EDITORIAL

EXAGGERATED EXERCISE, BLOOD PRESSURE AND LOWER LEVEL OF INFLAMMATION

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An abnormal rise in systolic blood pressure (BP) during exercise is observed in a portion of individuals with normal BP at rest. Such response has been associated with an increased risk for future hypertension and cardiovascular events. [1-2] The factors that influence this exaggerated rise in BP during exercise are not established. However, impaired endothelial function in the setting of excessive elevations in exercise BP has been reported recently. [3]

Endothelium, the innermost layer of all blood vessels, is not simply an inert barrier that separates blood from tissue but is a dynamic organ that plays a vital role in vasomotion and, consequently, blood flow regulation, coagulation, fibrinolysis, and inflammation. Intact endothelium is essential for maintaining normal vascular tone and relaxation. This is accomplished by a delicate balance between endothelium - derived vasodilating, mainly nitric oxide, and vasoconstricting agents. [4] During exercise, the increased blood flow increases the mechanical stress in the vessels (shear stress). This provides the stimulus for the intact endothelium to accelerate the release of nitric oxide, resulting in vasodilatation and a reduction in peripheral resistance. However, under the same exercise conditions, but with endothelial integrity compromised, nitric oxide bioavailability in response to shear stress (exercise) is reduced, resulting in an impaired vasodilatation and increased peripheral resistance. [5] Therefore, it is reasonable to assume that the excessive BP elevation observed during exercise may be caused by the impaired vasodilatation resulting from endothelial dysfunction. An association between an exaggerated BP response during exercise and impaired endothelial function has been reported. [3] Acute systemic inflammation has also been reported to profoundly impair endothelium - dependent vasodilatation in humans. [6]

In Journal of Cardiopulmonary Rehabilitation, Sae Young Jay and colleagues, [7] proposed that an exaggerated BP response may be the result of impaired endothelial function that can be detected by increased levels of low-grade inflammation markers. For this, they assessed exercise BP, C-reactive protein (CRP), and white blood cell (WBC) in 43 individuals with normal resting BP but an abnormal BP response at peak exercise. Another 42 individuals with normal BP

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at rest and during exercise served as controls. The investigators reported that the WBC count in those who exhibited an exaggerated BP response during exercise was significantly higher (approximately 15%) than that of the group with normal BP response. CRP levels were similar between the 2 groups and only a trend toward a positive association between CRP levels and WBC count was observed. They concluded that these findings suggest that low-grade inflammation may be associated with an exaggerated BP response at peak exercise.

As mentioned by the authors, increased WBC count has been associated with the development of hypertension (and perhaps inflammation). However, the lack of difference in CRP levels between the 2 groups in this study is disappointing and detracts from this conclusion. Because CRP is a much more sensitive and specific indicator of systemic inflammation than is the WBC count, one may ask why CRP levels were not increased in the group exhibiting an exaggerated BP response if inflammatory processes were involved. In addition, it is important to point out that the WBC count was very much within the reference range ($6.1x10^9$/L Vs $5.2x10^9$/L) for those with an exaggerated and normal BP response, respectively.

It would also be of interest to know if blood was drawn before or after the exercise test. If blood was drawn before exercise, the association between WBC count and inflammation is strengthened. However, if blood was drawn after the completion of the exercise test, it is possible that the higher WBC count was an acute response to the stressor induced by higher exercise BP and not the result of inflammation. Unfortunately, these data are not available to address this question. Despite these limitations, this study helps to generate some interesting prospects. The prospect that an exaggerated BP response during exercise may be an indicator for low-grade inflammation and impaired endothelial function is certainly enticing and should be explored further. Indeed, the additional information provided by an exaggerated BP response during a graded exercise test can have important clinical applications. Because exercise testing is already widely used, a wealth of information on exercise BP response exists. The information can then be used to examine associations between exercise BP responses and inflammatory markers. For future patients undergoing exercise testing, such information can easily be obtained during exercise testing at no additional cost because additional equipment or procedures are not required. Exercise BP response can be used as a non-invasive and relatively inexpensive screening tool for those at risk for hypertension, left ventricular hypertrophy, or other cardiovascular events. Individuals with an exaggerated BP response can then be referred for further evaluation. Those with low-grade inflammation can be followed closely and treated for conditions or factors that foster the inflammatory process.

The therapeutic potential of exercise should also be considered. Current criteria recommend that antihypertensive therapy should be initiated for patients with confirmed hypertension, defined as systolic BP ≥140 mm Hg or diastolic BP ≥90 mm Hg. Therefore, normotensive individuals who exhibit an exaggerated BP response during exercise are not likely to be treated with antihypertensive medication.

One study suggests that the exaggerated BP response during an acute bout of exercise can be attenuated by regularly performed, moderate-intensity exercises. They noted that moderate- and high-fit normotensive individuals exhibit lower BP at submaximal and maximum exercise levels, as well as 24-hour ambulatory BP compared with low-fit

individuals. Furthermore, low-fit individuals had higher left ventricular mass and greater likelihood of left ventricular hypertrophy than did their fit counterparts. [8] There was also a strong association between exercise BP and 24-hour BP, both strong predictors of left ventricular hypertrophy. In patients with severe hypertension, they reported that 16 weeks of low to moderate aerobic exercise resulted in significantly lower exercise BP at maximal and absolute submaximal exercise workloads. [9]

Collectively, these data suggest that exercise BP reflects the BP during routine daily activities. Regularly performed, moderate - intensity exercise is likely to attenuate an abnormal rise in daily BP and protect against associated health consequences. Is this accomplished by the restoration of endothelial function that is compromised by low-grade inflammation? We do not know yet. However, accumulating evidence suggests that increased physical activity is associated with significantly lower levels of inflammatory markers. [10]

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