Hypertension: The acute and chronic response to exercise

Monty C. Morales, MD, Neil L. Coplan, MD, Paul Zabetakis, MD, and Gilbert W. Gleim, PhD. *New York, N.Y.*

Hypertension is a significant risk factor for the development of cardiovascular disease,[1, 2] and lowering blood pressure in hypertensive individuals has been shown to reduce the likelihood of their developing secondary end-organ damage.[2] There has been increasing interest in the use of exercise as a nonpharmacologic method for treating hypertension. In addition, there is evidence that the exercise test may play a role in identifying normotensive patients at increased risk for developing hypertension over a long-term follow-up period.[3-5] This report reviews the acute and long-term effects of exercise on blood pressure, and examines the use of exercise as a diagnostic and therapeutic tool for patients with hypertension.

Acute blood pressure response to exercise. The acute blood pressure response to exercise depends on the type of exercise performed. It is important to distinguish between the blood pressure response during dynamic exercise training and the blood pressure response to isometric training. The initial response in endurance training is a linear increase in systolic blood pressure with increasing work intensity, which occurs secondary to increased cardiac output.[6] Concurrent with the increase in cardiac output there is a dilatation of the blood vessels in the exercising muscle, which causes a decrease in resistance to blood flow. This results in systolic blood pressure rising less than would be expected from the increase in cardiac output alone. The drop in resistance has an even more pronounced effect on diastolic blood pressure—diastolic pressure usually remains constant during aerobic exercise, and may even go down. Patients with hypertension have a less significant fall in peripheral vascular resistance during exercise, and cardiac function is decreased when compared with that in patients with normal blood pressure.[7]

During isometric exercise there is a dramatic increase in systolic blood pressure, along with an increase in diastolic blood pressure.[6] In one study[8] of five weight lifters (who had intraarterial lines to measure blood pressure during single arm curls, overhead presses, double and single leg presses), both systolic and diastolic blood pressure had extreme elevations during the 2 to 3 seconds required to move the weight, and blood pressure decreased to preexercise levels as the weight was lowered. Pressures reached progressively higher levels with each subsequent repetition, so that the highest recording occurred during the last completed attempt prior to failure. Increases in blood pressure were related to the size of the muscle mass involved in the contraction, with the highest absolute value from the double leg press measured at 480/350 mm Hg. Mean values for the exercises varied from 230/170 to 330/250 mm Hg.

The immediate rise in blood pressure during isometric exercise is the result of increased cardiac output secondary to increased heart rate, mechanical compression of blood vessels in exercising muscle, and to a lesser extent a reflex vasoconstriction in the vascular beds of the muscles not involved in exercise.[9] The rapid drop in blood pressure immediately after exercise is thought to be secondary to reactive hyperemia in the exercised muscle, as well as a transient pressure undershoot initiated by baroreceptor and cardiopulomonary reflexes.[8] These responses to the extreme elevation in blood pressure in the last repetition may contribute to dizziness or faintness occurring immediately after heavy lifting.

Blood pressure response during exercise testing. An exaggerated blood pressure response to exercise in normotensive patients may be a marker for the development of resting hypertension[3-5] and for left

From the Nicholas Institute of Sports Medicine and the Sections of Cardiology and Nephrology, Department of Medicine, Lenox Hill Hospital.

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Reprint requests: Neil L. Coplan, MD, NISMAT/Lenox Hill Hospital, 130 E. 77th St., New York, NY 10021.

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ventricular hypertrophy.[10] Wilson and Meyer[3] found that normotensive individuals with an elevated exercise blood pressure response (>225 mm Hg) followed for an average of 32 months had over twice the risk for the development of hypertension as subjects without this response. Dlin et al.[5] found that 10.6% of subjects with an exaggerated blood pressure response during exercise testing (defined as a systolic pressure >200 mm Hg or a rise of diastolic pressure of at least 10 mm Hg if this value exceeded 90 mm Hg) developed hypertension; none of the subjects who did not show an exaggerated exercise blood pressure response developed hypertension over the mean 5-year follow-up period.

These findings indicate that even in the absence of baseline hypertension, the blood pressure response during exercise testing may suggest an increased likelihood for developing hypertension in the future. However, the degree of exercise-related elevation in blood pressure that should be considered "exaggerated," and the predictive value of this finding remains to be determined.

The blood pressure response to exercise may also be a marker for left ventricular hypertrophy. Using echocardiography, Gottdiener et al.[10] showed that 14 of 22 normotensive subjects with exercise systolic blood pressure over 210 mm Hg had left ventricular hypertrophy; only 1 of 17 subjects with a lower exercise blood pressure had echocardiographic determined left ventricular hypertrophy. The left ventricular mass index was linearly correlated with maximum exercise blood pressure ($r = 0.65$, $p < 0.001$). There was no correlation between maximal oxygen consumption and left ventricular hypertrophy, suggesting that the relationship of left ventricular hypertrophy and increased maximal exercise blood pressure was unlikely to be due to a training effect. Whether the increased left ventricular mass in patients with normal blood pressure at rest who have an enhanced blood pressure response to exercise reflects the effect of intermittent elevated blood pressure integrated over long periods of time remains to be determined.

Long-term effect of exercise on hypertension and risk for cardiovascular disease. Several studies have shown that regular exercise is associated with a substantially lower over-all risk for sudden death, cardiovascular mortality, and all-cause mortality.[11-15] Siscovick et al.[13] demonstrated that exercise is associated with varying degrees of both short-term risk and long-term benefit, with the magnitude of risk decreasing and the benefit increasing as a function of performing increasing degrees of habitual exercise. In a study of 16,936 Harvard alumni, Paffenbarger et al.[16] showed that those individuals associated with a higher level of activity (>2000 kcal/week) had a lower risk of heart attack than more sedentary subjects. Paffenbarger and Hyde[11] found a proportionately greater drop in risk for all-cause mortality as physical activity increased among hypertensive alumni.

The causes for a decline in mortality in subjects who exercise more frequently is probably multifactorial; the effect on blood pressure may be one important factor. Epidemiologic studies suggest that increased activity is associated with lower blood pressure.[17] Paffenbarger and Wing[18] showed that the incidence of hypertension is lower in those who exercise compared with that in those who are more sedentary. Blair et al.[19] followed normotensive men and women from 1 to 12 years, and found that persons with low levels of physical fitness had an increased risk for the development of hypertension compared with highly fit people.

Aerobic exercise has also been shown to have a beneficial effect on lowering resting blood pressure in patients with established hypertension,[20-27] an effect that may contribute to reduction in cardiovascular morbidity and mortality. Harris et al.[26] found that after a 9-week circuit weight training period in borderline hypertensive subjects (140/90 to 160/95 mm Hg) there was no increase in resting or exercise blood pressure and a small decrease in diastolic blood pressure. Cade et al.[23] examined the effect of aerobic conditioning on 105 patient with established diastolic hypertension (<90 mm Hg). In 58 patients who were not taking medications in the preexercise period the mean blood pressure decreased 15 mm Hg; of the 47 patients receiving drug therapy, there was a mean decrease in blood pressure of 19 mm Hg, and 24 patients were able to discontinue all medications. Duncan et al.[24] evaluated the relationship between exercise, blood pressure, and plasma catecholamines; they further divided the exercise group into hyperadrenergic and normoadrenergic subgroups. After a 16-week aerobic exercise program, there was a reduction in systolic blood pressure of 6.3 mm Hg, 10.3 mm Hg, and 15.5 mm Hg for the control, normoadrenergic, and hyperadrenergic groups, respectively. Within the hyperadrenergic group the change in blood pressure was associated with a decrease in plasma catecholamine values.

Conclusions. Exercise has both a significant diagnostic and therapeutic role for hypertension. Patients with an exaggerated blood pressure response to exercise are at increased risk for the development of hypertension and may already have left ventricular hypertrophy; the therapeutic implications of this finding remain to be determined. Further work is

needed to determine the best method for integrating exercise into the diagnostic work-up of hypertension, and how to most effectively utilize exercise as a therapy for patients with an elevated blood pressure.

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