energy expenditure in leisure time physical time activity was calculated by multiplying the energy frequency (times/week) with the mean duration (hours/week) and the intensity (metabolic equivalent of basal metabolic rate). Basal metabolic rate was estimated with the Mifflin equation.[19]

The same physician performed 2-dimensionally controlled M-mode echocardiographic examinations on 100 and 89 participants in the intervention and control groups. Left ventricular wall thickness, interventricular septal thickness, relative wall thickness, and left ventricular mass index were determined according to established conventions and formulas.[20] Left ventricular echocardiograms were measured at or immediately below the tips of mitral leaflets and averaged over ≥3 heart cycles.

Statistical methods

Randomization was carried out using a random-number generator. One health center was allocated as the intervention site, and the other as the control site. With significance set at 5% and power at 85%, approximately 110–115 patients per treatment group had to be randomized to detect systolic/diastolic BP differences of 5/3 mm Hg, assuming an SD of 12.6/7.4, based on our earlier research on hypertensive patients at the same research center.[21]

Database management and statistical analyses were performed with SAS software, version 9.3 (SAS Institute, Cary, NC). The between-group differences of changes in continuous measurements were compared with *t* test for normally distributed variables and with Wilcoxon 2-sample test for non-normally distributed variables. With categorical variables, the between-group differences in proportions were compared with the χ^2 test, and within-group changes in proportions were compared with McNemar's test or with Bowker's test of symmetry. $P < 0.05$ was considered statistically significant.

RESULTS

Baseline characteristics of participants

A total of 244 patients were assessed for eligibility, 123 in the intervention health center, and 121 in the control health center (Figure 1). After exclusion of patients not suitable for the study design, 117 patients were assigned to the intervention group and 112 to the control group. Five patients in the intervention group and 4 patients in the control group did not complete the study. In total, 112 patients in the intervention group and 108 patients in the control group completed the study and were included in the analyses (Figure 1). The baseline characteristics were similar for both groups (Table 1).

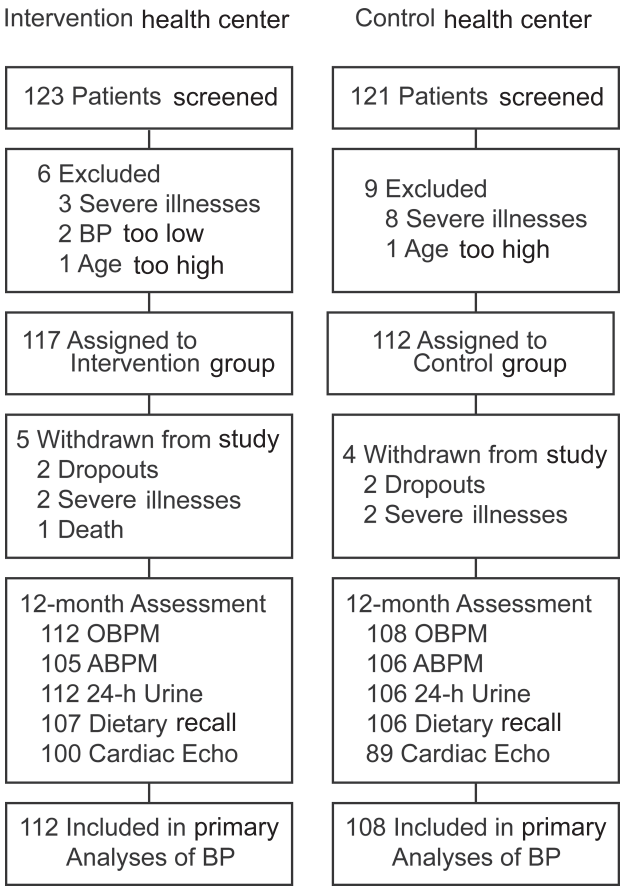

Figure 1. Participant flow in the study. Abbreviations: ABPM, ambulatory blood pressure measurement; BP, blood pressure; OBPM, office blood pressure measurement.

Table 1. Baseline characteristics by group

Characteristic	Intervention group (n = 112)	Control group (n = 108)
Age, mean (SD), y	62.9 (8.0)	61.5 (9.1)
Female	54 (48.2)	56 (51.9)
BMI, mean (SD), kg/m^2	28.5 (4.5)	28.4 (4.1)
Current smokers	13 (11.6)	11 (10.2)
Dyslipidemia[a]	49 (43.8)	44 (40.7)
Diabetes[b]	14 (12.5)	10 (9.3)
Antihypertensive treatment	84 (75.0)	80 (74.1)
>8 years of education	41 (36.6)	43 (39.8)
Office BP, mean (SD), mm Hg		
Systolic	146 (19)	148 (20)
Diastolic	87 (9)	87 (8)

Data are presented as no. (%) unless otherwise indicated.
Abbreviations: BMI, body mass index; BP, blood pressure.
[a]Total cholesterol ≥6.5 mmol/L or lipid-lowering treatment.
[b]Fasting glucose ≥7 mmol/L and/or antidiabetic treatment.

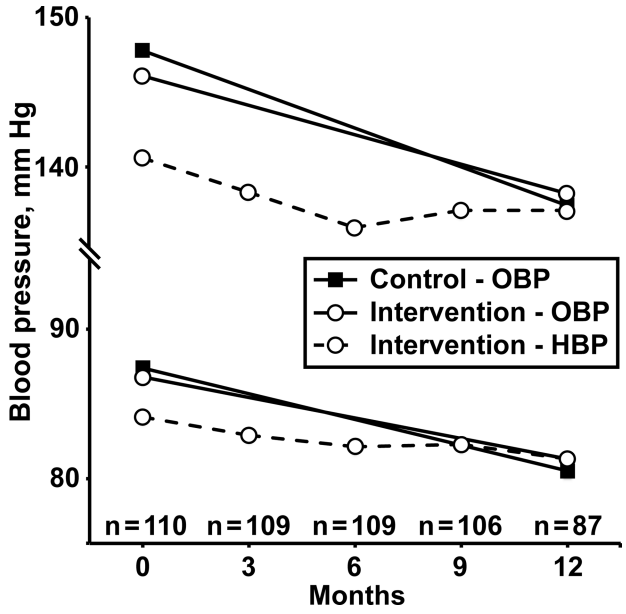

Figure 2. Mean systolic and diastolic blood pressure over time by randomized group. Upper lines indicate systolic blood pressure, and lower lines indicate diastolic blood pressure. Numbers indicate the amount of participants who successfully measured their home blood pressure at each time point in the intervention group. Abbreviations: HBP, home blood pressure; OBP, office blood pressure.

Intervention attendance and effects

Of the participants, 62.5% in the intervention group and 68.5% in the control group at baseline reported having received lifestyle guidance during the past 12 months. At the end of the 12-month follow-up, the corresponding percentages were 90.2% (P for within-group change < 0.001) and 60.2% (P for within-group change = 0.07). Of the patients in the intervention group, 45.5% took part in the group counseling sessions and 72.3% took part in the individual counseling sessions. The percentage of patients who had self-measured their BP increased from 18.8% to 92.0% in the intervention group during the 12-month follow-up (P for change < 0.001). In the control group, the percentage of patients who had self-measured their BP did not significantly change during follow-up (28.7% vs. 36.1%, P for change = 0.05). The number of participants in the intervention group who self-measured their BP at 0, 3, 6, 9, and 12 months is available in Figure 2.

After 12 months of follow-up, the intervention had only a small effect on the patients' lifestyle (Table 2). Body mass index, energy intake and overnight urinary excretion of sodium and potassium remained unchanged in both groups. The percentage of energy from carbohydrates increased, whereas the percentage of energy from saturated fat decreased in the intervention group. Reported alcohol intake, leisure physical activity, and the percentage of energy from protein increased in the control group. All between-group differences in the changes of the previously mentioned variables were nonsignificant at 12 months.

As shown in Table 3, there were no significant differences in the antihypertensive therapy between the control and intervention groups at baseline (P = 0.29). Antihypertensive therapy increased significantly in both groups (P = 0.004 for intervention group and P = 0.002 for control group). However, the between-group differences in antihypertensive therapy changes were nonsignificant (P = 0.63).

BP and left ventricular effects

Office BP declined during follow-up in both groups (Figure 2). From baseline to 12 months, mean reductions in systolic/diastolic office BP were 8 ± 17/6 ± 8 mm Hg in the intervention group, and 11 ± 17/7 ± 8 mm Hg in the control group. The pairwise differences in systolic/diastolic office BP reductions (3 ± 17/2 ± 8 mm Hg; P = 0.25/0.16) were nonsignificant. This finding was confirmed with ambulatory monitoring because the pairwise differences in systolic/diastolic 24-hour ambulatory BP reductions (2 ± 12/1 ± 7 mm Hg; P = 0.20/0.16) were also nonsignificant. In the intervention group, home BP was reduced from 141 ± 17/84 ± 7 (n = 110) to 137 ± 15/81 ± 6 (n = 87).

Figure 3 displays the percentage of hypertensive participants who reached their target office BP at baseline and 12 months. At 12 months, 52.7% had reached the target office BP of <140/85 mm Hg in the intervention group, whereas in the control group, this percentage was 60.2%. The between-group difference was nonsignificant (P = 0.26). No significant changes occurred in the echocardiographic parameters during follow-up.

DISCUSSION

In contrast with many previous studies, which have been performed in academic research settings, we decided to test the effects of a comprehensive intervention on hypertension control in a primary care setting. In our study, with the

Table 2. Intervention outcomes at baseline and at 12 months by group

Intervention outcome	Intervention group	Control group	*P* value
BMI, kg/m^2			
Baseline	28.5 (4.5)	28.4 (4.1)	
12 months	28.5 (4.6)	28.3 (4.4)	
Change	0.0 (−0.2 to 0.2)	−0.1 (−0.4 to 0.2)	0.86
Leisure physical activity, MJ/d			
Baseline	0.49 (0.45)	0.51 (0.44)	
12 months	0.49 (0.41)	0.58 (0.50)	
Change	0.00 (−0.08 to 0.07)	0.07 (0.01 to 0.13)	0.15
Alcohol intake, g/d			
Baseline	7.4 (12.7)	7.8 (12.6)	
12 months	7.3 (12.0)	11.1 (19.8)	
Change	−0.1 (−1.6 to 1.5)	3.2 (0.1−6.4)	0.06
Urine collections			
Sodium, mEq/8 h	n = 112	n = 106	
Baseline	46.5 (25.6)	48.6 (24.5)	
12 months	45.1 (22.8)	47.9 (25.4)	
Change	−1.4 (−6.1 to 3.4)	−0.9 (−6.4 to 4.6)	0.91
Potassium, mEq/8 h	n = 107	n = 95	
Baseline	17.9 (7.3)	19.1 (7.9)	
12 months	18.8 (9.0)	19.4 (8.1)	
Change	1.2 (−0.5 to 2.8)	0.3 (−1.7 to 2.2)	0.49
Dietary recalls	n = 107	n = 106	
Total energy, MJ/d			
Baseline	6.9 (2.3)	7.0 (1.9)	
12 months	6.7 (1.8)	6.9 (1.6)	
Change	−0.3 (−0.7 to 0.0)	−0.1 (−0.4 to 0.1)	0.37
Total carbohydrates, % MJ/d			
Baseline	45.1 (6.9)	45.7 (7.7)	
12 months	46.7 (6.7)	45.6 (6.5)	
Change	1.5 (0.3 to 2.7)	−0.0 (−1.3 to 1.3)	0.1
Total protein, % MJ/d			
Baseline	17.8 (3.7)	17.1 (2.8)	
12 months	17.8 (2.8)	17.8 (2.7)	
Change	0.0 (−0.6 to 0.7)	0.7 (0.2 to 1.2)	0.12
Total fat, % MJ/d			
Baseline	33.5 (6.0)	32.7 (5.8)	
12 months	32.6 (5.8)	32.6 (5.1)	
Change	−0.8 (−2.0 to 0.3)	−0.2 (−1.2 to 0.9)	0.41
Total saturated fat, % MJ/d			
Baseline	13.0 (2.8)	12.5 (2.9)	
12 months	12.3 (2.9)	12.4 (3.0)	
Change	−0.7 (−1.2 to −0.2)	−0.1 (−0.6 to 0.5)	0.11

P values refer to between-group comparisons in changes.

Table 3. Antihypertensive medications in the 2 groups

Characteristic	Intervention group	Control group
No therapy		
Baseline	25.0%	25.9%
12 months	14.3%	13.9%
Change	−10.7%	−12.0%
Single medication		
Baseline	43.8%	51.9%
12 months	48.2%	54.6%
Change	4.4%	2.7%
Combination therapy		
Baseline	31.2%	22.2%
12 months	37.5%	31.5%
Change	6.3%	9.3%

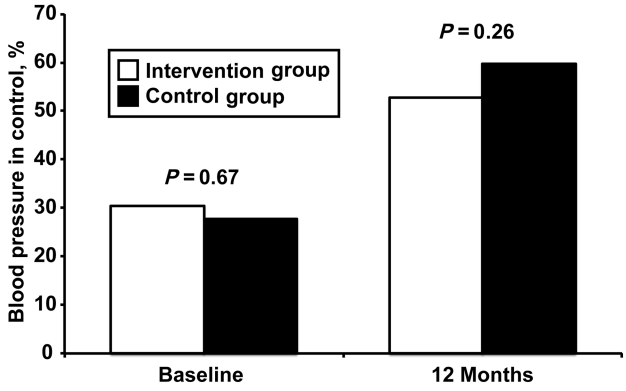

Figure 3. Percentage of participants with controlled hypertension (office blood pressure <140/85 mm Hg) at baseline and 12 months.

family practitioners and nurses serving as the interventionists, the added benefits of the intervention, as compared with standard care, were minimal.

Lifestyle changes are an important and effective part of treating hypertension. The Dietary Approaches to Stop Hypertension (DASH) trial demonstrated that a diet rich in fruits, vegetables, and low-fat dairy products reduced BP by 6/3 mm Hg more than a control diet during an 8-week follow-up.[22] In the DASH-Sodium trial, which added sodium restriction to the previously mentioned DASH intervention, reducing the sodium intake from high to intermediate level and from intermediate to low level during the DASH diet resulted in additional systolic BP reductions of 1 and 2 mm Hg, respectively.[6] The Diet, Exercise, and Weight Loss Intervention trial (DEW-IT) added exercise to the list of interventions, which resulted in mean net reductions of 10/5 mm Hg compared with the control group during a 9-week follow-up.[4] However, the results of these short-term studies cannot be generalized to typical primary care because experienced interventionists performed the studies, all meals consumed by the participants were provided by the study organization, and exercise sessions were performed under supervision.

In contrast to controlled-feeding studies, the PREMIER trial examined the effect of the DEW-IT intervention on BP in free-living participants. In this study, the intervention group underwent 18 face-to-face intervention contacts during the initial 6 months (14 group meetings and 4 individual counseling sessions). The effect of the intervention on systolic BP was not as marked as in the DASH and DEW-IT trials (4 mm Hg lower than in the control group after a 6-month follow-up), although the intervention was still implemented by trained, certified individuals in a university hospital setting.[3] The Trials of Hypertension Prevention (TOHP) trial also tested the effect of weight loss and reduction in sodium intake in free-living, overweight adults with high-normal BP during a relatively long 3-year follow-up.[5] In the TOHP trial, the intensive intervention phase consisted of weekly to monthly group sessions for 6 months. After the intensive phase, seasonal minimodules, consisting of 3 to 4 sessions each, were offered 6 times a year. Although the 2,382 patients were recruited and counseled by academic medical centers, the intervention resulted in a statistically significant, but clinically insignificant, reduction in systolic/diastolic BP (1/1 mm Hg). Because the rate of attrition was relatively low in our study (3.9%), the observed nonadherence to dietary and lifestyle interventions could have been because of the relatively long duration of the follow-up or the limited nature of the intervention. It could also be because of a lack of motivation either from the patients or the interventionists. Although 90% of the intervention group received lifestyle guidance during follow-up, the participation rates in the group and individual counseling sessions were suboptimal. The difference between interventions performed in the primary care and in the academic setting is best demonstrated by the results of our own study in which we, the research staff, acted as the interventionists instead of educators of the nonacademic interventionists. In this study, a 12-month dietary intervention based mainly on the reduction of sodium and saturated fat intake resulted in significant decreases in BP and left ventricular hypertrophy.[23,24] It can be therefore concluded that implementing lifestyle interventions on free-living patients is difficult. This applies especially when general practitioners administer the intervention with standard fiscal and personnel resources and without any financial or academic incentives.

We also aimed to improve BP control through initiation of home BP measurement. In a meta-analysis by Cappuccio *et al.*, BP control in people with hypertension and the proportion achieving targets were increased when home BP monitoring was used rather than standard BP monitoring in the healthcare system.[7] The reasons for this finding are not clear, although self-measurement might increase adherence to treatment by including the patients in their own care.[25] In addition, home BP measurements are the most acceptable method to patients and are preferred to either office measurements or ambulatory monitoring.[26] In our study, adherence to home BP monitoring was good, as 92% of the intervention group measured their home BP during the 12-month follow-up as compared with 36% in the control group. This, however, did not lead to additional BP reduction, as compared with the control group. One reason for

this finding may be that the patients themselves were not informed what their BP target should be.

In most corporate-sponsored trials on antihypertensive drugs, approximately 60%–70% of the patients usually reach the target BP.[27] In contrast, the BP control rates in the general population range from approximately 30% to 60%, with marked differences among countries.[1] It is thought that this is at least partly a result of clinical inertia, a "failure of health care providers to initiate or intensify therapy when indicated."[28] In the corporate-funded VIPER-BP study, clinical inertia was partly avoided by using a computer-assisted algorithm to apply intensive pharmacological therapy.[29] In this study, there was an 8.8% absolute difference in individual BP target achieved in favor of the intervention group compared with the usual-care group. Unfortunately, it appears that one of the best incentives to reward physicians for improved hypertension care is money. In a recent study by Petersen *et al.*, individual financial incentives, but not practice-level or combined incentives, resulted in greater BP control or appropriate response to uncontrolled BP.[30]

In addition to clinical inertia, adherence and persistence to antihypertensive therapy in general is poor. At 1 year, only 50% of patients are persistent with their prescribed antihypertensive regimen, and nonexecution—the proportion of doses omitted on each day of treatment—is 10%.[31] In our study, BP control rates improved from 30% to 53% in the intervention group. Because no significant lifestyle changes occurred, the improvements in BP control were mainly due to the observed increases in drug therapy. However, the between-group differences were nonsignificant because BP control improved similarly in the control group. Despite improvements in BP control, therapeutic inertia was observed in our study because approximately half of the patients were still on no therapy or monotherapy at 12 months, although only half of the patients had reached their BP target.

The results of our analyses must be interpreted within the context of their potential limitations. First, the sample size of our study was relatively small (220 patients with complete data), and the intervention was performed in a single clinic. Our results therefore need to be confirmed in a large-scale study with multiple study sites. Second, the nature of the lifestyle intervention was somewhat limited, with 3 counseling sessions at the beginning of the study and regular telephone contacts during follow-up. Third, the participation rates in the individual and group counseling sessions were suboptimal. However, we think that this reflects standard care because the treating physicians and nurses did not have financial or academic incentives, unlike in studies performed in academic centers. Fourth, although the participants were fairly healthy patients with essential hypertension, these results are not necessarily applicable to other populations because the Finnish population is relatively homogenous and white.

In conclusion, the added effects of a comprehensive intervention on hypertension control in the primary care setting, with family practitioners and nurses serving as the interventionists, were minimal because of therapeutic inertia and lack of compliance to lifestyle interventions. This could be because the interventionists were primary care physicians without any special incentives or because the population was relatively unselected. The results from intervention studies performed in tightly controlled academic settings by research personnel might in any case give an overly optimistic picture of the feasibility and effects of an intervention on BP control in primary care. Greater attention to education and financial incentives might be required in typical primary care settings to obtain better results. Because the majority of hypertension is treated in primary care, more public health efforts should be concentrated on educating general practitioners and the public of the dangers of hypertension and of the benefits of BP reduction. This action could reduce clinical inertia and improve the feasibility and effectiveness of lifestyle and pharmaceutical interventions.

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DISCLAIMER

The authors declared no conflict of interest.

REFERENCES

1. Wang YR, Alexander GC, Stafford RS. Outpatient hypertension treatment, treatment intensification, and control in Western Europe and the United States. *Arch Intern Med* 2007; 167:141–147.
2. Persell SD. Prevalence of resistant hypertension in the United States, 2003–2008. *Hypertension* 2011; 57:1076–1080.
3. Appel LJ, Champagne CM, Harsha DW, Cooper LS, Obarzanek E, Elmer PJ, Stevens VJ, Vollmer WM, Lin PH, Svetkey LP, Stedman SW, Young DR. Effects of comprehensive lifestyle modification on blood pressure control: main results of the PREMIER clinical trial. *JAMA* 2003; 289:2083–2093.
4. Miller ER, Erlinger TP, Young DR, Jehn M, Charleston J, Rhodes D, Wasan SK, Appel LJ. Results of the Diet, Exercise, and Weight Loss Intervention Trial (DEW-IT). *Hypertension* 2002; 40:612–618.
5. The Trials of Hypertension Prevention Collaborative Research Group. Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high-normal blood pressure. The Trials of Hypertension Prevention, phase II. *Arch Intern Med* 1997; 157:657–667.
6. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR, Miller ER, Simons-Morton DG, Karanja N, Lin PH. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *N Engl J Med* 2001; 344:3–10.
7. Cappuccio FP, Kerry SM, Forbes L, Donald A. Blood pressure control by home monitoring: meta-analysis of randomised trials. *BMJ* 2004; 329:145–149.
8. Sever PS, Messerli FH. Hypertension management 2011: optimal combination therapy. *Eur Heart J* 2011; 32:2499–2506.
9. Egan BM, Bandyopadhyay D, Shaftman SR, Wagner CS, Zhao Y, Yu-Isenberg KS. Initial monotherapy and combination therapy and hypertension control the first year. *Hypertension* 2012; 59:1124–1131.

10. Glynn LG, Murphy AW, Smith SM, Schroeder K, Fahey T. Interventions used to improve control of blood pressure in patients with hypertension. *Cochrane Database Syst Rev* 2010; 4:CD005182.
11. Working Group Appointed by the Finnish Hypertension Society. Hypertension: current care guideline. *Duodecim* 2002; 118:110–126.
12. Mancia G, Facchetti R, Bombelli M, Grassi G, Sega R. Long-term risk of mortality associated with selective and combined elevation in office, home, and ambulatory blood pressure. *Hypertension* 2006; 47:846–853.
13. Sega R, Cesana G, Milesi C, Grassi G, Zanchetti A, Mancia G. Ambulatory and home blood pressure normality in the elderly: data from the PAMELA population. *Hypertension* 1997; 30:1–6.
14. Bortolotto LA, Henry O, Hanon O, Sikias P, Mourad JJ, Girerd X. Validation of two devices for self-measurement of blood pressure by elderly patients according to the revised British Hypertension Society protocol: the Omron HEM-722C and HEM-735C. *Blood Press Monit* 1999; 4:21–25.
15. O'Brien E, Waeber B, Parati G, Staessen J, Myers MG. Blood pressure measuring devices: recommendations of the European Society of Hypertension. *BMJ* 2001; 322:531–536.
16. Hakala P, Marniemi J, Knuts LR, Kumpulainen J, Tahvonen R, Plaami S. Calculated vs analysed nutrient composition of weight reduction diets. *Food Chemistry* 1996; 57:71–75.
17. Kara PS, Erkoc R, Soyoral YU, Begenik H, Aldemir MN. Correlation of 24-hour urine sodium, potassium and calcium measurements with spot urine. *Eur J Gen Med* 2013; 10:20–25.
18. Klatsky AL, Armstrong MA, Kipp H. Correlates of alcoholic beverage preference: traits of persons who choose wine, liquor or beer. *Br J Addict* 1990; 85:1279–1289.
19. Mifflin MD, St Jeor ST, Hill LA, Scott BJ, Daugherty SA, Koh YO. A new predictive equation for resting energy expenditure in healthy individuals. *Am J Clin Nutr* 1990; 51:241–247.
20. Gottdiener JS, Bednarz J, Devereux R, Gardin J, Klein A, Manning WJ, Morehead A, Kitzman D, Oh J, Quinones M, Schiller NB, Stein JH, Weissman NJ; American Society of E. American Society of Echocardiography recommendations for use of echocardiography in clinical trials. *J Am Soc Echocardiogr* 2004; 17:1086–1119.
21. Jula A, Puukka P, Karanko H. Multiple clinic and home blood pressure measurements versus ambulatory blood pressure monitoring. *Hypertension* 1999; 34:261–266.
22. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, Bray GA, Vogt TM, Cutler JA, Windhauser MM, Lin PH, Karanja N. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med* 1997; 336:1117–1124.
23. Jula A, Rönnemaa T, Rastas M, Karvetti RL, Mäki J. Long-term nopharmacological treatment for mild to moderate hypertension. *J Intern Med* 1990; 227:413–421.
24. Jula AM, Karanko HM. Effects on left ventricular hypertrophy of long-term nonpharmacological treatment with sodium restriction in mild-to-moderate essential hypertension. *Circulation* 1994; 89:1023–1031.
25. van Onzenoort HA, Verberk WJ, Kroon AA, Kessels AG, Nelemans PJ, van der Kuy PH, Neef C, de Leeuw PW. Effect of self-measurement of blood pressure on adherence to treatment in patients with mild-to-moderate hypertension. *J Hypertens* 2010; 28:622–627.
26. Little P, Barnett J, Barnsley L, Marjoram J, Fitzgerald-Barron A, Mant D. Comparison of acceptability of and preferences for different methods of measuring blood pressure in primary care. *BMJ* 2002; 325:258–259.
27. Struijker-Boudier HA, Ambrosioni E, Holzgreve H, Laurent S, Mancia G, Ruilope LM, Waeber B. The need for combination antihypertensive therapy to reach target blood pressures: what has been learned from clinical practice and morbidity-mortality trials? *Int J Clin Pract* 2007; 61:1592–1602.
28. Phillips LS, Branch WT, Cook CB, Doyle JP, El-Kebbi IM, Gallina DL, Miller CD, Ziemer DC, Barnes CS. Clinical inertia. *Ann Intern Med* 2001; 135:825–834.
29. Stewart S, Carrington MJ, Swemmer CH, Anderson C, Kurstjens NP, Amerena J, Brown A, Burrell LM, de Looze FJ, Harris M, Hung J, Krum H, Nelson M, Schlaich M, Stocks NP, Jennings GL. Effect of intensive structured care on individual blood pressure targets in primary care: multicentre randomised controlled trial. *BMJ* 2012; 345:e7156.
30. Petersen LA, Simpson K, Pietz K, Urech TH, Hysong SJ, Profit J, Conrad DA, Dudley A, Woodard LD. Effects of individual physician-level and practise-level financial incentives on hypertension care: a randomized controlled trial. *JAMA* 2013; 310:1042–1050.
31. Vrijens B, Vincze G, Kristanto P, Urquhart J, Burnier M. Adherence to prescribed antihypertensive drug treatments: longitudinal study of electronically compiled dosing histories. *BMJ* 2008; 336:1114–1117.