Weight Loss and Reduction of Blood Pressure and Hypertension

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The article in the current issue of Hypertension by Aucott et al.\(^1\) raises some interesting questions. Does weight loss in the long term (ie, >3 years) result in a decrease in blood pressure (BP) levels? Is there a difference in the effect of weight loss on decreased systolic BP (SBP) versus diastolic BP (DBP)? They combined 8 clinical trials and 8 cohort studies and tried to evaluate the linear relationship between change in BP and weight change. Overall, there was only a 2.8-kg weight loss, which resulted in a 1.9-mm decrease in DBP and 2.9 mm for SBP, neither of which were significant. Most important, the association between decrease in SBP with weight loss dissipated substantially over time (that is, >3 years from the onset of the study). The conclusion is that in long term, weight loss had little effect on change in BP levels.

Weight loss has been a recommended nonpharmacological therapy for reducing BP levels for many years. Before the introduction of effective antihypertensive drug therapy, reduction of salt in the diet, weight loss, and mild sedation were recommended therapies for patients with nonmalignant hypertension.\(^2\)

Previous meta-analyses of clinical trials on the effects of weight reduction on BP concluded that weight loss is important in both the prevention and treatment of hypertension.\(^3\) Most of the studies in the meta-analysis had relatively small sample sizes and short duration, usually <1 year.

Is there really no health benefit of weight loss with decreased BP in the long term? To test such a hypothesis, participants would have to lose a substantial amount of weight (ie, >10 kg), have good BP measures before and after weight loss, a comparison group without substantial weight loss but having similar diet except for the differences in caloric intake or energy expenditure (ie, physical activity), and ideally not be on any antihypertensive drug therapy. Unfortunately, such a study does not exist. Long-term nonpharmacological intervention trials have been unsuccessful in maintaining weight loss (ie, >10 kg) for longer than 3 to 4 years after. Many of these trials have also included other dietary changes, especially sodium reduction, increases in fruits and vegetables (ie, potassium, etc.), and exercise. Thus it is difficult to disentangle the effects of changes in the quality versus the quantity of the diet or amount of exercise and weight loss.\(^4\) The most puzzling study has been the 8-year follow-up of the Swedish Obese Subjects study. Thirty-four-six obese participants had gastric surgery to reduce obesity and were matched with 346 obese controls that did not have surgery. Over 8 years, there was no weight loss in the controls and a substantial 20.1-kg weight loss for the surgical participants. There was a very substantial decrease in the risk of diabetes, as has been noted in other bariatric surgical studies. Surprisingly, however, there was no reduction in either the incidence of hypertension or BP levels over time between the 2 groups (ie, surgery and no surgery).\(^5\)

The Diabetes Prevention Program, a nonpharmacological weight loss exercise trial among prediabetics with BMIs averaging 33 kg/m\(^2\), reported that over an approximate 2.8-year follow-up that weight loss in the diet arm was about 5.6 kg, 2.1 kg in the metformin arm, and 0.1 in the placebo group. This resulted in a very substantial reduction in the risk of diabetes. There was a small 3.3 mm significant decrease in SBP and 3.1 mm of DBP in the diet arm, suggesting at least in the short term weight loss was associated with at least some decrease in BP.\(^6\) Longer-term follow-up from this study will be reported shortly. However, maintenance of the weight loss has been difficult as in other studies.

The PREMIER trial was a follow-up of the successful Dietary Approaches to Stop Hypertension (DASH) diet feeding trial that demonstrated substantial reductions in BP in the short term. At 18 months in the PREMIER trial, there was only a 2.3-kg difference in weight between the advice only and the active intervention groups and only about a 2-mm difference in SBP. The investigators, however, reported a linear association between the change in SBP and the change in weight, even with the small amount of weight loss. They also documented that weight loss was the most important determinant of the decrease in SBP.\(^7\) The largest of the trials, the Trials of Hypertension Prevention (TOHPS) II trial, at 36 months demonstrated only a 0.2-kg weight loss in the diet–weight loss arm versus a 1.8-kg increase in usual care (ie, an overall 2-kg difference in weight), similar to the results in the PREMIER trial above. The SBP decreased 0.8 mm in the diet–weight loss arm and increased 0.6 mm in the usual care, a 1.4-mm difference.\(^8\)

In the Women On the Move through Activity and Nutrition (WOMAN) Study, there was a 17-lb weight loss at 18 months in the lifestyle change (LC; intervention group) versus 3 lbs in the health education (HE) group.\(^9\) At 6 months there was a significant 5-mm decrease in SBP between the 2 groups, but by 18 months this had dissipated and the difference in SBP was no longer significant, 2.2 mm in the LC versus a decrease...
of 1.6 mm in the HE group in spite of the 17-lb weight difference. Recent long-term follow-up of this study to 48 months again documented a significant weight loss between the 2 groups but no significant difference in either SBP or DBP or use of antihypertensive drug therapy. Furthermore, even among a large number of women who lost more than 20 lbs over 4 years, there was little evidence of a substantial decrease in SBP or DBP (L.H. Kuller, personal communication, 2009).

How do we interpret these results? First, long-term substantial weight loss in nonpharmacological clinical trials has been extremely difficult to maintain for most participants. It is difficult to evaluate the effects of long-term weight loss on changes in BP. There is no question that weight loss reduces BP at least in the short term. There is a linear relationship between the amount of weight loss and the decrease in BP. This is most likely attributable to loss of both body water and salt and perhaps reduced sympathetic activity and insulin resistance. Unfortunately, with weight regain there is an increase in BP. Individuals who initially lose weight with a decrease in BP will then have a rise in their BP secondary to the increase in weight. Evaluating the effects of weight loss on BP change over the long term is very biased by whether the participants are losing or gaining weight or are weight stable in the short-term period before the BP measurement at end of study. Thus, an individual participating in a 5-year weight loss study who lost 50 lbs in the first 6 months and regained 20 lbs in the last 6 months before the terminal BP measurement, having a net 30-lb weight loss, may have a very different long-term change in BP baseline to 5 years from a similar participant who lost 6 lbs every year in the study, again a net of 30 lbs, but lost 6 lbs in the last year before the BP measurement. It is therefore practically impossible to evaluate the weight loss and BP change data that are provided in the literature without knowing the trajectories of weight change over time in relationship to the BP measurements of interest.

The long-term effects of weight loss on change in BP levels will be a function of the ratio of the decrease in BP with a reduction in weight as compared to the increase in BP with weight regain. It is unlikely that the slope of the BP changes for weight loss and regain are the same (ie, the amount of increase in SBP per unit increase or decrease in weight). Most likely, the composition of the diet, salt, potassium, amino acids, etc, physical activity, and host susceptibility (genetics) play important roles in determining the trajectory of BP change in relationship to both weight loss and weight regain. The key to the long-term effects of decrease in BP in relationship to weight loss may be being able to maximize substantial weight loss and prevent weight regain. Unfortunately, in the long term we have not developed effective approaches to prevent weight regain, and in almost all of the long-term studies there is substantial weight regain. Most studies do not have the resources to be able to maximize the intensive intervention necessary to continue the weight loss or to prevent weight regain over the long term. It is also possible that the long-term decrease in BP with weight loss is not really a function of the weight loss but rather the change in the diet, including the reduction of sodium or perhaps the ratio of sodium to potassium or other nutrients in the diet or exercise. There is little need for further short-term weight loss studies. All of the effort needs to be focused on how to maintain substantial weight loss in the long term and evaluate effect on risk factors (ie, BP) and clinical outcomes. Unfortunately, at the present time weight loss is not a viable option for the treatment, or even the prevention, of hypertension among most overweight and obese individuals.

Weight loss in overweight and obese individuals to prevent elevated BP has been a nice recommendation that has been around for the last 50 years. Is it time to admit defeat and recommend surgical pharmacological therapies? Probably not. Rather, it is time to focus the efforts on how to maintain substantial weight loss in the long term and hopefully demonstrate an effective approach to decrease BP. We know little more now than 50 years ago about long-term weight loss and decrease in BP.

Disclosures

None.

References


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Hypertension. published online August 24, 2009;
Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2009 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://hyper.ahajournals.org/content/early/2009/08/24/HYPERTENSIONAHA.109.138891.citation

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