



Nonpharmacologic management of hypertension: a multidisciplinary approach

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Purpose of review

Nonpharmacologic lifestyle modification interventions (LMIs), such as increasing physical activity, dietary modification, weight-loss, reducing alcohol consumption and smoking cessation, are effective strategies to lower resting blood pressures (BPs) in prehypertensive or hypertensive patients. However, the limited time shared between a physician and a patient is not adequate to instill an adoption of LMI. The purpose of this review is to therefore highlight evidence-based BP lowering, LMI strategies that can feasibly be implemented in clinical practices.

Recent findings

Interventions focusing on modifying physical activity, diet, weight-loss, drinking and smoking habits have established greater efficacy in reducing elevated BP compared with providing guideline recommendations based on national guidelines alone. Greater reductions in BP can be achieved through programmes that provide frequent contact time with exercise, nutrition and/or wellness professionals. Programmes that educate individuals to lead peer support groups can be an efficient method of ensuring compliance to LMI.

Summary

Evidence of a multidisciplinary approach to LMI is an effective and attractive model in managing elevated BP. This strategy is an attractive model that provides the necessary patient attention to confer lifestyle maintenance.

Keywords

alcohol consumption, diet, physical activity, smoking cessation, weight loss

INTRODUCTION

It is estimated that 31.5% of the global population or 1.4 billion people are diagnosed with hypertension (HTN) [1]. Its pervasive nature in the United States has contributed to the development of debilitating cardiovascular diseases (CVDs) and has attributed to an 8.2% increase in HTN-related deaths from 2003 to 2013 [2[•]]. Numerous studies have provided ample evidence supporting the blood pressure (BP)-lowering effects of lifestyle modifications (i.e. increased physical activity, dietary habits, weight-loss etc.), suggesting that impactful reductions in its incidence and prevalence should be achievable. Yet, the global incidence of HTN is on the rise in conjunction with increases in sedentary time and consumption of high caloric foods with low nutritional value, despite physician-guided recommendations against these habits [1]. As a result, physicians are left to rely on pharmacologic management of HTN that subsequently places an unprecedented burden on the healthcare system [2[•]].

The successful translation of evidence-based, lifestyle modification intervention (LMI) strategies to manage elevated BP in clinics has been challenging. Physicians are encouraged to outline current physical activity and nutrition guidelines to patients in a narrow window of time, which is also needed to

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KEY POINTS

- Untreated HTN is associated with increased risk of developing CVDs.
- Nonpharmacologic management of patients (i.e. increased physical activity, dietary habit modification and weight-loss) with elevated BPs can lower resting SBP and DBP by roughly 3–4 mmHg.
- Placing individuals with elevated BPs in programmes designed to promote lifestyle modification and maintenance by a multidisciplinary team is superior to making recommendations alone over 3–6-month period.
- Future directions need to explore the feasibility of implementing long-term (i.e. beyond 1 year) multidisciplinary interventions on managing and maintaining BP reductions.

diagnose and treat relevant symptoms at the time of the visit. This approach is by no means ideal for promoting behavior change and therefore new approaches must be considered and pursued [3[¶],4]. Adoption of a multidisciplinary team (i.e. clinical exercise physiologist, nutritionist, wellness coach etc.) approach is an attractive model that promotes a culture of health and facilitates patient compliance to nonpharmacologic methods to control BP [5[¶]]. The aim of this review is to therefore highlight evidence-based BP lowering, LMI strategies that can feasibly be implemented in a clinical practice.

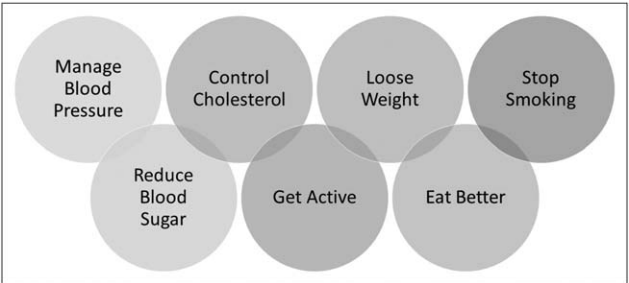


FIGURE 1. Life’s simple 7 programmes. See TIFF image.

GUIDELINES AND RECOMMENDATIONS FOR PURSUING NONPHARMACOLOGIC MANAGEMENT OF HYPERTENSION

According to recent Guideline documents, all individuals should be following the American Heart Association life’s simple 7 (LS7) programme (Fig. 1) for the prevention of CVD and HTN and to prevent borderline BP levels from progressing into established essential HTN. In patients without CVD and with SBP 140–159 mmHg and DBP 90–99 mmHg, 3 months of vigorous nonpharmacologic therapy with LMI, with the five-step approach of lifestyle modifications (Table 1) [6], including weight reduction, healthy dietary choices (Dietary Approaches to Stop Hypertension/DASH diet recommended, although other factors discussed below could be considered), lowering sodium intake (some evidence discussed below suggests that white sugar crystals may be even more toxic than the white salt crystals) and reducing alcohol intake into the low or

Table 1. Lifestyle modifications

Modification	Recommendation	Approximate SBP reduction (range) ^b
Reduce weight	Maintain normal body weight (BMI 18.5–24.9 kg/m ²)	5–20 mmHg/10 kg
Adopt DAHS ^a eating plan	Consume a diet rich in fruits, vegetables and low-fat dairy products with a reduced content of saturated and total fat	8–14 mmHg
Lower sodium intake	a. Consume no more than 2400 mg of sodium/day b. Further reduction of sodium intake to 1500 mg/day is desirable as it is associated with greater reduction in BP c. Reduce intake by at least 1000 mg/day as that will lower BP, even if the desired daily intake is not achieved	2–8 mmHg
Physical activity	Engage in regular aerobic physical activity such as brisk walking (at least 30 min per day, most days of the week)	4–9 mmHg
Moderation of alcohol consumption	Limit consumption to no more than two drinks (e.g. 24 oz beer, 10 oz wine or 3 oz 80-proof whiskey) per day in most men, and to no more than one drink per day in women and lighter weight persons	2–4 mmHg

^aDASH, dietary approaches to stop hypertension.
^bThe effects of implementing these modifications are dose-dependent and time-dependent, and could be greater for some individuals.
Adapted from [6].

at least moderate dose levels. In patients with established CVD or those with higher levels of SBP/DBP greater than 160/100 mmHg, vigorous LMI should be combined with immediate recommendations for pharmacologic intervention.

PHYSICAL ACTIVITY PROMOTION

An extensive body of literature has demonstrated the BP-lowering effects of physical activity interventions (i.e. 30–40 min of moderate to vigorous physical activity, ≥ 3 days per week) [7⁸,8–11]. Roughly, 3 months of regular exercise training can contribute to a 3–5 mmHg decline in SBP and 2–4 mmHg decline in DBP [12], although some reports suggest greater effects (6–10 mmHg). These seemingly minimal changes can translate to as much as 7–10% reductions in stroke and ischemic CVD events [13]. Furthermore, the BP-lowering effects of routine exercise can be more pronounced in HTN (6–8 mmHg and 5–6 mmHg reductions in SBP and DBP, respectively) and less in normotensives (2–3 mmHg and 1–2 mmHg reductions in SBP and DBP, respectively) [14]. Interventions that are initiated in early adulthood and are maintained throughout aging can significantly reduce the CVD burden in later life [13,15–17].

Much of the available data presenting the efficacy of lowering BP through physical activity interventions have been performed in highly controlled settings with regular and free access to exercise facilities, along with support and guidance from research assistants. Having access to similar programmes in a real-world setting may not be a financially feasible option for most patients, emphasizing the need for affordable and effective alternatives [18]. Initial evidence supports the concept of developing programmes that assist in forming social support networks, composed of groups with 6–7 participants [19]. Within each group, a volunteer is identified to stay in weekly contact with other group members to provide support and remind them to participate in upcoming community exercise sessions (40 min of walking). As compared to providing standard physical activity recommendations, community-based social support networks demonstrate 11 and 5 mmHg reductions in SBP and DBP, respectively, after 6 months of participation. What is not known is that if the improvements can be maintained for an extended period and if participation in a community support network is necessary to do so. A 16-month home-based exercise intervention [60–85% of age predicted (220-age) maximal heart rate, performed for 30 min, three times per week], with 2-month follow-up visits, has demonstrated a plateau in the reduction of

SBP (7 mmHg) starting between 6 and 8 months of intervention [20]. Indicating that reductions in resting BP in response to exercise may follow a dose–response relation, with greater intensity and/or duration of exercise needed to further reduce BP in those with elevated resting BP. However, it should be noted that the lack of a rise in BP during the intervention period is a favorable outcome that reaffirms home-based exercise, as an effective means of reducing and maintaining resting BP. As long as participants are held accountable by having frequent (i.e. 2 months) contact with clinical exercise physiologists (or like professionals) to monitor BP responses to the home-based exercise programme.

The clinical application of wearable technologies, aimed to mitigate sedentary time and increase time spent in moderate-to-vigorous physical activity intensities, has been gaining much attention [21]. Common pedometer feedback interventions aim to promote an increase in daily steps by 10% of baseline step values, each week step goals are achieved on 4 or more days of the week [22,23⁸]. This strategy may be cumbersome for patients to follow and a labor-intensive task for clinical staff to manage multiple patients. Alternatively, the use of an interactive website, designed to provide feedback, educational material, strategies to set and meet physical activity goals, and rewards, can facilitate the adoption of a physically active lifestyle. Implementation of a 12-week pedometer feedback and an interactive website intervention has been shown to reduce BP and improve vascular function in older adults with elevated BP [23⁸]. However, to our knowledge, there has not been a study that has examined this strategy's effects on long-term BP management and would require future studies to do so.

WEIGHT MANAGEMENT

Despite the evidence of an obesity paradox in CVD [24,25], weight-loss is a vital strategy to lower BP in patients with HTN [26–28], as it may reduce the reliance on BP-lowering drugs [29] and confer favorable effects on metabolic risk profile [30] and lower the risk of CVD-related deaths [26,27]. Although exact body composition levels have been debated [24,25], recent European and American clinical guidelines emphasize that the lowest all-cause mortality rates are of those with a BMI between 20 and 25 kg/m² [26,27]. In clinical practice, measuring a patient's BMI, while being mindful of the limitations of not being able to quantify the distribution of fat and muscle mass, can be used as an efficient method to identify patients who would benefit from a weight-loss programme. A goal of achieving a loss of 6–8% of one's body weight can

lead to a 5 and 4 mmHg decrease in SBP and DBP, respectively [31], and losses of approximately 10 kg may lower SBP by 5–20 mmHg [27].

There are many habitual behaviors (i.e. sedentary leisure and/or work lifestyle, frequent consumption of large portions of high calorie foods etc.) that can disrupt the balance between caloric expenditure and intake that favors caloric excess; making it difficult for patients to either maintain or lose weight. For many overweight or obese patients, losing weight without a personal or professional support group may be perceived as an unattainable task when receiving advice to lose weight from their physicians. There is currently limited evidence that commercial weight-loss programmes (i.e. Weight Watchers, Atkins Diet, Jenny Craig, SlimFast etc.) are effective in reducing BP [32,33]. However, there are little to no studies that have tested the BP-lowering effects of these programmes in overweight and/or obese hypertensive individuals. This brings attention to the need for future studies to further investigate this area research. Alternatively, referring patients to a structured lifestyle intervention programme, directed by health professionals (i.e. registered dietitians, wellness coaches etc.), may empower patients to adopt the skills and knowledge to balance meal plans, become physically active and achieve long-term weight-loss success [26,27,30].

Although short-term caloric restriction and exercise programmes [34] are generally effective at reducing weight and BP, greater attention should be given to developing and enrolling patients into long-term interventions. One such community-based weight-loss programme consists of three phases over a 2-year period. The first phase (1–6 months) had overweight, hypertensive patients who follow the ‘Med-South Diet’ (a modified version of the Mediterranean Diet) with regular sessions (four per month) with trained counselors [35^{***}]. Much of the emphasis was placed on dietary consultation, whereas the remaining time was spent developing goals and strategies to achieve at least 7500 steps/day or at least 30 min of walking at least 5 days/week. A telephone counseling option was also provided for participants who were unable to attend in person. Phase II (months 7–12) and phase III [14–22,23^{*},24,25] consisted of continuing in person and phone contact with participants to provide resources and strategies to lose weight and maintain LMI changes. At 12 months, the intervention group lowered their SBP and DBP by 9 and 7 mmHg, respectively, and 34% of the participants had a weight-loss of at least 5% body weight. At 24 months, participants were able to preserve declines in BP and generally maintained dietary behaviors and weight-loss.

DIETARY MODIFICATION

Excess sodium intake is purported to be a key driver of high BP, across all age, sex and ethnic groups [36^{*},37–40], although some have debated that sugar intake may be even more toxic on CVD and increasing BP than is salt [41]. Current clinical guidelines emphasize sodium intakes of no more than 2400 mg per day as initial treatment in prehypertensive individuals, and as a complementary aspect of pharmacological therapy in HTN adults [36^{*},38,40]. The preponderance of evidence that merits sodium reduction for HTN prevention was provided from the landmark DASH Sodium trial [38]. A feeding study involving three levels of sodium intake, 3600, 2300 and 1200 mg per day, reports increasingly lower BP results at each level [38]. Among various diet subtypes, the DASH diet, which emphasizes fruits, vegetables, fiber and low-fat dairy products, has shown to be associated with significant and greater reductions in SBP (–8 mmHg) and DBP (–4 mmHg), with magnitudes similar to earlier pharmacological monotherapy trials for mild HTN [40]. Despite the limited evidence demonstrating long-term concordance of a DASH diet for BP control, clinical guidelines recommend that adherence to a DASH diet-pattern is imperative (along with weight-loss and physical activity) for primordial prevention and management of BP [38,40]. Alternatively, DASH-style diets that resemble a Mediterranean diet including a 10% substitution of carbohydrates with unsaturated (primarily mono-unsaturated) fat, or protein [42], such as lean pork or chicken and fish [43], have also been investigated, yielding similar [43] or greater reductions in BP versus the conventional DASH diet [40, 42]. Such modifications may offer flexibility in food choices, and thus, greater adherence to effectively reduce sodium consumption.

Low compliance to a sodium-reduced diet has consistently been seen as a rate-limiting step in evaluating dietary effectiveness on BP control [44–46]. Partly explained by the fact that patients are insufficiently or not informed at all about dietary guidelines [45]; whereas, physicians have limited time during office visits to provide clinical nutrition counseling. Integrating nutritional counseling by registered dietitian nutritionists (RDNs) into primary care environments is one solution that may present an earlier opportunity to intervene in HTN and at-risk individuals. Recent evidence has shown that RDN-administered nutrition education during office visits or hospital stay significantly improved patient provision of nutrition density and quality and reduced complications associated with HTN [47,48]. RDNs are also able to assist physicians in regular BP monitoring and HTN risk

screening, and evaluate the effectiveness of evidence-based sodium reduction approaches in at-risk and HTN patients [47]. Partnerships between physicians and RDNs also appear to be essential in assisting patients with various levels of health and nutrition literacy levels, and increasing adherence to a tailored HTN prevention prescription [36,48]. Leveraging interdisciplinary collaborations between physicians, RDNs and other health providers to implement quality improvement in nutrition support may strengthen the current continuum care model, by delivering a more efficient, population-wide approach for HTN prevention and maintenance.

ALCOHOL CONSUMPTION

Alcohol consumption and its relation to a number of CVDs follows a J-shaped curve [49]. Light-to-moderate consumption of alcohol can confer CVD protection compared with nondrinkers, whereas chronic overconsumption exponentially increases risk of CVD and CVD events [49]. The acute, excess consumption of alcohol, or 'binge drinking', is associated with vascular oxidative stress, changes in endothelial and smooth cell function, and vascular reactivity [50]. Men and women reporting one or more episodes of binge drinking per week have been reported to have a prevalence ratio for pre-HTN of 1.26 [95% confidence interval (CI) 1.08–1.53] and 1.49 (95% CI 0.87–2.56), respectively [51]. This is particularly troubling considering that roughly 38 million US adults binge drink on four occasions per month [52] and those between the ages of 40 and 60 years with history of binge drinking have an elevated risk of stroke and myocardial infarction (MI) [53–55]. Furthermore, chronic excess consumption of alcohol increases the risk of HTN in a dose-dependent manner [49]. Analysis of alcohol consumption patterns and its relation to BP over time from the Women's Health Study and Physicians' Health Study identified four or more drinks per day for women and one or more drinks per day in men to significantly increase the risk of HTN [56] [with 'a drink' defined as a 12 oz of beer (~5% ethanol), 5 oz of table wine (~12% ethanol) or 1.5 oz of hard liquor or distilled spirits (about 40% ethanol)].

Much of our current understanding of the link between alcohol consumption and CVD risks/benefits has been derived from large-scale observational studies. The few investigations that examined the effects of alcohol reduction on BP have been limited by small sample sizes and/or short intervention durations [57]. A meta-analysis in this area of study noted significant decreases in SBP

(–3.3 mmHg) and DBP (–2.0 mmHg) in response to counseling programmes or low-alcohol beer substitution interventions [58]. These observations emphasize the potential BP-lowering effects of alcohol consumption interventions lead by LMI experts. What is less known, however, is the relation between elevated BP and its response to the start of light-to-moderate drinking habits (up to one drink per day for women and up to two drinks per day for men). Although there is observational evidence to support the cardioprotective effects of light-to-moderate drinking, the implications of recommending alcohol consumption with the purpose of lowering BP among nondrinkers on future drinking habits are unknown. Therefore, patients with elevated BP who drink above the recommended level should be referred to LMI programmes aimed at decreasing consumption, whereas nondrinkers pursue other LMIs discussed in this review [49].

SMOKING CESSATION AND BLOOD PRESSURE MANAGEMENT

Tobacco use is a leading preventable cause of preventable death in the United States [59]. Although today's smoking rate is lower than years past, progress may have reached a plateau. Furthermore, the increased use of noncigarette tobacco products and the introduction of new smoking products (e.g. e-cigarettes) maintain tobacco use as a strong lifestyle risk factor that impacts BP management [60]. Attempts to make the general public aware of the well-documented correlation between cigarette consumption has with cancer and CVD are still ongoing [61]. The Surgeon General's 1990 report showed a 50% reduction in death from CVD with complete cessation from smoking after 1 year [62] yet as of 2013, the Centers of Disease Control estimates that almost 18% of Americans remain chronic smokers [60]. Therefore, smoking is a complicated lifestyle risk factor in the sense that knowledge and awareness alone may not be enough to implement cessation as an LMI change. According to recent data from the Centers of Disease Control (2012–2013), over 60% of tobacco users are everyday users [63]. Cigarette smoke contains approximately 7000 various chemical substances, many of which are risk factors for CVD [59].

The INTERHEART study found that smoking even at relatively low doses 1–4 cigarettes per day had a 40% increased risk of MI compared with those who do not smoke [64]. Traditional cardiac rehabilitation programmes and healthy lifestyle promotion programmes (i.e. communities and workplaces) include smoking cessation components to reduce

and eliminate smoking for secondary prevention [65,66]. Obviously, public health messages and campaigns about the health dangers of smoking are also critical to implementation of BP control. However, recent work has found that novel uses of health education, mobile health and electronic communication strategies may be important in effective modern era smoking cessation programmes. For example, a recent meta-analysis found that comprehensive telephone interventions that included exercise education and smoking cessation, and dietary advice demonstrated reduced risk for hospitalization following MI and demonstrated more effective rates of smoking cessation [67]. Interestingly, there did not seem to be an effect on lipids [67]. In a recent clinical trial incorporating text messaging with health advice into a primary care-implemented smoking cessation programme, there was a benefit (24%) on effective 6-month cessation rates over those who received health information alone (11%) [68]. Another recent trial found that exercise interventions combined with smoking cessation counseling were more effective than health information and counseling alone after 52 weeks of follow-up following the intervention [69]. The addition of exercise may be critical to the implementation of comprehensive BP management programmes that include smoking cessation given the risks of weight gain that may mask some of the CVD risk reductions afforded by the reduction in tobacco use [69]. Interestingly, another study focusing on community health worker-implemented intervention for LMI change and CV risk reduction following MI found that the intervention which consisted of two in home visits focusing on patient diaries and health education was more effective for smoking cessation adherence compared with standard of care (85 versus 50%) [70]. Other lifestyle change outcomes such as exercise and drug adherence were improved in the intervention over the standard of care. After 1 year, the intervention group demonstrated lower BP and a significant reduction in body weight at the end of the study period [70]. There is very little known about the exact mechanism linking smoking cessation, the mode of smoking cessation and the benefits on BP reduction. Understanding the optimal mode of smoking reduction for the BP management across diverse populations will be an important area of clinical investigation.

CONCLUSION

In summary, LMI that promotes increases in physical activity, weight-loss, low sodium and low sugar diets, low or moderate alcohol and/or

smoking cessation can help manage elevated BP in most individuals. An effective method of implementing these changes can be achieved through forming multidisciplinary teams that can provide individual or group care, as well as help formulate community support networks that empower patients to adopt LMI changes that positively impact health.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

1. Mills KT, Bundy JD, Kelly TN, *et al.* Global disparities of hypertension prevalence and control: a systematic analysis of population-based studies from 90 countries. *Circulation* 2016; 134:441–450.
 2. Mozaffarian D, Benjamin EJ, Go AS, *et al.* Heart disease and stroke statistics—2015 update: a report from the American Heart Association. *Circulation* 2015; 131:e29–e322; 3.
- Published annually, this update is an excellent resource for cardiovascular statistics and associated risk factors.
3. Milani RV, Lavie CJ, Wilt JK, *et al.* New concepts in hypertension management: a population-based perspective. *Prog Cardiovasc Dis* 2016; 59:289–294.
- Outlines a model of integrating nonphysician personnel to provide personalized patient care for the management of HTN.
4. Phillips SA, Martino S, Arena R. Research opportunities and challenges in the era of healthy living medicine: unlocking the potential. *Prog Cardiovasc Dis* 2017. [Epub ahead of print]
 5. Fortuna RJ, Nagel AK, Rose E, *et al.* Effectiveness of a multidisciplinary intervention to improve hypertension control in an urban underserved practice. *J Am Soc Hypertens* 2015; 9:966–974.
- Highlights the efficacy of a multidisciplinary approach in HTN control in underserved settings.
6. Go AS, Bauman MA, Coleman King SM, *et al.* An effective approach to high blood pressure control: a science advisory from the American Heart Association, the American College of Cardiology, and the Centers for Disease Control and Prevention. *J Am Coll Cardiol* 2014; 63:1230–1238.
 7. Lavie CJ, Arena R, Swift DL, *et al.* Exercise and the cardiovascular system: clinical science and cardiovascular outcomes. *Circ Res* 2015; 117:207–219.
- Reviews acute and chronic physiological responses to exercise training and the effects of physical activity and cardiorespiratory fitness on CVD.
8. Ghadieh AS, Saab B. Evidence for exercise training in the management of hypertension in adults. *Can Fam Physician* 2015; 61:233–239.
 9. Donley DA, Fournier SB, Reger BL, *et al.* Aerobic exercise training reduces arterial stiffness in metabolic syndrome. *J Appl Physiol* 2014; 116:1396–1404.
 10. Leosco D, Parisi V, Femminella GD, *et al.* Effects of exercise training on cardiovascular adrenergic system. *Front Physiol* 2013; 4:348.
 11. Pescatello LS, Franklin BA, Fagard R, *et al.* American College of Sports Medicine position stand. Exercise and hypertension. *Med Sci Sports Exerc* 2004; 36:533–553.
 12. Nielson CM, Lockhart BD, Hager RL, *et al.* The effect of CardioWaves interval training on resting blood pressure, resting heart rate, and mind-body wellness. *Int J Exerc Sci* 2016; 9:89–100.
 13. Lewington S, Clarke R, Qizilbash N, *et al.* Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet* 2002; 360:1903–1913.

14. Wasfy MM, Baggish AL. Exercise dose in clinical practice. *Circulation* 2016; 133:2297–2313.
15. Allen NB, Siddique J, Wilkins JT, *et al.* Blood pressure trajectories in early adulthood and subclinical atherosclerosis in middle age. *JAMA* 2014; 311:490–497.
16. Gray L, Lee IM, Sesso HD, Batty GD. Blood pressure in early adulthood, hypertension in middle age, and future cardiovascular disease mortality: HAHS (Harvard Alumni Health Study). *J Am Coll Cardiol* 2011; 58:2396–2403.
17. Spring B, Moller AC, Colangelo LA, *et al.* Healthy lifestyle change and subclinical atherosclerosis in young adults: Coronary Artery Risk Development in Young Adults (CARDIA) study. *Circulation* 2014; 130:10–17.
18. Vuori IM, Lavie CJ, Blair SN. Physical activity promotion in the healthcare system. *Mayo Clin Proc* 2013; 88:1446–1461.
19. Chang SH, Chen MC, Chien NH, Lin HF. Effectiveness of community-based exercise intervention programme in obese adults with metabolic syndrome. *J Clin Nurs* 2016; 25 (17–18):2579–2589.
20. Farinatti P, Monteiro WD, Oliveira RB. Long term home-based exercise is effective to reduce blood pressure in low income Brazilian hypertensive patients: a controlled trial. *High Blood Press Cardiovasc Prev* 2016; 23:395–404.
21. Nes BM, Gutvik CR, Lavie CJ, *et al.* Personalized activity intelligence (PAI) for prevention of cardiovascular disease and promotion of physical activity. *Am J Med* 2016; 130:328–336.
22. Suboc TB, Strath SJ, Dharmashankar K, *et al.* Relative importance of step count, intensity, and duration on physical activity's impact on vascular structure and function in previously sedentary older adults. *J Am Heart Assoc* 2014; 3:e000702.
23. Suboc TB, Knabel D, Strath SJ, *et al.* Associations of reducing sedentary time with vascular function and insulin sensitivity in older sedentary adults. *Am J Hypertens* 2016; 29:46–53.
- Demonstrates the health benefits of reducing sedentary time with wearable technologies.
24. Lavie CJ, Sharma A, Alpert MA, *et al.* Update on obesity and obesity paradox in heart failure. *Prog Cardiovasc Dis* 2016; 58:393–400.
25. Lavie CJ, De Schutter A, Parto P, *et al.* Obesity and prevalence of cardiovascular diseases and prognosis: the obesity paradox updated. *Prog Cardiovasc Dis* 2016; 58:537–547.
26. Piepoli MF, Hoes AW, Agewall S, *et al.* 2016 European Guidelines on cardiovascular disease prevention in clinical practice. *Rev Esp Cardiol (Engl Ed)* 2016; 69:939.
27. Garvey WT, Mechanick JL, Brett EM, *et al.* American Association of Clinical Endocrinologists and American College of Endocrinology Comprehensive Clinical Practice Guidelines for medical care of patients with obesity. *Endocr Pract* 2016; 22 (Suppl 3):1–203.
28. Poorolajal J, Farbaksh F, Mahjub H, *et al.* How much excess body weight, blood sugar, or age can double the risk of hypertension? *Public Health* 2016; 133:14–18.
29. Hua K, Hao G, Li W. Cardiovascular outcomes of lifestyle intervention in hypertensive patients with antihypertensive agents. *Int J Cardiol* 2017; 227:751–756.
30. Mudaliar U, Zabetian A, Goodman M, *et al.* Cardiometabolic risk factor changes observed in diabetes prevention programs in us settings: a systematic review and meta-analysis. *PLoS Med* 2016; 13:e1002095.
31. Weiss EP, Albert SG, Reeds DN, *et al.* Effects of matched weight loss from calorie restriction, exercise, or both on cardiovascular disease risk factors: a randomized intervention trial. *Am J Clin Nutr* 2016; 104:576–586.
32. Mehta AK, Doshi RS, Chaudhry ZW, *et al.* Benefits of commercial weight-loss programs on blood pressure and lipids: a systematic review. *Prev Med* 2016; 90:86–99.
33. Vakili RM, Doshi RS, Mehta AK, *et al.* Direct comparisons of commercial weight-loss programs on weight, waist circumference, and blood pressure: a systematic review. *BMC Public Health* 2016; 16:460.
34. Swift DL, Johannsen NM, Lavie CJ, *et al.* The role of exercise and physical activity in weight loss and maintenance. *Prog Cardiovasc Dis* 2014; 56:441–447.
35. Keyserling TC, Samuel-Hodge CD, Pitts SJ, *et al.* A community-based lifestyle and weight loss intervention promoting a Mediterranean-style diet pattern evaluated in the stroke belt of North Carolina: the Heart Healthy Lenoir Project. *BMC Public Health* 2016; 16:732.
- Demonstrates the effectiveness of a community-based lifestyle modification program on lowering BP.
36. Davy BM, Halliday TM, Davy KP. Sodium intake and blood pressure: new controversies, new labels, new guidelines? *J Acad Nutr Diet* 2015; 115:200–204.
- Summarizes current sodium intake guidelines.
37. McLaren L, Sumar N, Barberio AM, *et al.* Population-level interventions in government jurisdictions for dietary sodium reduction. *Cochrane Database Syst Rev* 2016; 9:Cd010166.
38. Van Horn L. Dietary sodium and blood pressure: how low should we go? *Prog Cardiovasc Dis* 2015; 58:61–68.
39. Mozaffarian D, Fahimi S, Singh GM, *et al.* Global sodium consumption and death from cardiovascular causes. *N Engl J Med* 2014; 371:624–634.
40. Gay HC, Rao SG, Vaccarino V, Ali MK. Effects of different dietary interventions on blood pressure: systematic review and meta-analysis of randomized controlled trials. *Hypertension* 2016; 67:733–739.
41. DiNicolantonio JJ, O'Keefe JH. Hypertension due to toxic white crystals in the diet: should we blame salt or sugar? *Prog Cardiovasc Dis* 2016; 59:219–225.
42. Chiu S, Bergeron N, Williams PT, *et al.* Comparison of the DASH (Dietary Approaches to Stop Hypertension) diet and a higher-fat DASH diet on blood pressure and lipids and lipoproteins: a randomized controlled trial. *Am J Clin Nutr* 2016; 103:341–347.
43. Sayer RD, Wright AJ, Chen N, Campbell WW. Dietary Approaches to Stop Hypertension diet retains effectiveness to reduce blood pressure when lean pork is substituted for chicken and fish as the predominant source of protein. *Am J Clin Nutr* 2015; 102:302–308.
44. Jiang J, Liu M, Troy LM, *et al.* Concordance with DASH diet and blood pressure change: results from the Framingham Offspring Study. *J Hypertens* 2015; 33:2223–2230.
45. Kim H, Andrade FC. Diagnostic status of hypertension on the adherence to the Dietary Approaches to Stop Hypertension (DASH) diet. *Prevent Med Rep* 2016; 4:525–531.
46. Slade AN, Kim H. Dietary responses to a hypertension diagnosis: evidence from the National Health and Nutrition Examination Survey (NHANES)2007 2010. *Behav Med (Washington DC)* 2014; 40:1–13.
47. Lenders CM, Deen DD, Bistrian B, *et al.* Residency and specialties training in nutrition: a call for action. *Am J Clin Nutr* 2014; 99 (5 Suppl):1174S–1183S.
48. Nakano M, Eguchi K, Sato T, *et al.* Effect of intensive salt-restriction education on clinic, home, and ambulatory blood pressure levels in treated hypertensive patients during a 3-month education period. *J Clin Hypertens (Greenwich, Conn)* 2016; 18:385–392.
49. O'Keefe JH, Bhatti SK, Bajwa A, *et al.* Alcohol and cardiovascular health: the dose makes the poison...or the remedy. *Mayo Clin Proc* 2014; 89:382–393.
50. Piano MR, Mazzucco A, Kang M, Phillips SA. Cardiovascular consequences of binge drinking: an integrative review with implications for advocacy, policy, and research. *Alcohol Clin Exp Res* 2017; 41:487–496.
51. Fan AZ, Li Y, Elam-Evans LD, Balluz L. Drinking pattern and blood pressure among nonhypertensive current drinkers: findings from 1999–2004 National Health and Nutrition Examination Survey. *Clin Epidemiol* 2013; 5:21–27.
52. Kanny D, Liu Y, Brewer RD; Centers for Disease C, Prevention. Binge drinking:United States, 2009. *MMWR Suppl* 2011; 60:101–104.
53. Leong DP, Smyth A, Teo KK, *et al.* Patterns of alcohol consumption and myocardial infarction risk: observations from 52 countries in the INTERHEART case-control study. *Circulation* 2014; 130:390–398.
54. Marques-Vidal P, Arveiler D, Evans A, *et al.* Different alcohol drinking and blood pressure relationships in France and Northern Ireland: The PRIME Study. *Hypertension* 2001; 38:1361–1366.
55. Mukamal KJ, Maclure M, Muller JE, Mittleman MA. Binge drinking and mortality after acute myocardial infarction. *Circulation* 2005; 112:3839–3845.
56. Sesso HD, Cook NR, Buring JE, *et al.* Alcohol consumption and the risk of hypertension in women and men. *Hypertension* 2008; 51:1080–1087.
57. Hedayati SS, Elsayed EF, Reilly RF. Nonpharmacological aspects of blood pressure management: what are the data? *Kidney Int* 2011; 79:1061–1070.
58. Xin X, He J, Frontini MG, *et al.* Effects of alcohol reduction on blood pressure: a meta-analysis of randomized controlled trials. *Hypertension* 2001; 38:1112–1117.
59. How tobacco smoke causes disease: the biology and behavioral basis for smoking-attributable disease: a report of the surgeon general. Atlanta, GA: Publications and Reports of the Surgeon General; 2010.
60. Jamal A, Agaku IT, O'Connor E, *et al.* Current cigarette smoking among adults: United States, 2005–2013. *MMWR Morb Mortal Wkly Rep* 2014; 63:1108–1112.
61. Eyre H, Kahn R, Robertson RM, *et al.* Preventing cancer, cardiovascular disease, and diabetes: a common agenda for the American Cancer Society, the American Diabetes Association, and the American Heart Association. *Circulation* 2004; 109:3244–3255.
62. Novello AC. Surgeon general's report on the health benefits of smoking cessation. *Public Health Rep* 1990; 105:545–548.
63. Agaku IT, King BA, Husten CG, *et al.* Tobacco product use among adults: United States, 2012–2013. *MMWR Morb Mortal Wkly Rep* 2014; 63:542–547.
64. Teo KK, Ounpuu S, Hawken S, *et al.* Tobacco use and risk of myocardial infarction in 52 countries in the INTERHEART study: a case-control study. *Lancet* 2006; 368:647–658.
65. Lavie CJ, Arena R, Franklin BA. Cardiac rehabilitation and healthy life-style interventions: rectifying program deficiencies to improve patient outcomes. *J Am Coll Cardiol* 2016; 67:13–15.
66. Cahalin LP, Myers J, Kaminsky L, *et al.* Current trends in reducing cardiovascular risk factors in the United States: focus on worksite health and wellness. *Prog Cardiovasc Dis* 2014; 56:476–483.

67. Kotb A, Hsieh S, Wells GA. The effect of telephone support interventions on coronary artery disease (CAD) patient outcomes during cardiac rehabilitation: a systematic review and meta-analysis. *Plos One* 2014; 9:e96581.
68. Cobos-Campos R, Apinaniz Fernandez de Larrinoa A, Saez de Lafuente Morinigo A, *et al.* Effectiveness of text messaging as an adjuvant to health advice in smoking cessation programmes in primary care. A randomized clinical trial. *Nicotine Tob Res* 2016. [Epub ahead of print]
69. Bernard P, Ninot G, Cyprien F, *et al.* Exercise and counseling for smoking cessation in smokers with depressive symptoms: a randomized controlled pilot trial. *J Dual Diagn* 2015; 11 (3-4):205-216.
70. Xavier D, Gupta R, Kamath D, *et al.* Community health worker-based intervention for adherence to drugs and lifestyle change after acute coronary syndrome: a multicentre, open, randomised controlled trial. *Lancet Diabetes Endocrinol* 2016; 4:244-253.