Athletic Left Atrial Dilation
Size Matters?*

Aaron L. Baggish, MD

The cardiovascular system’s ability to generate high cardiac output is essential for athletic performance, and its failure underlies many forms of disease. During exercise, the heart and vessels circulate energy-rich substrate to exercising muscle and simultaneously remove the metabolic byproducts of muscular metabolism. This process occurs in an elegant and coordinated fashion whereby cardiac output is tightly linked to external work such that the energy demand-supply balance is maintained across the entire intensity spectrum of physical activity. During exercise, the cardiovascular system experiences changes in intravascular and intraventricular pressure and volume. In simplistic terms, endurance activities including running, cycling, and swimming are characterized by the generation and maintenance of high cardiac output and thereby represent a cardiovascular volume challenge. Strength activities, in contrast, are associated with repetitive surges in systemic blood pressure that translate into a cardiovascular pressure challenge. Repetitive exposure to these cardinal forms of hemodynamic stress stimulates the process of exercise-induced cardiac remodeling, which results in the myriad of structural and functional adaptations that often are broadly referred to as the “athlete’s heart” (1).

Forty years ago, a sentinel paper by Morganroth et al. (2) provided preliminary data suggesting that the heart, specifically the left ventricle (LV), remodels in a sport-dependent fashion. Despite some debate in the literature, the majority of subsequent cross-sectional studies (3) and more recent longitudinal work (4) confirm this notion. Specifically, endurance training promotes dilated LV geometry with or without mild hypertrophy, whereas strength training is associated with LV remodeling characterized by mild wall thickening in the absence of chamber dilation. Sport-specific right ventricular adaptations have similarly been documented (5). To date, the literature on the athlete’s heart has been dominated by studies that focused on ventricular characteristics, with atrial structure and function receiving comparatively little attention. Atrial metrics among athletes, with a few notable exceptions (6,7), have largely been reported as supporting data in papers focused on the ventricles. As such, comparatively little is known about atrial adaptations to exercise training.

In this issue of JACC, Iskandar et al. (8) present the results of a meta-analysis examining left atrial (LA) size in athletes. Using a systematic literature search and a priori retention criteria chosen to ensure capture of the intended study population (i.e., youthful elite athletes) and the desired dependent variables (i.e., LA diameter or volume), the authors amassed a robust study population consisting of 7,018 athletes and 1,044 control subjects. Regression analyses using both fixed and random effect variables were used to compare LA size between athletes and control subjects and to examine the relationships between LA dimensions, sex, and exercise training type. Results from this important effort are summarized as follows. First, athletes were found to have a pooled mean LA diameter that was 4.1 mm (95% confidence interval: 2.8 to 5.4 mm; p < 0.0001) greater and a pooled mean LA volume index that was 7.0 ml/m² (95% confidence interval: 2.3 to 11.6 ml/m²; p < 0.01) greater than sedentary control subjects. Second, LA dilation, relative to control subjects, tracked closely with physiological sporting discipline, with endurance athletes demonstrating the largest difference (4.6 mm; p < 0.0001), strength athletes the smallest difference (2.9 mm; p < 0.03), and mixed trained athletes falling in between (4.2 mm; p < 0.02). Finally, a comparison of male and female athletes found that LA diameter was 2.3 mm greater in men but that sex-based differences were eliminated when body surface area-indexed LA volumes were compared.

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Meta-analyses are most useful when single studies lack adequate statistical power to confirm or refute an observation or when aggregation of data permits meaningful subgroup analyses. The present report accomplishes both of these objectives. With a few notable exceptions, the majority of studies examining LA morphology in athletes are limited by factors including small sample size, lack of adequate control population, and focus on a single sex. Results from the present study confirm, in convincing fashion, that average LA size is larger in elite athletes than in sedentary people. This finding has direct clinical relevance, because LA dilation is a common feature of several cardiomyopathic conditions that may affect young athletes, including hypertrophic and hypertensive cardiomyopathy. Clinicians charged with the daunting task of differentiating adaptive remodeling from occult myopathy will benefit from an understanding that the presence of mild LA dilation is of minimal value in this diagnostic dilemma. In addition, the present paper demonstrates that LA size should be added to the list of exercise-induced cardiac adaptations, a list that currently includes LV chamber size, LV wall thickness, and right ventricular chamber size, that adhere to sport-specific remodeling patterns. This important insight represents a key step in our quest to understand how different forms of physical activity lead to changes in cardiac structure and function.

Having acknowledged the clinical and scientific advances afforded by the present study, we turn to several key questions it raises and leaves unanswered. Ventricular components of the athlete’s heart are typically regarded as adaptions that by definition confer a physiological advantage. For example, the LV dilation or eccentric LV hypertrophy that develops in experienced endurance athletes maximizes stroke volume reserve and thus cardiac output augmentation during exercise. Similarly, the concentric LV hypertrophy that is common among strength-trained athletes may, according to Laplace’s law, minimize transmural wall stress during brief but intense periods of LV pressure overload. Does LA dilation play a key role in exercise physiology as a determinant of exercise capacity or as a mechanism designed to offset the hemodynamic challenges of sport? Or is athletic LA dilation a physiologically inert byproduct of repetitive pressure and/or volume challenge that serves simply as a marker of prior exercise exposure? Further study coupling cardiac imaging with exercise physiology will be required to address this intriguing question.

The present study confirms in definitive fashion that mild to moderate LA dilation, measured echocardiographically as either major dimension or indexed volume, should be considered the norm, not the exception, among trained athletes. Does this mean that parameters of LA size hold no value for differentiating the athlete’s heart from cardiomyopathy? Returning to lessons learned from the athletic LV, it becomes clear that the data presently available, including those presented by Iskandar et al. (8), are insufficient to address this question. The key to resolving this issue will be to determine whether physiological LA dilation demonstrates a measurable and consistent upper limit of normal. If so, absolute or indexed LA dimensions may be as clinically useful as LV wall thickness, which has been shown to adhere to sex- and ethnicity-specific physiological limitations (9), for differentiating health from disease. If not, LA dimensions, much like LV chamber volumes, which often dilate markedly and without a clear normality limit because of exercise training (10), may hold no discriminatory value. Again, further work is needed to assign LA dimension to the correct physiological camp.

Finally, and perhaps most intriguingly, is the issue of clinical relevance with respect to atrial arrhythmia. As succinctly outlined in the present paper, a growing body of literature convincingly demonstrates an association between long-term athletic participation, specifically endurance sport training (11), and increased risk of atrial fibrillation (12). Although routine physical exercise may favorably impact many key determinants of cardiovascular disease (e.g., lipid profiles, blood pressure, and body mass), atrial fibrillation is the exception and is increasingly recognized as the Achilles heel of the aging master’s athlete (a group defined as competitors >40 years of age). Numerous underlying mechanisms, including those shared with the general, more sedentary population (e.g., undiagnosed hypertension, excessive alcohol consumption, sleep apnea), and some factors more specific to the aging competitive athlete, including resting vagotonia, chronic inflammation, and indeed LA dilation, have been proposed. Although it is tempting to assume that LA dilation, a risk factor for atrial fibrillation in the general population (13), carries similar prognostic implications among athletes, we should avoid this temptation until confirmatory studies are completed. In the interim, although it is safe to say that athletes have larger LAs than their sedentary counterparts, it remains uncertain whether or not size really matters.

REPRINT REQUESTS AND CORRESPONDENCE: Dr. Aaron L. Baggish, Cardiovascular Performance Program, Massachusetts General Hospital, Yawkey Suite 5B, 55 Fruit Street, Boston, Massachusetts 02114. E-mail: abaggish@partners.org.
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