Prevalence and Clinical Significance of Left Atrial Remodeling in Competitive Athletes

Antonio Pelliccia, MD,* Barry J. Maron, MD,† Fernando M. Di Paolo, MD,* Alessandro Biffi, MD,*
Filippo M. Quattrini, MD,* Cataldo Pisicchio, MD,* Alessandra Roselli, MD,* Stefano Caselli, MD,*
Franco Culasso, PhD‡

Rome, Italy; and Minneapolis, Minnesota

OBJECTIVES	In the present study we assessed the distribution and clinical significance of left atrial (LA) size in the context of athlete's heart and the differential diagnosis from structural heart disease, as well as the proclivity to supraventricular arrhythmias.
BACKGROUND	The prevalence, clinical significance, and long-term arrhythmic consequences of LA enlargement in competitive athletes are unresolved.
METHODS	We assessed LA dimension and the prevalence of supraventricular tachyarrhythmias in 1,777 competitive athletes (71% of whom were males), free of structural cardiovascular disease, that were participating in 38 different sports.
RESULTS	The LA dimension was 23 to 50 mm (mean, 37 ± 4 mm) in men and 20 to 46 mm (mean, 32 ± 4 mm) in women and was enlarged (i.e., transverse dimension ≥40 mm) in 347 athletes (20%), including 38 (2%) with marked dilation (≥45 mm). Of the 1,777 athletes, only 14 (0.8%) had documented, symptomatic episodes of either paroxysmal atrial fibrillation (n = 5; 0.3%) or supraventricular tachycardia (n = 9; 0.5%), which together occurred in a similar proportion in athletes with (0.9%) or without (0.8%; p = NS) LA enlargement. Multivariate regression analysis showed LA enlargement in athletes was largely explained by left ventricular cavity enlargement ($R^2 = 0.53$) and participation in dynamic sports (such as cycling, rowing/canoeing) but minimally by body size.
CONCLUSIONS	In a large population of highly trained athletes, enlarged LA dimension ≥40 mm was relatively common (20%), with the upper limits of 45 mm in women and 50 mm in men distinguishing physiologic cardiac remodeling ("athlete's heart") from pathologic cardiac conditions. Atrial fibrillation and other supraventricular tachyarrhythmias proved to be uncommon (prevalence <1%) and similar to that in the general population, despite the frequency of LA enlargement. Left atrial remodeling in competitive athletes may be regarded as a physiologic adaptation to exercise conditioning, largely without adverse clinical consequences. (J Am Coll Cardiol 2005;46:690–6) © 2005 by the American College of Cardiology Foundation

Left atrial (LA) enlargement is an independent predictor of atrial fibrillation in the general population and in patients with structural cardiac diseases, such as hypertrophic or dilated cardiomyopathies (1,2), often conveying adverse clinical consequences (3–6). Several previous investigations have suggested that trained athletes are predisposed to paroxysmal atrial fibrillation, possibly as a consequence of the cardiac remodeling associated with long-term exercise training (7–15). However, the prevalence of LA remodeling and the association with supraventricular tachyarrhythmias have not yet been systematically addressed in a large population of highly trained athletes.

In the present study involving a large cohort of Italian elite athletes engaged in a variety of competitive sports, we assessed the distribution and clinical significance of LA size in the context of athlete's heart and the differential diagnosis from structural heart disease, as well as the proclivity to supraventricular arrhythmias.

METHODS

Selection of the study population. All athletes consecutively examined at the National Institute of Sports Medicine from 1992 to 1995 initially were considered for inclusion in the study population. After clinical and echocardiographic assessment, 46 showed evidence of structural cardiac abnormalities and were excluded from further analysis. Therefore, the final study population comprised 1,777 highly trained athletes; of these, 1,566 had been examined as a part of our medical program for elite athletes (16) and 211 others initially had been referred for a suspected cardiovascular abnormality, which was eventually excluded.

Ages were 24 ± 6 years (range, 11 to 56 years); 1,298 (71%) were men. Body surface area was 1.86 ± 0.25 m² (range, 1.15 to 2.38 m²). Athletes participated in a wide variety of 38 different sporting disciplines. All were involved

Abbreviations and Acronyms

ECG = electrocardiogram/electrocardiographic/
           electrocardiography
LA = left atrial/atrium
LV = left ventricular

in vigorous training programs for substantial periods of time, from 2 to 28 years (mean, 6 years) before their evaluation at our institution. Of the 1,777 athletes, 390 (22%) had achieved international recognition in the Olympics or World Championships, and the remaining 1,387 competed at the national or regional level. Selected clinical data from these athletes have been presented in a previous report (17).

Echocardiography. Two-dimensional and Doppler echocardiographic studies were performed using a commercially available instrument (Sonos 2500 and 5500, Philips Technologies, Andover, Massachusetts). Measurements of end-diastolic and end-systolic left ventricular (LV) cavity dimensions and anterior ventricular septal and posterior free wall thicknesses were obtained from the M-mode echocardiogram, consistent with recommendations of the American Society of Echocardiography (18).

Maximum transverse LA dimension was measured from the M-mode echocardiogram during systole as the average of three consecutive cycles. Tracings were derived under direct anatomic visualization from the two-dimensional echocardiogram in the parasternal long-axis view and recorded at 100 mm/s. Because the focus of this study was the frequency with which athletes show LA enlargement in a range compatible with pathologic cardiac conditions, we relied primarily on absolute dimensions (as we have done in previous echocardiographic surveys of large athlete populations) (17,19), by selecting a threshold value that has been conventionally used in large epidemiologic studies (4,5) and can be placed directly in the context of clinical cardiovascular diagnosis. We chose the arbitrary cutoff of ≥40 mm to estimate enlarged transverse LA dimension (independent of body surface area or gender) because this partition value is widely used in clinical practice for defining LA enlargement (20).

Left ventricular mass was calculated using the formula proposed by Devereux (21). Relative wall thickness (h/r) was the ratio of the average of ventricular septal and posterior free wall thicknesses to the internal ventricular cavity radius (22). Parameters of LV filling were obtained with pulsed Doppler echocardiography, as previously described (23).

Echocardiograms were obtained in each athlete during periods of intense training; because of unavoidable practical considerations, echocardiography could not always be performed when athletes were at peak conditioning. When serial studies were available from the same athlete, the echocardiogram obtained at the first evaluation in our institution was used (17,19).

Interobserver variability for measurements of LA dimension was assessed in a subset of 90 subjects that were selected randomly from the present study group (and including 23 athletes with an LA ≥40 mm). Measurements were made independently by two investigators (A.P. and B.J.M.) without knowledge of the identity of the subjects, and concordance of measurements from the two observers was evaluated by intraclass correlation (17,24).

Electrocardiography (ECG). The ECG criteria of LA enlargement were based on the presence of prolonged P-wave duration of 0.12 s in standard leads I or II and/or inverted P-wave pattern with terminal negativity of >1 mm and ≥0.04 s in duration in lead V_1 (25).

Athletes with symptoms suggestive of supraventricular tachyarrhythmias underwent additional investigation, including symptom-limited exercise testing, Holter ECG monitoring (comprising a period of training similar to that performed typically by the athlete) and, in selected cases, electrophysiologic study (26).

Follow-up of athletes with LA enlargement. Athletes with enlarged LA in the absence of cardiac disease, including those with supraventricular arrhythmias, were allowed to continue in their training and competition (according to the Italian eligibility guidelines) (27), with the obligation of periodic (at least annual) cardiovascular evaluations. In each of these athletes, clinical status was reassessed in 2003, either by examination at our institution or by telephone interview.

Statistical methods. Data are expressed as mean ± standard deviation. Differences between mean values were assessed with the unpaired Student *t* test. Differences of proportions were assessed by the chi-square test. A two-tailed $p < 0.05$ was considered statistically significant.

Stepwise regression analysis was used to assess the impact that several variables (body surface area, age, blood pressure, heart rate, LV dimensions, LV filling indexes) had on LA size; those with statistically significant correlations were subsequently incorporated into the covariance analysis, together with gender and type of sport. Categorical variables were coded using a series of (N − 1) binary dummy variables, and their impact on atrial dimension was assessed after removing the effect of the continuous covariates, as previously reported (17). The statistical significance of variables in the covariance analysis was assessed by the Wald test (24).

RESULTS

LA dimensions. In the overall population of 1,777 athletes, transverse LA dimension ranged from 22 to 50 mm (mean, 35 ± 4 mm), and was greater in men (23 to 50 mm; mean, 37 ± 4 mm) than in women (20 to 46 mm; mean, 32 ± 4 mm; p < 0.001) (Fig. 1). The vast majority of athletes (i.e., 1,430 [80%]) had normal LA dimension <40 mm; the remaining 347 athletes (20%) showed increased LA size
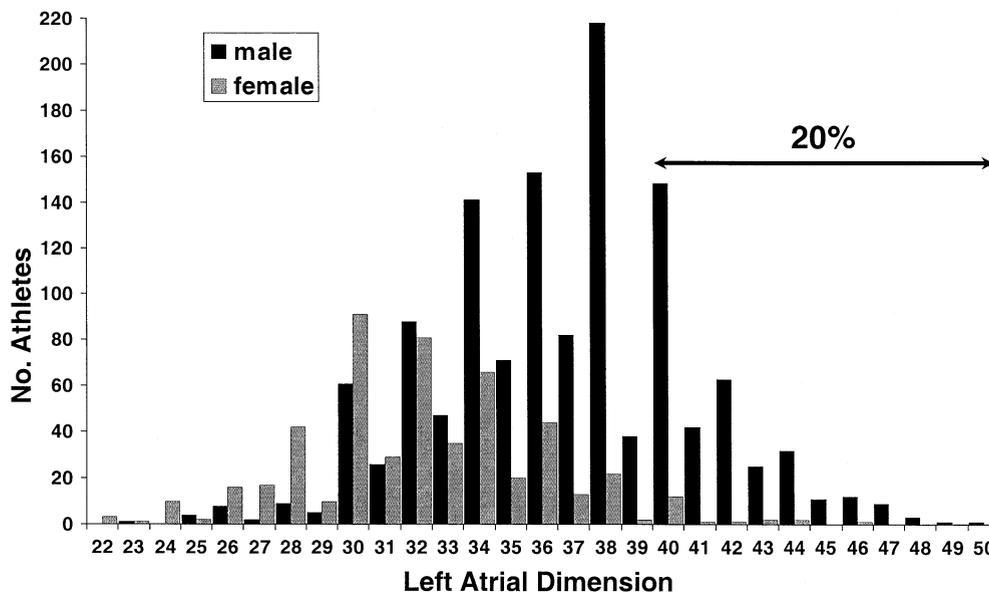
Figure 1. Distribution of transverse left atrium dimensions in 1,777 highly trained athletes. Data are shown separately for female **(grey bars)** and male **(black bars)** athletes. Twenty percent of athletes had an enlarged left atrium (range, 40 to 50 mm), including 2% with an atrial dimension ≥45 mm.

($\geq$40 mm), including 38 (2%) with a markedly dilated LA ($\geq$45 mm).

Other cardiac dimensions. Left ventricular end-diastolic cavity dimension ranged from 38 to 70 mm (mean, 54 ± 5 mm), and exceeded the clinically accepted upper partition value (i.e., ≥55 mm) (28) in a substantial proportion of athletes (786; 44%), including 251 (14%) with a markedly dilated LV cavity ≥60 mm. Maximum LV wall thickness (ventricular septum in each) was 6 to 16 mm (mean, 9 ± 1 mm) and exceeded normal limits (i.e., ≥12 mm) (28) in 17 (1%). Left ventricular mass normalized to body surface area was increased above normal limits (21) in 167 athletes (9%).

Determinants of LA dimension. In the overall group of 1,777 athletes, stepwise regression analysis showed that LA dimension was associated with LV end-diastolic cavity dimension (multiple R^2 = 0.53), suggesting that >50% of variability in atrial size was due to LV cavity size variability. By regression analysis, we calculated that in our athletes each increase of 1 mm in LV cavity dimension was associated with a 0.4-mm increase in LA diameter. Other variables, including maximum LV wall thickness, body surface area, and age, showed little additional independent impact on LA dimension, together increasing the multiple R^2 to 0.58.

Covariance analysis and the Wald test showed that the type of sport (as a categorical variable) significantly influenced the variability in LA dimension; specifically, cycling (p < 0.05), rowing/canoeing (p < 0.05), ice hockey (p < 0.01), and rugby (p < 0.01) had the largest influence on LA size. Gender had no significant additional impact on LA dimension.

Prevalence of supraventricular tachyarrhythmias. Of the 1,777 athletes, 14 (0.8%) had symptomatic and ECG documented episodes of supraventricular tachyarrhythmias at the entry of this study, including paroxysmal atrial fibrillation (n = 5; 0.3%), and supraventricular tachycardia (n = 9; 0.5%). None of these arrhythmic episodes were associated with embolic events.

Each of these 14 athletes had experienced prolonged palpitations either during exercise (n = 11) or at rest (n = 3), and symptoms were reproduced by occurrence of arrhythmia during either 24-h Holter ECG monitoring (n = 6), exercise testing or 12-lead ECG (n = 1 each), or induced at electrophysiologic study (n = 6) (Table 1).

Athletes with enlarged LAs. DEMOGRAPHICS AND SPORTING DISCIPLINES. The 347 athletes with enlarged LAs were predominantly men and had significantly larger body surface area compared with the other 1,430 athletes (Table 2). Athletes with enlarged LAs competed in a broad variety of 28 different sporting disciplines, with most engaged in rowing/canoeing (18%), cycling (10%), ice hockey (10%), rugby, and soccer (7%, each). A larger proportion of athletes with enlarged LAs had achieved an international level of performance and recognition (40% vs. 20% of athletes with normal-sized LAs; p < 0.001).

CARDIAC DIMENSIONS AND FUNCTION. These athletes also showed greater cardiac dimensional adaptation to training, including LV end-diastolic cavity ≥55 mm in 299 (86% vs. 34% in other athletes; p < 0.001) and increased LV mass ≥134 g (27% vs. 5%; p < 0.001) (Table 2, Fig. 2) (21). Patterns of transmitral flow-velocity were within normal limits in each athlete, and none had moderate or severe mitral regurgitation. Left ventricular systolic function was normal (i.e., ejection fraction >50%), and segmental wall motion abnormalities were absent in each (Table 2).

ECGS. The 12-lead ECG patterns were within normal limits in 117 athletes (34%). The remaining 230 athletes

Table 1. Demographic and Clinical Features of the 14 Athletes With Supraventricular Tachyarrhythmias Identified Among 1,777 Highly Trained Athletes

Athlete	Gender	Age (yrs)	Sport	Palpitations at	Diagnostic Testing*	Arrhythmia	LA (mm)
1	M	23	Rowing	Exercise	EPS	AF	38
2	M	32	Soccer	Rest	ECG	AF	38
3	M	16	Volleyball	Exercise	Holter	AVRT	32
4	M	20	Rowing	Exercise	Holter	AVNRT	37
5	M	36	Handball	Rest	EPS	AF	38
6	M	22	Cross-country skiing	Exercise	EPS	AVNRT	40
7	M	18	Pentathlon	Rest	Holter	AF	32
8	F	25	Field hockey	Exercise	Holter	AVNRT	32
9	M	15	Gymnastics	Exercise	Holter	AVNRT	36
10	F	28	Basketball	Exercise	EPS	AVNRT	36
11	M	18	Skating	Exercise	EPS	AVRT	33
12	M	23	Skating	Exercise	ECG (during exercise)	AVNRT	36
13	M	17	Basketball	Exercise	EPS	AF	44
14	F	28	Basketball	Rest plus Exercise	Holter	AVNRT	46

*Test during which the arrhythmia associated with symptoms was recorded.
AF = paroxysmal atrial fibrillation; AVNRT = atrio-ventricular nodal re-entrant tachycardia; AVRT = atrio-ventricular re-entrant tachycardia; ECG = 12-lead electrocardiogram; EPS = electrophysiologic study; Holter = 24-h Holter electrocardiographic monitoring; LA = left atrium transverse diameter.

demonstrated a variety of abnormal patterns, most commonly increased precordial lead voltages suggestive of LV hypertrophy (187; 54%), deep narrow Q waves (60; 17%), and T-wave inversion (56; 16%). Only 9 of the 347 athletes (3%) with an LA ≥40 mm on an echocardiogram met ECG criteria for LA enlargement.

SUPRAVENTRICULAR TACHYARRHYTHMIAS IN RELATION TO LA DIMENSION. Atrial fibrillation and supraventricular tachycardia, considered together, occurred with similar frequency in athletes with LA enlargement (n = 3; 0.9%) and with normal sized LAs (n = 11; 0.8%; p = NS). Paroxysmal atrial fibrillation occurred in only one athlete with enlarged LA and in four athletes with normal LA size (i.e., 0.3% prevalence in both) (Fig. 3). Individual subject analysis of the 38 athletes with most enlarged LAs of ≥45 mm showed that supraventricular tachyarrhythmias were exceedingly rare, occurring in only one of these athletes (Table 1).

FOLLOW-UP. Of the 347 athletes with an enlarged LA, clinical and echocardiographic data were obtained in 318 (92%) during a 1- to 10-year follow-up period (mean, 7 ± 3 years). Ages at most recent assessment were 29 to 62 years (mean, 33 ± 6 years). The vast majority of these athletes (i.e., 214, or 61%) had continued in their usual training schedule and sports competition during the follow-up period. Virtually all these athletes (312; 98%) had remained asymptomatic and without evidence of cardiovascular abnormalities during the follow-up. One former basketball player, a 33-year-old man, experienced brief episodes of supraventricular tachycardia without embolic events six years after withdrawing from training and competition (i.e., 0.04% year incidence). Of the remaining five athletes, the three with a history of atrial fibrillation or supraventricular tachycardia at the entry of this study have continued to experience paroxysmal episodes of tachyarrhythmias; two

Table 2. Clinical, Demographic, and Echocardiographic Findings in 1,777 Elite Athletes With Respect to Left Atrial Dimension

	LA Dimension		
Parameter	≥40 mm	<40 mm	p Value
No. athletes	347	1,430	—
Transverse LA dimension	41.7 ± 2.1	33.9 ± 3.4	—
No. (% male)	339 (95%)	959 (67%)	<0.001
Body surface area (m²)	2.06 ± 0.20 (1.51–2.67)	1.81 ± 0.23 (0.98–2.59)	<0.001
Heart rate (beats/min)	53 ± 10 (31–94)	58 ± 11 (31–112)	<0.001
LV end-diastolic dimension (mm)	58.9 ± 3.9 (48–70)	52.3 ± 4.9 (38–67)	<0.001
Max. LV wall thickness (mm)	10.4 ± 1.1 (7–16)	9.1 ± 1.3 (5–15)	<0.001
LV mass index (g/m²)	121 ± 21 (70–195)	95 ± 21 (44–170)	<0.001
h/r ratio	0.36 ± 0.04 (0.26–0.60)	0.35 ± 0.04 (0.23–0.60)	<0.004
Aorta (mm)	33.5 ± 2.7 (25–44)	30.3 ± 3.3 (20–43)	<0.001
E_{max} (mm/s)	68 ± 13 (35–109)	71 ± 14 (12–123)	NS
A_{max} (mm/s)	31 ± 9 (15–60)	32 ± 9 (12–108)	NS
E/A ratio	2.3 ± 0.8 (0.85–4.8)	2.4 ± 0.8 (0.4–6.2)	NS

Values are expressed as mean ± standard deviation and range in parentheses.
A_{max} = peak flow-velocity at late diastole, derived by pulsed Doppler; E_{max} = peak flow-velocity at early diastole, derived by pulsed Doppler; h/r = relative wall thickness (ratio of averaged ventricular septal and posterior free wall thicknesses to internal LV cavity radius); LA = left atrium; LV = left ventricular; Max. = maximum; NS = not significant.
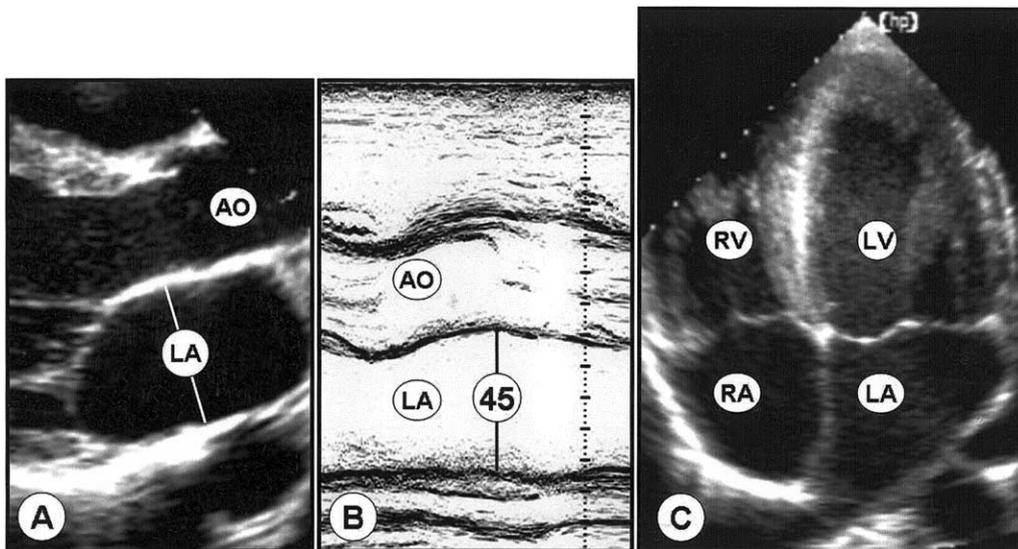
Figure 2. Stop-frame two-dimensional parasternal long-axis view **(A)**, M-mode echocardiogram **(B)**, and four-chamber **(C)** view from a 26-year-old elite rower, showing left atrial enlargement (transverse dimension = 45 mm). The left atrium is globally enlarged in both the transverse and longitudinal dimensions; left and right ventricular and right atrial chambers are also enlarged. AO = aortic root; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

other athletes died of noncardiac disease (automobile accident and cancer).

Interobserver variability. Left atrial transverse diameter measured by the first observer was 36 ± 6 mm (range, 23 to 55 mm) and by the second observer was 36 ± 5 mm (range, 25 to 51 mm). The mean interobserver difference was only 0.2 ± 2.1 mm (range 5 to −5, mm); p = 0.71. Concordance of measurements from the two observers, as assessed by intraclass correlation, was 93%.

DISCUSSION

The present study is relevant to the widespread clinical perception, based on previous observations in selected athlete groups (8–10,12,14), that lone atrial fibrillation (with

Figure 3. Prevalence of supraventricular tachyarrhythmias (i.e., paroxysmal atrial fibrillation or supraventricular tachycardia) before or at initial evaluation in our institute with respect to left atrial (LA) dimension, as assessed by echocardiography in 1,777 athletes. AF = paroxysmal atrial fibrillation; CV = cardiovascular; SVT = supraventricular tachycardia.

the potential for adverse clinical events) occurs disproportionately in highly trained and professional athletes (29), in the absence of demonstrable cardiac disease. Left atrial enlargement in the context of the physiologic cardiac remodeling in trained athletes (i.e., athlete's heart) has been considered responsible by itself for a proclivity to atrial fibrillation, especially when associated with marked sinus bradycardia (7–9).

The present data shows, however, that LA remodeling associated with intensive exercise and chronic athletic conditioning does not predispose per se to supraventricular tachyarrhythmias. In our systematic investigation of more than 1,700 elite athletes examined during a 3-year period, atrial fibrillation (and other supraventricular tachyarrhythmias) was distinctly uncommon at study entry (0.8%), a paradoxical finding given the large proportion (i.e., 20%) of athletes with LA enlargement. Furthermore, these arrhythmias occurred with similar frequency in athletes with enlarged and with normal LA size. The occurrence of atrial fibrillation in only 0.2% of our athlete population closely resembles the prevalence of paroxysmal atrial fibrillation (i.e., 0.2% to 0.3%) reported in general populations of comparable age and gender, with or without cardiac disease (30–32).

In addition, only 0.3% of those athletes with an enlarged LA on initial evaluation developed a supraventricular tachycardia during the average seven-year follow-up period (incidence 0.04% per year). We cannot exclude, however, the possibility that incidence of AF has been underestimated and may have been higher if routinely investigated with ECG monitoring (33).

Our data do not support, therefore, the clinical concern that LA enlargement in athletes may predispose them to
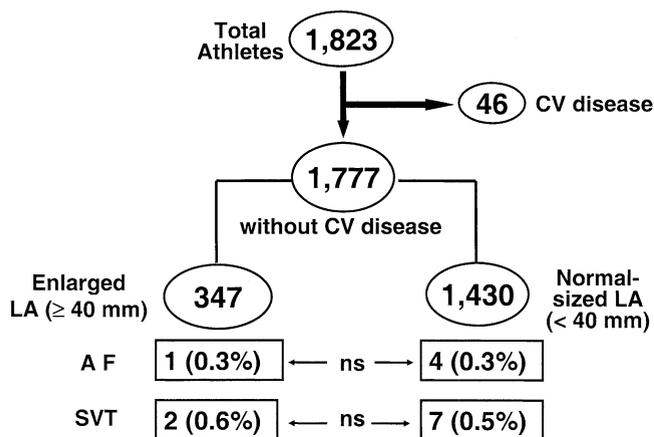
adverse clinical sequelae, such as stroke or other embolic events (3–5). In contrast to previous studies (7–14), our observations show that LA enlargement does not represent a preclinical abnormality in trained athletes but rather likely represents an innocent consequence of chronic and intensive exercise conditioning.

The present data also provide an assessment of LA dimension, with the purpose of defining the upper limits of atrial enlargement associated with intensive athletic training (i.e., athlete's heart). Recognition of the upper limits of atrial size in competitive athletes is of particular clinical relevance by offering the opportunity to distinguish physiologic and benign cardiac remodeling in athletes from structural heart diseases, such as hypertrophic or dilated cardiomyopathy (2,34). This differential diagnosis has important implications because the identification of these cardiomyopathies often represents the basis, in an effort to minimize the risk of disease progression or sudden death, for disqualifying athletes from competition (27,35).

In this regard, our data demonstrate that enlarged LA size (≥40 mm) is relatively common and present in 20% of a large cohort of competitive athletes, including marked dilatation ≥45 mm in 2% of our athletes, mimicking the atrial dilatation present in structural cardiac diseases (1,2). The upper range of LA enlargement in our athlete cohort was 46 mm in women and 50 mm in men. Therefore, transverse LA dimensions exceeding these outer limits in trained athletes are unlikely to represent the physiologic consequences of intensive exercise training and are more likely the expression of a primary pathologic condition.

The possible determinants of LA enlargement in our athlete population remain incompletely resolved. Multivariate regression analysis, however, showed that LA enlargement occurs in close association with LV cavity enlargement and that this morphologic change is minimally related to body size and largely dependent on the type of sport participated in by the athlete, with cycling, rowing, and canoeing showing the greatest impact. We have demonstrated that the same type of sports have the largest effect to enlarge also LV cavity dimension (17). Our observations suggest, therefore, that both LA and LV cavity remodeling observed in trained athletes represent the physiologic consequence of a global cardiac adaptation to the increased preload associated with intensive and chronic dynamic training (17).

This mechanism differs from certain pathologic conditions in which LA enlargement is the consequence of impaired LV compliance and/or mitral regurgitation (e.g., hypertrophic or dilated cardiomyopathy) (2,34), or increased afterload (e.g., systemic hypertension) (36). Because our athletes showed normal LV diastolic filling and systolic function, it is reasonable to conclude that their LA enlargement represents uniquely the physiologic consequence of chronic exercise training. Finally, ECG was largely inefficient in detecting LA enlargement because only a small minority (i.e., 3%) of our athletes with enlarged LAs by echocardiography had ECG criteria that was consistent with this diagnosis

Conclusions. Atrial fibrillation and other supraventricular tachyarrhythmias proved to be uncommon in our large population of highly trained athletes, despite a relatively high prevalence of LA enlargement. Therefore, LA enlargement in trained athletes can be regarded as a physiologic and benign adaptation to exercise conditioning and another component of the "athlete's heart." Absolute dimensional cut-offs of 45 mm in women and 50 mm in men may be useful for distinguishing physiologic cardiac remodeling from pathologic conditions such as cardiomyopathies.

Reprint requests and correspondence: Dr. Antonio Pelliccia, National Institute of Sports Medicine, Italian National Olympic Committee, Via dei Campi Sportivi, 46, 00197 Rome, Italy. E-mail: ant.pelliccia@libero.it.

REFERENCES

1. Olivotto I, Cecchi F, Casey SA, Dolora A, Traverse JH, Maron BJ. Impact of atrial fibrillation on the clinical course of hypertrophic cardiomyopathy. Circulation 2001;104:2517–24.
2. Gavazzi A, De Maria R, Renosto G, et al. The spectrum of left ventricular size in dilated cardiomyopathy: clinical correlates and prognostic implications. SPIC (Italian Multicenter Cardiomyopathy Study) Group. Am Heart J 1993;125:410–22.
3. Kopecky SL, Gersh BJ, McGoon MD, et al. The natural history of lone atrial fibrillation: a population-based study over three decades. N Engl J Med 1987;317:669–74.
4. Vaziri SM, Larson MG, Benjamin EJ, Levy D. Echocardiographic predictors of nonrheumatic atrial fibrillation: the Framingham Heart Study. Circulation 1994;89:724–30.
5. Benjamin EJ, D'Agostino RB, Belanger AJ, Wolf PA, Levy D. Left atrial size and the risk of stroke and death: the Framingham Heart Study. Circulation 1995;92:835–41.
6. Tsang TS, Barnes ME, Bailey KR, et al. Left atrial volume: important risk marker of incident atrial fibrillation in 1,655 older men and women. Mayo Clinic Proc 2001;76:467–75.
7. Coumel P. Autonomic influences in atrial tachyarrhythmias. J Cardiovasc Electrophysiol 1996;7:999–1007.
8. Furlanello F, Bertoldi A, Dallago M, et al. Atrial fibrillation in elite athletes. J Cardiovasc Electrophysiol 1998;9;S63–8.
9. Link MS, Wang PJ, Estes NA III. Cardiac arrhythmias and electrophysiologic observations in the athlete. In: Williams R, editor. The Athlete and Heart Disease. Philadelphia, PA: Lippincott Williams & Wilkins, 1998:197–216.
10. Jensen-Urstad K. High prevalence of arrhythmias in elderly male athletes with a lifelong history of regular strenuous exercise. Heart 1998;79:161–4.
11. Estes NA III, Link MS, Cannom D, et al. Report of the NASPE policy conference on arrhythmias and the athlete. J Cardiovasc Electrophysiol 2001;12:1208–19.
12. Mont L, Sambola A, Brugada J, et al. Long-lasting sport practice and lone atrial fibrillation. Eur Heart J 2002;23:477–82.
13. Coumel P. Atrial fibrillation: one more sporting inconvenience? Eur Heart J 2002;23:431–3.
14. Hoogsteen J, Schep G, van Hemel NM, van der Wall EE. Paroxysmal atrial fibrillation in male endurance athletes. A 9-year follow up. Europace 2004;6:222–8.
15. Toutouzas K, Trikas A, Pitsavos C, et al. Echocardiographic features of left atrium in elite male athletes. Am J Cardiol 1996;78:1314–7.
16. Pelliccia A, Maron BJ. Preparticipation cardiovascular evaluation of the competitive athlete: perspectives from the 30 year Italian experience. Am J Cardiol 1995;75:827–8.

17. Pelliccia A, Culasso F, Di Paolo FM, Maron BJ. Physiologic left ventricular cavity dilatation in elite athletes. Ann Intern Med 1999; 130:23–31.
18. Sahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. Circulation 1978;58: 1072–83.
19. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med 1991;324:295–301.
20. Feigenbaum H. Echocardiography. 4th edition. Philadelphia, PA: Lea & Febiger; 1986:622.
21. Devereux RB. Detection of left ventricular hypertrophy by M-mode echocardiography: anatomic validation, standardization, and comparison to other methods. Hypertension 1987;9 Suppl II:19–26.
22. Ford LE. Heart size. Circ Res 1976;39:297–303.
23. Maron BJ, Spirito P, Green KJ, Wesley YE, Bonow RO, Arce J. Noninvasive assessment of left ventricular diastolic function by pulsed Doppler echocardiography in patients with hypertrophic cardiomyopathy. J Am Coll Cardiol 1987;10:733–42.
24. Armitage P, Berry G. Statistical Methods in Medical Research. Oxford: McGraw-Hill Publications, 1994:277.
25. Pelliccia A, Maron BJ, Culasso F, et al. Clinical significance of abnormal electrocardiographic patterns in trained athletes. Circulation 2000;102:278–84.
26. Biffi A, Pelliccia A, Verdile L, et al. Long-term clinical significance of frequent and complex ventricular tachyarrhythmias in trained athletes. J Am Coll Cardiol 2002;40:446–52.
27. Organizing Cardiologic Committee on Eligibility for Sports (COCIS). Cardiologic Protocols on Determining Eligibility for Competitive Sports. Rome: CESI Publisher, 2003:1–114.
28. Henry WL, Gardin JM, Ware JH. Echocardiographic measurements in normal subjects from infancy to old age. Circulation 1980;62:1054–61.
29. "Atrial fibrillation" search query at home page of the National Basketball Association. Available at: www.nba.com. Accessed June 22, 2005.
30. Rose G, Baxter PJ, Reid DD, McCartney P. Prevalence and prognosis of electrocardiographic findings in middle-aged men. Br Heart J 1978;40:636–43.
31. Halperin JL, Hart RG. Atrial fibrillation and stroke: new ideas, persisting dilemmas. Stroke 1988;19:937–41.
32. Kannel WB, Abbott RD, Savage DD, McNamara PM. Epidemiologic features of chronic atrial fibrillation. The Framingham Study. N Engl J Med 1982;306:1018–22.
33. Fetsch T, Bauer P, Engberding R, et al. Prevention of atrial fibrillation after cardioversion: results of the PAFAC study. Eur Heart J 2004; 25:1385–94.
34. Maron BJ. Hypertrophic cardiomyopathy: a systematic review. JAMA 2002;287:1308–20.
35. Maron BJ, Zipes DP. 36th Bethesda Conference: eligibility recommendations for competitive athletes with cardiovascular abnormalities. J Am Coll Cardiol 2005;45:2–64.
36. Ganau A, Devereux RB, Roman MJ, et al. Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension. J Am Coll Cardiol 1992;19:1550–8.