Beyond salt: lifestyle modifications and blood pressure

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Introduction

Pharmacological treatment for hypertension (HTN) is effective in reducing both blood pressure (BP) and morbidity and mortality from cardiovascular and renal diseases. However, long-term pharmacological therapy can have adverse effects and requires continuous medical supervision. Lifestyle changes effect real and significant BP reductions. Salt intake reduction is supported perhaps by the greatest strength and diversity of evidence. This review attempts to help synthesize and clarify which of the many non-salt lifestyle modalities intended to reduce BP actually work, for whom, and to what extent.

The challenges are to substantiate in the minds of physicians and patients that lifestyle interventions for BP reduction are neither impractical nor ineffective, but rather simple, safe, and greatly consequential mediators of BP, and to design and implement public health initiatives.

Weight loss

A study of cause-specific deaths for the US population in 2004 revealed that obesity [body mass index (BMI) more than 30] was associated with 112,159 excess cardiovascular disease-related deaths and with 13,839 excess deaths from cancers considered obesity-related. Overweight (BMI 25–30) and obesity combined were associated with 61,248 excess deaths from diabetes and kidney disease.¹

Weight loss is recommended, in major guidelines, as an initial intervention in the treatment of hypertensive patients.² In the
TOHP-I trial, weight loss yielded a greater BP reduction at 6 months than did sodium intake reduction.9

Weight loss and blood pressure reduction: mechanism of action

It is hypothesized that visceral (as opposed to subcutaneous) adiposity and BP are linked through increased calorie, protein, and carbohydrate intake, resulting in increased plasma catechoamines, sympathetic nervous system activity, and insulin secretion.5 Consequently, renin–angiotensin–aldosterone system (RAAS) activation results in renal sodium retention through aldosterone synthesis, yielding increased cardiac output in the setting of insufficient adjustment in peripheral vascular resistance, all of which results in HTN (Figure 1).

Obese women had higher circulating levels of angiotensinogen, renin, aldosterone, and angiotensin-converting enzyme when compared with lean women.8 Decrease in BP following weight loss is associated with reduced sympathetic nervous system and RAAS activity, as well as accelerated natriuresis.9 With 10 kg weight loss, there was lowering of total circulating blood volume without change in peripheral vascular resistance, thus yielding decreased venous return and cardiac output.7

A possible link between weight and various physiological changes is leptin, which when secreted by adipocytes binds to a receptor on the hypothalamus and increases renal sodium and water excretion and alters vasoactive substances such as nitric oxide in the endothelium.10,11 In human adult populations, among the studies that report a positive association between leptin and BP, some report the association to be independent of adiposity, whereas others do not. A recent study illustrates that leptin accounts for a proportion of the variance in the association between BMI and BP, but that the association of leptin and BP is almost entirely explained by BMI.12

**Figure 1** Hypothetical mechanisms by which obesity may contribute to HTN. CNPS, cardia natriuretic peptide system; OSA, obstructive sleep apnoea; RAAS, renin–angiotensin–aldosterone system; SNS, sympathetic nervous system.

Relationship between weight loss and blood pressure

The relationship between weight loss and BP reduction appears to be linear (Figure 2).13 By and large, a reduction of 1 kg body weight is associated with 2/1 mmHg BP reduction.14 Reduction in BP due to weight loss is related to the decrease in visceral fat mass.15

Does it matter how one loses the weight?

The short answer is yes. Among the possible means of reducing body weight are lifestyle modifications, pharmacological interventions, and invasive or surgical interventions.

A 4 kg weight loss achieved with dietary treatment yielded a 6 mmHg systolic BP (SBP) reduction; the same 4 kg weight loss achieved with orlistat (decreases dietary fat absorption by inhibiting activity of pancreatic lipases) yields a lesser 2.5 mmHg reduction in SBP.16 An 8.4 kg reduction in weight using orlistat yielded a 4.0/3.0 mmHg reduction in BP, whereas an 8.3 kg weight reduction through the use of sibutramine (serotonin–norepinephrine reuptake inhibitor that acts as an appetite suppressant) did not cause a change in BP. Sibutramine may actually have a BP-raising effect that counteracts the BP reduction that comes with its weight loss effects. In the SCOUT trial, sibutramine was associated with a higher composite risk of heart attack, stroke, resuscitated cardiac arrest, or death.17

Bariatric surgical methods allow for the greatest degree of weight loss, ranging from 21 to 38%, 1-year post-operation depending on the type of surgery.18 For a weight loss at 2- and 10-year post-surgery of 23.4 and 16.1%, respectively, BP change was −4.4/−5.2 and +0.5/−2.6 mmHg. At 10-year post-surgery, although weight loss was sustained (though somewhat diminished), the BP-lowering effect was diminished.

Physical activity

A meta-analysis of randomized controlled trials found that aerobic exercise reduced BP by roughly 5/3 mmHg.19 This effect appears to be independent of the weight reduction associated with physical activity.20 In a study of normotensives followed for a mean of 4 years, increased physical fitness (a physiological response to regular physical activity) was significantly associated with decreased risk of developing elevated BP.21

Fitness was determined based on the duration each person was able to undergo a graded symptom-limited maximal exercise test.22 The association of fitness with incident HTN and other cardiovascular conditions is stronger than the association of physical activity with those same cardiovascular diseases. In the Coronary Artery Risk Development in Young Adults (CARDIA) Study, both baseline fitness and physical activity were inversely associated with incident HTN.23 The magnitude of association between physical activity and HTN was strongest and significantly inversely related with HTN incidence only among participants in the high-fitness category, whereas the magnitude of association between fitness and HTN was similar across tertiles of physical activity. The association of fitness or activity with incident HTN was modestly attenuated when BMI and waist circumference were included.
These findings may suggest that activity that does not raise fitness levels may not lower the likelihood of developing HTN.

The association between physical activity and BP seems to be dose-dependent, but only to a certain level of activity. Those who exercised 61–90 min/week gained more benefit than the 31–60 min/week group, but there was no further benefit beyond 61–90 min/week. It seems, therefore, that one does not need to train very aggressively to attain most of the fitness-related BP-reduction benefits.

Further, physical activity need not be equated with formal physical training, as in a gym or on a playing field. In a Japanese study, relative risk for HTN among middle-aged Japanese men decreased from 1.0 to 0.84 to 0.75 to 0.54 moving up from the first to fourth quartile of daily life energy expenditure. Daily life expenditure was calculated from the composite scores of such commonplace daily activities as sleeping, reading, driving, slow walking, taking a bath, and climbing stairs. The most active in this study, who incurred the greatest BP benefits, were doing such basic activities such as walking and climbing stairs. This study also showed that while exercise reduces the risk of HTN for all three levels of normotension (low normal, normal, and high normal), the benefit of activity in reducing HTN risk diminished with increasing baseline BP.

Exercise may diminish sympathoadrenergic activity and enhance prostaglandin mechanisms and augment endothelium-dependent vasodilatation through increasing production of nitric oxide. Exercise most effectively reduced BP in those with low-plasma renin activity at baseline.

Older persons may be resistant to BP-lowering effects of exercise. There was a lack of improvement in aortic stiffness with exercise which likely explains why there was no reduction in SBP and only modest reduction in diastolic BP (DBP) among elderly exercisers compared with elderly controls.

**DASH diet**

The landmark Dietary Approaches to Stop Hypertension (DASH) showed that a diet rich in fruits and vegetables with fat content typical for the US population lowered BP by 2.8/1.1 mmHg compared with a control diet. When such a diet also incorporated low-fat dairy products and low saturated and total fat (the DASH diet), BP decreased by 5.5/3.0 mmHg. The findings were even more pronounced in hypertensives (11.4/5.5 mmHg drop in BP for the DASH diet). That dietary intervention lowered BP in the normotensive participants implicates dietary intervention in primary prevention of HTN. In those already with high BP, dietary modification similar to the DASH diet has anti-hypertensive efficacy comparable with drug monotherapy for mild HTN.

Importantly, the reduction in BP began within 2 weeks and was maintained for the duration of the dietary intervention (an additional 6 weeks). The trial was not designed to assess the long-term effects of the diets on BP or clinical cardiovascular events.

Known diet-related determinants of BP—salt intake, weight, and alcohol—could not have accounted for the reductions in BP because differences in these were small and similar for all the diets. Further, multiple components of the DASH diet appear independently effective in reducing BP. Therefore, incorporating at least some of the components of the DASH diet into one’s own diet confers advantageous BP effects. A dietary intervention as in DASH is one the average person can realistically model. Adherence to the assigned diet was over 90% and not lower for the intervention arms compared with the control group.

**Dietary minerals and nutrients**

Significant inverse associations of BP with intake of magnesium, potassium, calcium, fibre, and protein have also been reported.
However, in trials that tested these nutrients, often as dietary supplements, the reduction in BP has typically been small and inconsistent. The effect of any individual nutrient in lowering BP may be too small to detect in trials. When several nutrients with small BP-lowering effects are consumed together, however, the cumulative effect may be sufficient for detection. The most convincing data for any single mineral in isolation exist for potassium.

**Potassium**

A meta-analysis found that for a 1.7 g increase in potassium intake, there was a 3.5/2.5 mmHg drop in BP for hypertensives and a 1.0/0.3 mmHg drop for normotensives. In a meta-analysis of 32 trials, potassium supplementation was associated with a fall in BP of 3.1/2.0 mmHg; the effect was more pronounced in patients who had higher salt intake (potassium has a powerful inhibitory effect on salt sensitivity). Increases in serum potassium hyperpolarize the endothelial cell through stimulation of the sodium pump, which results in decreased cytosolic calcium, which in turn promotes vasodilatation.

A recent analysis of the NHANES III study found that the sodium–potassium ratio in the highest (87.5) compared with lowest (12.5) quartiles was associated with a hazard ratio of 1.46, 1.46, and 2.15 for all-cause, cardiovascular disease, and ischemic heart disease mortality, respectively.

Several epidemiological studies show an association between increased dietary potassium intake and reduced stroke rate. An increase in potassium intake of 10 mmol/day led to a 40% reduction in stroke mortality. Increased intake of 1.64 g/day showed a statistically significant 21% risk reduction in stroke and trend towards lower cardiovascular disease. The relationship appears to be partly, but not entirely, mediated by the higher potassium intake effect on BP.

**Magnesium**

Magnesium deficiency raises BP in animals, and supplementation seems to prevent HTN in these models. Among trials in humans, although there are data linking low magnesium to incident coronary heart disease, there are no convincing data that magnesium deficiency is significantly related to increased BP, in a Framingham study cohort, for example, there was no association between baseline magnesium and incident HTN.

**Vitamin D (25(OH)D)**

25(OH)D is implicated in a great variety of clinical condition. Adolescents in NHANES II with the lowest 25(OH)D values had more than a two-fold greater odds of having elevated BP compared with the group of adolescents with higher 25(OH)D levels. Plasma 25(OH)D levels are inversely and independently associated with the risk of developing HTN. Women in the lowest compared with highest quartile of plasma 25(OH)D had an adjusted odds ratio for incident HTN of 1.66.

Nonetheless, a causal relationship between vitamin D and HTN (or many of the other cardiometabolic disease states) has not been established, and there are no convincing data as of yet that increasing vitamin D levels (via supplementation or increased sunlight exposure) may be effective for the primary prevention or treatment of HTN.

**Flavonoids**

Flavonoids, polyphenolic compounds found in high concentrations in foods such as fruits, vegetables, grains, legumes, tea, beer, and wines, are known for their antioxidant properties. Experimental evidence suggests that flavonoids exert beneficial BP effects by increasing endothelial-derived nitric oxide. One meta-analysis showed that some subclasses of flavonoids are associated with significant BP reductions: cocoa flavan-3-ols (found in cocoa beans and tea), for example, reduced BP by 5.9/3.3 mmHg. However, the design of the studies was such that the effect seen may be exaggerated and not exclusively attributable to the flavonoid components. Also, the quantity of flavonoids consumed by participants in the study was beyond the range typically consumed. One small study showed that eating a small amount of dark chocolate daily lowered BP 2.9/1.9 mmHg in those with pre-HTN or Stage 1 HTN. Another recent large prospective study of habitual intake of the various subclasses of flavonoids showed that a higher total anthocyanin (predominantly from blueberries and strawberries) intake decreased the rate of incident HTN after correcting for physical activity, BMI, and dietary factors.

**Caffeine and nicotine**

Caffeine has been shown to increase BP, but this effect resolves 4 h after ingestion. These effects are attenuated in habitual drinkers, suggesting tolerance to adrenergic effects. Habitual caffeine use has been shown to have a minimal effect on long-term BP. Nicotine seems to have divergent effects on BP; acutely, BP seems to rise while one is smoking a cigarette, but chronic tobacco use seems to be associated with lower BP, with one study showing an SBP reduction of 1.3 and 4.6 mmHg for light and heavy smokers, respectively.

**Biofeedback therapies**

Biofeedback training includes elements of cognitive therapy and relaxation training. Meditation, yoga, and breathing exercises are just three of the various types of relaxation therapy. A Cochrane meta-analysis comparing relaxation therapy to no active treatment among hypertensive individuals showed a 5.5/3.5 mmHg reduction in BP at 8 weeks; however, there were only nine trials with outcome assessment blinding and these trials yielded a nonsignificant 3.2 mmHg reduction in SBP. The authors concluded that the evidence favouring a causal relationship between relaxation therapy and BP reduction was weak.

‘Device-guided breathing’, through the use of commercially available FDA-approved electronic devices, is a form of biofeedback therapy that guides users to slower respiratory rates, which increases tidal volume, increases cardiopulmonary stretch receptor stimulation, and reduces sympathetic efferent discharge, thus resulting in vasodilatation. Such breathing has been shown to achieve modest BP reduction effects without adverse effects in observational and larger prospective-matched case–control
studies and prospective-randomized controlled trials. Studies, however, did not go beyond an 8-week follow-up period. It is currently considered adjunctive treatment, and more evidence is needed before it can be widely recommended as a treatment that provides predictable and meaningful BP reduction.

Alcohol

It has long been recognized that the problems with alcohol relate not to the use of a bad thing, but to the abuse of a good thing.

Abraham Lincoln

Sixteen per cent of all hypertensive disease is attributed to alcohol. What makes the issue of alcohol and cardiovascular risk intriguing and complicated is that unlike the effects on other organ systems, the cardiovascular effects of alcohol consumption are a mixed bag; some advantageous and others deleterious.

Magnitude and duration of effect

Longitudinal studies have shown a strong, linear relationship between alcohol consumption and BP. In one meta-analysis, an average 67% reduction in alcohol consumption yielded a net decrease in BP of 3.31/2.04 mmHg. Importantly, alcohol intake has paradoxically divergent effects on BP and coronary heart disease. Heavy drinking is associated with a higher prevalence of HTN, haemorrhagic stroke, and cardiomyopathy, whereas moderate drinking is associated with lower prevalence of coronary artery disease, ischaemic stroke, and sudden cardiac death.

Even in lower-risk individuals—men of mean age 57 who followed all four of the major healthy lifestyle behaviours (abstinence from smoking, maintaining a BMI 25 kg/m², exercising at least 30 min daily, and eating a healthy diet)—the consumption of one or two drinks per day was associated with a 40–50% decreased

Table I Relative blood pressure impacts of some of the major lifestyle modifications

<table>
<thead>
<tr>
<th>Modification</th>
<th>Recommendation</th>
<th>SBP reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>Maintain BMI 18.5–24.9</td>
<td>4.4 mmHg²</td>
</tr>
<tr>
<td>Adopt DASH eating plan</td>
<td>Consume a diet rich in fruits, vegetables, and low-fat dairy products with a reduced content of saturated and total fat</td>
<td>5.5–11.4 mmHg²</td>
</tr>
<tr>
<td>Dietary sodium reduction</td>
<td>Reduce dietary sodium intake to no more than 2.4 g sodium (or 6 g salt)</td>
<td>4–7 mmHg²</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Engage in aerobic physical activity 20–60 min/day, 3–5 days/week</td>
<td>5 mmHg²</td>
</tr>
<tr>
<td>Moderation of alcohol consumption</td>
<td>Limit to no more than 2 drinks per day (men) or 1 drink/day (women)</td>
<td>3 mmHg²</td>
</tr>
</tbody>
</table>

Figure 3 Estimated decrease in blood pressure mediated by non-pharmacological intervention in hypertension (modified from Messerli et al.).
risk of myocardial infarction. A meta-analysis of over 1 million individuals showed that consumption of one drink (1.5 oz of hard liquor, 5 oz of wine, or 12 oz of beer) daily by women and one or two drinks daily by men was associated with a reduction in total mortality of 18%. Intakes of two drinks daily in women and three drinks daily in men, however, were associated with increased mortality in a dose-dependent fashion.

**Pattern and type of alcohol consumption**

The frequency and setting of consumption was found consequential in more than one study. Data from the USA and Italy show that after adjustment for average amount of alcohol consumed over 30 days, drinking without food was associated with a strong significant increased risk of HTN, even in individuals with light-to-moderate alcohol intake (less than two drinks per day). There were no gender differences. The authors put forward possible explanations: the effect of food on absorption and metabolism of ethanol leading to lower peak of blood alcohol concentration; the effect of food on increased alcohol elimination rates; alcohol intake with meals represents a surrogate marker of health-related behaviours.

For predominant beverage preference, no consistent association with HTN risk was found across the various types of beverages considered (beer, wine, and liquor).

The cardioprotective effects of mild–moderate consumption are greatest when the consumption is spread out evenly over the course of a week. Binge drinking—linked to higher prevalence of cerebral thrombosis, cerebral haemorrhage, and coronary artery disease deaths—attenuates completely the decrease in cardiovascular mortality associated with moderate drinking.

**Why bother?**

To put lifestyle interventions into context, mean placebo-subtracted SBP reduction for drug monotherapy is in the range of 6.9–9.3 mmHg for four of the most common BP drug classes. Estimated SBP reductions for some of the major lifestyle modifications are in or near this range (Table 1).

Compliance with lifestyle modification can be difficult (of course, compliance with medication is also not perfect). When one sets out to lose weight or exercise, the goal is to succeed and sustain that success. Losing weight to regain it a year later would seem a failure to most. Why bother trying if one is unlikely to ‘succeed’? The answer is that trying, even when ultimately failing, may be better than not trying at all.

At 7-year follow-up of persons aged 30–54 who lost 3.5 kg or decreased salt intake by roughly 2 g/day, intervention effects on body weight and urinary sodium excretion had disappeared but the beneficial effect on HTN had not. In fact, the intervention group was heavier by 4 kg than the control group at 7 years; yet, average SBP and DBP were 1.8 and 1.3 mmHg lower, respectively.

It is likely that attempting to engage in one lifestyle modification can have risk-reduction effects even when the intended intervention is not fully achieved. The reason may be that it is hard to pursue one lifestyle intervention in isolation; when one sets out to lose weight, for example, one is likely to also pursue dietary improvements and/or increased physical activity. If one does not achieve weight loss goals, one may incur overall benefit from partial attainment of multiple lifestyle intervention goals. In fact, the strongest evidence supporting non-drug interventions for BP reduction is not for any single modality, but rather for the combining of several.

### Comparing and combining modalities

Patients who lost weight and reduced sodium intake delayed the onset of HTN more than those who only lost weight or only reduced sodium intake. Salt intake reduction confers a greater BP reduction in obese than in non-obese individuals. Furthermore, not only is BP more salt sensitive in the overweight, high-salt intake is a stronger and independent risk factor for cardiovascular disease and all-cause mortality in overweight people.

Combining DASH diet with low-salt intake is another example of how effective combination lifestyle interventions can be (Figure 3).

The PREMIERE trial further assessed multifaceted behavioural interventions: an ‘established behavioral intervention’ consisting of weight loss, physical activity, and limitations in salt and alcohol intake, ‘established behavioral intervention’ plus DASH diet, and one-time advice only. At 6 months, DASH plus ‘established
behavioral intervention’ and ‘established behavioral intervention’ alone reduced BP 4.3/2.6 and 3.7/1.7 mmHg, respectively (Figure 4).

The kind of decrease in BP obtained with some non-drug modalities would dramatically reduce incidence of and death from cardiovascular disease in populations. Although it may be hard to get 40 people to lose 40 pounds or reduce salt intake by 6 g, it may be less difficult to get 400 people to lose 4 pounds and reduce salt intake by 2 g, especially if public health interventions create an environment increasingly conducive to such efforts.

Intervening simultaneously to address smoking cessation, reduced dietary salt intake, and increased physical activity, for example, may be more effective than addressing each behaviour sequentially.34 Individuals with the most physical activity and dietary behaviour goals were the most likely to meet the most goals; attempting to meet one physical activity or dietary goal may actually enhance chances for attainment of a second such goal.35 And just as a combination approach to lifestyle interventions for BP reduction is greater than any single intervention, a moderate but sustained intervention is better than a more significant but short-lived one.

In conclusion, lifestyle changes safely and effectively delay or prevent HTN in non-hypertensives, delay or prevent medical therapy in Stage I hypertensives, and contribute to BP reduction in hypertensives already on medical therapy.

Conflict of interest: none declared.

References