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Impact of Therapeutic Lifestyle Changes in Resistant Hypertension

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Abstract

Hypertensive individuals are at an increased risk of developing heart disease and stroke. Adopting healthy lifestyles, such as being active on ≥4 days per week, weight-loss in the presence of obesity, consuming a diet rich in fruits and vegetables, and sodium below the recommended threshold, avoiding high alcohol consumption and refraining from smoking have been effective lifestyle therapies to prevent or control stage 1 hypertension (HTN). Among the 1 in 3 Americans who have HTN (systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥80 mmHg), 16% are diagnosed with resistant HTN (RHT). Although there are comparatively fewer studies examining the blood pressure lowering effects of a therapeutic lifestyle interventions in patients with resistant HTN, the available literature appears promising. This paper reviews key studies that quantify the blood pressure lowering effects of certain therapeutic lifestyles in patients with RHT and highlights areas needing more attention.

Keywords

Resistant hypertension; lifestyle change; physical activity; DASH diet

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Introduction

The growing number of economically developing countries that gradually adopt a Western lifestyle (i.e., sedentary behavior, physical inactivity, consumption of calorie dense, fatty foods, high sodium intake) is believed to be a major factor that is driving the increasing global prevalence of hypertension (HTN)[1]. Within the United States, under the 2014 guidelines, roughly 75 million or 1 in every 3 Americans were hypertensive (defined as ≥140/90 mmHg) and in 2015 HTN was the primary or contributing cause of death in over 427,631 individuals[2]. Recent findings of a nearly doubled risk of developing coronary heart disease and stroke in those with a systolic blood pressure (SBP) of 130–139 mmHg and diastolic BP (DBP) of 85–89 mmHg compared to normotensive individuals (<120/80 mmHg) prompted the lowering of the HTH threshold in the updated guidelines for the detection and management of HTN[3]. It is now estimated that 105.3 million Americans are categorized as being hypertensive[4, 5]. Of this proportion, 16% are categorized as having resistant HTN (RHT)[6]. RHT is defined as patients that are not at their BP goal (<130/80 mm Hg)[3] despite the use of at least three HTN agents of different drug classes at maximally tolerated doses, or at goal BP on four medications[7]. This is gravely concerning when considering that those with RHT are 50% more likely to suffer from a cardiovascular disease (CVD) related event compared to patients with controlled HTN[8].

The clinical management of patients in any stage of HTN requires both lifestyle (Table 1) and pharmacologic intervention. A prominent question in this area of clinical care relates to the respective efficacy of BP through medication and lifestyle changes such as exercise training. Recently Noone and colleagues[9] reported findings from one of the largest meta-analyses that compared the effects of exercise training and pharmacologic interventions on BP in individuals with HTN. While both interventions were found to be effective at lowering BP compared to control conditions, anti-HTN medications were found to be more effective. However, due to insufficient evidence, first-line anti-HTN medications were not shown to reduce BP to a greater extent than exercise training. These results suggest that non-pharmacologic therapy, such as exercise training, should be widely promoted as first-line therapy for stage 1 HTN in addition to pharmacotherapy.

Practicing healthy lifestyle behaviors (i.e., normal body mass index [BMI] and waist circumference, physically active on ≥4 days/week, nonsmoker, moderate alcohol consumption, following the Dietary Approaches to Stop Hypertension [DASH] diet) among individuals diagnosed with RHT has been found to have positive health benefits. Compared to RHT individuals with 0 or 1 healthy lifestyle factor, those that follow 2, 3 or 4 to 6 behaviors exponentially reduce their risk of future CVD events (0.91, confidence interval (0.68–1.21), 0.80 confidence interval (0.57–1.14), and 0.63 confidence interval (0.41–0.95), respectively)[10]. Fewer studies, however, have examined the extent to which initiating these lifestyle interventions modify BP in patients with RHT.

Accordingly, the ongoing Lifestyle Interventions in Treatment-Resistant Hypertension (TRIUMPH) study (NCT02342808) seeks to explore the effects of a 4-month, center-based lifestyle intervention consisting of aerobic exercise training (AET), DASH diet and weight management delivered by exercise and behavioral specialists compared to standard of care

for treating RHT in addition to education[11]. Upon its completion, it will be the most comprehensive study to date that explores the BP-lowering effects of therapeutic lifestyle changes in RHT and has the potential to help clarify expectations that ultimately help guide clinical therapy.

Identifying RHT

It is common to misdiagnose RHT due to measurement errors and patient non-adherence. Therefore, it is important to confirm patient medication adherence and proper BP measurement technique. It is useful to have a team-based approach including providers, pharmacists, and nurses when addressing the multifactorial problems that affect medication adherence. Medication cost, difficulty managing complex regimens, poor health literacy, and fear of adverse side effects can all adversely affect adherence to a BP regimen. Verifying refill history, pill counts, and patient self-report can help to confirm adherence. Additionally, attention to proper BP measurement technique, such as using the appropriate cuff size and patient positioning, is important and can have a significant impact on diagnosis. According to the 2017 American College of Cardiology (ACC)/American Heart Association(AHA) Guideline this should include an average of two different BP measurements on two separate occasions[3]. Ideally, ambulatory BP monitoring should be performed, but since this is not always available, 24-hour BP monitoring can provide additional support for a diagnosis. It is imperative that patients and caregivers are also educated on correct BP measurement technique, since home monitoring can help to rule out possible white coat HTN[7]. A physical examination should be performed to assess for end organ damage which can include: fundoscopic examination, assessment for renal or carotid bruits, a basic metabolic profile, and urinalysis to assess for proteinuria[12]. It is also important to rule out reversible secondary causes of RHT. This should include screening for primary aldosteronism, renal artery stenosis, as well as consideration of other less common endocrine disorders and referral to a specialist if indicated[12].

Pharmacological Management

The primary aim of this review is to highlight the extent to which lifestyle interventions impact BP in those with RHT. However, because pharmacologic treatment is essential in the management of RHT we will briefly summarize the latest clinical trials/recommendations in this population. For additional information regarding the pharmacologic/clinical management of RHT patients, we direct readers to more thorough reviews and guidelines statements[7, 13].

The Prevention And Treatment of Hypertension With Algorithm based therapY-2 (PATHWAY-2) study demonstrated that the addition of spironolactone can cause a mean decrease of 8 mmHg in SBP when compared to placebo in patients with RHT who are already on a three drug regimen[14]. Aldosterone antagonists do carry a risk of hyperkalemia that requires lab monitoring of potassium within 3–7 days after initiation or dose increase, and every 3–4 months once on chronic therapy. The risk of hyperkalemia is increased in patients with certain co-morbidities such as chronic kidney disease (CKD). Previous studies using spironolactone excluded patients with moderate to severe CKD due to this risk[14].

However, the recently published Spironolactone With Patiromer in the Treatment of Resistant Hypertension in Chronic Kidney Disease (AMBER) trial found that potassium binding agents such as patiromer can be safely used in this population to help mitigate this risk when using spironolactone, and can increase its use in patients with RHT and CKD[15]. If BP is still not controlled on a four-drug regimen or an aldosterone antagonist is not tolerated, the addition of a beta-blocker would be a reasonable next step[7].

There are a number of trials that are looking at renal denervation as a potential device-based intervention for RHT. The Renal Denervation With the Symplicity Spyral Multi-electrode Renal Denervation System in Patients With Uncontrolled Hypertension (SPYRAL)[16] and ReCor Medical Paradise System in Clinical Hypertension (RADIANCE)[17] studies are small sham-controlled trials that have looked at the safety and reductions in systolic pressure respectively for renal denervation using radiofrequency or ultrasound energy. Additionally, carotid baroreceptor activation therapy may also be a promising area of future research and trials are ongoing (NCT03730519, NCT02572024). Although these interventions have created a great deal of interest, there is currently not enough evidence to recommend their use outside of clinic trials[7, 18].

Lifestyle Therapeutic Interventions

Aerobic Exercise

Engaging in regular, moderate to vigorous physical activity (PA) is a powerful lifestyle behavior that reduces the risk of developing noncommunicable diseases and risk of adverse events in those with noncommunicable diseases[19]. Roughly 40% of individuals with RHT are physically inactive[20]. Moreover, PA levels are closely associated with cardiorespiratory fitness (CRF), which is widely considered the strongest prognosticator among commonly measured vital signs in clinical settings[21]. Even within patients diagnosed with RHT, CRF holds an inverse, independent, and graded association with all-cause mortality. In a large cohort of veterans, those stratified in to the highest quartile of CRF (average 8.8±1.1 metabolic equivalents [METs]) had a 62% lower risk of all-cause mortality compared to the lowest fit group (average 4.5±0.8 METs). Additionally, each 1-MET increase in exercise capacity contributed to a 18% reduction in mortality risk[22]. Provided that regular PA at moderate to vigorous intensities promotes increases in CRF, engaging in regular PA can therefore be a critical behavior that mitigates the risks of developing RHT, while promoting improvement of coexisting cardiovascular risk factors that commonly exist in RHT patients.

Comparatively, fewer studies have examined the effects of chronic AET in patients diagnosed with RHT than those with pre- or controlled HTN. The existing literature across HTN categories commonly report a graded reduction in resting SBP and DBP with higher resting BP. Individuals categorized as being pre-HTN commonly see decreases in SBP/DBP by 3–5/2–4 mmHg, whereas individuals with HTN can see a decrease in SBP/DBP as much as 5–8/2–4 mmHg (Table 1). Furthermore, AET performed when following optimum medication regimens is known to be safe without abnormal or exaggerated increases in the majority of individuals[23]. The acute effects of an AET bout have also been shown to persist throughout the day. This is of interest given the strong evidence supporting the prognostic utility of ambulatory versus resting or office-based BP[24]. Performing a 45-minute bout of

light (50% of maximal heart rate) or moderate (75% of maximal heart rate) cycle exercise has been shown to result in lower BP readings (−7.7±2.4 mmHg and −9.4±2.8 mmHg, respectively) over a 5 hour period after the exercise bout[25]. However, light intensity exercise may contribute to sustained (up to 19 hours) lower ambulatory SBP readings during the day and night compared to moderate intensity. This is particularly relevant to using exercise as precision medicine to target a particular outcome of interest, in this case ambulatory BP[26].

Few studies have examined the effects of chronic AET on resting and ambulatory BP in RHT patients. Dimeo and colleagues presented favorable outcomes when treadmill walking was performed 3 days per week for 8–12 weeks at an intensity just above the aerobic threshold (lactate concentration of 2.0±0.5 mmol/L in capillary blood)[27]. Office SBP and DBP decreased by 6.6±15.7 and 2.7±8.0 mmHg, respectively, with 24-hour ambulatory BP decreasing by 5.4±12.2 and 2.8±5.9 mmHg, respectively. Conversely, Kruk and colleagues[28] provided one-on-one, tailored PA recommendations to the patient's individual needs while educating them on the appropriate methods of increasing intensity of activity. Patients also received text message reminders on the benefits of regular PA three times per week. While significant decreases in office and ambulatory BP was observed after three months, improvements in SBP did not persist at the 6-month time period of the intervention. The implementation of mostly education guided PA therapy may therefore not be a sufficient strategy to treat RHT and would instead require structured exercise to experience significant decreases in office and/or ambulatory BPs.

Perhaps, one of the most successful BP lowering exercise interventions in RHT patients to date has been heated pool exercise[29, 30]. An initial pilot study examining the effects of 60-minute heated water (32°C) exercise training 3 times per week for 2 weeks contributed to significant decreases in SBP and DBP measured in the office (−18 mmHg and mmHg, respectively), 24-hour ambulatory (−9 and −9 mmHg, respectively), daytime (−16 and −10 mmHg, respectively) and nigh-time (−10 and −8 mmHg, respectively). These impressive decreases in BP across various measurement periods prompted a longer, 12-week training intervention following the same intervention of heated pool calisthenics and walking while a control group (n=16) maintained habitual activities. The longer duration of the intervention led to profound decreases in SBP and DBP measured in the office (−36 and −12 mmHg, respectively), 24-hour ambulatory (−17 and −9 mmHg, respectively), daytime (−21 and −11 mmHg, respectively) and nighttime (−15 and −8 mmHg, respectively). Furthermore, exercise training lead to improvements in peak oxygen consumption (25.0±4.6 to 27.9±4.0 ml/kg/min) with an associated decrease in peak SBP (198.3±33.3 to 175.1±28.0 mmHg), likely lowering the prevalence of exaggerated responses to submaximal or maximal exercise. The authors speculate that in addition to the decrease in sympathetic and up regulation of vagal activity, exercise in a heated pool may have further facilitated vasodilation, decreased renin, angiotensin II, aldosterone, renal sympathetic outflow and an increase in nitric oxide and arterial natriuretic peptide release. As these are the first studies to examine the effects of heated pool exercise in RHT patients, coupled with the promising outcomes, additional studies will be needed to confirm these findings.

Resistance Exercise

A growing body of evidence has recently highlighted the protective effects of resistance exercise training (RET) from developing the metabolic syndrome, morbidity and mortality[31, 32]. However, studies examining the effects of RET on BP have demonstrated conflicting results[33–37]. An overwhelming majority of studies that have examined the effects of RET on BP have been in pre-HTN or controlled HTN participants. To our knowledge, there has not been a study to date that has investigated the potential for RET to lower office, ambulatory and night time BP. Therefore, this section will provide a brief summary of well-designed studies that sought to characterize the effects of RET on BP.

Historically, patients with HTN were discouraged from performing RET due to the perceived negative effects of the Valsalva maneuver on BP[38]. However, an accumulation of evidence demonstrates that the RET benefits outweigh the risks in patients with HTN and accordingly has been included in the 2017 ACC/AHA guideline[3]. Furthermore, light RET has also been promoted in the AHA Scientific Statement on RHT and its management[7]. While it is safe for most RHT patients to perform RET, practitioners are encouraged to measure BP during exercise to rule out an exaggerated BP response. The benefits of RET have been shown to decrease BP after acute bouts and chronic RET. A wide range of acute decreases have been observed with as much as a 33/15 mmHg drop in SBP/DBP in response to high intensity (80% of 1 repetition max [RM]) in older adults[39], 8/6 mmHg in response to low intensity (40% of 1 RM)[40], as well as no difference at lower (40% of 1 RM) or higher (80% of 1 RM) intensities of RET[41]. Considering the BP effects of chronic RET, Moraes et al[42] studied the effects of 12 weeks of conventional RT at 60% 1 RM on BP in middle-aged men with elevated BP. They reported SBP reductions of 16 mmHg and mean DBP reductions of 12 mmHg[42]. Systolic BP reductions reported in this study are in line with other studies in elderly women with HTN[43]. Additionally, BP improvements were maintained during a 4-week detraining period and others have even demonstrated maintenance of BP improvements following RET for up to 14-weeks post-training[44].

In a meta-analysis, Cornelissen and Smart[45] compared the effects of different exercise training modalities on BP. All exercise modalities (AET, RET, and isometric training) were found to significantly lower SBP (−3.5 mmHg, −1.8 mmHg, and −10.9 mmHg, respectively) and DBP (−2.5 mmHg, −3.2 mmHg, and −6.2 mmHg, respectively). After a subgroup analysis, BP reductions after AET were shown to be more pronounced in men and participants with HTN, while groups of participants with elevated BP showed greatest reductions with RET[45]. In contrast to this finding, another recent meta-analysis by MacDonald et al[46] analyzed 64 randomized controlled studies and reported similar BP reductions with RET; however, individuals with HTN showed the greatest BP reductions (~ 6/5 mmHg) followed by those with elevated BP (~ 3/3 mmHg) compared to individuals with normal resting BP (~ 0/1 mmHg)[46]. Furthermore, it was found that BP reduction from RET was greater in nonwhite populations compared to white populations and this BP reduction was approximately double that previously reported from AET (10–14 vs 5–7 mmHg)[46]. The observation that the BP lowering effects of RET may be moderated by race/ethnicity may have significant clinical significance regarding exercise prescription if confirmed by future investigations.

Improvements in BP in response to RET may be related to adaptations as a result of the unique hemodynamic responses to RET characterized by periods of blood flow restriction during muscle contractions and periods of reactive hyperemia post contraction[47]. It is thought that this rhythmic pattern partly contributes to structural adaptations in arterial diameter and wall thickness as well as functional adaptations, such as improvements in arterial stiffness and endothelial function. When comparing the effects of 4-weeks of RET and AET on arterial stiffness in patients with HTN and elevated BP, Collier et al. reported improvements in SBP and DBP in both groups (−4.6 mmHg, −3.1 mmHg, respectively)[48]; however, participants undergoing RET showed significant increases in arterial stiffness, but was counterbalanced by greater increases in peak forearm blood flow and vascular conductance with RET compared to AET (52% vs. 19%). Thereby, indicating improvements in endothelial function and suggesting that the underlying physiological mechanisms responsible for BP reductions may be different between both modalities.

Improved endothelial function after RET has not been universally reported. Acute bouts of RET were found to have opposing effects on endothelial function in sedentary untrained individuals and trained individuals, with sedentary untrained individuals showing reduced endothelial function while trained individuals were protected from this adverse effect[49, 50]. Sedentary individuals have been shown to have decreased endothelium-dependent vasodilation, measured by flow-mediated dilation (FMD), in response to acute HTN (>170 mmHg) induced by strenuous resistance exercise[50, 51]. However, both endurance trained athletes and conditioned weightlifters alike show improved FMD responses to acute RET, unlike sedentary individuals[50]. Rather, FMD adaptations may require regular RET in order to improve endothelial function assessed via FMD[52]. Overall, the weight of the evidence supports positive vascular adaptations to both vascular structure and function and RET should be incorporated in exercise regimens aimed at reducing elevated BP[53].

Weight Loss

Obesity has been identified as a major contributor to the development of HTN[7, 54]. Data from the National Health and Nutrition Examination Survey, found that individuals with a body mass index (BMI) ≥30 kg/m^2 (i.e., obesity) have nearly double the risk of developing RHT compared to those with a normal BMI[55]. This risk is partly related to greater insulin resistance, inflammatory cytokines (i.e., tumor necrosis factor-α), sympathetic activity, renin-angiotensin and aldosterone activity, as well as markers of oxidative stress and reduced nitric oxide availability typically observed in obese individuals[56]. Although there are many pathophysiologic factors that lead to these observations, increased visceral adiposity has been identified as a major contributor[54]. Particularly, the adipose tissue located around the kidney and accumulated within the kidney can induce a physical renal compression, ultimately resulting in increase in BP[57]. Additionally, pharmacologic challenges in treating hypertension in obese RHT patients may occur due to excess adiposity and its impact on pharmacokinetics and pharmacodynamics[56]. Over recent years there has been an accumulation of data demonstrating the powerful health changes that occur after bariatric surgery mediated weight loss, especially in patients with type II diabetes[58, 59]. In addition to significantly lowering hemoglobin A1c in obese type II diabetic patients, Aminian and colleagues found a significant decrease in the use of noninsulin diabetes medications,

insulin, renin-angiotensin system blockers and other antiHTN medications, lipid-lowering therapies, and aspirin in patients receiving baritatric surgery compared to nonsurgical management[59]. Most recently, Schiavon and colleagues[60] examined 100 patients with a BMI between 30.0 to 39.9 kg/m^2 undergoing Roux-en-Y Gastric Bypass combined with medical therapy or medical therapy alone for 12 months. The prevalence of RHT was similar between the groups at baseline (10% compared to 16%, respectively). Participants in the medical therapy only group did not see a decrease in the prevalence of RHT (15%) after 12 months, however, all participants undergoing bariatric surgery were no longer categorized has RHT. Despite these compelling findings, its translation to clinical practice is limited as bariatric surgery for weight loss is accepted for those with a BMI >40.0 kg/m^2 and is not yet indicated to control hypertension. Instead, achieving weight loss through lifestyle modification has been effective in populations without RHT. One could expect to lower SBP and DBP by 5 and 4 mmHg with a 6–8% decrease in body weight[61] or as much as 5–20 mmHg decrease in SBP by losing 10 kg[62]. While, studies have not examined the effects of lifestyle induced weight loss in RHT patients, similar weight loss recommendations as pre-HTN or controlled HTN patients apply[7].

Dietary Modification

The American diet consists of large quantities of sodium, with the average American consuming greater than 3,400 mg per day. Easily exceeding the current dietary recommendations for Americans recommending a sodium intake to less than 2,300 mg per day. Reducing sodium intake in those with mild HTN has been effective at lowering BP (SBP by −8 mmHg and DBP −4 mmHg) to a similar magnitude as pharmacologic monotherapy trials[63]. In fact, a graded reduction in BP has been shown to be present with greater reductions in daily sodium intake[64, 65]. Individuals consuming sodium levels above the national recommendations have been shown to be at a greater risk of developing HTN in addition to CKD[66]. Decreasing the intake of dietary sodium in CKD patients with RHT has been shown to have profound BP lowering effects. Pimenta and colleagues[67] demonstrated this within 12 patients that participated in a randomized crossover study with 7 days of low (1,150 mg per day) and high (5,750 mg per day) sodium diets. Compared to baseline office BP (SBP, 145.8±10.8 and DBP, 83.9±11.2), the low sodium diet decreased office SBP and DBP by 22.7 and 9.1 mmHg, respectively, while the high sodium diet did not significantly differ. Within this population, the authors concluded that dietary sodium intake may be a significant contributor to anti-HTN medication resistance. Similar findings were observed in a double-blind randomized controlled crossover trail in 20 stage 3–4 CKD patients with HTN. Over a 2-week period, participants followed a low sodium diet (1,380 to 1,840 mg per day) or high sodium diet (4,140 to 4,600 mg per day) with a 1-week washout period between each dietary intervention[68]. The low sodium intervention contributed to statistically and clinically significant decreases in 24-hour ambulatory BP (SBP/DBP of −10/−4 mmgH) in addition to decreases in extracellular fluid volume, albuminuria and proteinuria. Considering the clinical implications of high BP and volume overload on the progression of CKD, achieving a decrease in fluid volume as well as ambulatory BP by 10/4 mmHg has been shown to significantly reduce mortality risk[69].

Other diets, such as the widely promoted DASH diet has been considered a key therapeutic dietary intervention that promotes BP control. This plan promotes a diet rich in fruits, vegetables, fiber and low-fat dairy products in addition to sodium reduction[65]. Importantly, the DASH diet also emphasizes the consumption of dietary potassium, which together with low-sodium content (<1,500 mg/day) can significantly contribute to BP reduction[65, 70].

However, no study to date has tested the effect of this specific intervention in RHT patients. Knowing the powerful effects of low sodium diet on BP management in RHT, it is perceivable that the DASH diet may also be an effect method of BP management with the caveat that patients comply with salt restriction. More recently, a Mediterranean dietary pattern has also been associated with improved BP and such effects have been at least partially attributed to the high content of oleic acid[71], a fatty acid highly prevalent in extra-virgin olive oil. As for the DASH diet, however, the role of the Mediterranean diet has not been explored in patients with RHT.

Conclusion

In examining the current literature that describes various therapeutic lifestyle interventions in RHT, it is evident that there is a considerable low volume of work that has been done in this area. However, increasing physical activity, weight-loss and modifying the quality of diet (i.e., DASH, Mediterranean diet) independent of body weight changes appear to be effective and promising strategies to achieve clinically meaningful reductions in office and ambulatory BP. In addition to the TRIUMPH trial, future investigations should seek to employ comprehensive lifestyle modification interventions to identify optimal strategies to modify the RHT diagnosis.

List of Abbreviations

ACC	American College of Cardiology
AET	aerobic exercise training
AHA	American Heart Association
AMBER	Spironolactone With Patiromer in the Treatment of Resistant Hypertension in Chronic Kidney Disease
BMI	body mass index
BP	blood pressure
CKD	chronic kidney disease
CRF	cardiorespiratory fitness
CVD	cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DBP	diastolic blood pressure

FMD	flow mediated dilation
HTN	hypertension
MET	metabolic equivalent
PA	physical activity
PATHWAY-2	Prevention And Treatment of Hypertension With Algorithm based therapY-2
RADIANCE	ReCor Medical Paradise System in Clinical Hypertension
RET	resistance exercise training
RHT	resistant hypertension
RM	repetition max
SBP	systolic blood pressure
SPYRAL-HTN	Renal Denervation With the Symplicity Spyral Multi-electrode Renal Denervation System in Patients With Uncontrolled Hypertension
TRIUMPH	Lifestyle Intervention in Treatment-Resistant Hypertension

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Table 1.

Nonpharmacological interventions for prevention and treatment of hypertension

	Nonpharmacological Intervention	Dose	Approximate Impact on SBP	
			Hypertension	Normotension
Weight loss	Weight/body fat	Best goal is ideal body weight but aim for at least a 1-kg reduction in body weight for most adults who are overweight. Expect about 1 mm Hg for every 1-kg reduction in body weight.	−5 mm Hg	−2/3 mm Hg
Healthy diet	DASH dietary pattern	Consume a diet rich in fruits, vegetables, whole grains, and low-fat dairy products, with reduced content of saturated and total fat.	−11 mm Hg	−3 mm Hg
Reduced intake of dietary sodium	Dietary sodium	Optimal goal is <1500 mg/d but aim for at least a 1000-mg/d reduction in most adults.	−5/6 mm Hg	−2/3 mm Hg
Enhanced intake of dietary potassium	Dietary potassium	Aim for 3500–5000 mg/d, preferably by consumption of a diet rich in potassium.	−4/5 mm Hg	−2 mm Hg
Physical activity	Aerobic	• 90–150 min/wk • 65%–75% heart rate reserve	−5/8 mm Hg	−2/4 mm Hg
	Dynamic resistance	• 90–150 min/wk • 50%–80% 1 rep maximum • 6 exercises, 3 sets/exercise, 10 repetitions/set	−4 mm Hg	−2 mm Hg
	Isometric resistance	• 4 × 2 min (hand grip), 1 min rest between exercises, 30%–40% maximum voluntary contraction, 3 sessions/wk • 8–10 wk	−5 mm Hg	−4 mm Hg
Moderation in alcohol intake	Alcohol consumption	In individuals who drink alcohol, reduce alcohol† to: • Men: ≤2 drinks daily • Women: ≤1 drink daily	−4 mm Hg	−3 mm Hg

*Type, dose, and expected impact on BP in adults with a normal BP and with hypertension.

†In the United States, one “standard” drink contains roughly 14 g of pure alcohol, which is typically found in 12 oz of regular beer (usually about 5% alcohol), 5 oz of wine (usually about 12% alcohol), and 1.5 oz of distilled spirits (usually about 40% alcohol).

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