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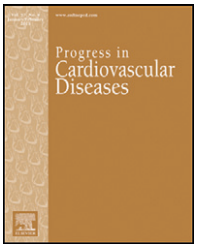

Exercise and Hypertension: Uncovering the Mechanisms of Vascular Control

Ahmad Sabbahi[a], *Ross Arena*[a], *Ahmed Elokda*[b], *Shane A. Phillips*[a,*]

[a]*Department of Physical Therapy and the Integrative Physiology Laboratory, College of Applied Health Sciences, University of Illinois at Chicago, Chicago, IL*

[b]*Department of Rehabilitation Sciences, Florida Gulf Coast University, Fort Myers, FL*

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ABSTRACT

Hypertension (HTN) has recently been determined to be the number one overall risk factor of disease. With direct and indirect costs amounting to $46.4 billion in 2011 and projections of six-fold increases by 2030, the importance of low-cost nonpharmacological interventions can be appreciated. Vascular structural changes, endothelial dysfunction, and sympathetic overstimulation are major contributing factors to the pathophysiology of HTN. Exercise training (ET) for blood pressure (BP) control has been shown to be an effective and integral component of nonpharmacological interventions for BP control. Different ET modalities (aerobic, resistance, and concurrent training) have contributed differently to BP reduction and control, driving scientific discourse regarding the optimum ET prescription (modality, volume, and intensity) for such effects; ET results in a multitude of physiological effects, with vascular and autonomic adaptations providing major contributions to BP control. Despite widespread acceptance of the role and importance of ET for BP reduction, only 15% of US adults have been found to meet ET/physical activity recommendations. The purpose of this review is to explore BP lowering effects of aerobic and resistance ET and the underlying physiological mechanisms that result in such effects. Further research is required to enhance our understanding of the proper ET prescription for BP control across different age groups and racial ethnicities. Furthermore, research into methods of improving awareness and adherence to ET recommendations proves to be equally important.

Exercise and hypertension

It has been estimated that more than one in every three Americans have one or more types of cardiovascular disease (CVD), and nearly the same percentage have hypertension (HTN) (identified as systolic blood pressure [BP;SBP] ≥140 mmHg and/or diastolic BP [DBP] ≥90 mmHg or being on anti-HTN medication).[1] When considering pre-HTN (SBP ≥120 to<140 mmHg; DBP ≥80 to<90 mmHg), nearly 70% of Americans experience elevated BP.[1,2] About 17.3% of those with HTN are not aware of their condition,[1] and only half of those receiving treatment have their BP under effective control.[1] Given these current trends, it is thus not surprising that in 2010, HTN rose from the fourth to the overall number one global risk factor for disease.[3] HTN has been found to amount to the loss of five years of CVD-free life in adults

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* Address reprint requests to Shane A. Phillips, PhD, PT, FAHA, Department of Physical Therapy, College of Applied Health Sciences, University of Illinois, Chicago, 1919 W. Taylor Street (MC 898) 60612.

E-mail address: shanep@uic.edu (S.A. Phillips).

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Abbreviations and Acronyms

Ach = Acetylcholine

ANS = Autonomic nervous system

AT_1-R = Angiotensin II type 1 receptor

BP = Blood pressure

CHD = Coronary heart disease

CO = Cardiac output

CVD = Cardiovascular disease

DBP = Diastolic blood pressure

ET = Exercise training

FMD = Flow mediated dilation

HF = Heart failure

HTN = Hypertension of hypertensive

IMT = Intima medial thickness

MAP = Mean arterial pressure

NO = Nitric oxide

PEH = Post-exercise hypotension

SBP = Systolic blood pressure

SNS = Sympathetic nervous system

TPR = Total peripheral resistance

30–59 years of age.[4] In 2011, the direct and indirect cost of HTN was $46.4 billion, and projected to increase almost six fold by 2030.[1] With the association of such substantial risk across the lifespan, the importance of relatively low-cost nonpharmacological interventions can thus be further appreciated.

Both obesity[5] and physical inactivity,[6] ranked 6th and 10th overall risk factors for disease respectively,[3] play an important role in complicating CVD risk with HTN. The importance of exercise as a cornerstone treatment modality can be viewed in light of its ability to reduce the impact of a multitude of the top 10 CVD risk factors.

Across a wide range of etiologies, exercise training (ET) has previously been shown to rival and occasionally surpass, pharmacologic therapy in terms of treatment effectiveness for HTN.[7] In a recent meta-analysis of ET and drug trials on mortality outcomes,[8] there was no statistically significant difference between the two interventions in terms of mortality outcomes, excluding patients with stroke and heart failure (HF), where ET was determined to be more effective than drug interventions in terms of mortality reduction in the former population and was surpassed by diuretic drug therapies in the latter.[8] In patients with stable coronary heart disease (CHD), a 12-month ET program resulted in higher event-free survival rates than a standard percutaneous coronary intervention at nearly half the cost.[9]

Regarding HTN, aerobic ET has been shown to decrease BP by 5–7 mmHg while resistance ET leads to a 2–3 mmHg decrease.[2] Reductions of the magnitude found with aerobic ET translate to a decrease in the risk of stroke by 14%, CHD by 9%, and total mortality by 7% (6%, 4%, 3% for resistance ET, respectively).[10] Aerobic ET has been widely accepted and recommended as the first line of treatment for BP reduction and control both nationally and internationally.[11–17] Even as well-established evidence favoring the benefits of ET on BP continues to grow, only 15% of US adults have been reported to meet ET recommendations and therefore a substantial number of adults are at risk of poor BP control.[18]

Unlike aerobic ET, the role of resistance ET with respect to BP lowering effects and whether or not it can be used as the primary intervention for BP lowering and control is less clear. This review will summarize current literature regarding the effects of different ET modalities and proposed physiological mechanisms of BP lowering in response to various exertional stimuli.

Aerobic ET and BP

Aerobic ET has been determined to have both acute and chronic effects on BP. Fitzgerald[19] was the first to describe the term post-exercise hypotension (PEH), which later became the tenant of acute physiological effects of ET on BP. This drop in BP post exercise has also been found to be dose-dependent,[20] with higher intensity ET resulting in greater reductions in BP. This observation can be further confirmed by results of a recent meta-anlysis[21] showing a dose–response relationship between aerobic ET intensity and improved endothelial function measured by flow-mediated dilation (FMD). Increased endothelial function is associated with and may contribute to lower peripheral vascular resistance. The magnitude of the BP drop has also been found to depend on pre-exercise BP.[12] People with higher baseline BP readings show greater PEH compared to normotensives. It was also shown that people with higher baseline BP readings show the greatest reductions after chronic ET.[22] PEH has been demonstrated to be effective and significant in patients receiving anti-HTN medication after a single bout of ET,[23] and has been found to be effective across all age groups, including those 80–90 years of age.[24] In patients with resistant HTN, defined as those unable to reach target BP readings despite receiving 3 or more anti-HTN medications one of which being a diuretic,[25] a treadmill walking protocol of 3×/week for 8–12 weeks at an intensity slightly above aerobic threshold was successful in reducing systolic and diastolic 24-h ambulatory BP by 6 ± 12 mmHg and 3 ± 7 mmHg, respectively.[25] Interestingly, the finding that PEH can occur effectively after ET durations as short as 15 minutes at low intensity (40% VO_{2peak}), in contrast to high-intensity interval ET,[26] is likely very clinically relevant to HTN individuals unable to perform long bouts of ET.[27] When compared to a single 30-min ET bout, three 10-min bouts at 60%–65% VO_{2peak}were shown to be equally effective in lowering 24-h ambulatory SBP.[28] Furthermore, in a randomized crossover trial,[29] 10-min exercise bouts of treadmill walking at 50% VO_{2peak} performed once an hour over four hours showed equivalent BP reductions to a single 40-min exercise session and lasted longer (up to 3 hours) before returning to baseline[29]; BP was reduced for approximately 10 hours in the group receiving intermittently accumulated ET compared to 7 hours for the continuous ET group.

Nonetheless, it was previously identified that nearly 24% of people with elevated BP do not show reductions in BP post exercise.[30]A study by Liu et al,[31] showed strong correlations ($r = 0.89$, $P < 0.01$) between acute and chronic BP reduction responses. Participants who demonstrated the greatest BP reductions to acute exercise had the greatest chronic