female population, this group was excluded from this analysis. The 37-month matrix for the distribution of events and the presence of coronary calcium for asymptomatic men is detailed in Table 1.

Finally, assuming a sensitivity of 30%, a specificity of 98%, and a 10-year event risk of 10%, the positive predictive value of a non-0 calcium score would be 13.5%, and the negative predictive value of a 0 calcium score would be 99%, numbers in line with published data.

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REPLY

In reply to Dr. Boyar’s letter, using data from Figure 2 in our study (1) and rounding off, we calculated a sensitivity of 0.94 and a specificity of 0.33 for all calcium scores ≥ 0.1. The values in our Figure 2, 0.91 and 0.39, respectively, differ slightly because the computer program that generated Figure 2 grouped all square roots less than the whole number with the previous whole number. Thus, calcium scores of 1, 2, and 3, with square roots of 1.0, 1.4, and 1.7, respectively, were all plotted as corresponding to a value of 1.0 on the abscissa. Calcium scores of 4 to 8, with square roots of 2.0 to 2.8, were lumped together as 2, and so on. We apologize for any confusion created by Figure 2 in our report (1).

We are not aware of any standard that a threshold means > the threshold value rather than ≥ the threshold value. In the case of calcium scores of 0, we chose ≥ 0 because this makes a useful reductio ad absurdum point about the test, and because we believed the rest of the graph makes better sense if the threshold values were included.

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REFERENCE


Left Atrial Remodeling in Competitive Athletes

Pelliccia et al. (1), as so frequently in the past, have added to our knowledge of “athlete’s heart.” One can only agree with their conclusions, but I believe their quantitative results might have been significantly different had they not restricted the search for prolonged P-wave duration to leads I, II, and V1. Except for V1, use of the limb leads only puts us in the anachronistic standards of the 1920s when there were only three leads (2). (Curiously, some current textbooks still rely on lead II.) We have shown several times that one needs to evaluate all 12 leads of the standard electrocardiogram (ECG) to get true P-wave durations. Indeed, if we had relied on lead II only, we would have recognized only just over one-half of the prolonged P waves despite utilizing calibrated magnifying graticules (2,3). Indeed, leads V3 and V4 gave substantially more prolonged P waves than did lead II. Another quantitative effect of the protocol may have occurred because the investigators used M-mode echocardiography when it is quite clear that, when assessing the left atrium volumetrically, two-dimensional echocardiography would have significantly been more sensitive (4).

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Competitive Athletes and Left Atrial Remodeling

In a recent issue of the Journal, Pelliccia et al. (1) assessed the prevalence and clinical significance of left atrial (LA) enlargement in competitive athletes. Enlarged LA size was common and present in 20% of examined athletes, and we agree with the investigators that the possible determinants of these changes remain incompletely resolved. They found that LA enlargement occurred in association with left ventricular (LV) enlargement and were largely dependent on the type of sport practiced, with cycling, rowing, and canoeing showing maximal impact. In their opinion these changes are due to the increased preload as they revealed normal resting LV diastolic filling and systolic function.

Rowing and cycling represent typical strength and endurance sports involving combined dynamic and static exercise of large
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 consequence 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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