Cardiac adaptation to training and decreased training loads in endurance athletes: a systematic review

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Introduction: Changes in echocardiographic standard measurements as a consequence of training and detraining in elite or subelite athletes have not been comprehensively studied.

Methods: We identified 200 potentially relevant studies from 1966 to June 2006 and eliminated 187 studies that did not fulfil the objectives of the study. We identified 13 studies with echocardiographic assessment of heart adaptation following variation in training loads in elite or subelite endurance-trained athletes. We performed a meta-analysis by studying the changes in the left ventricular end-diastolic (LVEDD), left ventricular end-systolic (LVESD), left ventricular posterior wall (LVPW) and interventricular septum thickness (IVST) dimensions induced by training.

Results: A significant positive overall effect size on echocardiographic outcomes was found following training, using the fixed effect model on LVPW, LVEDD, LVESD and IVST. LVPW and LVEDD were significantly higher following training.

Conclusion: Studies reported an increase in LVEDD and LVPW, following endurance training. However, the heterogeneity of the studies and the sensitivity of echocardiography technique can be two reasons, for which the results do not allow to state unequivocally that the adaptation to endurance training of highly trained hearts stems from increments of diastolic diameter of the left ventricle and lateral wall of the left ventricle (LVPW).

Keywords: training/detraining/cardiac dimensions/echocardiography
Introduction

Echocardiography has been used for more than 30 years to evaluate training-induced cardiac adaptation in athletes.\textsuperscript{1–6} These studies confirmed the original concepts of cardiac chambers enlargement and hypertrophy of Henschen.\textsuperscript{7} The increase in the thickness of the posterior wall of the left ventricle and that of the interventricular septum, and the maintenance of the physiological relationship between septum and posterior wall, indicate physiological hypertrophy.\textsuperscript{6,8} This relationship does not change with training versus detraining state.

Relatively few studies focused on the adaptation of cardiac dimension from the perspective of physical training, aiming instead to characterize the physiological adaptation of the cardiac muscle in cardiomyopathies. We used a meta-analytical approach to review the results of studies that have investigated the cardiac changes taking place as a consequence of athletic training.

Materials and methods

Identification of studies and inclusion criteria

We searched two electronic databases (Medline and Sports Discus) from 1966 to June 2006. Two reviewers independently reviewed the literature searches, selected the articles and extracted data. We searched for all reports of the training effect on heart structure in athletes with the following restrictive terms: (left ventricular mass OR left ventricular dimensions OR left ventricular OR echocardiographic dimensions OR echocardiographic OR cardiac dimensions OR cardiac structure) AND (Athletes OR Swimm*OR Cycli*OR Runn*OR rowers OR Oarsmen OR conditioning OR deconditioning OR Training) with limits ‘In human’, ‘Title/Abstract’ by age ‘Adolescent: 13–18 years, Adult: 19–44 years, Middle Aged + Aged: 45 + years’. We identified potentially relevant studies and selected reports for inclusion in the review (Fig. 1).

Data extraction

The following data were extracted and entered into a standardized electronic form: number of athletes included, age and echocardiographic outcomes for trained (T) and detrained (D) states. The echocardiographic outcome measures considered were left ventricular end-diastolic (LVEDD), left ventricular end-systolic (LVESD), left ventricular posterior wall (LVPW) and interventricular septum thickness.
(IVST) dimensions. The data of the study by Snoeckx et al.\textsuperscript{9,10} were obtained from the figures, because the authors did not report these values in the tables. We contacted one author,\textsuperscript{11} as the echocardiography dimension data were expressed in volume units. The author granted us access to use his raw data. In this way, we transformed the volume data into linear units for echocardiographic outcomes for each of the studied athletes. To solve the equation by Teichholz et al.\textsuperscript{12} (equation 1), the diameters of the left ventricle were obtained in two moments of the heart cycle using the program Matlab v 7.1.0.246 (MathWorks, Inc., Natick, MA, USA) to solve equation (2)\textsuperscript{1}

\[
V = \left(\frac{7.0}{2.4 + d}\right)d^3
\]  \hspace{1cm} (1)
where \( V \) is the volume in millilitres and \( d \) is the diameter in millimetres

\[
-7d^3 + Vd + 2.4V = 0
\]

(2)

Statistics

We used the kappa statistics to assess agreement between two reviewers. The effect size was computed as the mean difference (detraining minus training) in the changes for each of echocardiography outcome dimensions. A negative effect size implies an increase in the cardiac dimension from training. To calculate the overall effect size, each study was weighted by the reciprocal of its variance. We analysed heterogeneity between studies by using the \( Q \)-test.

We used a fixed effect model for analysis. If this evidenced a statistically significant effect size, the analyses were repeated using a random effect model.

A funnel plot was drawn, and asymmetry was measured to assess the possible influence of biases. The intercept of linear regression, where the effect size divided by the standard error is regressed against the reciprocal of the standard error, provides a measure of asymmetry. To quantify the size of the effect, we calculated weighted mean and standard deviation between training and detraining periods for each outcome measure when the relevant data were available. Analyses were conducted using Comprehensive Meta-analysis® (shareware version) (www.meta-analysis.com) and significance was set at \( \alpha = 0.05 \).

Results

We identified 13 publications with echocardiographic assessment of heart adaptation to following variation in training loads (two groups trained and detrained) in the elite or subelite endurance-trained athletes (Table 1). These studies included at least one of the following outcome measures: LVEDD, LVESD, LVPW and IVST.

Figures 2 and 3 show the descriptive statistics for the two echocardiographic outcomes that showed the greatest significant differences between training and detraining (T and D).

A significant positive overall effect size on echocardiographic outcomes was found with training using the fixed effect model: LVPW, LVEDD, LVESD and IVST. LVPW and LVEDD were significantly higher \((P < 0.05)\). In 9 and 12 studies, respectively, LVPW \((-0.162; -2.364)\) and LVEDD \((-0.724; -10.682)\) were significantly higher in the training state \((P < 0.05)\). The standard mean differences for IVST \((-0.259; -3.776)\) were also significantly higher in the training state in nine studies. LVESD \((0.006; 0.714)\) were significantly increased in four studies.
The overall effect size at 95% confidence interval was significant in the fixed effect model, but the studies showed strong heterogeneity for LVEDD \( (Q = 61.015) \), LVESD \( (Q = 32.311) \), LVPW \( (Q = 178.526) \) and IVST \( (Q = 84.657) \) data. High values of \( Q \) did not allow to perform a random effect analysis. When we used a random effect model, we found a significant and positive effect for LVEDD, LVPW and IVST. To quantify the size of the effect, we analysed the weighted means by variance mean difference and standard deviation between detraining and training for each outcome. The results are given in Figures 2 and 3 for LVPW and LVEDD, respectively.

### Discussion

The studies conducted in humans on cardiac adaptations and their effect on aerobic power \( (V/O_2) \) following endurance training confirm...
Fig. 2 Results for LVPW between trained and detrained states.
Fig. 3 Results for LVEDD between trained and detrained states.

<table>
<thead>
<tr>
<th>Study</th>
<th>Standard mean differences (fixed at 95% CI)</th>
<th>Weight (%)</th>
<th>Standard mean differences (fixed at 95% CI)</th>
<th>Z-value</th>
<th>P-value</th>
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</thead>
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<tr>
<td>Maron et al.¹²</td>
<td></td>
<td>4.09</td>
<td>−0.500</td>
<td>−1.491</td>
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</tr>
<tr>
<td>Ehsani et al.¹¹</td>
<td></td>
<td>2.04</td>
<td>2.000</td>
<td>−4.216</td>
<td>0.000</td>
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<tr>
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<td></td>
<td>1.75</td>
<td>−1.806</td>
<td>−3.521</td>
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<tr>
<td>Valdés et al.²⁷</td>
<td></td>
<td>8.18</td>
<td>−1.000</td>
<td>−4.216</td>
<td>0.000</td>
</tr>
<tr>
<td>Legaz Arrese¹¹</td>
<td></td>
<td>19.79</td>
<td>−1.250</td>
<td>−8.201</td>
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<td>Shaphiro et al.</td>
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<td>7.25</td>
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<td>Martin et al.³³</td>
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<td>1.93</td>
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<td>Lamont²⁶</td>
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<td>8.78</td>
<td>−0.667</td>
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<td>0.004</td>
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<td>1.000</td>
</tr>
<tr>
<td>Fagard et al.³⁵</td>
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<td>9.20</td>
<td>0.000</td>
<td>0.000</td>
<td>1.000</td>
</tr>
<tr>
<td>Snoeckx et al.³⁵</td>
<td></td>
<td>11.27</td>
<td>−0.200</td>
<td>−0.990</td>
<td>0.322</td>
</tr>
<tr>
<td>Snoeckx et al.³⁰</td>
<td></td>
<td>10.90</td>
<td>0.500</td>
<td>−2.434</td>
<td>0.015</td>
</tr>
<tr>
<td>Total (95% CI)</td>
<td></td>
<td>100</td>
<td>−0.724</td>
<td>−10.682</td>
<td>0.000</td>
</tr>
</tbody>
</table>
the results in animals\(^{13–20}\) that endurance training exerts a beneficial effect on the heart, particularly in sedentary subjects. However, this effect is more difficult to study in athletes, who exhibit a high level of cardiac adaptation, who have trained for much longer and in whom selection and genetic bias may exist.

The present study showed that echocardiographic variables are influenced by endurance training. The increase in the cardiac dimension as a consequence of training results both in enlargement (LVEDD) and in hypertrophy (LVPW). However, the results from various studies are not homogeneous. Indeed, the weighted heterogeneity of the echocardiographic outcomes is a considerable limitation of the review. Even by eliminating those studies with a high standard deviation, it was not possible to decrease the value of \(Q\). In contrast, there was great heterogeneity in the various studies regarding the characteristics of the training and detraining: this may lead to confusing results. For example, although Ehsani \textit{et al.}\(^{21}\) have demonstrated similar decreases in the LVEDD and LVPW, Maron \textit{et al.}\(^{22}\) have not detected a symmetrical regression of these two variables. These authors report the same changes in six Olympic athletes when they stopped training for \(6–34\) weeks (mean \(13\) weeks): LVPW decreased from \(13.8\) to \(10.5\) mm (24% change) and LVEDD practically did not change from \(57.8\) mm when trained to \(57.1\) mm when detrained (1.21% change).

The discrepancies in the effects of training on echocardiographic outcomes may be at least partially explained by the methodology of the studies and the resolution power of echocardiography.

\textbf{Methodology}

In some studies, the period of inactivity was relatively long (3–12 weeks).\(^{21–23}\) In other studies, the athletes underwent echocardiography at different stages of their training season,\(^{10,24–28}\) or over several seasons,\(^{11}\) so that their grade of inactivity was relatively difficult to assess, if at all present, as elite athletes only have a short period of relative rest after the end of the sports season (3–4 weeks). As the detraining effect on the heart manifests at about that time, the results could be interpreted in an incorrect fashion.

The training load at the time of echocardiography is often not characterized in sufficient details. In some studies,\(^{11,21,22,24,27}\) the two main characteristics of training, intensity and volume, are not described fully. The details refer to the daily or weekly frequency, total volume of training or years of training, but neither training methods nor intensities are described. In other studies,\(^{9,10,25,26,28}\) the type of training is indicated, but not its intensity.
Most studies have investigated only short-term training and detraining. Only Wieling et al.\textsuperscript{28} and Snoeckx et al.\textsuperscript{9,10} carried out long-term studies. Wieling et al.\textsuperscript{28} measured the echocardiographic outcomes in five training moments: initial, 2 weeks and 1, 2, 4 and 7 months. They observed significant differences for LVEDD, but not for LVPW. Likewise, Snoeckx et al.\textsuperscript{9,10} analysed the echocardiographic outcomes in four periods of training. Although they did not analyse their results statistically, LVPW in long-distance runners increases in the first three periods of training. The time course of the changes in LVEDD is different between long-distance runners and cyclists. In the former, the highest LVEDD values were recorded in the first two training periods, whereas in the latter, the highest LVEDD values were in the second and third periods. Hence, it is not clear what the changes of heart morphology are when athletes move, for example, from general to specific sports training.

Performance and resolution of echocardiography

All studies describe the methodology of echocardiography in detail. However, if the objective is to detect small variations in echocardiographic variables, these descriptions are probably insufficient. First, the studies were performed between 1978 and 2000: in this period, hardware and software have evolved considerably. In addition, the actual standards of echocardiographic recording have changed during this time. Despite software advances, it may be difficult to quantitate echocardiographic images.\textsuperscript{29,30} The general range of change is $\sim 2\sim 3$ mm, and the differences during a training period are of the order of a few tenths of millimetres. Therefore, echocardiography may not be sensitive enough to detect changes in heart dimensions taking place in trained athletes during detraining.\textsuperscript{32,33}

Despite the afore-mentioned reasons, this meta-analysis confirms that endurance training is associated with a positive effect on echocardiographic outcomes in highly trained athletes, who exhibit both chamber enlargement and hypertrophy. Although only two studies investigated athletes during a whole training season, probably echocardiography is not powerful enough to detect the possible heart changes because of training. Therefore, to characterize better cardiac modifications during training, echocardiography should be performed by two or three board-certified experts in echocardiography, who regularly compare their results with each other. Probably, inter- or intra-tester differences in cardiac measures $>5\%$ should prompt a further, possibly joint, echocardiography examination. Finally, the studies should better describe the characteristics of training in terms of intensity (for
example, heart rate and percentage of the maximum heart rate), volume (in each microcycle or mesocycle), and frequency.

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**References**