Effect of Drug and Diet Treatment of Mild Hypertension on Diastolic Blood Pressure

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The Trial of Antihypertensive Interventions and Management is a multicenter randomized trial designed to examine the diastolic blood pressure response of various combinations of pharmacological and dietary interventions in the treatment of mild hypertension (diastolic blood pressure 90–100 mm Hg). Eight hundred and seventy-eight participants at 110–160% of ideal weight were randomly allocated to nine drug/diet treatment groups receiving either a placebo, chlorthalidone (25 mg), or atenolol (50 mg), combined with a usual, a weight loss, or a low sodium/high potassium diet. The primary outcome was diastolic blood pressure change from baseline to 6 months. Seven hundred and eighty-seven participants had follow-up data. The mean baseline diastolic blood pressure was 93.8 mm Hg; 55.9% of the participants were male, and the weight loss diet group lost an average of 4.7 kg. Multiple comparisons were accounted for in the analysis. A significantly greater lowering of diastolic blood pressure (12.4 mm Hg) was achieved in the atenolol group compared with either the low sodium/high potassium diet group (7.9 mm Hg, \( p = 0.001 \)) or weight loss group (8.8 mm Hg, \( p = 0.006 \)). Adding weight loss to chlorthalidone significantly enhanced blood pressure lowering (15.1 mm Hg) when compared with the diuretic alone (10.8 mm Hg, \( p = 0.002 \)), but adding a low sodium/high potassium diet (12.2 mm Hg, \( p = 0.029 \)) did not. In the short-term treatment of mild hypertension where diastolic blood pressure is the sole consideration, drugs outperform diet, and weight loss is beneficial, especially with diuretics. (Hypertension 1991;17:210–217)

Mild hypertension is a significant risk factor for cardiovascular mortality and morbidity.1 Treatment of mild hypertension significantly reduces this mortality and morbidity,2–5 but the cost/benefit ratio has been questioned.6 How best to treat mild hypertension is undecided and remains a controversial point.7,8 The symptomatic and biochemical side effects, need for long-term use and cost of pharmacological treatment for all hypertensive persons have led to suggestions that dietary therapy may be a preferable alternative therapy for the person with mild blood pressure elevation.9 Also, there is continuing controversy as to the most desirable medication for initiating treatment in the mild hypertensive individual.10

The Trial of Antihypertensive Interventions and Management (TAIM) is a randomized, controlled trial among three clinical centers involving persons between 21 and 65 years of age with diastolic blood pressure between 90 and 100 mm Hg without other substantial disease. TAIM was undertaken to assess the relative value of the most commonly used approaches to drug therapy (thiazides and \( \beta \)-blockers) and diet therapy (weight loss and a restricted sodium, increased potassium intake) applied singly and in combination. This report, based on the first 6 months of intervention, describes changes in blood pressure for the various combinations of therapy. The trial is continuing, and long-term results will be available in a few years.

Methods

Participants were recruited from January 24, 1985, to June 30, 1987, from the communities of three
clinics, which included the Albert Einstein College of Medicine, Bronx, N.Y.; University of Alabama School of Medicine, Birmingham, Ala.; and the University of Mississippi School of Medicine, Jackson, Miss. There was a nutrition and behavioral core located at Albert Einstein College of Medicine and a coordinating center located at the University of Texas School of Public Health, Houston, Tex. The protocol was reviewed and approved by the Institutional Review Boards at all the centers. The rationale, experimental design, and selected baseline results of TAIM have been reported elsewhere. All the participants were male and female volunteers aged 21–65 years.

Eligibility criteria for TAIM at a preliminary screen were 1) a diastolic blood pressure of 100 mm Hg or less for participants currently taking antihypertensive medication or 2) a diastolic blood pressure between 90 and 104 mm Hg for those on no treatment. In addition, participants had to be between 110% and 160% of their ideal weight by recall. At the second screen or first clinic visit, all potentially eligible participants had to be on no antihypertensive medication and had to have a diastolic blood pressure between 90 and 100 mm Hg and again be between 110% and 160% of their ideal weight by clinical measurement. To become eligible for TAIM by this second screen, those patients on prior antihypertensive therapy had their medication reduced, then discontinued over a time period of up to 8 weeks. Determination of blood pressure eligibility was made 2 or more weeks after medication was discontinued.

Participants were excluded who had a history or other evidence of myocardial infarction, stroke or bronchial asthma, a creatinine level of 180 μmol/l or greater, diabetes requiring insulin therapy, allergy to thiazides or β-blockers, actual or contemplated pregnancy, or likelihood of difficulty in complying with the interventions.

There were 10,148 participants screened for TAIM. Of these, 1,949 were seen at the first clinic visit and 881 went on to a second clinic visit. The major reasons for not proceeding to the first or second visit were ineligibility due to blood pressure, age, or weight. At the second visit, 878 people were randomly allocated into the TAIM study.

**Randomization**

All eligible persons signed an informed consent statement. On completion of the baseline evaluation, designated clinical center personnel contacted the coordinating center, which randomly assigned each participant to one of nine possible drug/diet treatment groups. Random allocation was stratified within clinical center and by race. Medications were identified by preassigned code to maintain the double-blind design.

**Intervention**

Within 1–2 weeks of randomization, most TAIM participants were started on their interventions. The study was a 3×3 factorial design consisting of three drug and three diet groups. Participants received either a diuretic (chlorthalidone 25 mg), a β-blocker (atenolol 50 mg), or a placebo. All pills were designed to appear identical and were administered in double-blind fashion. Two types of active dietary intervention were used: weight loss and low sodium/high potassium diets. Also, participants could be assigned to a usual (no change) diet. The goal for the weight loss group was a reduction of 10% of baseline weight or 4.54 kg, whichever was greater. Sodium and potassium goals were individualized by weight and ranged from 52 to 100 mmol/day for sodium (average 87 mmol/day), and from 62 to 115 mmol/day for potassium (average 103 mmol/day). To attain these dietary goals, nutritional counseling oriented to behavioral change was provided in group sessions held weekly for 10 weeks. Thereafter, individual sessions were held every 6–12 weeks. This approach was a modification of the one used in the Dietary Intervention Study of Hypertension.

Participants randomly assigned to TAIM protocol groups had clinical visits every month for the first 6 months and then quarterly. Long-term follow-up data beyond 6 months are still being acquired. Dietary intervention was begun for the low sodium/high potassium and weight reduction groups within 3 weeks of the baseline visit. Participants who failed to achieve adequate blood pressure control were stepped up to additional therapy at 6 months or sooner if emergency failure criteria were met. The additional step-up therapy was administered in a double-blind fashion. Either 25 mg chlorthalidone or 50 mg atenolol was given to placebo failures (allocated in a 1:1 random fashion); combined 25 mg chlorthalidone–50 mg atenolol therapy was given to the chlorthalidone or atenolol failures. Medication was increased during the first 6 months if diastolic blood pressure was equal to or greater than 100 mm Hg for three visits at 2-week intervals, greater than 105 mm Hg at two visits a week apart, or equal to or greater than 115 mm Hg at any visit. If the additional step-up therapy did not adequately control the diastolic blood pressure, open-label therapy (known antihypertensive medication) was used.

**Trial End Points**

The primary outcome of the study was the 6-month change in diastolic blood pressure. Fourteen pairwise comparisons of this change among the nine treatment groups were formulated by the TAIM investigators and are presented in detail elsewhere. Essentially, diet therapy was compared with low dose drug therapy and combined diet and drug treatment was compared with diet alone or drug alone. Other outcomes compared among the treatment groups included change in biochemical variables, changes in Framingham cardiovascular risk scores, and changes in quality of life measures.
Statistical Methods

The sample size was calculated to detect (with an \( \alpha \) of 0.05 and a \( \beta \) of 0.20) differences of at least 4–5 mm Hg of 6-month diastolic blood pressure changes for the 14 selected pairwise comparisons. The sample size calculations are described elsewhere.11

Comparisons of baseline variables for all nine intervention groups were performed using \( \chi^2 \) tests for categorical variables and one-way analysis of variance for continuous variables. The mean differences for the 14 selected questions were compared using the Bonferroni multiple comparison procedures.17 All analyses were by intention to treat. Because of multiple comparisons, a \( p < 0.0036 \) (0.05/14) was considered statistically significant.

Multiple linear regression analyses were also performed to account for imbalances among the treatment groups in baseline factors and to explore possible interaction effects.17

Measurement

Blood pressures were measured, using American Heart Association guidelines, by staff who were trained and certified according to the Hypertension Detection and Follow-up Program criteria.18 The random zero mercury sphygmomanometer was used to minimize observer bias for cutpoints. Blood pressure observers were blinded to participants’ diet assignments in one out of the three clinical centers. All observers were blinded to drug therapy as previously explained. The reasons for incomplete diet blinding were due to logistical and budgetary considerations. Blood pressures were measured after the participant had been seated quietly for at least 5 minutes. The average of two readings of the fifth-phase diastolic blood pressure was used in all analyses. Participants normally took their medication in the morning and made their clinic visits at various times during the day.

Dietary intake was assessed with 3-day food records at baseline and 6 months. Participants were instructed in keeping an accurate record and completed records were reviewed in detail by nutritionists. Additional assessment of sodium and potassium intake was provided by data from 24-hour urine samples collected at baseline and at 6 months.

Participants underwent a standard battery of tests at baseline and 6 months including the measurement of glucose, uric acid, creatinine, cholesterol, high density lipoprotein (HDL) cholesterol, triglycerides, and electrolytes. These analyses were performed at a central laboratory. In addition, at baseline and 6 months serum potassium levels were monitored to evaluate the need for potassium supplements.

Results

At baseline, all nine groups were compared for almost all the major variables measured (Table 1). There were no statistically significant differences between groups with regard to these variables except gender. There were more men than women in each group except the usual diet/placebo and weight loss diet/chlorthalidone groups.

Participation Status

Participation was at a high level throughout the first 6 months of the trial for clinic attendance, completion of food records, procedures completed, and medication taken. The end point blood pressure was obtained after 6 months of intervention (range, 5–8 months; mean, 6.3 months). The mean times from the baseline visit and randomization to the end point blood pressure determination were 7.5 and 6.8 months, respectively. Twenty-four participants had
TABLE 2. Change in Selected Variables From Baseline to 6 Months by Treatment Group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Usual</th>
<th>Weight loss</th>
<th>Low Na/high K</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P</td>
<td>C</td>
<td>A</td>
</tr>
<tr>
<td>Change* in weight (kg)</td>
<td>-0.71</td>
<td>-1.52</td>
<td>-0.53</td>
</tr>
<tr>
<td>Mean</td>
<td>90</td>
<td>87</td>
<td>87</td>
</tr>
<tr>
<td>Total SD=4.67</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change* in urinary sodium (mmol/day)</td>
<td>9.06</td>
<td>12.05</td>
<td>-4.56</td>
</tr>
<tr>
<td>Mean</td>
<td>82</td>
<td>72</td>
<td>76</td>
</tr>
<tr>
<td>Total SD=72.68</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change* in urinary potassium (mmol/day)</td>
<td>0.78</td>
<td>12.08</td>
<td>-0.65</td>
</tr>
<tr>
<td>Mean</td>
<td>82</td>
<td>72</td>
<td>76</td>
</tr>
<tr>
<td>Total SD=30.36</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change* in systolic blood pressure (mm Hg)</td>
<td>-10.34</td>
<td>-17.41</td>
<td>-15.06</td>
</tr>
<tr>
<td>Mean</td>
<td>90</td>
<td>87</td>
<td>87</td>
</tr>
<tr>
<td>Total SD=15.95</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

P, placebo;  C, chlorthalidone;  A, atenolol.

*Change equals 6-month value minus baseline value.

withdrawn or been lost to follow-up by the first month visit and 67 participants had their blood pressures measured outside the 5-8-month window. Therefore, 787 participants had available blood pressure data. The main reason for withdrawal or lost to follow-up was patient refusal (about 90%).

Compliance with drug therapy was assessed by the use of monthly pill counts. By the end of the 6 months, 92% of the participants with pill counts were taking more than 80% of their assigned medication. However, all the 787 participants did not remain on their initial drug assignment over the 6-month study. Among the placebo-assigned participants, 20.0% of those in the usual diet group, 10.0% of those in the weight loss group and 16.5% of those in the low sodium/high potassium group were on additional therapy (double-blind step-up or open-label medication) by the end of 6 months. For those participants assigned to chlorthalidone and atenolol, 2.7% and 3.0%, respectively, were on additional therapy and 3.4% and 2.6%, respectively, were on no active medication.

Attainment of Dietary Goals

Table 2 presents the change in weight and 24-hour urinary sodium and potassium for each of the nine subgroups. There was an average loss of 4.7 kg in the total weight loss group with considerable variation among the three drug subgroups. The low sodium/high potassium group had an average decrease in urinary sodium of 27.4 mmol/day and an average increase of urinary potassium of 10.9 mmol/day. The placebo subgroup within the low sodium/high potassium diet group had the greatest reduction in urinary sodium and the greatest increase in urinary potassium, compared with the chlorthalidone and atenolol subgroups within that diet.

Changes in Diastolic Blood Pressure

Table 3 presents the change in diastolic blood pressure from baseline to 6 months for the six main groups (in the margin) and all nine subgroups (cells).
The differences among marginal changes are displayed in Table 4. The largest subgroup changes were observed in the weight loss/chlorthalidone and weight loss/atenolol groups. The diet/placebo subgroups all exhibited about the same drop in blood pressure. Two-way analysis of variance shows that the main effects of drug and diet were highly significant \((p<0.001)\), but there was no significant interaction effect.

TAIM was designed to look at diastolic blood pressure changes. Analysis of the systolic blood pressure changes showed similar trends. (See Table 2 for results.) The percent of participants at or below selected levels of diastolic blood pressure by 6 months was also analyzed. The greatest percent below 90 mm Hg were in the weight loss/chlorthalidone groups (93%) and the weight loss/atenolol groups (92%). The usual diet/placebo group had a response rate of 71% at or below 90 mm Hg by 6 months. If all participants placed on an active anti-hypertensive drug during therapy were removed from this group, the response rate would be 69%.

Table 4 presents the drug and diet comparisons. To achieve statistical significance here, \(p=0.05/3=0.017\) must be achieved. In terms of blood pressure reduction, the weight loss diet was significantly better than usual diet, and either diuretic or \(\beta\)-blocker was significantly better than placebo. The weight loss diet lowered blood pressure more than the low sodium/high potassium diet \((p=0.019)\). The usual diet and low sodium/high potassium diet were statistically indistinguishable in terms of blood pressure reduction as was the diuretic and \(\beta\)-blocker.

The first set of comparisons were designed to compare diet with low dose drug therapy. \(\beta\)-Blocker therapy alone achieved a significantly greater reduction of diastolic blood pressure than did a low sodium diet alone \((\text{difference}=4.5 \text{ mm Hg}, \ p=0.001)\) and achieved a substantial reduction of diastolic blood pressure when compared with a weight loss diet alone \((\text{difference}=3.7 \text{ mm Hg}, \ p=0.006)\), although it was not significant at the prespecified level. Diuretic therapy was statistically equivalent to either a low sodium diet \((\text{difference}=2.9 \text{ mm Hg}, \ p=0.03)\), or a weight loss diet \((\text{difference}=2.0 \text{ mm Hg}, \ p=0.13)\), although the blood pressure reduction was greater than in either diet.

The next set of comparisons asked whether dietary therapy adds anything to a pharmacological regimen. Weight loss in combination with diuretic therapy added significantly to the lowering of diastolic blood pressure \((\text{difference}=4.3 \text{ mm Hg}, \ p=0.002)\). Weight loss in combination with \(\beta\)-blocker therapy also added to blood pressure-lowering \((\text{difference}=2.4 \text{ mm Hg}, \ p=0.07)\) but less so, and the result was not significant. The low sodium/high potassium diet had little blood pressure-lowering effect when added to diuretic therapy and essentially none when added to \(\beta\)-blocker therapy.

For participants receiving diuretic therapy, a weight loss diet helped lower blood pressure more than a low sodium/high potassium diet \((\text{difference}=2.9 \text{ mm Hg}, \ p=0.03)\). Also, for participants on \(\beta\)-blocker therapy, a weight loss diet also helped lower blood pressure more than a low sodium/high potassium diet \((\text{difference}=2.1 \text{ mm Hg}, \ p=0.12)\).

For participants on dietary therapy, adding a diuretic or \(\beta\)-blocker was equivalent in terms of blood pressure reduction. For participants on both active drug and active diet, weight loss diet plus either diuretic or \(\beta\)-blocker appeared to be better at lowering blood pressure than a low sodium/high potassium diet plus diuretic \((\text{difference}=2.6 \text{ mm Hg}, \ p=0.05)\) or \(\beta\)-blocker \((\text{difference}=2.3 \text{ mm Hg}, \ p=0.08)\), but neither of the results was statistically significant. All the above comparisons were examined by clinical center and by excluding crossovers (see Participation Status), and no substantial differences from the main results were noted.

To account for possible imbalances in the baseline covariates, a multiple linear regression analysis was performed. This analysis used 6-month change in blood pressure as the dependent variable and the following as independent variables: baseline diastolic blood pressure, age, sex, race, history of prior anti-hypertensive medication use, atenolol group, chlorthalidone group, weight loss group, and the low sodium/high potassium group.

Baseline diastolic blood pressure, sex, race, and assignment to either of the two active drug groups or

<table>
<thead>
<tr>
<th>Comparisons</th>
<th>Difference of means (mm Hg)</th>
<th>95% confidence intervals (mm Hg)</th>
<th>(p) value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diet</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Usual as compared with weight loss</td>
<td>2.48</td>
<td>(0.66, 4.30)</td>
<td>0.001</td>
</tr>
<tr>
<td>Usual as compared with low sodium/high potassium</td>
<td>0.71</td>
<td>(-1.11, 2.53)</td>
<td>0.347</td>
</tr>
<tr>
<td>Low sodium/high potassium as compared with weight loss</td>
<td>1.77</td>
<td>(0.05, 3.59)</td>
<td>0.019</td>
</tr>
<tr>
<td><strong>Drug</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo as compared with chlorthalidone</td>
<td>4.44</td>
<td>(2.62, 6.26)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Placebo as compared with atenolol</td>
<td>5.10</td>
<td>(3.28, 6.92)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Chlorthalidone as compared with atenolol</td>
<td>0.67</td>
<td>(-1.15, 2.49)</td>
<td>0.38</td>
</tr>
</tbody>
</table>

Difference of means is the difference of first and second groups listed in Comparisons column. Positive numbers indicate second group had greater reduction in diastolic blood pressure. Confidence intervals take into account multiple comparisons, \(n=787\).
the weight loss group were significant factors in determining 6-month blood pressure change. Blood pressure decreased in women more than men and in whites more than blacks. Controlling for all other variables in the model, assignment to weight loss group yielded a decrease of 2.3 mm Hg. Again, controlling for all other variables in the model, a person with a baseline diastolic blood pressure of 100 mm Hg would have a 5.9 mm Hg greater decrease in diastolic blood pressure when compared with a person with a baseline diastolic blood pressure of 90 mm Hg. A similar regression analysis for systolic blood pressure was performed, and this showed age as well as baseline systolic blood pressure, sex, race, and assignment to either of the two active drug groups or the weight loss group to be significant factors in determining 6-month systolic blood pressure change.

Further regression modeling revealed a race-diet-drug interaction and a sex-drug interaction. Blacks on the weight loss/chlorthalidone intervention reduced their diastolic blood pressure 5.2 mm Hg more than whites in the same intervention. Women taking chlorthalidone reduced their diastolic blood pressure by 5.0 mm Hg more than men on chlorthalidone.

Discussion

The desirability of antihypertensive therapy for mild hypertensive patients is emphasized by the recent report from the Tecumseh Blood Pressure Study.19 Abnormalities in target organs and coronary risk factors were significantly more frequent in individuals with borderline hypertension than in normotensive individuals. The question of which drug or diet, or combination of the two, was best for treatment of mild hypertension was the focus of the TAIM study. It was designed to compare changes in blood pressure among nine programs for the treatment of mild hypertension. Both chlorthalidone and atenolol were effective blood pressure-lowering agents in the study. Assignment to the weight reduction group also lowered blood pressure. Assignment to the sodium restriction/increased potassium diet group did not substantially affect blood pressure.

The addition of weight loss to diuretic therapy provided the clearest evidence of benefit from combined diet and drug therapy. In addition, the weight loss/atenolol group had greater reduction in blood pressure than the usual diet/atenolol group. The attained 6-month blood pressure change of the participants on the weight loss/atenolol combination was the same as the 6-month blood pressure change of those on the weight loss/chlorthalidone combination. Yet, atenolol produced a somewhat greater blood pressure drop as sole therapy than chlorthalidone did. A partial explanation for the difference in the additive effects of weight loss between chlorthalidone and atenolol may be found in the differences in weight loss among these groups. Those assigned to weight loss plus chlorthalidone lost an average of 6.9 kg, whereas those assigned to weight loss plus atenolol lost 3.0 kg. Part of the difference in weight loss between the two groups may be due to the volume reduction effect from the diuretic.

One interesting result in TAIM should be noted. The placebo/usual diet group had a mean drop in diastolic blood pressure of 8.0 mm Hg. This could be due to a combination of the following: 1) 20% of this group was on step-up or open-label therapy by the end of the 6 months, 2) regression to the mean, and 3) habituation to the site of blood pressure determination.

The low sodium/high potassium diet did not lower diastolic blood pressure as much as the weight loss diet. This could be due to the lack of effect of the former diet. Another possible explanation is that the TAIM participants were healthy volunteers with a relatively low baseline urinary sodium of 133 mmol/day (compared with 180 mmol found in Framingham20). In addition, over the 6-month period, those assigned to the low sodium/high potassium diet achieved only a modest decrease in sodium intake and excretion and a minimal change in potassium intake and excretion.

The relatively minimal diastolic blood pressure reduction in the low sodium/high potassium group in TAIM is similar to the effect noted by Logan,21 whose participants lowered their sodium excretion from 164 mmol/day to 121 mmol/day but did not experience a change in blood pressure. However, other studies wherein the sodium excretion rate was reduced to 50–90 mmol/day did demonstrate a blood pressure-lowering effect. The Australian National Health and Medical Research Study22 showed that lowering the sodium excretion rate to 90 mmol/day had a significant lowering effect on diastolic and systolic blood pressure. McGregor et al23 showed that long-term reduction of the urinary sodium to about 50 mmol/day resulted in a significant drop in blood pressure. However, to get to 50 mmol/day requires special dietary reduction such as salt-free products that are not generally available. Therefore, the inconclusive results of the low sodium/high potassium diet may relate to two factors: 1) present day volunteers for a hypertension study may already have made major changes in their sodium intake; and 2) it is very difficult to achieve a sodium intake below 100 meq/day without major modification of the usual American diet.

Previous studies have shown that weight reduction can lower diastolic blood pressure. MacMahon et al24 reported an average weight loss of 6.8 kg to be an effective blood pressure–reducing maneuver. A pooled analysis of five weight loss diet trials25 showed a 3.1 mm Hg drop in diastolic blood pressure with an average weight loss of 9.2 kg. However, participants in the study by Haynes et al26 lost an average of 4.2 kg and had no change in blood pressure compared with control patients at 6 months. TAIM participants assigned to the weight loss group lost an equivalent amount, 4.7 kg, without significant hypotensive benefit in the placebo-assigned participants. However, this loss combined with a diuretic or ß-blocker low-
ered blood pressure more than either drug treatment alone or weight reduction alone.

The overall picture that emerges from the TAIM study of diet change and antihypertensive medication is 1) a successful weight loss program provided additional major benefit to thiazide or β-blocker antihypertensive therapy in terms of blood pressure reduction; 2) a diet that yielded a modest reduction in sodium intake and a minimal increase in potassium intake in a group with a low salt intake resulted in little additional blood pressure reduction compared with the diet plus active drug; 3) in a regression analysis, weight loss alone produced a beneficial effect on blood pressure, although the beneficial effect was less marked than the addition of weight loss to thiazide-type diuretics; and 4) the evaluation of therapy for mild hypertension requires consideration of three components—blood pressure, risk factors, quality of life—and, if possible, cost. The long-term implications of these observations are currently being assessed.

Appendix

Participating Institutions and Investigators

Birmingham Clinical Center (University of Alabama at Birmingham): Albert Oberman, MD (Principal Investigator); James M. Racynski, PhD; James R. Kitts, PA-C; Heidi Hataway, MS, RD; Gary R. Cutter, PhD, Glenn Hughes, PhD.

Jackson Clinical Center (University of Mississippi): Herbert G. Langford, MD (Principal Investigator); Sheila Corrigan, PhD; Lori Danks, BS, RN; Stephanie Jennings, MS, RD.

New York Clinical Center (Albert Einstein College of Medicine): M. Donald Blaufox, MD, PhD (Principal Investigator); Maureen Magnani, RN; Judy Stern, MS, RD; Gail Miller, RN.

Nutrition and Behavioral Core (Albert Einstein College of Medicine): Sylvia Wasserthiel-Smoller, PhD (Director); Judith Wylie-Rosett, EdD, RD; Charles Swencionis, PhD; Yvonne P. Raiford.

Coordinating Center (University of Texas Health Science Center at Houston, School of Public Health): C. Morton Hawkins, ScD (Principal Investigator); Barry R. Davis, MD, PhD; Neal Zimbaldi, BS; Maura O’Connell Knerr, MS.

Central Laboratory (SmithKline Bio-Science Laboratories, Van Nuys, Calif.): Sue Bird.

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Plasma Renin Activity Laboratory (Albert Einstein College of Medicine): Hyo-Bok Lee, MS.

Trial of Antihypertensive Interventions and Management Policy Advisory Committee: Lloyd Filer, MD; W. Gordon Walker, MD; Max Halperin, PhD (deceased); Elisabeth McSherry, MD; Pat Elmer, MS; Thomas P. Blaszkowski, PhD.

National Heart, Lung, and Blood Institute: Thomas P. Blaszkowski, PhD; Jeffrey Cutler, MD.

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