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Editorial

Is Exercise Safe in Hypertension?

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Hypertension is a major risk factor for the development of stroke, heart failure, coronary heart disease, peripheral artery disease, and renal disease [1,2]. Currently, 67 million (~30%) Americans are diagnosed with hypertension and less than half of these individuals are receiving effective antihypertensive therapy [3]. When left untreated, hypertension often leads to these more detrimental diseases, thereby exponentially increasing the financial burden of hypertension treatment. Regular physical activity is often considered the cornerstone of preventing and managing hypertension; however, individuals with hypertension exhibit marked exercise intolerance [4] characterized by an exaggerated increase in blood pressure during physical activity (i. e. exercise pressor reflex) [5-7]. This excessive exercise pressor reflex and concomitant reduction in muscle blood flow, increase the risk of adverse cardiovascular events such as myocardial infarction, cardiac arrest, or stroke during or immediately after exercise [6,7] and is an established risk factor for cardiovascular morbidity and mortality [8].

The exercise pressor reflex originates at the skeletal muscle and involves group III (mechanoreceptors) and group IV (metaboreceptors) afferent fibers that participate in the modulation of sympathetic nervous system activity during exercise [9,10]. Muscular contraction results in mechanical distortion and metabolic stimuli which activate the group III and IV afferents in a graded fashion to increase heart rate (HR), blood pressure (BP), and vascular resistance [11,12]. We recently reported that in healthy individuals continuous feedback from the afferent fibers is obligatory for the appropriate hemodynamic and metabolic responses to both passive and dynamic exercise in humans [13,14]. In hypertension these receptors may be overactive owing to exaggerated increases in BP and HR which may facilitate symptoms contributing to exercise intolerance in these individuals [4]. In a rat model of hypertension the pressor response to muscle contraction was positively correlated to the baseline mean arterial pressure (MAP). Additionally, during activities of a relatively non-strenuous nature (i. e. low levels of tension development) the increase in MAP was enhanced in hypertension [15,16]. This increased sensitivity of the afferent fibers may be directly related to reduced skeletal muscle perfusion and impaired oxygen delivery during exercise with hypertension [17]. Impaired blood flow during exercise in hypertension would result in an accelerated accumulation of metabolic by products thus stimulating the group IV metabolically sensitive afferents.

Human hypertension research implicates the metaboreceptors as a major contributor to the augmented exercise pressor reflex [18], while animal models of hypertension also reveal a contribution of over sensitive mechanoreceptors to the exaggerated exercise pressor reflex [12,15,16,19]. Delineation of the respective contribution of the group III and group IV afferents is important in the development of therapeutic strategies aimed at attenuating or abolishing the excessive exercise pressor reflex in hypertension. Currently, the mechanisms responsible for the heightened sensitivity of afferent fibers are not known, however elevated free radicals and oxidative stress may contribute to this phenomenon. In fact, elevated free radical production and the accompanying increase in oxidative stress may be a common denominator in the etiology of this disease [20-22]. Oxidative stress contributes to reductions in vascular function with hypertension and aging and may play a role in the reduced blood flow present in this condition [17]. Additionally, free radicals have been reported to directly activate group IV muscle afferents at rest and during exercise [23], potentially contributing to the excessive exercise pressor reflex observed in this pathology. Specifically, animal studies have identified that free radicals, when applied directly to the group IV skeletal muscle afferents, provide a potent stimulus to the muscle metaboreceptors to increase firing frequency and raise blood pressure. Conversely, pre-treatment with superoxide dismutase (SOD) effectively abolished the stimulatory impact of the free radicals [23], providing convincing evidence for the direct involvement of oxidative stress in the exercise pressor reflex. Interestingly, the attenuated skeletal muscle blood flow during exercise associated with aging is, at least in part, ameliorated by the acute administration of an oral antioxidant cocktail (Vit C, E, and alpha lipoic acid), improving muscle perfusion and O_2 availability [24].

At this point we are left with the question, "is exercise safe in hypertension"? On one hand we know that exercise reduces blood pressure while simultaneously improving the function of multiple organ system. Unfortunately the exaggerated exercise pressor reflex is a real phenomenon that directly places a hypertensive patient at increased cardiovascular risk during or after exercise. Therefore, identifying the mechanisms associated with the augmented exercise pressor reflex and impaired blood flow response to exercise will be critical in the development of novel strategies aimed at improving exercise tolerance and reducing risks associated with exercise in hypertension. In order for exercise to be a viable and safe treatment for hypertension the causes and consequences of the exaggerated exercise pressor reflex must be clearly understood.

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