Remodelling of the left ventricle in athlete’s heart: a three dimensional echocardiographic and magnetic resonance imaging study

S De Castro, A Pelliccia, S Caselli, E Di Angelantonio, F Papetti, E Cavarretta, I Carbone, M Francone, R Passariello, N G Pandian, F Fedele

Intensive long term athletic training is associated with morphological cardiac changes, which have extensively been described as “athlete’s heart”. These changes are considered to be physiological adaptations to increased haemodynamic overload induced by chronic and intensive exercise.1–3

For many years, morphological assessment of athlete’s heart and its differentiation from pathological cardiac conditions have been based on two dimensional and M mode echocardiography. The formulas used with these methods are based on geometric assumptions and are possible causes of inaccuracy.

The objective of our study was, therefore, to validate and assess the pattern of left ventricular (LV) remodelling in a population of highly trained athletes by using three different techniques—conventional two dimensional echocardiography, three dimensional echocardiography, and magnetic resonance imaging (MRI)—and to explore the potential advantages and limitations of these techniques.

METHODS

Thirty subjects were studied: 18 male top level athletes, who were members of the Italian Olympic rowing team (> 3 consecutive years’ long term exercise), and 12 untrained sedentary male subjects. All subjects signed an informed consent form.

Each patient underwent two dimensional echocardiography (Sonos 5500; Philips). LV mass was calculated by the Devereux formula, and LV volumes and ejection fraction (EF) were calculated by the modified Simpson’s rule. Three dimensional echocardiography was performed with a Philips Sonos 7500 equipped with the X-Matrix probe (2–4 MHz). Images were acquired by the “full volume” technique, which consists of a wide angle three dimensional pyramid built on four smaller parallel scans gated to the ECG during patient breath holding.

Two cardiologists (SDC, SC) blinded to the imaging mode analysed three dimensional data offline with dedicated software (4D Cardio View RT; TomTec). LV mass and LV end diastolic (LVEDV) and end systolic (LVESV) volumes were obtained by tracing the endocardial and epicardial borders through eight equidistant planes. We also calculated an LV remodelling index (LVRI), expressed as the ratio of LV mass to LVEDV.

Two experienced observers (IC, MF) blinded to echocardiographic data quantitatively analysed MRIs (1.5 T Magnetom Vision; Siemens) offline with dedicated software. Manual tracing was used to outline endocardial and epicardial borders for the evaluation of LV mass and volumes.

All analyses were based on data normalised to body surface area. LV parameters, assessed by two dimensional and live three dimensional echocardiography, were correlated with those obtained by MRI, regarded as the reference standard technique. Pearson’s correlation was performed to compare two dimensional echocardiography versus MRI and three dimensional echocardiography versus MRI. The differences between the results of two and three dimensional echocardiography and MRI were tested by a one sample t test and the 95% limits of agreement were calculated with adjustment for small sample size.4

Intraobserver and interobserver variabilities were calculated by using the intraclass correlation coefficient (ICC). Differences in cardiac dimensions between athletes and controls were assessed with the unpaired t test. All data were analysed with Stata 8 software (StataCorp, College Station, Texas, USA). Data are presented as mean (SEM).

RESULTS

Of the 18 athletes enrolled in this study, most had ECG abnormalities, including increased R/S wave precordial voltages (> 30 mm) in 12 athletes, an abnormal repolarisation pattern with a flat or inverted T wave in precordial leads V4–V6 or in standard leads II, III, and aVF in four, and a Q wave in one athlete. None of these athletes had echocardiographic, clinical, or familial evidence of hypertrophic cardiomyopathy.

M and B mode echocardiographic measurements were routinely assessed and showed a range of LV wall thicknesses from 11–14 mm in athletes and from 7–10 mm in controls.

Two dimensional echocardiography allowed for online acquisition and very quick (two minute) offline quantification of volumes, EF, and mass. Three dimensional echocardiography required 8 (2) seconds for volumetric acquisition and 9 (2) minutes for offline analysis, and MRI needed 51 (10) minutes for acquisition and 20 (3) minutes for quantitative analysis.

Table 1 shows the comparisons between two and three dimensional echocardiography and MRI. Two dimensional measurements significantly but mildly underestimated LVEDV, LVESV, and LV mass relative to MRI. Conversely, a good agreement was found between three dimensional echocardiography and MRI for all the parameters. Interobserver and intraobserver variabilities were negligible for three dimensional echocardiography (ICC between 0.89 and 0.96 for all parameters) as opposed to two dimensional echocardiography (ICC between 0.78 and 0.85 for all parameters).

Cardiac values from the athletes were then compared with those from controls by three dimensional echocardiography.

Abbreviations: EF, ejection fraction; ICC, intraclass correlation coefficient; LV, left ventricular; LVEDV, left ventricular end diastolic volume; LVESV, left ventricular end systolic volume; LVRI, left ventricular remodelling index; MRI, magnetic resonance imaging
TLV volumes increased significantly in athletes compared with controls. In particular LVEDV and LVESV were significantly increased in athletes by 58.7% (95.2 (16.1) v 60.0 (5.1) ml/m² in controls, p < 0.001) and 65.7% (38.6 (6.3) v 23.3 (3.9) ml/m², p < 0.001). EF (athletes, 61.3 (4.8)% v controls, 59.3 (4.0%), p = 0.214) did not differ between the two groups. LV mass was greater in athletes than in controls by 74.3% (104.6 (20.7) and 60.0 (10.2), respectively, p < 0.001). However, the LVRI was similar in athletes (1.11 (0.17) g/ml) and controls (1.00 (0.19) g/ml, p = 0.130).

DISCUSSION

The present investigation shows that evaluation of cardiac dimensions in athletes by three dimensional echocardiography is reliable and shows very good agreement with MRI. Even though MRI mildly overestimated the values, as reported from previous studies, this difference was more evident for two dimensional than for three dimensional echocardiographic data.1 In particular, the difference between three dimensional echocardiography and MRI may be due to different tracing methods. The MRI contouring procedure excludes the trabeculae of the LV as opposed to three dimensional echocardiography, where these contours are usually included. Also, MRI software determines cardiac volumes from short axis cross sections with a disk summation method as opposed to three dimensional echocardiography software, which uses long axis images for endocardial borders. Indeed, the low interobserver and intraobserver variabilities observed in three dimensional echocardiographic studies suggest that offline analysis is highly accurate and that the operator dependence of live three dimensional echocardiography is very low.

Our study furthermore suggests that intensive, long term athletic conditioning is associated with morphological LV remodelling. In rowing athletes, this remodelling consists of a substantial increase in LV cavity dimension, wall thickness, volume, and mass compared with controls.2 In our study, we measured the LVRI to describe the pattern of LV remodelling in athletes. An increased LVRI is consistent with concentric hypertrophy, whereas a reduced LVRI is consistent with eccentric hypertrophy. Our athletes’ LVRI was similar to that of controls, suggesting that the LV remodelling associated with intensive athletic conditioning does not alter LV geometry. For this reason, we define “symmetric” remodelling as those conditions in which an increased cavity dimension and volume are accompanied by an increased thickness and mass of the ventricles. This finding is consistent with previous investigations we performed with two dimensional echocardiography and Fourier analysis, in which we found a normal LV cavity shape despite an increased absolute dimension in top level endurance athletes.3

In conclusion, LV systolic function was normal in top level athletes and did not differ from that of controls. Therefore, according to previous studies, LV morphology is substantially remodelled in athletes in the absence LV systolic dysfunction.

| Table 1 Comparison of two (2D) and three dimensional (3D) echocardiography with magnetic resonance imaging (MRI) |
|-----------------------------------------------|-----------------------------------------------|
| 2D echocardiography versus MRI | 3D echocardiography versus MRI |
| r | Difference | 95% limits of agreement | r | Difference | 95% limits of agreement |
| LVEDV (ml/m²) | 0.93 | -7.42 (1.58)** | -10.53, -4.06 | 0.97 | -0.97 (1.09) | -3.24, 1.30 |
| LVESV (ml/m²) | 0.85 | -5.51 (0.98)** | -7.54, -3.46 | 0.98 | -0.99 (0.40)* | -1.82, 0.16 |
| EF [%] | 0.64 | 3.19 (0.71)** | 1.72, 4.68 | 0.81 | 0.80 (0.54) | -0.32, 1.92 |
| LV mass [g/m²] | 0.97 | -7.65 (0.76)** | -9.18, -6.02 | 0.99 | -2.55 (0.44)** | -3.52, -1.68 |

Data are mean (SEM).
*p<0.05; **p<0.001.
EF, ejection fraction; LV, left ventricular; LVEDV, left ventricular end diastolic volume; LVESV, left ventricular end systolic volume; r, product-moment correlation coefficient.

REFERENCES


Authors’ affiliations

S D Castro, C Casesi, F Papetti, E Cavarretta, F Fedele, Department of Cardiovascular and Respiratory Sciences, “La Sapienza” University of Rome, Rome, Italy
A Pelliccia, National Institute of Sports Medicine, Rome, Italy
E D Angelantonio, Department of Internal Medicine, La Sapienza University of Rome, Rome, Italy
I Carbone, M Francone, R Passariello, Department of Radiology, La Sapienza University of Rome, Rome, Italy
N G Pandian, New England Medical Center, Tufts University, Boston, Massachusetts, USA

Correspondence to: Dr Stefano De Castro, Department of Cardiovascular and Respiratory Sciences, “La Sapienza” University of Rome, Viale del Policlinico 155, 00161 Rome, Italy; stefano.decastro@uniroma1.it

Accepted 17 October 2005