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Abstract

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Cardiac Dimensions Over 5 Years in Highly Trained Long-Distance Runners and Sprinters

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Abstract: Aims We assessed the changes in cardiac morphology between elite endurance-trained runners (n = 42) and elite sprinters (n = 34) over a 5-year period. In addition, we studied the relationship between heart size and maximum oxygen consumption (VO₂ max). Methods At the beginning of 5 consecutive seasons, all athletes underwent an incremental running test to determine VO₂ max and a color-coded pulsed Doppler examination to determine baseline echocardiographic variables. We hypothesized that cardiac morphology had reached its upper limit in elite athletes, and showed only minor changes during 5 years of regular training. Results Although all echocardiographic variables remained stable in nearly all sprinters studied, in the endurance runners (who presented higher cardiac cavity dimensions compared with sprinters), variations in heart morphology became evident from the third season, and were within established physiological limits. Conclusion Only 6 (17%) endurance runners and 3 (9%) sprinters showed a left ventricular internal diameter of > 60 mm (the threshold pathological value) at end diastole at some point during the observational period. Moreover, no statistically significant association was detected between changes in VO₂ max and changes in heart size. After 5 years of intense training, the changes of the echocardiographic variables examined remained different between endurance runners and sprinters.

Keywords: echocardiography; adaptation; performance; left ventricle; maximum oxygen consumption

Introduction
Noninvasive cardiology techniques (eg, echocardiography, Doppler echocardiography, and nuclear magnetic resonance) allow accurate assessment of cardiac adaptations to training. The enlargement of the heart chambers, increase of the end-diastolic interventricular septal thickness (IVSTd), end-diastolic posterior wall thickness (PWTd), and maintenance of the IVSTd/PWTd ratio document physiological cardiac hypertrophy as a consequence of training.1–5 One of the distinct features of physiological adaptations versus pathological adaptations in heart morphology is the tendency to return toward baseline values with cessation of or decrease in training, although the process of adaptation to training is more obvious than adaptation to detraining.6 The increase in heart dimensions (expressed as the increase in the size of the left ventricular internal diameter at end diastole [LVIDd]) and hypertrophy (expressed as the increase in the PWTd) are adaptations to high-load training.2,7–9 The pathological threshold of these adaptations has been established as 60 mm for LVIDd.5,9,10,11 The cardiac adaptations to different types of training are still not defined clearly.6,12–14 We performed a longitudinal echocardiographic study of nationally and internationally ranked runners over 5 seasons to determine intense training-induced heart adaptations in elite endurance runners and elite sprinters. Additional aims were to: 1) evaluate whether heart dimensions of elite runners increase above physiological levels; 2) identify any differences in cardiac adaptation between endurance-trained runners and sprint-trained runners; and 3) detect any relationship between echocardiographic variables measured at rest and peak performance (ie, at the time of reaching maximum oxygen consumption [VO₂ max]), which was measured via an incremental treadmill running test.

Materials and Methods
Participants
This study was approved by the local ethics committee. All participants were informed of the nature of the tests, and all provided written informed consent to participate in the study. From 1995 to 2005, a cohort of 76 men who had been training for ≥ 10 years, including competing for ≥ 5 years at national and international levels, completed 1 test per year during 5 consecutive years. The athletes were grouped into sprint-trained runners (n = 34; distance ≤ 400 m; age, 23.2 ± 2.4 years; height, 178.1 ± 10.0 cm; body mass, 75.0 ± 6.3 kg; absolute VO₂ max, 4.7 ± 0.5 L·min⁻¹; VO₂ max/kg, 62.6 ± 3.5 mL·min⁻¹·kg⁻¹) and endurance-trained runners (n = 42; distance, > 400 m; age, 24.5 ± 3.2 years; height, 181.1 ± 8.2 cm; body mass, 73.2 ± 4.0 kg; VO₂ max, 4.9 ± 0.5 L·min⁻¹; absolute VO₂ max/kg, 66.9 ± 3.5 mL·min⁻¹·kg⁻¹).

Study Protocol
As part of their yearly medical preparticipation screening examination, the athletes underwent an echocardiographic examination and an incremental treadmill exercise test to exhaustion at the beginning of each season over the 5 consecutive seasons.

Echocardiography
The echocardiographic examinations were performed by 2 experienced physicians using a Toshiba SSH-140 ultrasonograph (Toshiba Medical Systems SA, Madrid, Spain). This equipment allows for 1- and 2-dimensional images (M-mode and 2-dimensional, respectively), and provides continuous and color-coded pulsed Doppler examinations. The 25-MHz electronic transducer used employed phased array technology, with an axial resolution of 0.6 mm. The equipment uses its own software to measure and calculate all conventional echocardiographic variables. Both M-mode and 2-dimensional images were at a 45° angle, with the athletes in the left semilateral decubitus position; conventional planes were visualized by placing the transducer in the corresponding positions. The IVSTd, PWTd, and the diameter of the ventricles in the 2 phases of the cardiac cycle were determined via the long parasternal projection axis in M-mode, guided by real time 2-dimensional images and the electrocardiographic (ECG) signal obtained from a CM5 lead. Diastolic measurements were taken at the moment coinciding with the start of the QRS complex; systolic measurements were taken at the moment of greatest posterior displacement of the septum. When measuring thickness, special care was taken not to include echocardiograms from trabeculated areas (ie, the cordae tendinae of the left ventricle), or the subvalve apparatus and moderator band of the right ventricle. All measurements were made according to the norms of the American Society of Echocardiography. End-diastolic volume (EDV) and end-systolic volume (ESV) were calculated using the Teichholz equation. Subsequently, stroke volume was calculated as the difference between EDV and ESV. Body surface area was calculated with the DuBois equation, and left ventricular mass was calculated using the Deveraux equation.

To determine the reproducibility of echocardiographic measurements, 35 subjects were randomly selected and evaluated by both operators, and interobserver variability was determined using the following parameters: left ventricular internal diameter at end systole (LVIDs), LVIDd, IVSTd, and PWTd. In addition, the coefficient of variation between the results of these observers and those of a highly experienced expert was calculated to ensure that the 2 observers’ measurements were correct.

Incremental Test
All athletes (42 endurance runners and 34 sprinters) who enrolled in the study underwent an incremental treadmill running test (H/P/COSMOS 3P 4.0®, H/P/Cosmos Sports & Medical, Nussdorf-Traunstein, Germany) at a 1% slope. After a 3-minute warm-up period at a speed of 6 km·h\(^{-1}\), the speed was increased 1 km·h\(^{-1}\) every minute until exhaustion. During this test, gas analysis was performed using the Jaeger Oxycon Pro gas analyzer (Erich Jaeger, Viasys Healthcare, Germany). Maximum oxygen consumption was determined as the mean of the 2 highest values recorded at the maximum treadmill speed reached by each subject.

Statistical Analysis
Differences between groups were analyzed using the Student’s t-test for independent samples. Repeated measures were then performed using 2-way analysis of variance (ANOVA) for season and training type, with echocardiography and VO\(_2\) max serving as the dependent variables. Post hoc Bonferroni analysis identified in what specific season these differences began. The relationship between the LVIDd and VO\(_2\) max was determined by calculating the Pearson’s product-moment correlation coefficient. All calculations were performed using SPSS v.13.0 software (SPSS, Inc., Chicago, IL). Significance was set at \(P < 0.05\).

Results
Interobserver Coefficient of Variation of Echocardiographic Measurements
The interobserver coefficient of variation was < 2% for all of the measures considered, and never reached statistical significance.

Echocardiographic Variables of Endurance Runners and Sprinters
Table 1 demonstrates the mean values of the variables measured for the 2 groups of athletes for the whole study. The endurance runners were found to have a larger LVIDs and LVIDd compared with the sprinters. Differences were also evident regarding the left atrial anteroposterior diameter at end diastole (LADd), both in absolute terms and relative to the body surface area. The endurance runners had a significantly larger right ventricular internal diameter and PWTd relative to the body surface area compared with the sprinters. Finally, stroke volume, ESV, EDV, and left ventricular mass were found to be significantly greater than those of the endurance runners (Table 1).

View: [Table 1] - Echocardiographic Variables in Sprinters Versus Endurance Runners Over the 5-Year Study Period
Changes in Cardiac Morphology
The heart structure in most sprinters remained stable over the 5 seasons (Table 2; Figure 1). However, changes were noticeable in some variables for endurance runners from the beginning of the third season. The LVIDs remained stable until the beginning of the fourth season, when a significant increase was seen compared with the LVIDs from the first season. A significant difference was also seen between the first and fifth seasons.

**View:** (Table 2) - Changes in Echocardiographic Variables Over the 5-Year Study Period

**View:** (Figure 1) - Changes in LVIDs, LVIDd, and VO₂ max over the 5 seasons in both groups of athletes (STR and ETR).

The LVIDd exhibited a similar change, with significant differences recorded from the third to fifth seasons (Figure 1). Only 3 sprinters and 6 endurance runners showed values > 60 mm, which are dimensions that are normally considered pathological at some point during the 5-year experimental period. In the third and fourth seasons, the endurance runners showed a reduction in the LADd in absolute terms compared with the first year. When LADd was expressed as relative to body surface area, significant differences were seen in the third, fourth, and fifth seasons compared with the first season. Finally, the left ventricular ejection fraction decreased significantly in the fourth season (Table 2).

**Discussion**

In our article, we report the results of a large longitudinal study of echocardiographic features in elite runners and sprinters. Our cohort was unique in terms of performance abilities, and we also had a 100% follow-up rate for all athletes in the original cohort. This reflects the dedication of the research team as well as the fact that all athletes were required to undergo a compulsory preparticipation screening to train and compete. The cardiac morphology of highly trained athletes was assessed with echocardiography at physiological levels, with LVIDd never being measured beyond physiological limits. In the long term, the cardiac adaptation to training was found to be similar in endurance runners and sprinters. The sprinters showed no significant variations in their echocardiographic dimensions in the study period. We also identified no evidence of an association between heart dimensions and VO₂ max. Figure 1 shows that in endurance runners, the LVIDd changes significantly between the third and fifth seasons. In the present study, only 6 runners in the endurance runners group showed LVIDd values of > 60 mm (ie, above the pathological threshold). However, this is similar to findings reported by other authors who investigated middle- and long-distance runners. An athlete’s athletic career is divided into 3 stages: basic training, specialization and achievement of top performance, and maintenance of top performance. Our subjects fit into the last category. Therefore, their apparent lack of cardiac adaptation may result from the absence of any further margin for changes in cardiac dimensions. The lack of significant change in LVIDd in sprinters over time may result from the different type of training they undertake compared with endurance runners. During training, a sprinter’s heart is subjected to intense, brief exercise, whereas an endurance runner’s heart provides increased ventricular function over long periods.

The LVIDd is significantly smaller in sprinters (100- and 200-m sprinters) compared with endurance runners (n = 29; 50.4 ± 2.15 mm vs n = 58; 54.9 ± 4.36 mm), although other authors found no differences between such athletes (55.9 ± 4.8 mm vs 53.9 ± 3.8 mm). The mechanism responsible for the differences in the heart morphology of endurance runners and sprinters might be the increase in venous return during the recovery phase.

In all subjects studied, both the IVSTd and PWTd decreased more in endurance runners than in sprinters (Table 2). This may be because the endurance runners experienced both a reduction in the PWTd and an increase in left ventricular volume. In addition, the IVSTd/PWTd ratio remained within normal limits in both the sprinters and endurance runners. At no point were IVSTd or PWTd values recorded at > 11 mm, and the ratio remained around 1.

The correlation shows a lack of association between cardiac adaptation (expressed as the change in the LVIDd) and the change in performance (expressed as the percentage change in VO₂ max) in both endurance runners and sprinters. Other authors report a significant correlation (r = 0.67; P < 0.001) between changes in LVIDd and performance, expressed as the percentage change in the best speed achieved during competition in different seasons. However, the same authors report a lack of a relationship between the change in VO₂ max and performance (r = 0.27; P > 0.05). This discrepancy and the absence of a relationship in the present study might be related to the fact that VO₂ max tends to stabilize in highly trained athletes. Therefore, VO₂ max is also a poor indicator of any adaptations...
made, or improvement in performance in highly trained runners. Further, it is measured at the moment of maximum exertion, whereas echocardiographic variables are measured at rest. If VO₂ max stabilizes in highly trained athletes and performance improves, this improvement must result from other factors, such as running economy, increase in anaerobic threshold, or better mechanical efficiency. Maximum oxygen consumption is limited by the capacity to capture, pump, transport, and use oxygen; therefore, it is not solely a result of the Fick equation, but also depends on Fick’s Principle of Oxygen Diffusion. In horses, a relationship has been observed between the heart size and VO₂ max. However, in homogeneous samples of highly trained athletes, there is a loss of linearity in the relationship between VO₂ max and cardiac output. This makes VO₂ max a poor indicator of performance or improvements achieved through training. It would be interesting to study other submaximal performance variables, such as running economy, and anaerobic, ventilatory, and lactate thresholds to determine whether these can offer an explanation for the improvements achieved.

The athletes who participated in the present investigations were all males. Therefore, our results can only be applied to male athletes. Further study would be needed to assess and compare results in an equally high ranking group of female endurance and sprint runners. The results of the present study demonstrate that heart dimensions of elite runners do not increase above physiological levels, since the values are maintained in the normal range along 5 years of intense training. Therefore, the heart dimensions evaluated by echocardiography do not reach pathological values after intense training.

Conclusion
Our study determined that elite male athletes reach an upper limit of heart adaptation. Over 5 seasons of intense training, the changes produced in the echocardiographic variables of male endurance runners and sprinters were different, although they did not exceed physiological adaptation limits. Our results also demonstrate that in sample of highly trained athletes, VO₂ max remained stable during the 5 seasons, and changes in the heart size are not accompanied by changes in VO₂ max.

Conflict of Interest Statement
Francisco Javier Calderón, PhD, Victor Díaz, PhD, Ana B. Peinado, PhD, Pedro J. Benito, PhD, and Nicola Maffulli, MD, MS, PhD, FRCS(Orth) disclose no conflicts of interest.

References


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