Prevalence and Clinical Significance of Aortic Root Dilation in Highly Trained Competitive Athletes

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Background—Few data are available that address the impact of athletic training on aortic root size. We investigated the distribution, determinants, and clinical significance of aortic root dimension in a large population of highly trained athletes.

Methods and Results—Transverse aortic dimensions were assessed in 2317 athletes (56% male), free of cardiovascular disease, aged 24.8±6.1 (range, 9 to 59) years, engaged in 28 sports disciplines (28% participated in Olympic Games). In males, aortic root was 32.2±2.7 mm (range, 23 to 44; 99th percentile=40 mm); in females, aortic root was 27.5±2.6 mm (range, 20 to 36; 99th percentile=34 mm). Aortic root was enlarged ≥40 mm in 17 male (1.3%) and ≥34 mm in 10 female (0.9%) subjects. Over an 8-year follow-up period, aortic dimension increased in these male athletes (40.9±1.3 to 42.9±3.6 mm; P<0.01) and dilated substantially (to 50, 50, and 48 mm) in 3, after 15 to 17 years of follow-up, in the absence of systemic disease. Aortic root did not increase significantly (34.9±0.9 to 35.4±2.1 mm; P=0.11) in female athletes. Multiple regression and covariance analysis showed that aortic dimension was largely explained by weight, height, left ventricular mass, and age (R²=0.63; P<0.001), with type of sports training having a significant but lower impact (P<0.003).

Conclusions—An aortic root dimension ≥40 mm in highly conditioned male athletes (and ≥34 mm in female athletes) is uncommon, is unlikely to represent the physiological consequence of exercise training, and is most likely an expression of a pathological condition, mandating close clinical surveillance. (Circulation. 2010;122:698-706.)

Key Words: aorta ■ competitive athletes ■ echocardiography ■ remodeling

Over the last 2 decades, several studies have reported morphological cardiac changes induced by athletic conditioning, including left ventricular (LV) and left atrial remodeling,1-5 relevant to the differential diagnosis of athlete’s heart from structural cardiac disease.6 Intuitively, enlargement of the aortic root may be expected to occur in response to the hemodynamic overload associated with the athletic lifestyle. At present, however, few data are available in regard to the impact of exercise training on aortic root dimension or whether enlargement of the aorta occurs as a physiological adaptation to chronic exercise.

Clinical Perspective on p 706

These issues are of clinical relevance given that the Bethesda Conference No. 36 recommendations7 for competitive athletes with aortic enlargement are based on an arbitrary dimensional cutoff of ≥40 mm, which is also used in clinical practice.8,9 Therefore, the present study was designed to investigate the upper normal limits of aortic remodeling associated with intense and chronic athletic training, as well as the prevalence and significance of aortic root dilatation in a large cohort of competitive athletes.

Methods

Study Population

The Institute of Sports Medicine and Science is a medical division of the Italian National Olympic Committee, responsible for the medical evaluation of members of the Italian national teams, from which Olympic athletes are selected. This program includes a cardiovascular evaluation with history, physical examination, 12-lead and exercise ECG, and echocardiography.10 During 1992–1995, 2362 consecutive athletes were evaluated in our institute and considered for inclusion in the study group. On the basis of clinical and echocardiographic assessment, 45 of these athletes showed structural cardiac abnormalities and were excluded. Therefore, the final study population comprised 2317 athletes free of cardiovascular disease: 1300 (56%) male and 1017 (44%) female. Of the 2317 athletes, 659 (28%) were elite, by participating in the Olympic Games and/or World Championships, and the remaining 1658 competed at a national or regional level. Athletes were engaged in a spectrum of 28 different disciplines. Males participated primar-
Echocardiography11); measurements were averaged from 3 consec-
tutive cycles (Figure 1). In athletes with enlarged aortic root (see
transverse aortic root dimension (ie, 34 mm). LA indicates left
atrium; Ao, aorta.

Definition of Aortic Enlargement
Because our primary focus was to determine the frequency with
which athletes demonstrate aortic enlargement compatible with
pathological conditions, we chose an arbitrary cutoff of the 99th
percentile in our study population of 2317 athletes, determined
separately for males and females.

Athletes identified at study entry with aortic root dimension >99th
percentile had cardiovascular, skeletal, and ocular investigation
in accordance with Ghent criteria for Marfan syndrome.14 Mitral valve
prolapse was diagnosed on the basis of echocardiographic evidence
in the parasternal long-axis plane of systolic displacement of
thickened mitral valve leaflets into the left atrium.15

Power Versus Endurance Athletes
We performed subgroup analysis by comparing aortic and other
cardiac dimensions in athletes engaged in power disciplines known
to be associated with transient increases in blood pressure and aortic
stress (eg, weight lifting, discus, javelin, shot putting, bobsledding,
and wrestling) (n = 65 males, 30 females) versus those in endurance
disciplines (eg, marathon running, rowing, canoeing, road cycling,
and swimming) (n = 174 males, 99 females) of similar age, height,
and body surface area.

Follow-Up
Athletes with aortic root dimension >99th percentile value had serial
clinical and echocardiographic reassessment over an extended period
of 8.5 years (range, 2 to 17; median, 12) in males and 8±7 years
(range, 1 to 17; median, 8) in females.

Control Athletes
For the purpose of comparison with those athletes demonstrating
aortic enlargement, we selected a subset of 115 athletes (71 males, 44
females) with normal aortic diameter (<99th percentile value)
evaluated in our institute during the same time period (1992–1995).
From the database of 2362 athletes, we selected every consecutive
athlete with normal aortic root dimension; those aged 15 or
under with follow-up duration >2 years were excluded.

The 71 normal male controls had similar age (24±3 versus 25±6
years; P = 0.12), height (188±8 versus 189±11 cm; P = 0.56), and
weight (86±10 versus 91±17 kg; P = 0.15) compared with males

Table 1. Demographics and Cardiac Dimensions in 2317
Trained Athletes According to Sex

<table>
<thead>
<tr>
<th></th>
<th>Males (n = 1300)</th>
<th>Females (n = 1017)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>24.8±6.1 (11 to 59)</td>
<td>22.6±6 (9 to 53)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>1.9±0.2 (1.3 to 2.7)</td>
<td>1.6±0.2 (1.0 to 2.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Aortic root, mm</td>
<td>32.2±2.7 (23 to 44)</td>
<td>27.6±2.6 (20 to 36)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Aortic root/BSA, mm/m²</td>
<td>16.5±1.5 (12 to 22)</td>
<td>16.9±1.7 (12 to 25)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AVS, mm</td>
<td>9.8±1.2 (7 to 16)</td>
<td>8.0±1.0 (5 to 12)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVd, mm</td>
<td>55.6±0.1 (42 to 70)</td>
<td>48.6±3.9 (38 to 66)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Left atrium, mm</td>
<td>36.8±4.0 (23 to 50)</td>
<td>32.1±3.6 (20 to 46)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV mass index, g/m²</td>
<td>108.6±0.6 (61 to 195)</td>
<td>79.4±16.7 (45 to 148)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Vmax, cm/s</td>
<td>109.6±19.6 (68 to 208)</td>
<td>108.5±16.8 (76 to 173)</td>
<td>0.154</td>
</tr>
<tr>
<td>Emax, cm/s</td>
<td>68.3±13.7 (33 to 117)</td>
<td>74.1±13.6 (35 to 123)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>EF slope, cm/s²</td>
<td>463±128 (115 to 990)</td>
<td>518±130 (125 to 999)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Amax, cm/s</td>
<td>31.3±8.7 (12 to 83)</td>
<td>31.9±9.4 (12 to 76)</td>
<td>0.112</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>2.3±0.8 (0.8 to 5.9)</td>
<td>2.5±0.8 (0.9 to 6.2)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are expressed as mean±SD (range). BSA indicates body surface area; AVS, anterior ventricular septum; LVd, LV end-diastolic diameter; Vmax, aortic peak flow velocity; Emax, early diastolic peak flow velocity; EF slope, deceleration time of the early diastolic peak flow velocity; Pmax, late diastolic (atrial) peak flow velocity; and E/A ratio, ratio of early to late diastolic peak flow velocities.

Echocardiography
Two-dimensional and Doppler echocardiographic studies were per-
formed with the use of commercially available instruments (Sonos
2500, Sonos 5500, and iE33, Philips). Images of the heart were
obtained in multiple cross-sectional planes with the use of standard
descan images, 2 to 3 cm distal to the aortic valve plane.

Other cardiac dimensions were assessed as reported previously.12

Aortic regurgitation was evaluated by continuous wave and
color-flow Doppler echocardiography according to the width of the
regurgitant aortic jet at the level of the aortic leaflets in the left
parasternal long-axis plane and also by the length of the regurgitant
jet in the apical 5-chamber view.12 Peak aortic flow velocity was
assessed by continuous wave Doppler in the apical views. Parame-
ters of LV filling were obtained with pulsed Doppler echocardiog-
raphy, as described previously.11
Aortic Root Dimensions

Distributions of aortic root dimensions within the cohort of 1300 male and 1017 female athletes are shown in Figure 2A and 2B.

In male athletes, mean aortic root was $32.2 \pm 2.7$ mm (range, 23 to 44), with 99th percentile $40$ mm. In female athletes, mean aortic root was $27.5 \pm 2.6$ mm (range, 20 to 36), with 99th percentile $34$ mm. Overall, the aortic root was $<40$ mm in 1283 males and was $\geq 40$ mm in only 17 (1.3%) (Figure 2A). The aortic root was $<34$ mm in 1007 female athletes and was $\geq 34$ mm in 10 (0.9%) (Figure 2B).

Normalized to body surface area, aortic root in males was $16.5 \pm 1.5$ mm/m² (range, 12.0 to 22.3), with 99th percentile $=20.7$; aortic root in females was $16.9 \pm 1.7$ mm/m² (range, 11.7 to 25.3), with 99th percentile $=21.4$. Other cardiac dimensions and derived indexes of systolic and diastolic LV function are reported in Table 1.

Determinants of Aortic Dimensions

In the overall athlete cohort, multiple regression analysis showed that aortic root dimension was associated with sex, age, height, weight, and LV mass ($R^2=0.64$), together accounting for $>60\%$ of the variability in aortic size (Table I in the online-only Data Supplement). In particular, height was closely correlated with aortic dimensions as assessed by linear regression analysis ($R^2=0.674$; $P<0.01$) (Figure 3). Covariance analysis and Wald test for each variable confirmed the statistical significance of LV mass, height, weight, age, and sex (for each, $P<0.001$) and also showed that sports participation had a significant, although lesser, impact ($P<0.003$) on the variability of aortic dimension (Table II in the online-only Data Supplement). Specifically, sports such as cycling ($P=0.01$), swimming ($P=0.01$), water polo ($P=0.02$), and basketball ($P=0.04$) showed a significant impact on aortic root dimension (Figure 4).

Power Versus Endurance Athletes

Aortic dimensions were larger in male endurance athletes compared with male power athletes ($33.2 \pm 2.2$ versus

Results

Statistical Analysis

Data are expressed as mean±SD. Differences between mean values were compared with the unpaired or paired Student $t$ test, as appropriate. A 2-tailed $P<0.05$ was considered statistically significant.

Multiple regression analysis was used to assess the impact that several variables (height, weight, age, systolic and diastolic blood pressure, LV mass) had on aortic dimension, and those significantly correlated were subsequently incorporated into the covariance analysis, together with sex and type of sport.

The impact of type of sport, adjusted for other covariates, was then calculated by the formula $I_{i}-I_{ref}$, where $I_i$ is the impact factor of the candidate sport and $I_{ref}$ is the impact factor of the reference sport (ie, shooting, in our population). Only sport disciplines with $>30$ athletes were included in the analysis. The statistical significance of each of the variables entered into the covariance analysis was analyzed by the Wald test.
31.7±2.2 mm; P<0.01). In addition, LV cavity dimension, wall thickness, and mass were greater in male endurance athletes. No significant differences were evident, however, for aortic root dimensions in female endurance versus female power athletes (28.9±2.5 versus 28.7±2.3 mm; P=0.67) (Table 2).

**Athletes With Aortic Enlargement**

**Males**

Seventeen male subjects had enlarged aortic root ≥40 mm; they were aged 25±7 years, were of substantial height (188±10 cm [74±4 inches]) and body surface area (2.17±0.25 m²), and competed most commonly in rowing/canoeing (n=5), basketball (n=3), and volleyball (n=2) (Table 3).

None of these 17 athletes satisfied the Ghent criteria for the diagnosis of Marfan syndrome, although 1 basketball player (height, 2.08 m [81.9 inches]) had an arm span/height ratio 1.08, and 2 others had positive thumb and/or wrist sign. No athlete had major chest deformities (pectus carinatum or excavatum), severe scoliosis, displacement of medial malleolus (pes planus), dislocated lens, or family history for Marfan syndrome or premature sudden cardiac death. Only 1 of the 17 had bicuspid aortic valve.

Fifteen (of the 17) male athletes with aortic root ≥40 mm at study entry had serial evaluations with echocardiography over a period of 8±5 years. Of these 15 athletes, 4 had 2 evaluations over a period of 2 to 5 years; 10 had 3 evaluations over 2 to 16 years, and 1 had 11 evaluations in 12 years. Of them, 8 remained athletically active, and 7 had terminated their athletic careers at the most recent evaluation. None experienced cardiac symptoms or incurred cardiovascular events. In these 15 athletes, mean aortic root dimensions increased significantly, from 40.9±1.3 to 42.9±3.6 mm (change of 5%; P<0.01). The increase was modest during the period they remained physically active (41.4±1.7 to 41.9±2.2 mm; P=0.04) but was more substantial after they terminated their athletic careers (41.9±2.2 to 45.1±4.1 mm; P=0.02) (3.2±2.9-mm increase). Similar dimensional increase was observed in the ascending aorta (35.3±2.3 to 37.1±3.8 mm; P<0.03) (Table 3 and Figure 5).

Among these 15 athletes, 2 former basketball players showed substantial aortic root dilatation, from 40 to 50 mm and from 42 to 50 mm, after 15 and 16 years (at 45 and 38 years of age), respectively. Both of these athletes had genetic testing negative for FBN1 mutations. A third athlete (a former elite canoeist) had aortic dimensional increase from 43 to 48 mm after a 17-year follow-up (at 35 years of age). None of...
Table 2. Demographics and Cardiac Dimensions in Power and Endurance Athletes According to Sex

<table>
<thead>
<tr>
<th></th>
<th>Male Athletes (n=124)</th>
<th>Female Athletes (n=99)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>26±4 (20 to 41)</td>
<td>25±4 (20 to 43)</td>
<td>0.79</td>
</tr>
<tr>
<td>Height, cm</td>
<td>178.5±8 (163 to 190)</td>
<td>180±7.5 (162 to 190)</td>
<td>0.07</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>1.97±0.18 (1.64 to 2.54)</td>
<td>1.94±0.12 (1.65 to 2.24)</td>
<td>0.07</td>
</tr>
<tr>
<td>HR, bpm</td>
<td>57±10 (31 to 84)</td>
<td>51±9 (34 to 80)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>124±9 (100 to 145)</td>
<td>120±10 (90 to 150)</td>
<td>0.02</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>78±9 (70 to 90)</td>
<td>75±7 (60 to 95)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Aorta, mm</td>
<td>31.7±2.2 (27 to 37)</td>
<td>33.2±2.2 (27 to 39)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Max WT, mm</td>
<td>9.5±0.9 (8 to 12)</td>
<td>10.7±1.1 (8 to 15)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVd, mm</td>
<td>54.0±3.3 (48 to 60)</td>
<td>59.0±3.8 (48 to 70)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVM/BSA, g/m²</td>
<td>97.1±12.8 (71.1 to 132.5)</td>
<td>129.2±21.1 (78.9 to 195.2)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LAD, mm</td>
<td>35.8±2.9 (30 to 41)</td>
<td>38.9±3.9 (30 to 48)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>FS, %</td>
<td>35.3±25 (25 to 44)</td>
<td>35.4±21 (20 to 50)</td>
<td>0.46</td>
</tr>
<tr>
<td>Vmax, cm/se</td>
<td>111±19.8 (78 to 168)</td>
<td>109±19.6 (68 to 190)</td>
<td>0.43</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>2.5±0.8 (1.3 to 4.4)</td>
<td>2.4±0.8 (0.9 to 5.5)</td>
<td>0.77</td>
</tr>
</tbody>
</table>

Values are expressed as mean±SD (range). BSA indicates body surface area; HR, heart rate; BP, blood pressure; Max WT, maximum LV wall thickness; LVd, LV end-diastolic diameter; LVM/BSA, LV mass normalized to body surface area; LAD, left atrial transverse diameter; FS, fractional shortening; Vmax, aortic peak flow velocity; and E/A ratio, ratio of early to late diastolic transmural peak flow velocities.

Interobserver Variability
Aortic dimension measured by observer 1 was 29.4±2.8 mm (range, 38 to 24 mm) and was 29.1±3.4 mm (range, 38 to 22 mm) as measured by observer 2. Mean interobserver difference was 0.25±1.28 mm (range, 4 to −2) or 1% of measured values. Concordance of measurements by the 2 observers, as assessed by intraclass correlation, was 93%.

Discussion
The aorta is subject to substantial hemodynamic load during exercise; for instance, systolic pressure exceeds 200 mm Hg during rowing and cycling.17, 18 and even higher values are attained during weight training.19, 20 It is plausible, therefore, that the aorta undergoes a remodeling as a consequence of intensive athletic conditioning.21

Furthermore, marked aortic dilatation occurs in the Marfan syndrome or bicuspid aortic valve, potentially leading to the lethal complications of aortic dissection and rupture.14,22,23 Indeed, famed athletes have died of aortic rupture, including Olympians Alexander Belov, Luciano Vendemini, and Flo Hyman.24–26 Risk for aortic dissection increases with the degree of aortic root enlargement,8,14,27 and, in fact, the Bethesda Conference No. 36 recommends close monitoring with echocardiography in athletes with aortic root dimension ≥40 mm and Marfan syndrome and disqualification from most intensive competitive sports.8

At present, few data are available regarding the occurrence, extent, and determinants of aortic dimensional changes in athletes.28 Therefore, in the present study we investigated the upper normal limits of aortic remodeling, as well as the prevalence and clinical significance of aortic dilatation in highly trained athletes.

We took advantage of the database of the Institute of Sports Medicine and Science, where competitive athletes are

these 3 athletes developed significant aortic regurgitation. Each has undergone serial clinical and echocardiographic surveillance, but no definitive decision regarding prophylactic surgical intervention has been made to date.

**Females**
Ten athletes had enlarged aortic root ≥34 mm, ranging from 34 to 36 mm. They were aged 25±4 years (range, 20 to 36 years), were tall (height, 177±1 cm; range, 160 to 200), and participated in basketball (n=3), athletics (n=2), cross-country skiing (n=2), swimming, tennis, and handball (each, n=1) (Table 4).

None of the 10 female athletes satisfied the Ghent criteria for the diagnosis of Marfan syndrome, although a 27-year-old basketball player had mitral valve prolapse and dorsal scoliosis. Seven had a follow-up of 1 to 17 years (mean, 8±7; median, 8) and, at the most recent evaluation, aortic root dimension had not changed significantly (34.8±0.9 to 35.4±2.1 mm; P=0.11) (Table 4). Ascending aorta dimension also remained unchanged (32.0±1.4 to 32.3±1.5 mm; P=0.23).

**Control Athletes**
The 71 male control athletes had serial clinical and echocardiographic evaluations over a 9±4-year period (range, 3 to 16; median, 10), during which they continued to engage in training and competition. During this period, the aortic root showed a mild increase of 0.7 mm, a 2% change (33.2±2.1 to 33.9±2.5 mm; P<0.01). At the most recent observation, the aortic dimension was still <40 mm in all male controls, except in 2 (in whom the aorta was 40 mm).

The 44 female controls had serial evaluations over an 8±3-year period (range, 2 to 18; median, 8) while engaged in training and competition. Aortic dimension did not change significantly (29.4±2.0 to 29.5±1.9 mm; P=0.77).
Of note, our study shows that in only a small subset of athletes (ie, 1.3% in males and 0.9% in females) was the aortic root enlarged over the threshold values of 40 mm in female athletes (and ≥34 mm in male athletes), with the largest increases occurring in midlife, well after their competitive athletic careers had ended. Furthermore, aortic dilatation was substantial in 20% of this subset, progressing into a range sufficient to justify consideration for prophylactic surgical treatment. These data also underscore our recommendation for serial clinical and echocardiographic evaluations when the aortic root exceeds the threshold of 40 mm in males.

The partition value we identified in male athletes (ie, 40 mm in males) corresponds to the threshold for aortic enlargement in the Bethesda Conference No. 36 recommendations and is widely implemented in current clinical practice. No threshold value for aortic dimension has been defined separately for female athletes. These values may be used as the upper normal limits for aortic root dimension in highly trained athletes for the purpose of preparticipation evaluation to exclude cardiovascular abnormalities.

The partition value that in our cohort corresponded to 40 mm in male athletes and 34 mm in female athletes shows that (independent of body size and type of sport) routine examination by echocardiography, and focused our attention on those subjects with aortic root dimensions exceeding the 99th percentile, a partition value that in our cohort corresponded to 40 mm in male athletes and 34 mm in female athletes. These values may be used as the upper normal limits for aortic root dimension in highly trained athletes for the purpose of preparticipation evaluation to exclude cardiovascular abnormalities.

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In contrast, similar close surveillance would not appear to be required in male athletes with normal aortic dimension, in whom only trivial dimensional changes were observed over long-term follow-up, not significantly different from those occurring in the general (untrained) population. In female athletes, the aortic dimensional increase was either absent or trivial over the long-term period of follow-up and consequently did not raise clinical concern.

The cause of the disproportionate aortic enlargement observed over time in a few male athletes remains elusive. None of the 3 athletes with greatly enlarged aorta at the most recent evaluation (potentially candidates for prophylactic surgery) showed clinical (or genotyping in 2) evidence of Marfan syndrome, bicuspid aortic valve, or any systemic disease. Therefore, this study demonstrates that aortic enlargement can occur occasionally in competitive athletes in the absence of apparent etiology and that large body size and intensive exercise training are unlikely to explain this substantial aortic remodeling. Recent investigations have suggested the possibility of a familial and inherited susceptibility to aortic

Table 4. Demographics and Cardiovascular Dimensions in Female Athletes With Enlarged Aortic Root at Initial and Most Recent Evaluation

<table>
<thead>
<tr>
<th>No.</th>
<th>Sport/Level</th>
<th>Age 1, y</th>
<th>Age 2, y</th>
<th>Prior Training, y</th>
<th>Follow-Up, y</th>
<th>T/U</th>
<th>Aortic Root Dimension 1, mm</th>
<th>Aortic Root Dimension 2, mm</th>
<th>Ascending Aorta 1, mm</th>
<th>Ascending Aorta 2, mm</th>
<th>LVMI 1, kg/m²</th>
<th>LVMI 2, kg/m²²</th>
<th>Cardiovascular Family History</th>
<th>PE</th>
<th>Clinical Findings</th>
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<td>1</td>
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<td>T</td>
<td>36</td>
<td>34</td>
<td>80</td>
<td>...</td>
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<td>Normal</td>
<td>None</td>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>Shot putting/Olympic</td>
<td>23</td>
<td>29</td>
<td>5</td>
<td>6</td>
<td>T</td>
<td>35</td>
<td>36</td>
<td>30</td>
<td>32</td>
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<td>95</td>
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<tr>
<td>3</td>
<td>Basketball/national</td>
<td>22</td>
<td>...</td>
<td>5</td>
<td>0</td>
<td>T</td>
<td>36</td>
<td>33</td>
<td>...</td>
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<td>0</td>
<td>T</td>
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<td>Normal</td>
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<td>5</td>
<td>Basketball/national</td>
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<td>U</td>
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<td>Normal</td>
</tr>
<tr>
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<td>51</td>
<td>18</td>
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<td>U</td>
<td>34</td>
<td>35</td>
<td>32</td>
<td>31</td>
<td>126</td>
<td>88</td>
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<tr>
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<td>42</td>
<td>7</td>
<td>18</td>
<td>U</td>
<td>34</td>
<td>35</td>
<td>30</td>
<td>30</td>
<td>100</td>
<td>88</td>
<td>None</td>
<td></td>
<td>Systolic murmur</td>
</tr>
</tbody>
</table>

| Mean  | 25.5 | 34.7* | 8.5  | 8.8  | 34.8 | 35.4 | 32.0 | 32.3 | 94.2 | 89.9 |
| SD    | ±4.6 | ±11.2 | ±2.1 | ±7.5 | ±0.9 | ±2.1 | ±1.4 | ±1.5 | ±15.7 | ±14.8 |

1 indicates initial evaluation; 2, most recent evaluation; prior training, years before first echocardiogram; T/U, trained/untrained at most recent evaluation; LVMI, LV mass index; PE, physical examination; MVP, mitral valve prolapse; and AF, paroxysmal atrial fibrillation.

*Age 1 vs age 2: P<0.02.
remodeling in apparently healthy individuals, but investigation of this possibility was well beyond the present study design.

Guidelines regarding competitive sports participation in individuals with enlarged aortic root in the absence of Marfan syndrome or bicuspid aortic valve are currently lacking. Although none of our athletes with diluted aorta incurred adverse clinical events, nevertheless, whether to restrict selectively these individuals from the most intensive competitive sports to reduce the risks associated with accelerated aortic enlargement should be appropriately addressed in future consensus recommendations.

Our analysis showed that sports per se had only limited influence on aortic dimension, including the most intense endurance disciplines (eg, cycling and swimming), which is different from our previous observations that such sports have substantial impact on LV wall and cavity dimensions. In addition, our findings demonstrated that power disciplines (eg, weight lifting, throwing events) have only trivial, if any, impact on aortic remodeling, consistent with our prior observation showing lack of significant LV chamber remodeling in power athletes. Such findings were somewhat unexpected given that power athletes had significantly higher blood pressures than endurance athletes, who paradoxically demonstrated, by comparison, greater aortic remodeling.

Finally, the results of the present investigation are derived from a selected population of Italian athletes and may not be directly comparable to other athletic populations with different ethnic backgrounds or engagement in different training and competitive sports activities.

In conclusion, aortic root enlargement (≥40 mm in males and ≥34 mm in females) is particularly uncommon in highly trained athletes and does not appear to be an expression of physiological cardiac remodeling in the context of athlete’s heart. Over long-term follow-up, enlarged aortas in male athletes showed further dimensional increases, which were occasionally marked, underscoring the importance of continued clinical surveillance with echocardiography.

Sources of Funding
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Disclosures
None.

References
The aorta is exposed to hemodynamic stress during endurance or power exercise training, which is associated with substantial increases in cardiac output and blood pressure. It is therefore plausible that significant aortic root remodeling may occur in highly trained athletes. The distribution of aortic dimension, prevalence, and clinical significance of aortic dilatation were investigated in 2317 highly trained athletes (56% male) free of cardiovascular disease. In males, aortic root was 23 to 44 mm (32.2 ± 2.7), with 99th percentile of 40 mm; in females, aortic root was 20 to 36 mm (27.5 ± 2.6), with 99th percentile of 34 mm. Only 1.3% of male and 0.9% of female athletes showed aortic root enlargement above the threshold values of 40 mm and 34 mm, respectively. Multivariable analysis showed that sports per se had only a limited influence on aortic dimension, whereas a more substantial impact was attributable to age, height, weight, and sex. In male athletes with enlarged aorta (> 40 mm), further dimensional increase (40.9 ± 1.3 to 42.9 ± 3.6 mm; P < 0.01) occurred over an 8±5-year follow-up, including 3 athletes in whom the aorta dilated substantially (to 50, 50, and 48 mm) after 15 to 17 years of follow-up in the absence of systemic disease, underscoring the importance of continued clinical surveillance. In female athletes, changes in aortic dimension over time were trivial and did not raise clinical concern. In conclusion, aortic root dimension ≥40 mm (and ≥34 mm in females) is very uncommon, occurring in only ~1% of a large population of highly trained athletes, and does not represent an expression of physiological cardiac remodeling (ie, athlete’s heart).
Prevalence and Clinical Significance of Aortic Root Dilation in Highly Trained Competitive Athletes

Antonio Pelliccia, Fernando M. Di Paolo, Elvira De Blasiis, Filippo M. Quattrini, Cataldo Pisicchio, Emanuele Guerra, Franco Culasso and Barry J. Maron

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Appendix Table 1. Multivariable regression analysis.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Std error</th>
<th>T</th>
<th>P</th>
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<tbody>
<tr>
<td>Gender</td>
<td>-1.328</td>
<td>0.137</td>
<td>-9.66</td>
<td>&lt;0.001</td>
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<tr>
<td>Age</td>
<td>0.090</td>
<td>0.008</td>
<td>11.07</td>
<td>&lt;0.001</td>
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<td>Height</td>
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<td>0.008</td>
<td>6.23</td>
<td>&lt;0.001</td>
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<tr>
<td>Weight</td>
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<td>0.007</td>
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<tr>
<td>LV Mass</td>
<td>0.020</td>
<td>0.001</td>
<td>16.13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SBP</td>
<td>-0.003</td>
<td>0.005</td>
<td>-0.58</td>
<td>0.56</td>
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<tr>
<td>DBP</td>
<td>0.012</td>
<td>0.008</td>
<td>1.38</td>
<td>0.17</td>
</tr>
</tbody>
</table>

**Abbreviations:** LV = Left Ventricle; SBP = systolic blood pressure; DBP = diastolic blood pressure
### Appendix Table 2. Wald tests of significance of fixed effects and covariates

<table>
<thead>
<tr>
<th>Variable</th>
<th>Degree of freedom</th>
<th>$\chi^2$</th>
<th>P</th>
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<tr>
<td>LV Mass</td>
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<td>Sport</td>
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</tr>
<tr>
<td>Weight</td>
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<td>&lt;0.001</td>
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<td>SBP</td>
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<tr>
<td>DBP</td>
<td>1</td>
<td>1.83</td>
<td>0.18</td>
</tr>
</tbody>
</table>

**Abbreviations:** LV = Left Ventricle; SBP = systolic blood pressure; DBP = diastolic blood pressure.