

Is Eccentric Exercise Is an Effective Way to Reduce Blood Pressure?

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Abstract: Physical activity is considered to be a non - pharmacological strategy for control of hypertension in the form of phenomenon which is called post exercise hypotension (PEH). It characterizes with reduction in resting blood pressure, sympathetic neural activity (SNA) and peripheral vascular resistance in the minutes and hours following an exercise bout. Literary based on population research the magnitude of the PEH can be translated into prevention and/or treatment of hypertension. It is well-documented that an unaccustomed eccentric exercise bout incorporates a high degree of eccentric muscle contractions commonly can lead to the muscle damage and delayed onset muscle soreness (DOMS). During eccentric contractions the muscle will lengthen, absorb mechanical energy with high force and small changes in oxygenation and hemodynamic values will be interpreted to represent lower muscle activation. Eccentric exercise elevates pain and inhibits short-term recovery of muscle function, it may result in a positive healing process to reduce high blood pressure.

Key words: Hypertension • Hypotension • Eccentric Exercise • Blood Pressure

INTRODUCTION

Sustained Post- Exercise Hypotension: Over a century investigations have exhibited that there is a sustained reduction in blood pressure after a single bout of exercise results in a post - exercise hypotension (PEH) [1-3]. The expression of PEH first has been documented by Hill [4] during the 90 min following a 400 yard dash whereby gained the importance as a medical entity in the potential antihypertensive benefits of exercise.

PEH since has been observed in response to several types of dynamic exercises, which use large muscle groups continuously, rhythmical and aerobic in nature [5] including walking [6, 7], jogging [8] running [9], leg cycling [10] and swimming [11]. Similarly, PEH has been elicited by resistant exercise modality [12, 13]. Electrical stimulation through the activation of muscle afferents in isometric contractions also has been shown to induce PEH in spontaneously hypertensive rats (SHRs), which is defined as post-stimulation hypotension (PSH) [14]. Additional confirmatory by Halliwill's group suggests post-exercise hypotension becomes more apparent especially after prolonged exercise that likely produce injury and inflammation in which might be important contributor to this phenomenon [15].

The decreased hemodynamic pattern following PEH is manifested by precipitous decreases in systolic (SBP) and/or diastolic blood pressure (DBP) to below pre-exercise resting values [16, 17] which has been recounted as indicators of decreased cardiac output (\dot{Q}) total peripheral resistance (TPR) or both [18].

These associate to changes in several physiological conditions such as a shift in the baroreflex control [19, 20] a reduction in the alpha-adrenergic responsiveness, an impaired vasoconstrictor responses and sympathetic withdrawal [21, 22] as well as to an increased secretion of humoral [23] and local vasodilator substances in response to exercise bout [24, 25]. Concomitantly these heterogeneous hypotensive responses mediate by a handful of central pathway [26]. For instance, gene expression of preproenkephalin has been shown to increase in the brainstem during post - exercise hypotension, after treadmill exercise in SHRs [27]. Central opioid system which activates by mechanosensitive afferent nerve fibers (Group III) from contracting skeletal muscle also contributes significantly to reductions in sympathetic outflow during PEH [28] and more studies by Boone *et al.* [29] have displayed opioid sensitive receptors appear to be involved in post - systolic blood

pressure reduction following a single bout of submaximal exercise in normotensive humans. Indeed, Yao *et al.* [30] have shown depletion of brain serotonin with parachlorophenylalanine attenuate the blood pressure lowering effect of sciatic stimulation in spontaneously hypertensive rats, suggesting the involvement of a central serotonergic system to cardiovascular depressor response. On the whole, these findings provided insights into the possibility that interactive mechanisms account for the blood pressure lowering effect in PEH.

The salutary effect of exercise on blood pressure reduction during post-exercise hypotension can persist for sometimes by several hours [31]. Relatively, the prolonged reductions followed by post-exercise hypotension to some extent depend upon the health status of the individual [32, 33]. Although, PEH has been confirmed in both normo- and hypertensive subjects [34-36], the depressor response to exercise is greater in magnitude and duration in hypertensive [37] perhaps because of the higher baseline blood pressure level whereby clinically and statistically is more significant [38].

Eccentric Exercise and Post Exercise Hypotension:

During eccentric exercise (e.g., downhill locomotion) the whole body oxygen consumptions ($\dot{V} O_2$) is lower than during level locomotion [39, 40]. Consequently, the reduced oxygen uptake of subjects performing negative work while resisting applied forces could, therefore, be attributed not just to a reduction in the number or frequency of excitation of active muscle fibres required to do the work, but also to a reduced average rate of oxygen uptake per fibre [41]. It also has been reported that when a muscle is stretched the energy requirement of the fibre, measured from both its rate of metabolic heat production and its rates of ATP and phosphocreatine break-down, falls substantially [42, 43]. These general systemic measurements indicate that eccentric actions are metabolically less demanding [44, 45]. Additionally, the effect of lower oxygen consumption as well as lower ATP per fibre during an eccentric exercise such as downhill exercise on muscle perfusion would result lower blood pressure due to compensatory vasodilatation mechanisms (e.g., adenosine) [46, 47]. In this respect, Halliwill [48] has proposed that the reduction of oxygen ($\downarrow O_2$) and ATP act as the potential vasodilators in post-exercise hypotension process to elevate blood flow.

Furthermore, other vascular control are mechanisms involved in the regulation of skeletal muscle blood flow that could be differentially affected by eccentrically biased exercise, such as sympathetic nerve activity

and the mechanical effects of the muscle pump [49]. For instance, Delp *et al.* [50] have demonstrated lower heart rate (HR) and higher blood flow to the spleen and stomach than muscle blood flow during downhill walking, an eccentrically based exercise rather than during concentrically biased exercise in rat, suggesting lower sympathetic outflow during eccentric contractions. These findings suggest that during a bout of dynamic exercise, when both muscle actions are performed with the same workload and for a similar period of time, eccentric actions probably would contribute to a lesser extent to the activation of the muscle metaboreflex and central command and thereby to the lesser increase in muscle sympathetic nerve activity (MSNA), than would concentric actions. It is possible, however, that the lower activation of the muscle metaboreflex and central command by eccentric actions may be compensated for by a greater activation of the mechanosensitive muscle afferents as a result of the stretching of the muscle [51]. Evidence also suggests that reductions in muscle sympathetic vasoconstrictor outflow and blunted vasoconstrictor responses to sympathoexcitation following dynamic large muscle-mass exercise contribute to the vasodilatation which increases the magnitude of PEH [52, 53].

On the other hand, researchers use an unaccustomed eccentric exercise as a model for acute inflammation with an assumption that blood flow increases right away as it would in acute injury [54, 55]. The release of myocellular enzymes into the blood as the result of sarcolemmal disruption can generally be perceived as an indication of muscle injury [56] which results in leakage of serum creatine kinase (CK), myoglobin (Mb) and lactate dehydrogenase (LDH) enzyme into bloodstream coming after eccentric exercise [57-59]. These biochemical markers, lead to the normal inflammatory response and increase chemical mediators such as histamine, bradykinin, prostaglandin and serotonin from damaged endothelial cells, causing pain and swelling. This is followed with an increase the capillary membrane permeability and change in blood vessel diameter [60, 61]. One may speculate hence, that there is a straight relation between the production of endothelial substances derived from exercise and the magnitude of post-exercise hypotension [62]. For instance, among these chemical substances, histamine is recently well-recognized for its potential vasodilator prospects during PEH in humans and evidence has shown the sustained post-exercise vasodilation and post-exercise hypotension after a single bout of dynamic large muscle-mass exercise is

predominately histamine H₁- and H₂-receptors mediated [63-65]. Thus, the increase in endothelial function is highly correlated with post-exercise changes in peripheral conductance and is therefore implicated as a mechanism of PEH [66].

Glossary: The important finding from this manuscripts indicates that eccentric exercise which is followed by muscle damage and inflammation after a single bout is likely evoke post – exercise hypotension more than other type of concentric or isometric exercise. However there is no consequence of eccentric exercise within the literature on blood pressure control. Therefore, new experimental researches are required as new approach to prevent and /or control high blood pressure in particular population such as hypertensive.

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