CSD Grand Rounds

Sex Differences in the Effects of Aerobic and Anaerobic Exercise on Blood Pressure and Arterial Stiffness

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ABSTRACT

Exercise can be used as an antihypertensive prescription for individuals with pre- to stage-1 essential hypertension, but the outcomes differ between the sexes and depend on the mode of exercise employed. Both aerobic exercise and resistance training lower blood pressure in men and women; however, resistance training lowers diastolic blood pressure to a greater extent in women but increases arterial stiffness to a greater extent in men. These studies emphasize the need for further research on the effect of resistance exercise training on blood pressure and arterial stiffness so that sex differences in response to exercise treatment programs are fully taken into account when devising individual antihypertensive treatment regimens.

INTRODUCTION

Cardiovascular disease (CVD) is ubiquitous and is the leading cause of morbidity and mortality in the United States in both sexes. In fact, CVD accounts for 28% of total deaths in this country.[1] Essential hypertension, defined as a systolic blood pressure (SBP) >140 mm Hg and a diastolic blood pressure (DBP) >90 mm Hg, is closely associated with coronary artery disease, stroke, renal disease, and all-cause mortality.[2] Left untreated, chronic hypertension increases a person's risk for developing CVD and stroke by 3-fold and 7-fold, respectively.[2,3] For several decades now, hypertension has been a prevalent form of CVD and a growing source of all-cause mortality.[4]

Prehypertension is considered the precursor state for stage-1 essential hypertension and is defined as an SBP of 120 to 139 mm Hg or a DBP in the range of 80 to 89 mm Hg. In a report from the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure,[2] prehypertension is also considered a self-accelerating condition, in which the development of arteriolar hypertrophy and endothelial dysfunction facilitates the transition between prehypertension and essential hypertension. The prevalence rate of hypertension is increasing in the US population, but unfortunately, the rate of diagnosis is not keeping pace with this increase in incidence, and the management of this disease remains suboptimal.

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In elderly populations, hypertension is not only more prevalent in women than in men, it is also more severe and less well controlled.

For most of human life, sex differences exist in blood pressure (BP), in the incidence of hypertension, and in associated CVD. Women have lower SBP than do men through the fifth decade of life. As women become older, their BP approaches that of age-matched men, and after the sixth decade of life, women have higher BP than do same-aged men. In elderly populations (aged >70 years), hypertension is not only more prevalent in women, it is also more severe and less well controlled than in men.[5–7] In 2004 in the United States alone, 39 million of the 72 million people with hypertension were female; thus, women accounted for ~54% of all hypertension. Furthermore, hypertensive women have a higher risk of dying from this disease and its associated complications than do hypertensive men.[8] Of the 54,000 deaths in the US population attributed to hypertension, 31,400 (~58%) were female. CVD mortality is also higher in women than in men in this elderly population.[7] Furthermore, elevated BP is more directly linked to cardiovascular mortality in women than in men. The age-adjusted relative risk of death from coronary artery disease for women is twice the risk of that for men for every 20–mm Hg increase in SBP above normal levels. Given these sex differences in BP, the incidence of hypertension, and the severity and consequences of associated CVD, it is imperative that we understand the influence of biological sex on treatments for hypertension.

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AVAILABLE TREATMENTS

Currently, treatment options for managing and reducing high BP include drugs, lifestyle modification, and psychotherapy. Pharmacologic treatment is widely prescribed by physicians for treating hypertension; however, pharmacologic intervention is the subject of great debate in the treatment of individuals identified as prehypertensive. The primary aim of the Trial of Preventing Hypertension (TROPHY) was to determine whether pharmacologic treatment with candesartan, an angiotensin type 1 receptor antagonist, reduced the incidence of hypertension for up to 2 years after the discontinuation of treatment.[9] In this 4-year, multicenter, randomized study, 809 prehypertensive subjects (aged 30–65 years with a mean age of 49 years), who had never been treated with antihypertensives, were randomized to treatment with candesartan or placebo for 2 years and then followed up for an additional 2 years after treatment. The study found a 26% reduction in new onset of hypertension in the candesartan treatment group compared with the placebo group. From these findings, the research team concluded that pharmacologic treatment of prehypertension with candesartan prevents and postpones (carryover effect) the development of hypertension for up to 2 years after discontinuation of treatment.

Debate has ensued over the conclusions of the TROPHY study, because pharmacologic treatment of prehypertension would expose >25 million Americans to a myriad of potential health risks associated with antihypertensive drug treatment and have enormous financial impact on the public health care system.[10] Moreover, concerns were raised about the study's test design of the carryover effect, because the results from the carryover period may have been confounded by premeasures in the treatment period. Lumley et al[11] concluded that data from the TROPHY study were consistent with a lack of carryover effect of candesartan on postponing the development of hypertension.

Pharmacologic treatment of prehypertension would expose millions of Americans to the potential health risks associated with drug treatment and financially stress the public health care system.

Grassi[10] noted that the trend of increasing BP with aging was directly related to age-associated changes in lifestyle and diet choices. In fact, the cornerstone for treating prehypertension and preventing the development of hypertension has been lifestyle modification, of which a large part is the prescription of aerobic exercise. Moderate-intensity aerobic exercise, which is defined as reaching and sustaining 65% of peak oxygen consumption (VO_{2peak}) for 30 minutes 3 times a week, is often prescribed as a nonpharmacologic treatment regimen for high BP, because it has been found to reduce both SBP and DBP in hypertensive individuals.[12] In contrast, moderate-intensity resistance exercise, which is defined as 1 to 3 sets at 65% of an individual's 10-repetition maximum, is not typically recommended as a BP-lowering treatment strategy and is suggested only as a complement to aerobic-based programs.[13,14]

Increasing age is associated with irreversible damage to the vasculature as a result of hypertrophy or atherosclerosis. These vascular changes are believed to diminish the long-term responsiveness to antihypertensive drugs.[11,15] Thus, even if we do initiate drug treatment at the prehypertensive stage, this treatment strategy is unlikely to prevent the development and progression of hypertension in later years if obesity and sedentary lifestyle choices are not addressed as well. The somewhat promising effect of psychotherapy on reducing hypertension[16,17] is also greatly increased when combined with exercise.[18]

It is unlikely that pharmacologic treatment of prehypertension will prevent the development and progression of hypertension in later years if obesity and sedentary lifestyle choices have not been addressed.

EXERCISE AS AN INTERVENTION

Exercise is a valuable lifestyle modification that can be used to help prevent or control hypertension. It is well known that poor cardiorespiratory fitness is a strong and independent risk factor for CVD and all-cause mortality. Studies have shown that physical activity levels positively correlate with cardiorespiratory fitness; increased physical activity leads to improved cardiorespiratory fitness and reduced BP in many individuals.[19] A report by Green et al[20] suggests that exercise may confer a vascular conditioning effect that improves the vascular wall by reducing existing endothelial dysfunction. Conditioning increases the bioavailability of nitric oxide, a powerful vasodilator. Nitric oxide improves endothelial function and has antiatherogenic effects through its ability to reduce platelet aggregation in the vessel wall. The improved endothelial function and antiatherogenic properties are associated with reductions in cardiovascular risk.

Exercise is associated with a marked decrease in cardiovascular events such as myocardial infarctions. In fact, the 30% decrease in cardiac events associated with a standard aerobic exercise program (30 minutes 3 times per week) rivals the 26% to 28% reduction in risk associated with lipid-lowering and antihypertensive pharmacologic treatment regimens.[21] In addition to reducing the risk for a cardiovascular event, a meta-analysis of exercise-based cardiac rehabilitation programs suggests that aerobic exercise may reduce mortality by 20% to 32%.[22]

The percent decrease in cardiac events associated with a standard aerobic exercise program has been found to be approximately equivalent to the reduction in risk associated with lipid-lowering and antihypertensive pharmacologic treatment regimens.

The vast majority of studies investigating the effect of exercise on BP have shown that aerobic exercise lowers BP. Taylor et al[22] observed that aerobic exercise resulted in an 8– to 17–mm Hg reduction in SBP and a 6– to 13–mm Hg reduction in DBP in individuals with essential hypertension. In contrast, the effect of resistance exercise training has been underinvestigated; only a few published studies have examined the effect of such training on BP. Kelley[23] reported a meta-analysis of 9 studies examining the effect of resistance exercise training on BP in >250 adults. The pro-

grams ranged from 6 to 26 weeks of training at an average frequency of 3 days per week utilizing several muscle groups. The meta-analysis showed that resistance exercise training resulted in a 4.6–mm Hg reduction in resting SBP and a 3.8–mm Hg reduction in DBP; however, these studies were primarily undertaken in normotensive populations. Thus, the effect of resistance training on BP in individuals with hypertension is not well understood.

Resistance exercise training has been recommended by the American Heart Association only as a "complement" to an aerobic-based exercise program for hypertensive populations.[14] Consequently, clinicians may be reluctant to prescribe this mode of exercise in a patient's treatment regimen. However, there are many benefits of resistance training that should be considered. In contrast to a typical aerobic exercise routine, an effective resistance exercise program can be completed with very little sweating, making it possible to exercise without necessarily having to shower, which may therefore be more convenient for individuals with a busy schedule. In addition, resistance training can be accomplished even if the individual has physical impairments that make walking, running, or cycling difficult or impossible. In women, resistance training has the added benefit of inhibiting osteopenia and osteoporosis.

Although there are many advantages to using resistance training to help lower BP, one study found that this mode of exercise can lead to increases in heart rate and arterial stiffness,[24] effects that are, at the very least, disconcerting. My research group has been studying the effect of 4 weeks of moderate-intensity aerobic (30 minutes at 65% of VO_{2peak}) and resistance (3 sets at 65% of a 10-repetition maximum) exercise training 3 times per week on hemodynamics in men and women in their late 40s with pre- to stage-1 essential hypertension (**Table I**).[25] We found that both aerobic and anaerobic exercise training lowered resting SBP and DBP by 3 to 4 mm Hg (**Table II**). We also observed that aerobic exercise was associated with a 5-beats-per-minute decrease in resting heart rate, whereas resistance exercise was associated with a 2-beats-per-minute increase in resting heart rate.

Table I. Characteristics of participants with pre- to stage-1 essential hypertension in a study of arterial stiffness and resting hemodynamics following 4 weeks of moderate-intensity aerobic or resistance exercise training (n = 30). Data are presented as mean (SE). Reproduced with permission.[25]

Characteristic	Aerobic	Resistance
Age, y	49.8 (1.6)	47 (2)
Height, cm	171.2 (2.7)	172.5 (2.7)
Weight, kg	86 (5)	90 (5)
BMI, weight/height^2	29 (1.8)	30 (1.2)
Body fat, %	32.9 (2.3)	31.8 (1.7)

BMI = body mass index.

Table II. Effect of 4 weeks of moderate-intensity aerobic or resistance exercise training on blood pressure and heart rate in participants with pre- to stage-1 hypertension (n = 30). Data are presented as mean (SE). Reproduced with permission.[25]

	Aerobic		Resistance	
Variable	Pre	Post	Pre	Post
SBP, mm Hg	141.2 (3.4)	136.6 (3.4)*	136.4 (3.4)	132 (3.4)*
DBP, mm Hg	80 (1.47)	76.9 (1.63)*	78.2 (1.47)	74.1 (1.63)*
MAP, mm Hg	103.5 (1.8)	100.3 (2)*	100.8 (1.8)	96 (2)*
HR, bpm	72.5 (2.9)	67.5 (2.8)*†	67.3 (2.9)	69.3 (2.8)*†

Pre = pre-exercise; Post = postexercise; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate; bpm = beats per minute.
*Significant interaction between training modes ($P < 0.05$).
†Significant change between pre- and postexercise groups ($P \leq 0.05$).

To further examine the renal hemodynamic effects of aerobic and resistance exercise, we measured pulse wave velocity (PWV) and blood flow after 15 minutes of rest in a supine position before and after the 4-week aerobic and anaerobic exercise programs.[25] PWV is the classic evaluation of arterial distensibility and is considered a powerful predictor of cardiovascular risk in hypertensive patients. Blood flow is another valuable marker of cardiovascular risk, and low blood flow is strongly associated with an increased risk for cardiovascular events. We found a statistically significant interaction between modes for central ($P < 0.001$) and peripheral ($P = 0.013$) PWV. Resistance exercise led to 9% increases in central (carotid to femoral) and peripheral (femoral to dorsalis pedis) PWV, whereas in contrast, aerobic exercise led to 10% decreases in both central (**Figure 1**) and peripheral PWV (**Figure 2**). Although the subjects for this study were unmedicated pre- to stage-1 patients with essential hypertension, we have also observed a similar acute increase in central PWV with young normotensive subjects after moderate-intensity resistance training.[26] These data suggest that anaerobic exercise may increase arterial stiffness, whereas aerobic exercise may reduce it. Our findings also suggest that arterial remodeling may not be the mechanism by which the pulse wave transit time is decreased after resistance exercise, because distensibility decreased after acute resistance training and returned to preexercise levels in ~60 minutes.

We also analyzed baroreflex sensitivity with data obtained from the finger arterial pressure waveform using the linear spontaneous sequence technique, which involves determination of the time between 2 consecutive R waves of the electrocardiogram (R-R intervals) and beat-to-beat systolic arterial pressure measurements. Whereas baroreflex sensitivity was reduced by 10% after resistance exercise, aerobic exercise had the opposite effect and increased baroreflex sensitivity.[27]

Reactive hyperemia is a transient increase in organ blood flow that occurs after a period of tissue ischemia and is a measure of resistance artery endothelial function. We measured reactive hyperemia noninvasively with a mercury-in silastic strain gauge, which determines forearm blood flow after the release of a 5-minute blood flow occlusion from an upper-arm cuff.[25] We found

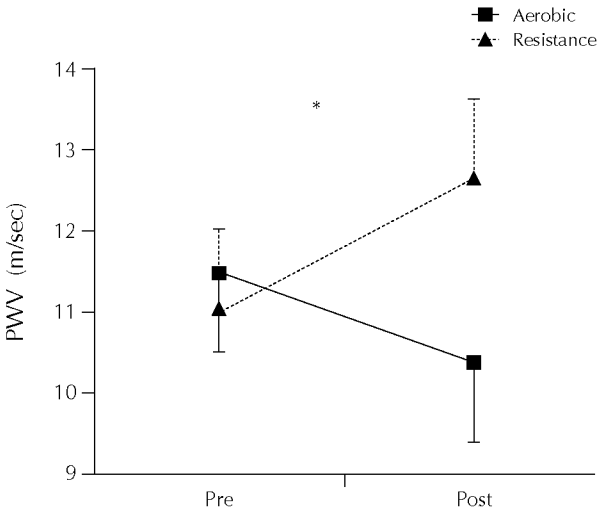

Figure 1. Mean (SE) central (carotid to femoral arteries) pulse wave velocity (PWV) measured pre- and postexercise in aerobic or resistance training groups (n = 30). *Significant interaction for central PWV ($P < 0.001$). Reproduced with permission.[25]

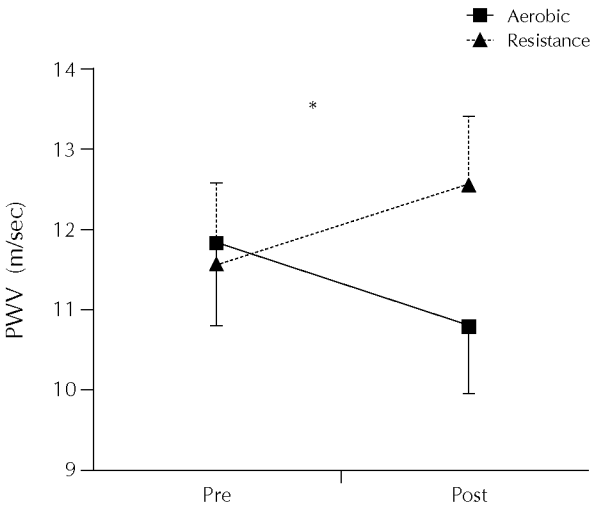

Figure 2. Mean (SE) peripheral (femoral to dorsalis pedis arteries) pulse wave velocity (PWV) measured pre- and postexercise in aerobic or resistance training groups (n = 30). *Significant interaction for peripheral PWV ($P = 0.013$). Reproduced with permission.[25]

that resistance training increased forearm blood flow in response to reactive hyperemia to a greater extent than did aerobic exercise, suggesting that resistance exercise may increase the vasodilatory capacity of arterial vessels (**Figure 3**).

Based on these findings, we concluded that the aerobic and anaerobic training–induced changes in arterial stiffness, as indicated by changes in PWV, were unrelated to changes in BP or vessel vasodilatory capacity. Furthermore, we concluded that body mass had no influence on the BP decreases observed in this study, because the mean body mass index was not affected by either training regimen and did not significantly differ between the 2 treatment groups.[28]

Resistance training increased forearm blood flow in response to reactive hyperemia to a greater extent than did aerobic exercise, suggesting that resistance exercise may increase the vasodilatory capacity of arterial vessels.

SEX DIFFERENCES IN THE EFFECTS OF AEROBIC AND ANAEROBIC EXERCISE ON HEMODYNAMICS

When our research group compared the effects of exercise training in hypertensive men and women, we found sex differences in resting hemodynamic changes and vascular distensibility.[29] We studied the effects of 4 weeks of aerobic and resistance exercise training in 21 men and 13 women. Whereas both aerobic and resistance training were associated with decreased resting SBP and DBP in both sexes, women exhibited greater decreases in resting DBP after resistance exercise training than did men. We also noted a sex difference in the PWV response to resistance training: whereas men exhibited a 17% decrease in central PWV, resistance exercise training had no effect on PWV in women.

To our knowledge, ours is the first study to directly compare the hemodynamic effects of aerobic and resistance exercise training in men and women in unmedicated prehypertensive individuals. These data suggest that although both men and women may benefit from the BP-lowering effects of resistance training, hypertensive women have an added advantage compared with hypertensive men, in that resistance exercise does not increase arterial stiffness in women to the same extent that has been shown in men. Thus, resistance exercise training may be a useful treatment regimen for prehypertensive women, not only because it lowers BP, but also because it inhibits the development of osteoporosis in women. Furthermore, resistance training may be of additional value in treating hypertensive women in light of a meta-analysis of the effects of antihypertensive drugs on BP, which showed that commonly used antihypertensive drugs are less beneficial in lowering BP in women than in men.[30]

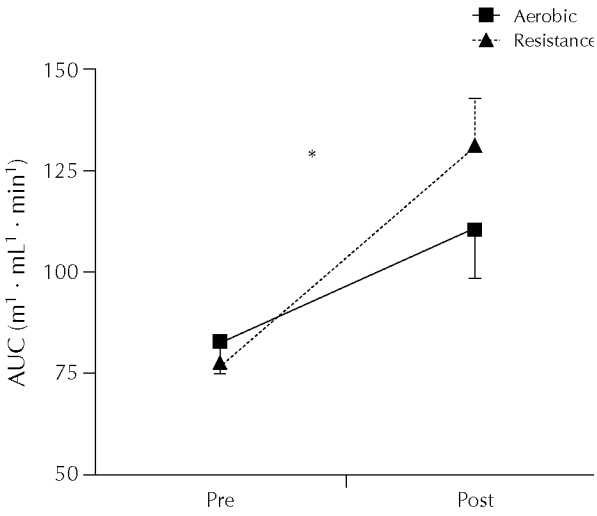

Figure 3. Reactive hyperemia blood flow measured pre- and postexercise in aerobic or resistance training groups (n = 30). *Significant interaction for blood flow (AUC) in response to arterial occlusion ($P < 0.05$). AUC = area under the curve. Reproduced with permission.[25]

FUTURE STUDIES

It will be important in future studies to determine

Although both men and women may benefit from its blood pressure–lowering effects, resistance exercise training does not increase arterial stiffness in women to the same extent as in men.

the mechanisms underlying male and female differences in the effects of aerobic and resistance exercise training on BP and arterial stiffness. In this regard, it will be worthwhile to investigate the role of the renin-angiotensin-aldosterone system (RAAS) in these sex differences, because the RAAS plays a critical role in the control of BP and the microcirculation. Angiotensin II, the key player of the RAAS, has been found to attenuate baroreflex control both of heart rate and sympathetic activity.[31] Neurohumoral systems modulate the RAAS, which in turn can regulate BP by modulating sympathetic tone.[32]

Leptin, produced by adipose tissue, is another molecule that may play an important role in sex differences in the effects of exercise on hemodynamics. This hormone exerts a myriad of effects in many tissues that could influence cardiovascular function, for example, by increasing platelet aggregation and thrombosis. Hyperleptinemia is an independent risk factor for coronary artery disease

When recommending antihypertensive treatment regimens, sex differences in response to exercise treatment programs must be considered.

and a strong predictor of myocardial infarction.[33] Moreover, studies suggest that leptin can directly elevate BP, because its overexpression has been found to augment sympathoexcitation.[33,34]

In conclusion, the effect of resistance exercise training on arterial stiffness and BP should be further investigated in both men and women so that sex differences in response to exercise treatment programs are fully taken into account when devising individual antihypertensive treatment regimens. Furthermore, research should focus on a translational approach to investigating how exercise attenuates the activity of vasoconstrictive agents and improves the microcirculation in hypertensive individuals. Animal modeling will reveal the specific mechanisms responsible for exercise modulation of vascular properties while clinical studies will elucidate how the cardiovascular system is modulated by exercise training.

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