Aortic Root Size in Elite Athletes
When No Change Matters

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It is known that sport-specific remodeling of the heart may occur with exercise. Although some overlap exists, exercise activity can be segregated into 2 forms: isotonic exercise (endurance training) and isometric exercise (strength training), with defining hemodynamic differences. Increased cardiac output leads to a volume challenge that occurs with isotonic exercise that affects all 4 heart chambers and may cause an increase in left ventricular (LV) mass, biventricular hypertrophy and reduction in LV diastolic function. Conversely, isometric exercise (strength training) will increase the peripheral vascular resistance and cause a pressure load on the heart. This results in concentric LV hypertrophy and reduction in LV diastolic function. Although an athlete may have expertise in one particular sport, usually elite athletes train with combined isotonic and isometric components (weight training, plyometrics, speed drills, running, etc.) and may not fit into one specific category of training.

There is limited data on the effect of exercise training on the aorta and whether aortic dilation occurs because of the hemodynamic effects of chronic exercise training. One could hypothesize that with isotonic exercise, there is a transient increase in peripheral vascular resistance and systolic hypertension that leads to increased aortic wall tension and chronically may result in aortic dilation. A meta-analysis performed by Iskandar and Thompson showed minor enlargement associated with training in elite athletes at the level of the aortic valve annulus and the sinus of Valsalva compared with controls. The degree of enlargement at the aortic valve annulus was less than at the level of the sinuses suggesting that the fibrous skeleton of the heart may have a protective effect. Another study of 100 elite, strength-trained athletes showed that all levels of the ascending aorta were greater in size when compared with healthy, age- and height-matched controls. They also found that the duration of the high-intensity training to be the strongest predictor of aortic root enlargement. Similarly, in a study of 615 patients, D’Andrea et al found the body surface area (BSA), exercise type and duration, and LV circumferential end-systolic stress were independent predictors of the aortic root diameter at all levels. Although the aorta may be enlarged compared with controls, it is unclear how and if this minor aortic enlargement will affect the athlete clinically.

In this issue of Circulation: Cardiovascular Imaging, Boraita et al evaluated 3281 elite athletes and found that only 1.8% of the athletes had dilated aortas (>40 mm in men or 34 mm in women) and also found that age, LV mass, and BSA were the main predictors of aortic dimensions. These results are similar to the percent of athletes (elite and nonelite) who were found to have aortic dilatation in the study by Pellicca et al using similar cut-off values for aortic dilatation. These findings suggest that ascending aortic dilation in elite athletes is rare and if found on echocardiogram, work up of underlying aortopathy should be considered.

The inclusion of male and female athletes is important and allowed the authors to examine the effect of sex on cardiac dimensions in their populations. Similar cardiovascular adaptations to exercise training occur in men and women; however, unique adaptations to exercise in women have been described. Both men and women demonstrate an increase in heart chamber sizes and muscle mass, and when adjusted for BSA, the findings are not as substantial in women when compared with men. In this study, 1242 female athletes were included, and after BSA adjustment, LV end-diastolic diameter and atrial size remained larger in women compared with men. Similar findings were seen, albeit on a smaller scale of female patients, in a longitudinal study published by Baggish et al. Sex differences were also seen in the aortic diameters that were adjusted for BSA. Larger aortic diameters were found at all levels of the aorta in men except at the sinotubular junction and the ascending aorta. These sex differences are intriguing and potentially implicate a hormonal effect on vascular remodeling in females. Further studies evaluating the hormonal effects on the aorta in elite female athletes would be an interesting future investigational study.

A few limitations should be considered in the study by Boraita et al including that it is a cross-sectional study that comes with its inherent limitations, including the inability to exclude the possibility that the findings are from innate changes that may have occurred without training. A longitudinal study design that assesses athletes before and after training would be more informative, however challenging with such a large cohort of patients. Given that echocardiographic imaging is mandatory in many European countries, a study examining serial changes in cardiac dimensions during the course of years in athletes would be truly original and helpful.
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Disclosures

None.

References


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