

Achieving Weight Loss and Hypertension Control Among Obese Adults: A US Multidisciplinary Group Practice Observational Study

Aaron K Ho,¹ Christie M. Bartels,^{1,2} Carolyn T. Thorpe,^{3,4} Nancy Pandhi,^{2,5} Maureen A. Smith,^{2,5-7} and Heather M. Johnson^{1,2}

BACKGROUND

Among adults with hypertension, obesity independently contributes to cardiovascular disease. Weight loss and hypertension control are critical to reduce cardiovascular events. The purpose of this study was to evaluate rates and predictors of achieving weight loss among adults who achieved hypertension control within 1 year of developing incident hypertension.

METHODS

Retrospective electronic health record analysis was performed of ≥ 18 year olds with a body mass index ≥ 30.0 kg/m², who received regular primary care from 2008 to 2011 and achieved hypertension control. Exclusions were less than 60 days follow-up, prior hypertension diagnosis, prior antihypertensive prescription, or pregnancy. The primary outcome was clinically significant weight loss (≥ 5 kg); the secondary outcome was modest (2.0–4.9 kg) weight loss. Multinomial logistic regression identified predictors of achieving weight loss (≥ 5 or 2.0–4.9 kg) compared to no significant weight loss (< 2 kg).

RESULTS

Of the 2,906 obese patients who achieved hypertension control, 72% ($n = 2,089$) did not achieve at least 2.0 kg weight loss. Overall, 12% ($n = 351$) achieved ≥ 5 kg weight loss. Young adults (18–39 year olds; odds ratio (OR): 2.47, 95% confidence interval (CI): 1.63–3.47), middle-aged adults (40–59 year olds; OR: 2.32, 95% CI: 1.59–3.37), and patients prescribed antihypertensive medication (OR: 1.37, 95% CI: 1.07–1.76) were more likely to achieve clinically significant weight loss and hypertension control. Age remained a significant predictor for 2.0–4.9 kg weight loss.

CONCLUSIONS

Despite achieving hypertension control, the majority of obese patients did not achieve clinically significant weight loss. Effective weight loss interventions with dedicated hypertension treatment are needed to decrease cardiovascular events in this high-risk population.

Keywords: blood pressure; hypertension; obesity; primary care; weight management.

doi:10.1093/ajh/hpw020

The relationship between obesity and the development of incident hypertension has been recognized since the Framingham Heart Study nearly 50 years ago.^{1,2} In the United States, the prevalence of adult obesity (body mass index (BMI) ≥ 30 kg/m²) has increased steadily over the past 3 decades. Currently, more than one third of US adults are now classified as obese, with over \$200 billion in health-care costs attributable to obesity and its resultant complications.^{3,4} It is estimated that at least two thirds of hypertension cases are directly attributed to obesity.⁵

Hypertension and obesity are each well-established independent risk factors for morbidity (e.g., heart failure and stroke) and mortality.^{6,7} Cardiovascular disease–related and all-cause mortality rates are increased in obese adults compared to normal-weight adults, with an estimated 5 years of life lost among patients with a higher BMI.^{8–10} However, weight loss of at least 5 kg has been shown to significantly reduce systolic and diastolic blood pressure by an average of 4.44 and 3.57 mm Hg, respectively.¹¹ For smaller weight reductions, there is an estimated average decrease of

Correspondence: Heather M. Johnson (hm2@medicine.wisc.edu).

Initially submitted January 7, 2016; date of first revision January 25, 2016; accepted for publication February 7, 2016; online publication February 24, 2016.

¹Department of Medicine, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin, USA; ²Health Innovation Program, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin, USA; ³Health Services Research and Development, Veterans Affairs Pittsburgh Healthcare System, Pittsburgh, Pennsylvania, USA; ⁴Department of Pharmacy & Therapeutics, School of Pharmacy, University of Pittsburgh, Pittsburgh, Pennsylvania, USA; ⁵Department of Family Medicine, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin, USA; ⁶Department of Population Health Sciences, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin, USA; ⁷Department of Surgery, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin, USA.

© American Journal of Hypertension, Ltd 2016. All rights reserved.
For Permissions, please email: journals.permissions@oup.com

1.05 mm Hg (systolic) and 0.92 mm Hg (diastolic) per kilogram of weight loss.¹¹

An ongoing focus of lifestyle modifications is needed to lower blood pressure and address negative hemodynamic and metabolic abnormalities among obese patients with hypertension.^{12–14} Even with initial hypertension control, patients may require more and/or higher doses of medication to maintain control with ongoing obesity and possibly greater weight gain. Therefore, the purpose of this study was to evaluate rates and predictors of achieving weight loss among an obese population that achieved hypertension control within 1 year of developing incident hypertension. Understanding characteristics that contribute to successful weight loss will inform the development of targeted interventions for this high-risk population.

METHODS

Sample

This study was approved by the University of Wisconsin–Madison Health Sciences Institutional Review Board with a waiver of consent. For our sample, we first identified patients who met established criteria from the Wisconsin Collaborative for Healthcare Quality (WCHQ)^{15,16} for being “currently managed” in a large, midwestern, multidisciplinary academic group practice between 1 January 2008 and 31 December 2011. Per WCHQ criteria, patients had to have ≥ 2 billable office encounters in an outpatient, nonurgent, primary care setting or 1 primary care and 1 office encounter in an urgent care setting (regardless of diagnosis code)

in the 3 years prior to study enrollment, with at least one of those visits in the prior 2 years.¹⁷ To achieve a sample with *incident hypertension*, patients’ records were then evaluated for the first date that Seventh Report of the Joint National Committee (JNC 7) on Prevention of blood pressure criteria for a hypertension diagnosis¹⁸ were met based on the Tu criteria (401.x (essential hypertension), 402.x (hypertensive heart disease), 403.x (hypertensive renal disease), 404.x (hypertensive heart and renal disease), 405.x (secondary hypertension)).^{19,20} Blood pressure eligibility criteria were based on electronic health record data: (i) ≥ 3 elevated outpatient blood pressure measurements (systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg) from 3 separate dates, ≥ 30 days apart but within a 2-year span¹⁸ or (ii) 2 elevated blood pressures (systolic blood pressure ≥ 160 mm Hg or diastolic blood pressure ≥ 100 mm Hg), ≥ 30 days apart but within a 2-year period. The blood pressures did not need to be from billable encounters. If more than one blood pressure was taken at a visit, the average was used.¹⁸ A total of 14,974 adults met both the WCHQ and incident hypertension diagnosis criteria (Figure 1).

Our study focus in this analysis was to assess weight loss within 1 year (12 months) of developing incident hypertension and achieving hypertension control; therefore, we excluded patients who did not achieve hypertension control within 12 months of developing incident hypertension (Figure 1). A separate analysis will address weight management among adults not achieving hypertension control. Hypertension control was defined as 3 consecutive normal blood pressures ($<140/90$ mm Hg for all patients)¹⁸ on 3 separate ambulatory clinic visit dates.^{6,23} To account for blood

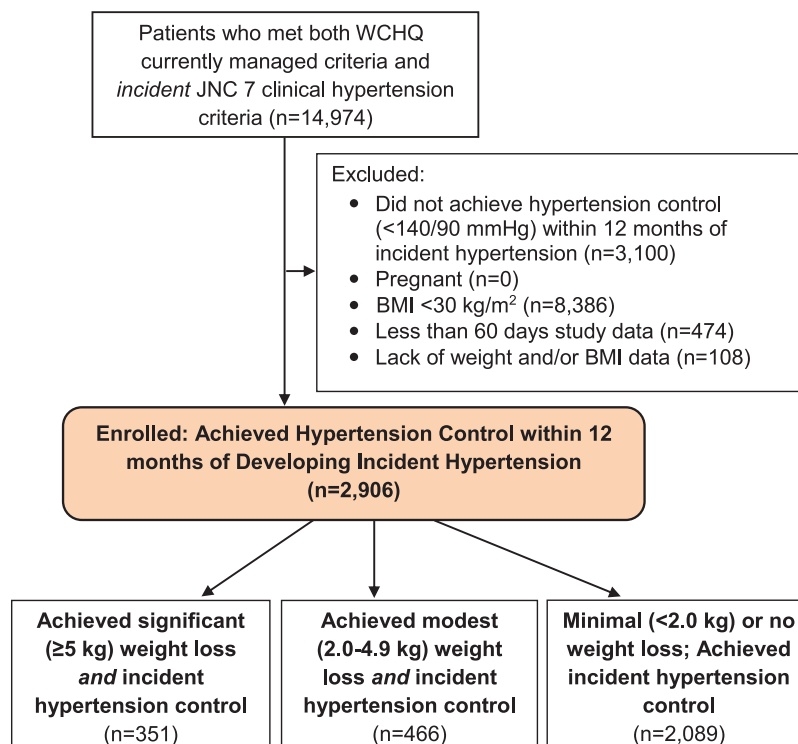


Figure 1. Study sample: enrollment and analysis. Abbreviations: JNC, Joint National Committee; WCHQ, Wisconsin Collaborative for Healthcare Quality.

pressure variability and the unavailability of 24-hour ambulatory blood pressure readings, we used clinic blood pressure readings across consecutive visits.

Patients who were pregnant during the study period and/or 1 year prior to study entry were excluded using a modified Manson approach.²⁴ Additional exclusion criteria included patients with a BMI $<30 \text{ kg/m}^2$ (i.e., nonobese), lack of weight and/or BMI data at baseline or during follow-up (at least 3 ambulatory visits with recorded weights were required during baseline and follow-up), or less than 60 days of study follow-up data. A minimum of 60 days follow-up was required to assess weight loss in this obese population, based on the American Heart Association's recommendations for a gradual weight loss of 1–2 pounds per week until a healthy weight is achieved.²⁵ All patients had up to 365 days (1 year) of follow-up time to achieve weight loss. After applying these exclusion criteria, 2,906 patients met study criteria. The 365 days prior to study enrollment was considered the “baseline period” where patients' baseline comorbidities and utilization were assessed.

Outcome variables

The primary outcome variable of weight loss within 1 year of developing incident hypertension was constructed based on the prior data regarding clinically significant outcomes associated with weight loss of varying degrees. Prior research has demonstrated that a net weight reduction of 5.1 kg reduced systolic blood pressure by 4.44 mm Hg and diastolic blood pressure by 3.57 mm Hg.¹¹ On a population level, these blood pressure reductions decrease population rates of mortality from stroke and coronary artery disease.¹⁸ Smaller weight losses of 2–5 kg may also produce mortality benefits.^{26,27} Thus, our outcome variable was defined as a 3-category variable: (i) achievement of $\geq 5 \text{ kg}$ within 12 months of developing incident hypertension, (ii) achievement of 2.0–4.9 kg of weight loss within 12 months of developing incident hypertension, or (iii) no clinically significant weight loss within 12 months.

Explanatory variables

Patient and provider explanatory variables were selected based on prior studies.^{11,28} Patient-related factors included sociodemographics (age, sex, marital status, race/ethnicity, primary spoken language, Medicaid use during the baseline or study period), behavioral risk factors (baseline tobacco use, BMI, weight at study entry), and JNC 7 hypertension stage (Stage 1: 140–159/90–99 mm Hg; Stage 2: $\geq 160/100 \text{ mm Hg}$). Patients' comorbidities were assessed using established algorithms including: hyperlipidemia,²⁹ diabetes mellitus,³⁰ and a combined variable of anxiety³¹ and/or depression.^{32,33}

Patients' morbidity burden can predict healthcare utilization, which in turn may influence hypertension control rates.^{34,35} Therefore, we used the Johns Hopkins Adjusted Clinical Group (ACG) Case-Mix System (version 10.0), which assesses morbidity burden based on patient age, gender, and patterns of disease in the electronic health record to predict future healthcare resource utilization.³⁵

Additional measures of utilization included the number of baseline face-to-face primary care, specialty, and urgent care visits. Primary care visits included those to family medicine/family practice, internal medicine, and a combined category of lower prevalence specialties (obstetrics/gynecology, pediatrics/adolescent medicine) with a physician (faculty, resident, fellow), nurse practitioner, or physician assistant.

Provider explanatory variables included age, specialty, and gender. Patients were assigned to the primary care provider they saw most frequently in outpatient face-to-face Evaluation and Management visits, as reported in professional service claims.¹⁷ Provider's age, practice (primary care or specialty care), and gender were obtained from the provider group's human resource office and/or the American Medical Association (AMA) 2011 Masterfile data.

Statistical analysis

Analyses were conducted using SAS 9.1.3 (SAS Institute, Inc., Cary, NC) and Stata/MP 13.1 (StataCorp LP, College Station, TX). Only patients with complete covariates were included. Categorical variables were summarized using percentages. Continuous variables were summarized using means (SD). We compared patient- and provider-descriptive characteristics based on the presence or absence of antihypertensive medication using chi-square and Wilcoxon rank-sum tests. Multinomial logistic regression models estimated adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for achieving each outcome: (i) significant ($\geq 5 \text{ kg}$) weight loss and (ii) modest (2.0–4.9 kg) weight loss, within 12 months of an incident hypertension diagnosis, compared to no significant weight loss ($<2 \text{ kg}$). Robust estimates of the variance were used to account for clustering at the provider level. Given the relationship between age and BMI,³⁶ interaction testing was performed between age and BMI, with and without adjustment for antihypertensive medication use.

RESULTS

Sample characteristics

A total of 2,906 patients met inclusion criteria (Figure 1). Table 1 summarizes the study population according to achieving hypertension control, with or without blood pressure medication, within 12 months of an incident hypertension diagnosis. Overall, 33% ($n = 956$) achieved hypertension control without antihypertensive medication; among this group, 70% ($n = 669$) had Stage 1 (mild) hypertension and 47% were obese Class I (BMI: 30.0–34.9 kg/m^2). Patients requiring blood pressure medication to achieve hypertension control were more likely older, had a higher BMI, and a diabetes mellitus diagnosis (all $P < 0.05$). Patients with a higher morbidity burden (ACG score) or more primary care, specialty, and urgent care visits were more likely to be prescribed antihypertensive medication. Provider factors (age, specialty, gender) were not significantly associated with the presence or absence of antihypertensive medication to achieve hypertension control.

Table 1. Patient and provider characteristics of obese adults achieving incident hypertension control (*n* = 2,906)

	All patients, <i>n</i> = 2,906	Without antihypertensive medication, <i>n</i> = 956	With antihypertensive medication, <i>n</i> = 1,950	<i>P</i> value
Baseline patient characteristics				
Age, years, <i>M</i> (SD)	48 (13)	50 (13)	47 (13)	<0.001
18–39 years, <i>n</i> (%)	822 (28)	204 (21)	618 (32)	<0.001
40–59 years, <i>n</i> (%)	1,524 (52)	538 (56)	986 (51)	<0.001
≥60 years, <i>n</i> (%)	560 (19)	214 (22)	346 (18)	<0.001
Female, <i>n</i> (%)	1,669 (57)	523 (55)	1,146 (59)	0.04
Marital status, <i>n</i> (%)				0.26
Married/partnered	1,736 (60)	585 (61)	1,151 (59)	
Not married	1,170 (40)	371 (39)	799 (41)	
Race/ethnicity, <i>n</i> (%)				0.76
White	2,525 (87)	828 (87)	1,697 (87)	
Non-White ^a	381 (13)	128 (13)	253 (13)	
Primary spoken language, <i>n</i> (%)				0.77
English	2,817 (97)	928 (97)	1,889 (97)	
Non-English	89 (3.1)	28 (2.9)	61 (3.1)	
On Medicaid ever ^b , <i>n</i> (%)	356 (12)	129 (13)	227 (12)	0.15
Tobacco use, <i>n</i> (%)				0.33
Current tobacco use	456 (16)	141 (15)	315 (16)	
Never/former tobacco use	2,450 (84)	815 (85)	1,635 (84)	
BMI, kg/m ² , <i>M</i> (SD)	37 (6.1)	37 (6.5)	36 (5.9)	0.02
BMI obesity classes, <i>n</i> (%)				0.10
Obese Class I (BMI 30.0–34.9 kg/m ²)	1,453 (50)	452 (47)	1,001 (51)	
Obese Class II (BMI 35.0–39.9 kg/m ²)	777 (27)	275 (29)	502 (26)	
Obese Class III (BMI ≥ 40.0 kg/m ²)	676 (23)	229 (24)	447 (23)	
Weight, kg, <i>M</i> (SD)	107 (21)	108 (21)	106 (20)	0.07
JNC 7 stage of hypertension at baseline, <i>n</i> (%)				<0.001
Stage 1: 140–159/90–99 mm Hg	2,327 (80)	645 (67)	1,682 (86)	
Stage 2: ≥160/≥100 mm Hg	579 (20)	311 (33)	268 (14)	
Baseline comorbid conditions, <i>n</i> (%)				
Hyperlipidemia	703 (24)	233 (24)	470 (24)	0.87
Diabetes mellitus	258 (8.9)	102 (11)	156 (8.0)	0.02
Anxiety and/or depression	622 (21)	149 (16)	473 (24)	<0.001
ACG score, young, <i>M</i> (SD)	1.7 (1.7)	1.9 (1.8)	1.5 (1.3)	<0.001
Ambulatory visits in baseline period, <i>M</i> (SD)				
Primary care	3.1 (2.6)	3.3 (2.7)	2.5 (2.2)	<0.001
Specialty care	2.7 (3.3)	3.1 (3.5)	2.0 (2.6)	<0.001
Urgent care	0.64 (1.2)	0.72 (1.3)	0.47 (0.93)	<0.001
Baseline provider characteristics				
Female provider, <i>n</i> (%)	1,412 (49)	474 (50)	938 (48)	0.40
Provider age ^c , <i>M</i> (SD)	45 (11)	45 (10)	45 (11)	0.55

Table 1. *Continued*

	All patients, <i>n</i> = 2,906	Without antihypertensive medication, <i>n</i> = 956	With antihypertensive medication, <i>n</i> = 1,950	<i>P</i> value
Provider practice, <i>n</i> (%)				0.59
Primary care ^d	2,592 (89)	857 (90)	1,735 (89)	
Specialty care ^e	314 (11)	99 (10)	215 (11)	

Bold values denote statistical significance ($P < 0.05$) in comparison with the reference. Abbreviations: ACG, Adjusted Clinical Group Case-Mix Assessment System; BMI, body mass index; JNC 7 stage of hypertension, Joint National Committee, severity of blood pressure elevation at study entry.

^aNon-White (% is of entire study population): African–American (5.95%); Hispanic–Latino (2.65%); Asian (0.83%); Native Hawaiian–Pacific Islander (0.72%); American Indian–Alaskan Native (0.45%); Unknown (2.51%). ^bOn Medicaid at any point during the baseline or study period. ^cAMA (American Medical Association) is the source for the raw physician data (provider ages only); statistics, tables, or tabulations were prepared by User-Customer (M.A.S.; PI: H.M.J.) using 2011 AMA Masterfile data. ^dPrimary care = family medicine/family practice, internal medicine, pediatrics/adolescent medicine, obstetrics/gynecology. ^eSpecialty care = endocrine, cardiology, nephrology.

Table 2. Predictors of achieving hypertension control weight loss, adjusted ORs, and 95% CIs (*n* = 817)

Variable	>5 kg weight loss, <i>n</i> = 351		2.0–4.9 kg weight loss, <i>n</i> = 466	
	Adjusted OR (95% CI)	<i>P</i> value	Adjusted OR (95% CI)	<i>P</i> value
Patient age, categories				
18–39 years	2.47 (1.63–3.74)	<0.001	1.79 (1.27–2.51)	0.001
40–59 years	2.32 (1.59–3.37)	<0.001	1.84 (1.36–2.49)	<0.001
≥60 years (reference)	1.00 (–)	–	1.00 (–)	–
Female	0.93 (0.73–1.18)	0.56	1.13 (0.92–1.40)	0.25
Race/ethnicity				
White	0.98 (0.70–1.37)	0.91	0.96 (0.71–1.29)	0.79
Non-White ^{a,b} (reference)	1.00 (–)	–	1.00 (–)	–
BMI tertiles				
BMI tertile 1 (reference)	1.00 (–)	–	1.00 (–)	–
BMI tertile 2	1.09 (0.82–1.44)	0.56	0.92 (0.72–1.17)	0.49
BMI tertile 3	0.98 (0.73–1.31)	0.89	0.69 (0.53–0.89)	0.004
ACG score, young	0.98 (0.91–1.05)	0.62	0.99 (0.93–1.05)	0.76
JNC 7 stage of hypertension				
Stage 1: 140–159/90–99 mm Hg (reference)	1.00 (–)	–	1.00 (–)	–
Stage 2: ≥160/≥100 mm Hg	1.02 (0.76–1.36)	0.91	0.82 (0.63–1.08)	0.16
Prescribed antihypertensive medication	1.37 (1.07–1.76)	0.01	1.03 (0.82–1.29)	0.80
Ambulatory visits in baseline period	0.97 (0.93–1.02)	0.27	1.00 (0.96–1.04)	0.85

Bold values denote statistical significance ($P < 0.05$) in comparison with the reference. Abbreviations: ACG, Adjusted Clinical Group Case-Mix Assessment System; BMI, body mass index; JNC 7 stage of hypertension, Joint National Committee, severity of blood pressure elevation at study entry.

^aNon-White (% is of study population with >5 kg weight loss): African–American (7.41%); Hispanic–Latino (2.28%); Asian (0.28%); Native Hawaiian–Pacific Islander (0.85%); American Indian–Alaskan Native (0.85%); Unknown (2.28%). ^bNon-White (% is of study population with 2.0–4.9 kg weight loss): African–American (6.44%); Hispanic–Latino (3.43%); Asian (0.64%); Native Hawaiian–Pacific Islander (0.43%); American Indian–Alaskan Native (0.64%); Unknown (2.36%).

Predictors of achieving hypertension control and significant (≥5 kg) weight loss

Overall, 12% (*n* = 351) of patients achieved ≥5 kg weight loss and hypertension control within 12 months of a hypertension diagnosis. After adjustment (Table 2), young adults (18–39 year olds; OR: 2.47, 95% CI: 1.63–3.47) and

middle-aged adults (40–59 year olds; OR: 2.32, 95% CI: 1.59–3.37) were more likely to achieve clinically significant weight loss and blood pressure control compared to older adults (≥60 year olds). Patients prescribed antihypertensive medication were also more likely to achieve weight loss and blood pressure control within 12 months of an incident hypertension diagnosis compared to patients without a prescription

(OR: 1.37, 95% CI: 1.07–1.76). Additionally, patients achieving ≥ 5 kg weight loss had the greatest decrease in diastolic blood pressure (≥ 6 mm Hg) compared to patients without significant weight loss ($P = 0.021$); a significant difference was not noted with systolic blood pressure ($P = 0.381$). Socioeconomic factors (Medicaid), tobacco use (current/former/never), and provider factors were not significant predictors. There was also not a significant interaction between age and BMI ($\chi^2 = 2.76$; $P = 0.598$).

Predictors of achieving modest (2.0–4.9 kg) weight loss and hypertension control

The majority (72%, $n = 2,089$) of this obese population did not achieve at least 2.0 kg weight loss. Among patients that achieved 2.0–4.9 kg weight loss, age remained a significant predictor (Table 2). Young (18–39 year olds; OR: 1.79, 95% CI: 1.27–2.51) and middle-aged (40–59 year olds; OR: 1.84, 95% CI: 1.36–2.49) adults were more likely to achieve smaller amounts of weight loss and hypertension control within 12 months of a hypertension diagnosis. Interestingly, patients in the highest BMI tertile (≥ 40 kg/m²) were less likely to achieve the smaller range of weight loss, despite achieving hypertension control. Further analysis demonstrated that patients in the highest BMI tertile were more likely to be younger (18–39 year olds, $P < 0.001$); however, we adjusted for age in the models so the significant relationship between BMI tertile and weight loss is not completely explained. Socioeconomic factors, tobacco use, and provider factors were not significant predictors. There was not a significant interaction between age and BMI ($\chi^2 = 3.08$; $P = 0.545$).

DISCUSSION

This is the first US study to assess predictors associated with both hypertension control and clinically significant weight loss in an obese population. This retrospective observational cohort analyzed comorbid cardiovascular disease risk management (i.e., obesity and hypertension) in a primary care population. Interestingly, approximately one third of patients achieved hypertension control without antihypertensive medication, but this result reflected the subpopulation in the lowest obesity class and with mild (Stage 1) hypertension. Since patients in this study had to have multiple elevated blood pressures on separate dates for study eligibility and to determine hypertension control, this finding likely demonstrates the positive health impact of lifestyle modifications rather than an incorrect diagnosis. Additionally, patients with significant weight loss (≥ 5 kg) had the greatest decrease in diastolic blood pressure (≥ 6 mm Hg). These data support our findings that young and middle-aged adults were more likely to achieve significant weight loss, which reflects a population with a higher prevalence of diastolic hypertension.³⁷

A significant concern is that the large majority (72%) of this obese population did not achieve at least 2.0 kg weight loss. Less than 20% achieved clinically significant weight loss (≥ 5 kg). Young and middle-aged patients were more likely

to achieve weight loss compared to ≥ 60 year olds. This was an encouraging finding since young adults were more likely to be in the highest BMI tertile (≥ 40 kg/m²). However, the negative health effects of hypertension and obesity have been established in older populations.^{38,39} In addition, patients with a BMI ≥ 35 kg/m² or BMI ≥ 40 kg/m² have demonstrated the highest cardiovascular mortality.⁴⁰

Another important finding is that patients who are prescribed antihypertensive medication are more likely to achieve both clinically significant weight loss and hypertension control within 12 months of a hypertension diagnosis. This finding likely reflects that patients prescribed antihypertensive medications have more frequent blood pressure-focused visits to check labs (e.g., creatinine with angiotensin-converting enzyme inhibitors), address possible medication side effects, and follow-up blood pressure readings to evaluate medication response. However, primary care visits are less likely to focus solely on lifestyle modifications.²⁸ Interestingly, our data demonstrated that patients in the highest BMI tertile were less likely to achieve smaller amounts of weight loss. Our results may have missed small variations in weight between visits; however, the data also highlight that any small weight change was unable to be maintained.

Provider (age, specialty, gender) and patient socioeconomic factors were not significant predictors of weight loss with hypertension control. These findings highlight that across primary care clinics, the focus should not just be on achieving hypertension control but should also include an ongoing focus on modifiable risk factors that contribute to the development and progression of disease. Primary care providers and healthcare systems need interventions to address effective, timely delivery of lifestyle modification counseling, as well as ongoing monitoring tailored to patient's specific comorbidities.

Interestingly, we did not find a significant association between achieved weight loss and baseline tobacco status, dyslipidemia, or diabetes mellitus. The lack of association among patients with diabetes mellitus may be secondary to statistical power, as less than 10% of the entire sample had a baseline diagnosis. Among patients with current tobacco use and/or dyslipidemia, there was not a significant difference in baseline antihypertensive medication prescriptions and only 10 patients self-reported tobacco cessation during follow-up.

The primary strength of this study was the ability to analyze an adult population receiving regular primary care in a large multispecialty group practice. One limitation is the use of data from a single healthcare system, which limits the generalizability of the findings. However, this healthcare system is one of the 10 largest physician practices in the United States, including over 300 primary care physicians and 43 primary care clinics. Another limitation is the retrospective use of administrative data, which raises the potential for misclassification of diagnoses or lack of documentation in the health record. However, previously established algorithms were used to identify hypertension and other comorbidities. There are limitations of using a 365-day baseline period to assess comorbidities, but our goal was to include patients who were currently managed by the healthcare system prior to assessing comorbidities.

Among patients with a baseline diagnosis of dyslipidemia and/or diabetes mellitus, we did not have data to assess their level of control (e.g., low-density lipoprotein-cholesterol and HgbA_{1c}) and its association with achieved weight loss. In this study, we were unable to determine if patients participated in a formal weight loss program or received dietary counseling. However, our previous research demonstrated that among young adults (18–39 year olds) who received any hypertension lifestyle counseling, dietary topics (e.g., low sodium and portion control) were discussed during only 55% of the counseling sessions.²⁸ Unfortunately, we did not have prescription medication data to evaluate weight changes according to antihypertensive medication regimen. We also did not have ambulatory blood pressure data or motivational factors. Future studies would be beneficial with this additional data to guide lifestyle and pharmacologic management of hypertension in obese populations.

Despite achieving incident hypertension control, the majority of obese patients did not achieve modest or clinically significant weight loss. Combined cardiovascular risk factor management (i.e., obesity and hypertension) is needed to decrease risks for cardiovascular morbidity and mortality in this high-risk population.

ACKNOWLEDGMENTS

The authors gratefully acknowledge Katie Ronk, BS and Patrick Ferguson, MPH for data preparation and Jamie LaMantia, BS and Colleen Brown, BA for manuscript preparation. This original research was supported by the Clinical and Translational Science Award program, previously through the National Center for Research Resources (NCR – UL1RR025011) and now by the National Center for Advancing Translational Sciences of the National Institutes of Health (NIH) under award number U54TR000021. H.M.J. is supported by the National Heart, Lung, and Blood Institute of the NIH (K23HL112907) and also by the University of Wisconsin (UW) Centennial Scholars Program of the University of Wisconsin School of Medicine and Public Health. C.M.B. is supported by the National Institute of Arthritis and Musculoskeletal and Skin Diseases of the NIH (K23AR062381). N.P. is supported by the National Institute on Aging of the NIH (K08AG029527). The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIH. Additional funding for this project was provided by the UW Health Innovation Program and the UW School of Medicine and Public Health from The Wisconsin Partnership Program. Some preliminary findings reported in this manuscript were presented in poster form at the American Heart Association 2014 Annual Scientific Sessions in Chicago, IL on 15–19 November 2014.

DISCLOSURE

A.K.H., C.M.B., N.P., and H.M.J. have clinical appointments with the academic group practice that has a financial

interest in delivering care to the general population from which study subjects were drawn. The remaining authors declared no conflicts of interest.

REFERENCES

1. Kannel WB, Brand N, Skinner JJ Jr, Dawber TR, McNamara PM. The relation of adiposity to blood pressure and development of hypertension. The Framingham study. *Ann Intern Med* 1967; 67:48–59.
2. Gelber RP, Gaziano JM, Manson JE, Buring JE, Sesso HD. A prospective study of body mass index and the risk of developing hypertension in men. *Am J Hypertension* 2007; 20:370–377.
3. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. *JAMA* 2014; 311:806–814.
4. Centers for Disease Control and Prevention. Adult Obesity Facts <http://www.cdc.gov/obesity/data/adult.html> 2015. Accessed 17 February 2016.
5. Krauss RM, Winston M, Fletcher RN, Grundy SM. Obesity: impact of cardiovascular disease. *Circulation* 1998; 98:1472–1476.
6. James PA, Oparil S, Carter BL, Cushman WC, Dennison-Himmelfarb C, Handler J, Lackland DT, LeFevre ML, MacKenzie TD, Ogedegbe O, Smith SC Jr, Svetkey LP, Taler SJ, Townsend RR, Wright JT Jr, Narva AS, Ortiz E. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA* 2014; 311:507–520.
7. Poirier P, Giles TD, Bray GA, Hong Y, Stern JS, Pi-Sunyer FX, Eckel RH. Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation* 2006; 113:898–918.
8. Borrell LN, Samuel L. Body mass index categories and mortality risk in US adults: the effect of overweight and obesity on advancing death. *Am J Public Health* 2014; 104:512–519.
9. Masters RK, Powers DA, Link BG. Obesity and US mortality risk over the adult life course. *Am J Epidemiol* 2013; 177:431–442.
10. Whitlock G, Lewington S, Sherliker P, Clarke R, Emberson J, Halsey J, Qizilbash N, Collins R, Peto R. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. *Lancet* 2009; 373:1083–1096.
11. Neter JE, Stam BE, Kok FJ, Grobbee DE, Geleijnse JM. Influence of weight reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension* 2003; 42:878–884.
12. Bramlage P, Pittrow D, Wittchen HU, Kirch W, Boehler S, Lehnert H, Hoefler M, Unger T, Sharma AM. Hypertension in overweight and obese primary care patients is highly prevalent and poorly controlled. *Am J Hypertens* 2004; 17:904–910.
13. Calhoun DA, Jones D, Textor S, Goff DC, Murphy TP, Toto RD, White A, Cushman WC, White W, Sica D, Ferdinand K, Giles TD, Falkner B, Carey RM. Resistant hypertension: diagnosis, evaluation, and treatment: a scientific statement from the American Heart Association Professional Education Committee of the Council for High Blood Pressure Research. *Circulation* 2008; 117:e510–526.
14. Sharma AM, Pischon T, Engeli S, Scholze J. Choice of drug treatment for obesity-related hypertension: where is the evidence? *J Hypertens* 2001; 19:667–674.
15. Hatahet MA, Bowhan J, Clough EA. Wisconsin Collaborative for Healthcare Quality (WCHQ): lessons learned. *WMJ* 2004; 103:45–48.
16. Sheehy A, Pandhi N, Coursin DB, Flood GE, Kraft SA, Johnson HM, Smith MA. Minority status and diabetes screening in an ambulatory population. *Diabetes Care* 2011; 34:1289–1294.
17. Thorpe CT, Flood GE, Kraft SA, Everett CM, Smith MA. Effect of patient selection method on provider group performance estimates. *Med Care* 2011; 49:780–785.
18. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ. The Seventh Report of the Joint National Committee on Prevention,

- Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* 2003; 289:2560–2572.
19. Johnson HM, Thorpe CT, Bartels CM, Schumacher JR, Palta M, Pandhi N, Sheehy AM, Smith MA. Undiagnosed hypertension among young adults with regular primary care use. *J Hypertens* 2014; 32:65–74.
 20. Tu K, Campbell NR, Chen ZL, Cauch-Dudek KJ, McAlister FA. Accuracy of administrative databases in identifying patients with hypertension. *Open Med* 2007; 1:e18–e26.
 21. Myers MG, Tobe SW, McKay DW, Bolli P, Hemmelgarn BR, McAlister FA. New algorithm for the diagnosis of hypertension. *Am J Hypertens* 2005; 18:1369–1374.
 22. Schmittiel J, Selby JV, Swain B, Daugherty SL, Leong TK, Ho M, Margolis KL, O'Connor P, Magid DJ, Bibbins-Domingo K. Missed opportunities in cardiovascular disease prevention?: low rates of hypertension recognition for women at medicine and obstetrics-gynecology clinics. *Hypertension* 2011; 57:717–722.
 23. Weber MA, Schiffrin EL, White WB, Mann S, Lindholm LH, Kenerson JG, Flack JM, Carter BL, Materson BJ, Ram CV, Cohen DL, Cadet JC, Jean-Charles RR, Taler S, Kountz D, Townsend RR, Chalmers J, Ramirez AJ, Bakris GL, Wang J, Schutte AE, Bisognano JD, Touyz RM, Sica D, Harrap SB. Clinical practice guidelines for the management of hypertension in the community: a statement by the American Society of Hypertension and the International Society of Hypertension. *J Clin Hypertens (Greenwich)* 2014; 16:14–26.
 24. Manson JM, McFarland B, Weiss S. Use of an automated database to evaluate markers for early detection of pregnancy. *Am J Epidemiol* 2001; 154:180–187.
 25. American Heart Association. 5 Goals to Losing Weight http://www.heart.org/HEARTORG/GettingHealthy/WeightManagement/LosingWeight/5-Goals-to-Losing-Weight_UCM_307260_Article.jsp#.Vjef2k2FN8w 2015. Accessed 17 February 2016.
 26. Jackson CL, Yeh HC, Szklo M, Hu FB, Wang NY, Dray-Spira R, Brancati FL. Body-mass index and all-cause mortality in US adults with and without diabetes. *J Gen Intern Med* 2014; 29:25–33.
 27. Kritchevsky SB, Beavers KM, Miller ME, Shea MK, Houston DK, Kitzman DW, Nicklas BJ. Intentional weight loss and all-cause mortality: a meta-analysis of randomized clinical trials. *PLoS One* 2015; 10:e0121993.
 28. Johnson HM, Olson AG, LaMantia JN, Kind AJ, Pandhi N, Mendonça EA, Craven M, Smith MA. Documented lifestyle education among young adults with incident hypertension. *J Gen Intern Med* 2015; 30:556–564.
 29. Borzecki AM, Wong AT, Hickey EC, Ash AS, Berlowitz DR. Identifying hypertension-related comorbidities from administrative data: what's the optimal approach? *Am J Med Qual* 2004; 19:201–206.
 30. Hebert PL, Geiss LS, Tierney EF, Engelgau MM, Yawn BP, McBean AM. Identifying persons with diabetes using Medicare claims data. *Am J Med Qual* 1999; 14:270–277.
 31. Marciniak MD, Lage MJ, Dunayevich E, Russell JM, Bowman L, Landbloom RP, Levine LR. The cost of treating anxiety: the medical and demographic correlates that impact total medical costs. *Depress Anxiety* 2005; 21:178–184.
 32. Elixhauser A, Steiner C, Harris DR, Coffey RM. Comorbidity measures for use with administrative data. *Med Care* 1998; 36:8–27.
 33. Fawcett J, Kravitz HM. Anxiety syndromes and their relationship to depressive illness. *J Clin Psychiatry* 1983; 44:8–11.
 34. Campbell NR, So L, Amankwah E, Quan H, Maxwell C. Characteristics of hypertensive Canadians not receiving drug therapy. *Can J Cardiol* 2008; 24:485–490.
 35. Starfield B, Weiner J, Mumford L, Steinwachs D. Ambulatory care groups: a categorization of diagnoses for research and management. *Health Serv Res* 1991; 26:53–74.
 36. Meeuwssen S, Horgan GW, Elia M. The relationship between BMI and percent body fat, measured by bioelectrical impedance, in a large adult sample is curvilinear and influenced by age and sex. *Clin Nutr* 2010; 29:560–566.
 37. Franklin SS, Jacobs MJ, Wong ND, L'Italien GJ, Lapuerta P. Predominance of isolated systolic hypertension among middle-aged and elderly US hypertensives: analysis based on National Health and Nutrition Examination Survey (NHANES) III. *Hypertension* 2001; 37:869–874.
 38. Folsom AR, Kushi LH, Anderson KE, Mink PJ, Olson JE, Hong CP, Sellers TA, Lazovich D, Prineas RJ. Associations of general and abdominal obesity with multiple health outcomes in older women: the Iowa Women's Health Study. *Arch Intern Med* 2000; 160:2117–2128.
 39. Dixon JB. The effect of obesity on health outcomes. *Mol Cell Endocrinol* 2010; 316:104–108.
 40. Hainer V, Aldhoon-Hainerová I. Obesity paradox does exist. *Diabetes Care* 2013; 36(Suppl 2):S276–S281.