Reduction in left ventricular wall thickness after deconditioning in highly trained Olympic athletes

Barry J Maron, Antonio Pelliccia, Antonio Spataro, Maristella Granata

Abstract

Background—Clinical distinction between athlete’s heart and hypertrophic cardiomyopathy in a trained athlete is often difficult. In an effort to identify variables that may aid in this differential diagnosis, the effects of deconditioning on left ventricular wall thickness were assessed in six highly trained elite athletes who had competed in rowing or canoeing at the 1988 Seoul Olympic Games. Each of these athletes exhibited substantial ventricular septal thickening associated with training (13–15 mm) which resembled that of hypertrophic cardiomyopathy.

Methods—The athletes voluntarily reduced their training substantially for 6–34 weeks (mean 13) after the Olympic competition. Echocardiography was performed at peak training and also after deconditioning, and cardiac dimensions were assessed blindly.

Results—Maximum ventricular septal thickness was 13.8 (0.9) mm in the trained state and 10.5 (0.5) mm in the deconditioned state (p < 0.005) (change 15–33%).

Conclusions—The finding that deconditioning may be associated with a considerable reduction in ventricular septal thickness in elite athletes over short periods strongly suggests that these athletes had a physiological form of left ventricular hypertrophy induced by training. Such a reduction in wall thickness with deconditioning may help to distinguish between the physiological hypertrophy of athlete’s heart and primary pathological hypertrophy (for example, hypertrophic cardiomyopathy) in selected athletes with increased left ventricular wall thickness.

Patients and methods

SELECTION OF ATHLETES

Between 1986 and 1988, 947 elite athletes were evaluated at the Institute of Sports Science (Rome, Italy); 16 showed considerable physiological left ventricular wall thickening (≥13 mm). Six of these 16 were selected for the present study because each had qualified for or participated in the 1988 Seoul Olympic Games and had undergone a substantial period of deconditioning immediately after that competition. All those selected were men (aged 19–29 years, mean 24).

Five athletes were rowers (double or quadruple sculls) and one was a canoeist. At the Olympic Games three of the rowers won gold medals and the canoeist was a finalist in seventh place. None of these athletes had consistent increases in blood pressure or evidence of hyperthyroidism or use of anabolic steroids.

ECHOCARDIOGRAPHY

Cross sectional echocardiographic studies were performed with the athlete in a supine position with a commercially available Hewlett-Packard instrument (77020AC) and 3-5 MHz transducer. Images of the heart were obtained in multiple cross sectional planes and the extent and distribution of left ventricular hypertrophy were assessed as described elsewhere.24 M mode echocardiograms were derived from cross sectional images under direct anatomical visualisation and recorded at 100 mm per second.

In the parasternal short axis plane the left ventricle was divided into four segments: the anterior and posterior ventricular septum and the anterolateral and posterior free wall in both the proximal and distal portions of the ventricle. The diastolic left ventricular wall thickness in these regions was measured directly from the television monitor with the aid of calipers and a
calibration scale produced by the instrument. The thickness of the anterior ventricular septum was assessed by an integrated analysis of the cross sectional and M mode recordings. We made an effort to exclude overlying trabeculations when we measured the thickness of the ventricular septal and left ventricular free wall. Left ventricular cavity dimensions were measured according to published recommendations.⁹ The left ventricular mass was calculated from maximum end diastolic wall thickness and cavity dimension.¹⁰

Echocardiograms were initially obtained during peak training (12 intense training sessions per week) 8-40 weeks (mean 25) before the Olympic Games. After the Olympic competition each athlete had a complete deconditioning period of 6-34 weeks (mean 13) when they did not train in rowing. Their only physical activity was occasional recreational soccer or tennis. A second echocardiogram was subsequently recorded in five of the six athletes after a short period of light retraining involving rowing and low density conditioning in boats (six sessions per week for 6-8 weeks). In the sixth athlete, who retired from training and competition immediately after the Olympic Games, the second echocardiogram was obtained after 34 weeks of complete deconditioning.

Echocardiograms were interpreted by one observer (BJM) without knowledge of the identity of the athlete or the state of conditioning. To test interobserver variability, these measurements were compared with those made independently by another observer (AP). For the maximum left ventricular wall thickness, interobserver mean (SD) variability was 1-0 (0-7) mm. Intraobserver variability was tested by one observer (AP) from blinded assessments a year apart: variability was 1-0 (0-4) mm.

### Statistical Analyses
Data are expressed as mean values (SD). Mean differences in variables assessed at peak training and after deconditioning were analysed by Student’s paired t test; a two-tailed p value of <0.05 was regarded as statistically significant.
LV regression in Olympic athletes

<table>
<thead>
<tr>
<th>Blood pressure (mm Hg)</th>
<th>ECG voltage score</th>
<th>Duration deconditioning (wk)</th>
<th>Sport</th>
<th>Olympic achievement</th>
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<tbody>
<tr>
<td>Pk De</td>
<td>Pk De</td>
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</tr>
<tr>
<td>140/80 120/70</td>
<td>67 66</td>
<td>141 120 13</td>
<td>Rowing</td>
<td>Gold medal</td>
</tr>
<tr>
<td>125/80 140/90</td>
<td>48 50</td>
<td>159 166 8</td>
<td>Rowing</td>
<td>Gold medal</td>
</tr>
<tr>
<td>120/70 130/80</td>
<td>47 49</td>
<td>207 173 6</td>
<td>Rowing</td>
<td></td>
</tr>
<tr>
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<td>166 112 34</td>
<td>Canoeing</td>
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<tr>
<td>140/80 140/90</td>
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<td>159 124 8</td>
<td>Rowing</td>
<td>Gold medal</td>
</tr>
<tr>
<td>135/80 140/90</td>
<td>55 58</td>
<td>128 110 8</td>
<td>Rowing</td>
<td>Gold medal</td>
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| NS | <0.05 | <0.05 |

<table>
<thead>
<tr>
<th>LV mass (g)</th>
<th>A</th>
<th>B</th>
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<tr>
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<td>p &lt; 0.01</td>
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<table>
<thead>
<tr>
<th>A</th>
<th>B</th>
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<tbody>
<tr>
<td>Maximum LV wall thickness (mm)</td>
<td>LV mass (g)</td>
</tr>
<tr>
<td>Trained</td>
<td>Deconditioned</td>
</tr>
</tbody>
</table>

![Figure 1](image1.png) Changes in left ventricular wall thickness (A) and mass (B) associated with deconditioning in six Olympic athletes. Open symbols represent the athlete who retired from training and competition after the 1988 Olympic Games and had been deconditioned for 34 weeks at the time of the most recent echocardiographic study.

![Figure 2](image2.png) Serial M mode echocardiograms just below mitral valve level recorded in a 26 year old Olympic rower at peak training (A) and after more than eight months of complete deconditioning (B). The ventricular septal (VS) thickness had decreased from 15 to 10 mm. In addition there are also small decreases in the posterior free wall (PW) thickness and left ventricular end diastolic cavity dimension (LVED). The large calibration marks are 1 cm apart.

striking decrease in wall thickness. This feature of the study design may also partly explain why left ventricular cavity size measured after deconditioning was not significantly different from peak training values. These findings resemble those reported by others who studied cardiac dimensions and maximum oxygen uptake in athletes after a reduction or short-term cessation in training.

The findings of this study are relevant to the differential diagnosis of athlete’s heart and hypertrophic cardiomyopathy. The importance of this diagnostic distinction is underlined by the fact that athlete’s heart is characterised by physiologically induced left ventricular hypertrophy, while hypertrophic cardiomyopathy is probably the most common cause of sudden death in young competitive athletes. In highly trained athletes such as ours the thickness of the septum (13-15 mm) is similar to that in patients with mild structural expressions of hypertrophic cardiomyopathy. Because we found that left ventricular wall thickness was considerably reduced after deconditioning, hypertrophy in our athletes was likely to be physiological and related to training. If wall thickness had not been reduced by deconditioning a static pathological process such as hypertrophic cardiomyopathy would have been more likely. Consequently, a brief period of forced deconditioning combined with serial echocardiography may be useful in distinguishing between physiological and pathological left ventricular hypertrophy in selected athletes with absolute increases in ventricular septal thickness. We should point out that this diagnostic distinction could also be made in other ways, including identification of the familial occurrence of hypertrophic cardio-
myopathy, a normal Doppler left ventricular filling waveform, or differences in left ventricular end diastolic cavity dimension or ultrasonic myocardial reflectivity (ie, backscatter signal).

Previous studies of serial echocardiographic studies in trained athletes also showed rapid changes (within 4–8 weeks) in calculated left ventricular mass in response to conditioning and deconditioning. However, these earlier studies differ from the present investigation in certain important respects. For example, the previously reported athletes did not show an absolute increase in left ventricular wall thickness at peak training and the observed changes associated with deconditioning were virtually confined to alterations in end diastolic cavity dimension. Furthermore, the earlier studies of deconditioning did not independently test the interobserver variability of the echocardiographic measurements, nor were assessments of cardiac dimensions before and after deconditioning always made in a rigorously blinded fashion. Therefore, the data in the present study show for the first time that increased left ventricular wall thickness associated with athletic training may be altered substantially by deconditioning.

We thank Roberto Colli and Gianpietro Michelangelo for their substantial contributions.

This work was partly supported by the Italian National Olympic Committee related to work of the National Council for Research project FATMA.

1 Maron BJ. Structural features of the athlete’s heart as defined by echocardiography. J Am Coll Cardiol 1986;7:190–203.