groups of muscles with marked increases in heart rates and systolic blood pressure up to 200 mm Hg during exercise (2). Thus, changes observed in this recent study may not only be due to the increased preload but may be a consequent to the combined volume and pressure overload. I find it hard to agree with the investigators’ claim that as their athletes showed normal LV diastolic filling “it is reasonable to conclude that their LA enlargement represents uniquely the physiologic consequence of chronic exercise training” as opposed to LA enlargement seen in certain pathologic conditions associated with impaired LV compliance or increased afterload (1). We have demonstrated that while athletes may show normal resting LV filling patterns, some, such as weightlifters, may reveal an abnormal response to isometric stress with reduction in E and marked increase in peak A velocity, resembling that seen in the hypertensive group (3). However, in runners the filling pattern remained normal during isometric stress as well (3,4).

Hence, isometric and isotonic training affect LV filling differently as revealed during isometric stress testing, although resting patterns may be normal. Therefore, one cannot claim that LA enlargement is due solely to changes in preload, as concluded in the present study, relying only on resting LV filling patterns. Moreover, the involvement of afterload changes during exercise may have determined which of their athletes showed the greatest impact on the LA size depending on the type of exercise they performed.

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Physiological Upper Limits of Left Atrial Diameter in Highly Trained Adolescent Athletes

We have read the study by Pelliccia et al. (1) in which the investigators characterized the prevalence and upper limits of left atrial (LA) enlargement in highly trained adult athletes. The data are of great interest and provide yet another parameter for aiding the differentiation of physiological adaptation (athlete’s heart) from hypertrophic or dilated cardiomyopathy, which are recognized causes of sudden death in athletes. Currently, there are few data on structural cardiac changes in adolescent athletes in whom deaths from cardiomyopathy are more prevalent than in adults (2). Data derived from adult athletes cannot be extrapolated to younger athletes for the purposes of differentiating athlete’s heart from cardiomyopathy. This is because adolescent athletes are physically less mature and have trained for a shorter duration than have adult athletes.

The death of some high-profile athletes has put pressure on sporting organizations in the United Kingdom to implement screening of apprentice athletes (usually aged 14 to 18 years) before recruitment for competition (3). Our establishment performs cardiovascular evaluation in junior athletes (aged 14 to 18 years) from the British Lawn Tennis Association, premier soccer and rugby league junior squads, and the national cycling, swimming, boxing, and triathlon squads. Between 2002 and 2005 we evaluated 1,000 highly trained adolescent athletes (75% male; mean body surface area 1.75 ± 0.16 m²) using echocardiography. Cardiac measurements and Doppler indices were measured by conventional methods.

Adolescent athletes had a mean LA diameter of 31 ± 4.8 mm. Based on upper limits derived from an age-, gender-, and size-matched sedentary control group, 120 (12%) athletes had a greater than predicted LA diameter. Although, 368 athletes had left ventricular wall thickness (4) or cavity dimension (5) measurements exceeding predicted upper limits and consistent with that seen in patients with morphologically mild hypertrophic or dilated cardiomyopathy, respectively, none of the athletes had a left atrial diameter >45 mm. We considered all athletes with enlarged cardiac dimensions to have physiological cardiac enlargement based on the absence of symptoms or family history of cardiomyopathy and the presence of normal indices of diastolic and systolic function.

Our observations suggest that in highly trained adolescent athletes, LA diameter is modestly increased; however, a diameter >45 mm is exceedingly rare. We conclude that, during assessment of adolescent athletes, LA diameter measurement >45 mm should be applied as a cut-off point in differentiating physiological enlargement from cardiomyopathy in an adolescent athlete with a wall thickness or cavity size exceeding upper limit. This figure is considerably less (5 mm; 11%) than that recommended for adult athletes from the study by Pelliccia et al. (1).

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REPLY

We thank Dr. Basavarajaiah and colleagues for their comments regarding our study (1) recently published in JACC. We also appreciate their prior contributions to the left atrial (LA) remodeling in adolescent trained athletes (2,3) by establishing an appropriate threshold value, which is different from what we found in adult athletes.

The investigation by Dr. Basavarajaiah and colleagues have shown that atrial remodeling associated with athletic conditioning differs in young and adolescent athletes because of their incomplete body maturation and less strenuous conditioning programs. From this input, we know that transverse LA dimension rarely exceeds 45 mm in younger athletes (2) but may be >45 mm (and up to 50 mm) in adult trained athletes (1). These cut-off values can be used in clinical practice to differentiate the physiologic atrial enlargement associated with intensive athletic conditioning from the pathologic atrial remodeling associated with cardiomyopathies.

We also thank Dr. Spodick for his insights and wisdom regarding proper interpretation of the scalar electrocardiogram (ECG) in relation to LA enlargement. Dr. Spodick underscores the opportunity in our study to assess the presence of interatrial block (identified as P-wave duration ≥120 ms), which may be associated with LA enlargement and represents a marker of atrial dysfunction and risk for atrial arrhythmias (such as atrial fibrillation) (4,5). We do not dispute this suggestion, but we wish to emphasize that the primary focus of our study in highly trained athletes was the assessment of LA dimensional changes by echocardiography and the relation to clinical profile and course (including development of atrial fibrillation). Our data show that LA enlargement is not responsible, per se, for proclivity to atrial arrhythmias, including atrial fibrillation in trained individuals (6).

Dr. Spodick is also correct in suggesting that measurement of LA volume would be more sensitive than that of transverse LA dimension, which we have reported in the present study (and also in previous investigations) (1,7). However, our study design intentionally incorporated this quantitative measure of transverse LA size, which is most commonly used in the clinical practice, and conventionally employed in large epidemiologic studies (6,8) so as to be consistent with customary echocardiographic laboratory interpretation and the published literature in this area.

Finally, Dr. Abinader’s comments regarding the mechanisms of LA enlargement in trained athletes are of interest. We agree with Dr. Abinader that rowing/canoeing and cycling involve both dynamic and static exercise of large muscle groups, which are responsible for an increase in both preload and afterload during prolonged training sessions, with the consequence of combined volume and pressure overload (9). Therefore, the atrial remodeling we have reported in the present study (1) and in previous investigations (7) are likely to be the consequence of this combined hemodynamic overload, as also suggested by the close relation we have demonstrated between LA and left ventricular dimensional changes (1).

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