Journal of the American College of Cardiology
© 2010 by the American College of Cardiology Foundation
Published by Elsevier Inc.

Vol. 55, No. 15, 2010
ISSN 0735-1097/10/$36.00
doi:10.1016/j.jacc.2009.10.068

Long-Term Clinical Consequences of Intense, Uninterrupted Endurance Training in Olympic Athletes

Antonio Pelliccia, MD,* Norimitsu Kinoshita, MD,† Cataldo Pisicchio, MD,* Filippo Quattrini, MD,* Fernando M. DiPaolo, MD,* Roberto Ciardo, MD,* Barbara Di Giacinto, MD,* Emanuele Guerra, MD,* Elvira De Blasiis, MD,* Maurizio Casasco, MD,* Franco Culasso, PHD,‡ Barry J. Maron, MD§

Rome, Italy; Yokohama, Japan; and Minneapolis, Minnesota

Objectives The aim of this study was to assess incidence of cardiac events and/or left ventricular (LV) dysfunction in athletes exposed to strenuous and uninterrupted training for extended periods of time.

Background Whether highly intensive and uninterrupted athletic conditioning over a long period of time might be responsible for cardiac events and/or LV dysfunction is unresolved.

Methods We assessed clinical profile and cardiac dimensions and function in 114 Olympic athletes (78% male; mean age 22 ± 4 years), free of cardiovascular disease, participating in endurance disciplines, who experienced particularly intensive and uninterrupted training for 2 to 5 consecutive Olympic Games (total, 344 Olympic events), over a 4- to 17-year-period (mean 8.6 ± 3 years).

Results Over the extended period of training and competition, no cardiac events or new diagnoses of cardiomyopathies occurred in the 114 Olympic athletes. Global LV systolic function was unchanged (ejection fraction: 62 ± 5% to 63 ± 5%; $p = NS$), and wall motion abnormalities were absent. In addition, LV volumes (142 ± 26 ml to 144 ± 25 ml; $p = 0.52$) and LV mass index (109 ± 21 g/m^2 to 110 ± 22 g/m^2; $p = 0.74$) were unchanged, and LV filling patterns remained within normal limits, although left atrial dimension showed a mild increase (37.8 ± 3.7 mm to 38.9 ± 3.2 mm; $p < 0.001$).

Conclusions In young Olympic athletes, extreme and uninterrupted endurance training over long periods of time (up to 17 years) was not associated with deterioration in LV function, significant changes in LV morphology, or occurrence of cardiovascular symptoms or events. (J Am Coll Cardiol 2010;55:1619–25) © 2010 by the American College of Cardiology Foundation

The long-term clinical consequences of cardiac remodeling in trained athletes and whether chronic exposure to highly intensive athletic conditioning might ultimately be responsible for abnormal cardiac function and/or adverse events are not completely resolved. Occurrence of "cardiac fatigue" with acute and transient left ventricular (LV) systolic dysfunction and segmental wall motion abnormalities have been reported as an effect of prolonged endurance events lasting 3 to 17 h—such as Iron-man triathlon races (1–4)—suggesting that athletic training, when particularly strenuous, might be responsible for deterioration in LV function. We have previously shown persistent LV cavity enlargement after prolonged deconditioning in former elite athletes (5), and other investigators have reported LV dysfunction in professional bicyclists of middle-age (6). Indeed, more than 10% of elite cyclists participating in serial Tour de France races unexpectedly showed systolic dysfunction with marked

See page 1626

LV cavity dilation, suggesting a possible diagnosis of dilated cardiomyopathy (7). More recently, ominous ventricular tachyarrhythmias of right ventricular origin—associated with mild reduction in systolic function—have been reported in highly trained cyclists (8), suggesting that excessive physical training might have adverse electrophysiologic consequences.

From the *Institute of Sports Medicine and Science, Italian National Olympic Committee, Rome, Italy; †Sports Medicine Research Center, Keio University, Yokohama, Japan; ‡Department of Experimental Medicine, University La Sapienza, Rome, Italy; and §The Hypertrophic Cardiomyopathy Center, Minneapolis Heart Institute Foundation, Minneapolis, Minnesota.

Manuscript received January 13, 2009; revised manuscript received October 27, 2009, accepted October 27, 2009.

JACC Vol. 55, No. 15, 2010
April 13, 2010:1619–25

Abbreviations and Acronyms

E/A ratio = early-to-late diastolic peak flow velocity ratio

ECG = electrocardiography

LV = left ventricular

To address this important clinical issue, we have taken the opportunity to assess a unique population of Olympic athletes, who had been exposed to strenuous and uninterrupted training for extended periods of time, including participation in up to 5 consecutive Olympic Games.

Methods

Study population. The Institute of Sports Medicine and Science in Rome is a medical division of the Italian National Olympic Committee, responsible for the physiologic and medical evaluation of elite athletes who are members of the national teams, from which the participants in the Olympic Games are selected every 4 years. The medical program for Olympic athletes routinely includes cardiovascular evaluation with history, physical examination, 12-lead and exercise electrocardiography (ECG), and 2-dimensional and Doppler echocardiography (9).

For the purpose of this investigation, we selected elite athletes engaged in those endurance sports known to be associated with the most substantial cardiac remodeling (10,11), who had been training uninterruptedly at the highest competitive level in at least 2 consecutive Summer or Winter Olympic Games from 1992 or 1994, respectively, and in the World Championships in the alternate years.

Of the 9,630 athletes evaluated from 1992 to 2008 in our Institute, 2,330 were examined within the Olympic program; from this population, 460 Italian athletes participating in particularly intense endurance sports disciplines (i.e., cycling, long-distance running, cross-country skiing, swimming, rowing/canoeing, triathlon) were considered for inclusion in the study. Among these 460 athletes, 114 had participated in 2 or more consecutive Olympic Games and had technically satisfactory echocardiograms and therefore were selected for the final study population.

These 114 Italian athletes (89 male and 25 female), age 22 ± 4 years at the study entry and with mean body surface area (BSA) of 1.9 ± 0.2 m^2, were engaged in rowing/canoeing (n = 55), cycling (n = 19), middle-distance running (800 m; 3,000 m steeple-chase; and 5,000 m; n = 8), long-distance running (10,000 m and marathon; n = 9), cross-country skiing (n = 15), swimming (400 to 1,500 m; n = 6), and triathlon (n = 2). Before their initial Olympic team selection, the 114 athletes had been involved in regular endurance training and competition at the national level for a period of 1 to 15 years (mean 6.9 ± 3.8 years) and, after Olympic selection, trained uninterruptedly for an additional 4 to 17 years (mean 8.6 ± 3.7 years), for a total training period of 15.5 ± 4.1 years. Training schedules in the pre-Olympic phase usually included 2 daily sessions, each of ≥2-h duration, including specific work (e.g., on boat for rowers/canoeists, on track for runners) and technical aspects and/or general muscle conditioning programs (see detailed training schedule in Online Appendix).

The period of observation extended from the time each athlete underwent our evaluation before first participation in the Olympic Games to the time the athlete participated in the most recent Olympics (or World Championships). The average time duration between these 2 examinations was 8.6 ± 3.7 years (range 4 to 17 years).

Each subject included in this analysis was judged to be free of structural cardiovascular disease at initial evaluation on the basis of medical history, physical examination (with blood pressure consistently <140/90 mm Hg), exercise ECG testing, and 2-dimensional and Doppler echocardiography. In athletes with LV wall thickness exceeding normal limits (≥13 mm) and/or markedly dilated LV cavity (≥60 mm), physiologic remodeling of the athlete's heart was distinguished from hypertrophic cardiomyopathy and dilated cardiomyopathy, respectively, according to previously described criteria (11–14). Six athletes presented markedly abnormal repolarization patterns on ECG, with inverted T waves of >2 mm in >2 precordial/standard leads in the absence of structural cardiac disease (15).

Comparison group. To exclude the possibility that changes in cardiac function could have precluded athletes not included in our study group from continued participation in consecutive Olympic Games; we assembled a comparison group of elite athletes engaged in endurance sports on the basis of these criteria: 1) participation in only 1 Olympic Games; and 2) subsequent 4-year period during which systematic training continued but the athletes failed to be selected as members of Italian teams for the subsequent Olympic Games.

From our database of athletes engaged in endurance sports, who were examined within our Olympic program from 1992 to 2000, we identified 97 who met these criteria. These athletes were comparable to the study group of the 114 with regard to age (23.5 ± 4.1 years vs. 22.1 ± 3.8 years) and type of sport participated in (73% vs. 78% engaged in rowing/canoeing, cycling, and long-distance running).

Echocardiography. Two-dimensional and Doppler echocardiographic studies were performed with commercially available Philips Sonos 1500 or Sonos 5500 (Philips Medical System, Bothell, Washington) instruments. Images of the heart were obtained in multiple cross-sectional planes by using standard transducer positions. M-mode echocardiograms were derived from 2-dimensional images under direct anatomic visualization and were recorded at 100 mm/s. Measurements of end-diastolic and end-systolic LV cavity dimensions and anterior ventricular septal and posterior free wall thicknesses were obtained in accordance with recommendations of the American Society of Echocardiography (16,17). The LV mass was calculated with the formula proposed by Devereux et al. (18) and was indexed to BSA. Relative wall thickness was the ratio of the average ventric-

ular septal and posterior free wall thickness to the radius of the ventricular cavity.

Ejection fraction was assessed from end-diastolic and -systolic LV volumes, in the apical 4-chamber view and quantified according to modified Simpson rule (19). In addition, the blood pressure/end-systolic volume index ratio was used to characterize LV systolic function (20). Parameters of LV filling were obtained with pulsed Doppler echocardiography, as previously described (21).

This study was funded by the Italian National Olympic Committee and the design was approved by the institutional review board. Written informed consent was waived for all athletes undergoing a standard clinical evaluation pursuant to Italian law and institute policy. All the records and clinical data on these athletes are kept in a database maintained by the Institute.

Statistical analysis. Data were expressed as mean ± SD. Differences between means were assessed with paired or unpaired Student *t* test, as appropriate. Differences between proportions were assessed with chi-square test. A 2-tailed p value <0.05 was considered evidence of statistical significance. Statistical analyses were carried out with the BMDP Statistical software (22).

Results

Athlete's career. Of the 114 athletes included in this investigation, 42 had participated in 2 consecutive Olympic Games, 40 athletes in 3 Olympics, 20 athletes in 4 Olympics, and 12 athletes in 5 Games. Overall, the athletes in this study participated in 344 Olympic Games and 862 World Championships, and 42 (37%) had been medalists 1 or more times.

Cardiac functional changes. Echocardiographic-derived indexes of LV function are reported in Table 1. Global systolic function did not change over time (ejection fraction: 62 ± 5% to 63 ± 5%; p = 0.09), and no athlete showed decreased LV systolic function (i.e., ejection fraction <50%) or regional wall motion abnormalities at most recent evaluation. The blood pressure/systolic volume index ratio was within normal limits in each athlete and did not change over the period of observation (4.09 ± 0.9 to 4.31 ± 1.1; p = 0.32).

Doppler-derived late diastolic (atrial) peak A-wave velocity increased during the follow-up (32.3 ± 10.0 cm/s to 42.3 ± 9.4 cm/s; +31%; p < 0.001), and early diastolic peak E-wave velocity did not change. Therefore, the E/A ratio decreased (2.5 ± 0.7 to 1.8 ± 0.4; −28%; p < 0.001). However, at the most recent evaluation each athlete showed a normal filling pattern (with E/A >1). Deceleration time increased during the observation period from 165 ± 33 to 193 ± 39 (p < 0.05).

Cardiac dimensional changes. Cardiac dimensions in Olympic athletes at initial and most recent evaluation are reported in Table 2. No significant changes (by paired *t* test) were observed for LV cavity dimensions, wall thickness, relative wall thickness, or mass index over the long-term period of training and competition (Fig. 1). A modest but statistically

Table 1 Indexes of LV Function at Initial and Most Recent Evaluation in the 114 Olympic Athletes

	Male (n = 89)			Female (n = 25)			Total Athletes (n = 114)		
	Initial	Most Recent	p Value	Initial	Most Recent	p Value	Initial	Most Recent	p Value
EF (%)	61 ± 5 (50–71)	62 ± 5 (53–72)	0.04	64 ± 5 (57–78)	64 ± 4 (55–74)	0.96	62 ± 5 (50–78)	63 ± 5 (53–74)	0.09
FS (%)	35 ± 4 (27–47)	36 ± 4 (26–46)	0.10	37 ± 3 (30–43)	39 ± 4 (33–48)	0.01	35 ± 4 (27–47)	37 ± 4 (28–48)	0.02
Peak E (cm/s)	74.0 ± 14.8 (46–118)	74.3 ± 13.4 (43–107)	0.79	73.6 ± 14.2 (42–98)	81.8 ± 19.3 (55–145)	0.05	74.1 ± 14.7 (42–116)	76.5 ± 15.1 (43–145)	0.15
Peak A (cm/s)	32.0 ± 10.1 (15–56)	41.8 ± 9.3 (24–72)	0.001	33.6 ± 9.9 (19–56)	44.4 ± 9.8 (26–74)	0.001	32.3 ± 10.0 (15–56)	42.3 ± 9.4 (24–76)	0.001
E/A ratio	2.5 ± 0.7 (1.2–5.1)	1.8 ± 0.9 (0.8–3.7)	0.001	2.33 ± 0.8 (1.3–4.7)	1.9 ± 0.3 (1.2–2.6)	0.005	2.5 ± 0.7 (1.2–5.1)	1.8 ± 0.4 (1.1–3.7)	0.001
E slope (cm/s^2)	461 ± 122 (140–839)	397 ± 122 (158–779)	0.005	467 ± 121 (271–695)	457 ± 117 (320–737)	0.86	463.1 ± 112.6 (140–839)	414.1 ± 122.4 (158–779)	0.005
DT (ms)	166.0 ± 34.9 (84.6–284.1)	196.1 ± 34.9 (111.3–316.7)	0.000	161.6 ± 26.8 (126.5–213.9)	183.0 ± 31.4 (123.0–243.0)	0.004	165.1 ± 33.3 (84.6–284.1)	193.3 ± 39.9 (111.3–316.7)	0.001
PSP/ESVi (mm Hg/ml)	3.98 ± 0.82 (2.03–6.05)	4.17 ± 0.99 (2.42–7.16)	0.08	4.47 ± 1.01 (3.00–6.37)	4.81 ± 1.14 (3.25–7.67)	0.11	4.09 ± 0.88 (2.03–6.37)	4.31 ± 1.05 (2.42–7.67)	0.32

Values are reported as mean ± SD (range). Statistical differences assessed by paired *t* test and by chi-square test for proportions.
DT = deceleration time; E/A ratio = early-to-late diastolic peak flow velocity ratio; EF = ejection fraction; E slope = early diastolic peak flow velocity deceleration rate; FS = fractional shortening; peak A = late (atrial) diastolic peak flow velocity; peak E = early diastolic peak flow velocity; LV = left ventricular; PSP/ESVi = ratio of the peak systolic pressure/end-systolic volume index.

JACC Vol. 55, No. 15, 2010
April 13, 2010:1619–25

Table 2 Cardiac Dimensions at Initial and Most Recent Evaluation in the 114 Olympic Athletes

	Male (n = 89)			Female (n = 25)			Total Athletes (n = 114)		
	Initial	Most Recent	p Value	Initial	Most Recent	p Value	Initial	Most Recent	p Value
AVS (mm)	10.8 ± 1.1 (8–15)	10.9 ± 1.0 (8–13)	0.35	9.4 ± 0.9 (8–11)	9.2 ± 0.8 (8–11)	0.51	10.5 ± 1.2 (8–15)	10.6 ± 1.2 (8–13)	0.73
PFW (mm)	10.4 ± 0.9 (8–14)	10.6 ± 0.9 (8–13)	0.15	8.9 ± 0.7 (8–10)	9.0 ± 0.7 (8–10)	0.77	10.1 ± 1.1 (8–14)	10.2 ± 1.1 (8–13)	0.19
LVDd (mm)	58.8 ± 3.4 (50–70)	59.0 ± 3.1 (53–67)	0.56	52.2 ± 3.2 (45–60)	52.4 ± 3.1 (46–59)	0.73	57.3 ± 4.3 (45–70)	57.5 ± 4.1 (46–67)	0.47
LVDs (mm)	38.2 ± 3.2 (32–49)	37.8 ± 3.7 (30–46)	0.31	32.9 ± 2.9 (28–38)	32.0 ± 3.1 (27–38)	0.13	37.1 ± 3.9 (28–49)	36.5 ± 4.3 (27–46)	0.16
RWT	0.36 ± 0.03 (0.27–0.58)	0.36 ± 0.03 (0.27–0.45)	0.47	0.35 ± 0.03 (0.27–0.42)	0.35 ± 0.03 (0.27–041)	0.74	0.36 ± 0.04 (0.27–0.58)	0.36 ± 0.03 (0.27–0.45)	0.79
LAD (mm)	38.6 ± 3.5 (30–47)	39.6 ± 2.9 (34–46)	0.01	34.7 ± 2.6 (30–40)	36.6 ± 3.0 (31–45)	0.007	37.8 ± 3.7 (30–47)	38.9 ± 3.2 (31–46)	0.001
Ao (mm)	33.3 ± 2.1 (30–39)	34.0 ± 2.7 (28–40)	0.01	29.3 ± 2.0 (24–32)	29.1 ± 2.0 (25–34)	0.70	32.4 ± 2.7 (24–39)	32.9 ± 3.3 (25–40)	0.04
EDV vol (ml)	172.7 ± 23.6 (118.2–255.4)	173.7 ± 21.1 (135.3–231.36)	0.61	131.6 ± 18.7 (92.4–180.0)	132.7 ± 18.2 (97.3–173.2)	0.74	163.6 ± 28.3 (92.4–255.4)	164.7 ± 26.6 (97.3–231.4)	0.53
ESV vol (ml)	63.6 ± 13.1 (41.0–112.8)	62.2 ± 14.2 (35.0–97.34)	0.38	44.4 ± 9.6 (29.5–61.9)	41.5 ± 9.8 (27.0–61.9)	0.14	59.4 ± 14.7 (29.5–112.8)	57.7 ± 15.9 (27.0–97.3)	0.23
LVM/EDV (g/ml)	1.51 ± 0.20 (1.05–2.59)	1.53 ± 0.17 (1.05–1.94)	0.35	1.33 ± 0.15 (1.05–1.60)	1.32 ± 0.14 (1.04–1.60)	0.78	1.47 ± 0.20 (1.05–2.59)	1.48 ± 0.18 (1.04–1.94)	0.61
LVM index (g/m^2)	128.9 ± 18.7 (89.7–194.6)	130.4 ± 20.5 (78.1–188.9)	0.46	105.6 ± 14.1 (80.3–134.0)	106.2 ± 18.0 (76.2–138.3)	0.86	123.8 ± 20.2 (80.3–194.6)	125.1 ± 22.3 (76.2–188.9)	0.24

Values are reported as mean ± SD (range). Statistical differences assessed by paired *t* test.
Ao = aortic root dimension; AVS = anterior ventricular septum thickness; EDV vol = end-diastolic left ventricular volume; ESV vol = end-systolic left ventricular volume; LAD = left atrial dimension; LVDd = end-diastolic left ventricular cavity dimension; LVDs = end-systolic left ventricular cavity dimension; LVM/EDV = ratio of left ventricular mass to left ventricular volume; LVM index = left ventricular mass normalized to body surface area; PFW = posterior free wall thickness; RWT = relative wall thickness.

significant dimensional increase was found for left atrial dimensions in both men (38.6 ± 3.5 mm to 39.6 ± 2.9 mm; +3%; $p < 0.01$) and women (34.7 ± 2.6 mm to 36.6 ± 3.0 mm; +5%; $p < 0.007$). Mild increase of the aortic root dimension was observed in men (33.3 ± 2.1 mm to 34.0 ± 2.7 mm; +2%; $p < 0.01$) but not in women ($p = 0.70$).

Clinical findings. In the overall population of Olympic athletes, no cardiovascular symptoms or events occurred during the extended period of training and competition, and each performed exercise testing to ≥85% of predicted maximum heart rate without symptoms at the most recent evaluation. None of the athletes with abnormal repolarization patterns showed either clinical or morphologic evidence of cardiac disease.

However, 5 athletes had abnormal clinical findings, specified here. Two of these athletes had ventricular tachyarrhythmias observed at initial evaluation, including frequent premature ventricular beats with left bundle branch block pattern, 1 couplet, and 1 burst of nonsustained ventricular tachycardia (4 beats with R-R interval of 250 ms) observed on exercise testing in 1; and rare premature ventricular beats, rare couplets, and 1 short run of nonsustained ventricular tachycardia (3 beats with R-R interval 450 ms) recorded on 24-h ambulatory (Holter) ECG in the other. Echocardiography and cardiovascular magnetic resonance were negative for structural or functional cardiac abnormalities. The first, a 33-year-old male rower, who participated in 3 consecutive Olympic Games, remained asymptomatic and without recurrence of arrhythmias after successful radiofrequency ablation. The latter, a 32-year-old male cross-country skier, who participated in 2 consecutive Olympic Games, showed disappearance of the ventricular arrhythmias over the follow-up while undergoing periodical evaluations in our institute.

Three other athletes, including a 35-year-old male marathon runner, a 28-year-old male rower, and a 32-year-old female skier, showed T-wave inversion and ≥2-mm ST-segment depression on routine exercise ECG, at workloads of ≥300 watts, in the absence of cardiac symptoms. Presence of anomalous origin or course of coronary arteries and atherosclerotic coronary artery disease were excluded by computed tomography scan and/or coronary angiography and morphologic cardiac abnormalities by echocardiography and/or cardiovascular magnetic resonance. These 3 athletes were eventually cleared for competition and have remained asymptomatic over the follow-up period encompassing ≥2 Olympic Games.

Comparison group. Demographic and cardiac dimensional and functional characteristics of the 97 athletes comprising the comparison group, who participated in 1 Olympic Games, trained over the subsequent 4 years, but eventually were not selected for the next Olympic Games, are reported in Table 3. None of these athletes reported cardiac symptoms or incurred clinical events. No changes were evident in cardiac dimensions, including LV volume and LV mass index, LV systolic function (ejection fraction:

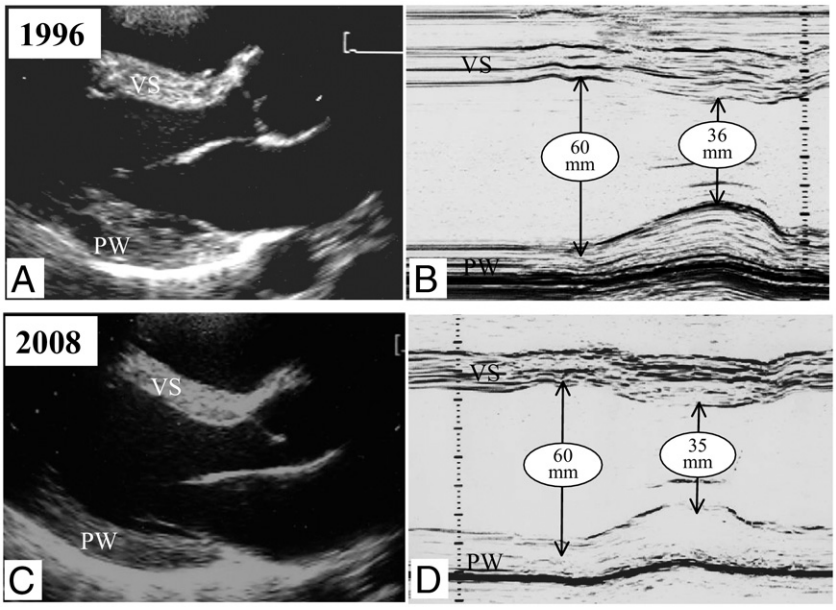

Figure 1 **Serial Echocardiographic Views of the LV in an Elite Italian Marathon Runner**

Serial echocardiograms obtained from an elite male marathon runner, at the time of selection for participation in the 1996 Olympic Games **(A, B)** at age 24 years and at the most recent evaluation before participation in the 2008 Olympic Games **(C, D)** at age 36 years. **(A)** Two-dimensional parasternal long-axis view; **(B)** M-mode tracing; transverse left ventricular (LV) cavity diameter is 60 mm in end-diastole and 36 mm in end-systole. **(C)** Parasternal long-axis view; **(D)** M-mode tracing of the LV; transverse LV cavity diameter remains 60 mm in end-diastole and 35 mm in end-systole. PW = posterior wall; VS = ventricular septum.

63 ± 6% to 64 ± 6%; p = 0.39), blood pressure/systolic volume index ratio, or LV filling pattern as assessed by transmitral Doppler echocardiography.

Discussion

The long-term clinical consequences of cardiac remodeling in athletes have periodically been questioned and remain largely unresolved. Most studies describing cardiac remodeling in elite athletes have been cross-sectional in design and underpowered to clarify the clinical implications and long-term consequences of the "athlete's heart" (10,11,23–28). In the present study we have addressed this question, by assessing a unique population of highly trained, particularly elite, and largely world-class athletes, who had been engaged in endurance training and competition at the highest level uninterruptedly for more than 8 years on average,

Table 3 **Demographic, Dimensional, and Cardiac Functional Characteristics of the 97 Athletes Comprising the Comparison Group Who Participated in Only 1 Olympic Games**

	Initial Evaluation	Most Recent Evaluation	p Value
Age (yrs)	23.1 ± 4.2 (15–34)	27.1 ± 4.4 (19–38)	0.001
BSA (m^2)	1.86 ± 0.20 (1.48–2.26)	1.86 ± 0.21 (1.48–2.27)	0.84
AVS (mm)	9.9 ± 1.2 (7–13)	9.9 ± 1.2 (7–13)	1.0
PFW (mm)	9.7 ± 1.1 (7–12)	9.7 ± 1.2 (7–13)	0.94
LVDd (mm)	53.9 ± 4.4 (45–64)	54.3 ± 4.1 (46–65)	0.47
LV volume (ml)	142.1 ± 26.3 (92–208)	144.6 ± 25.2 (97–216)	0.51
LVM index (g/m^2)	108.8 ± 20.6 (57.4–157.1)	109.9 ± 22.4 (60.6–166.9)	0.73
LA (mm)	36.1 ± 4.0 (25–38)	36.8 ± 3.7 (29–48)	0.18
EF (%)	63.4 ± 5.8 (50–77)	64.2 ± 6.0 (51–81)	0.39
E wave (mm/s)	79.7 ± 16.4 (46–131)	80.3 ± 15.7 (47–127)	0.79
A wave (mm/s)	39.9 ± 8.8 (24–82)	42.2 ± 8.5 (23–66)	0.08
E/A ratio	2.1 ± 0.6 (1.1–3.6)	1.9 ± 0.5 (1.0–3.3)	0.12
SBP/ESV	4.4 ± 1.0 (1.9–7.6)	4.7 ± 1.3 (2.5–8.6)	0.21

Values are reported as mean ± SD (range).

A wave = peak late (atrial) diastolic filling velocity; AVS = anterior ventricular septum; BSA = body surface area; E wave = peak early diastolic filling velocity; EF = ejection fraction; LA = left atrium; LV = left ventricular; LVDd = left ventricular end-diastolic diameter; LVM index = left ventricular mass normalized to body surface area; PFW = posterior free wall; SBP/ESV = ratio of systolic blood pressure to normalized end-systolic volume.

JACC Vol. 55, No. 15, 2010
April 13, 2010:1619–25

including participation in up to 5 consecutive Olympic Games. We regarded this unique athletic population, comprising top competitors in endurance disciplines (known to be associated with the most marked cardiac remodeling) (10,11), as an appropriate model to assess the possibility of adverse cardiovascular consequences of a particularly intensive and chronic exercise training.

The most important finding of our study was the demonstration that, despite exposure to the most vigorous training schedule required to compete at the highest (i.e., Olympic) level, prolonged for an substantial period of time, no cardiovascular events occurred; nor was deterioration in global LV systolic function or wall motion abnormalities evident in these athletes. Furthermore, we did not observe changes in LV systolic function or incidence of cardiovascular abnormalities in our comparison group of athletes who competed in 1 Olympic Games but failed to qualify for the subsequent Games 4 years later.

Therefore, contrary to a previous investigation showing LV dysfunction in professional cyclists participating in Tour de France races (7), our data show that strenuous and chronic exercise training is not responsible, per se, for deterioration of LV systolic function in healthy athletes. Furthermore, we believe that the systematic anti-doping policy implemented in recent years had a major impact in preventing potential harmful effects of doping, including possible LV dysfunction. Definitely, the Italian Olympic athletes included in this study had been cleared at serial doping controls in the periods preceding and during the Olympic Games. Finally, despite certain morphologic similarities of cardiac remodeling between our chronically trained athletes and patients with dilated cardiomyopathies, none of our athletes with marked LV cavity enlargement experienced cardiac symptoms or impaired physical performance over the long-term observation period of up to 17 years (15).

As an incidental finding, we observed a modest (although statistically significant) increase in left atrial dimension in our athletes. This finding was not entirely unexpected, because we have previously reported enlargement of the left atrium as an effect of long-term, intensive training in competitive athletes (28). Such atrial remodeling as well as LV cavity enlargement likely represents a physiologic adaptation to increased preload associated with chronic exercise and ultimately a mechanism by which LV filling is normal in trained athletes. Indeed, the physiologic nature of the atrial enlargement is underscored by the preserved systolic emptying (shown by normal Doppler-flow pattern) and the absence of atrial arrhythmias. However, given the youthful age of our athletes at most recent assessment, we cannot exclude the possibility that enlargement of the left atrium might be of clinical relevance over the long-term period and when athletes achieve more advanced age.

We wish to underscore that the population of elite athletes who comprise the present investigation is unique by virtue of their particularly prolonged and successful commitment to an intensive training schedule over many years. Because of the very select nature of this subset of athletes, however, results derived from the present investigation might not be directly comparable to the natural course of cardiac functional changes in all competitive athletes undergoing chronic and intensive conditioning.

In conclusion, this study of uniquely trained Olympic athletes shows that intensive conditioning over many years was not associated with inappropriate LV remodeling or dysfunction or with adverse clinical events, onset of symptoms, or new diagnosis of cardiomyopathies. Our findings support the benign nature of physiologic heart remodeling in trained athletes and the safety of long-term and intense sports participation, even at the highest competitive level.

Reprint requests and correspondence: Dr. Antonio Pelliccia, Institute of Sports Medicine and Science, Largo Piero Gabrielli 1, 00197 Rome, Italy. E-mail: antonio.pelliccia@coni.it.

REFERENCES

1. Douglas PS, O'Toole ML, Hiller WD, Hackney K, Reichek N. Cardiac fatigue after prolonged exercise. Circulation 1987;76:1206–13.
2. Whyte GP, George K, Sharma S, et al. Cardiac fatigue following prolonged endurance exercise of differing distances. Med Sci Sports Exerc 2000;32:1067–72.
3. Neilan TG, Januzzi JL, Lee-Lewandroski E, Ton-Nu T, Yoerger DM, Jassal DS, et al. Myocardial injury and ventricular dysfunction related to training levels among nonelite participants in the Boston marathon. Circulation 2006;114:2325–33.
4. Rifai N, Douglas PS, O'Toole M, Rimm E, Ginsburg GS. Cardiac troponin T and I, echocardiographic wall motion analyses and ejection fractions in athletes participating in the Hawaii Ironman Triathlon. Am J Cardiol 1999;83:1085–9.
5. Pelliccia A, Maron BJ, De Luca R, Di Paolo FM, Spataro A, Culasso F. Remodeling of left ventricular hypertrophy in elite athletes after long-term deconditioning. Circulation 2002;105:944–9.
6. Nishimura T, Yamada Y, Kawai C. Echocardiographic evaluation of long-term effects of exercise on left ventricular hypertrophy and function in professional bicyclists. Circulation 1980;61:832–40.
7. Abergel E, Chatellier G, Hagege AA, et al. Serial left ventricular adaptations in world-class professional cyclists: implications for disease screening and follow-up. J Am Coll Cardiol 2004;44:144–9.
8. Ector J, Ganame J, van der Merwe N, et al. Reduced right ventricular ejection fraction in endurance athletes presenting with ventricular arrhythmias: a quantitative angiographic assessment. Eur Heart J 2007;28:345–53.
9. Pelliccia A, Maron BJ. Preparticipation cardiovascular evaluation of the competitive athlete: perspectives from the 30-year Italian experience. Am J Cardiol 1995;75:827–9.
10. 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.
11. Pelliccia A, Culasso F, Di Paolo FM, Maron BJ. Physiologic left ventricular cavity dilatation in elite athletes. Ann Intern Med 1999;130:23–31.
12. Maron BJ, Pelliccia A, Spirito P. Cardiac disease in young trained athletes: insights into methods for distinguishing athlete's heart from structural heart disease, with particular emphasis on hypertrophic cardiomyopathy. Circulation 1995;91:1596–601.
13. Maron BJ. Distinguishing hypertrophic cardiomyopathy from athlete's heart: a clinical problem of increasing magnitude and significance. Heart 2005;91:1380–2.
14. Gavazzi A, De Maria R, Renosto G, et al. The spectrum of left ventricular size in dilated cardiomyopathy: clinical correlates and prognostic implications. Am Heart J 1993;125:410–22.

15. Pelliccia A, Di Paolo FM, Quattrini F, et al. Outcomes in athletes with marked ECG repolarization abnormalities. N Engl J Med 2008;358:152–61.
16. 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.
17. Schiller NB, Shah PM, Crawford M, et al., American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. J Am Soc Echocardiogr 1989;2:358–67.
18. Devereux RB, Alonso DR, Lutas EM, et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol 1986;57:450–8.
19. Mercier JC, Sessa TGD, Jarmakani JM, et al. Two-dimensional echocardiographic assessment of left ventricular volumes and ejection fraction in children. Circulation 1982;65:962–9.
20. Ginzton LE, Laks MM, Brizendine M, Conant R, Mena I. Noninvasive measurement of the rest and exercise peak systolic pressure/end-systolic volume ratio: a sensitive two-dimensional echocardiographic indicator of left ventricular function. J Am Coll Cardiol 1984;4:509–16.
21. Spirito P, Maron BJ. Doppler echocardiography for assessing left ventricular diastolic function. Ann Intern Med 1988;109:122–6.
22. Armitage P, Berry G. Statistical Methods in Medical Research. Oxford: McGraw-Hill, 1998:157–75.
23. Huston P, Puffer JC, Rodney WM. The athletic heart syndrome. N Engl J Med 1985;315:24–32.
24. Pelliccia A, Maron BJ, Culasso F, Spataro A, Caselli G. Athlete's heart in women: echocardiographic characterization of highly trained elite female athletes. JAMA 1996;276:211–5.
25. Pluim BM, Zwinderman AH, van der Laarse A, van der Wall EE. The athlete's heart: a meta-analysis of cardiac structure and function. Circulation 2000;101:336–44.
26. Scharhag J, Schneider G, Urhausen A, Rochette V, Kramann B, Kindermann W. Athlete's heart: right and left ventricular mass and function in male endurance athletes and untrained individuals determined by magnetic resonance imaging. J Am Coll Cardiol 2002;40:1856–63.
27. Maron BJ, Pelliccia A. The heart of trained athletes. Cardiac remodeling and the risks of sports, including sudden death. Circulation 2006;114:1633–44.
28. Pelliccia A, Maron BJ, Di Paolo FM, et al. Prevalence and clinical significance of left atrial remodeling in competitive athletes. J Am Coll Cardiol 2005;46:690–6.

Key Words: intense athletic training ■ left ventricular function ■ long-term follow-up ■ Olympic athletes.

APPENDIX

For the supplementary Methods section, please see the online version of this article.