Left Ventricular Myocardial Velocities and Deformation Indexes in Top-Level Athletes

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Background: The aim of this study was to define the range of left ventricular (LV) velocities and deformation indexes in highly trained athletes, analyzing potential differences induced by different long-term training protocols.

Methods: Standard echocardiography, pulsed-wave tissue Doppler echocardiography, and two-dimensional strain echocardiography of the interventricular septum and lateral wall were performed in 370 endurance athletes and 280 power athletes. Using pulsed-wave tissue Doppler, the following parameters of myocardial function were assessed: systolic peak velocities (S_m), early (E_m) and late (A_m) diastolic velocities, and the E_m/A_m ratio. By two-dimensional strain echocardiography, peaks of regional systolic strain and LV global longitudinal strain were calculated.

Results: LV mass index and ejection fraction did not significantly differ between the two groups. However, power athletes showed an increased sum of wall thicknesses ($P < .01$) and relative wall thickness, while LV stroke volume and LV end-diastolic diameter ($P < .001$) were greater in endurance athletes. By pulsed-wave tissue Doppler analysis, E_m and E_m/A_m at both the septal and lateral wall levels were higher in endurance athletes. By two-dimensional strain echocardiography, myocardial deformation indexes were comparable between the two groups. E_m/A_m ratios ≥ 1 were found in the overall population, while 90 % of athletes had an $E_m \geq 16$ cm/sec, $S_m \geq 10$ cm/sec, and global longitudinal strain $\leq -16\%$. Multivariate analyses evidenced independent positive association between Em peak velocity and LV end-diastolic volume ($P < .001$) and an independent correlation of global longitudinal strain with the sum of LV wall thicknesses ($P < .005$).

Conclusions: This study describes the full spectrum of systolic and diastolic myocardial velocities and deformation indexes in a large population of competitive athletes. (J Am Soc Echocardiogr 2010;23:1281-8.)

Keywords: Athlete's heart, Tissue Doppler, Two-dimensional strain, Left ventricular hypertrophy, Endurance, Power, Training

Athlete's heart is a cardiac adaptation to long-term, intensive training including increased cavity diameters, wall thickness, and left ventricular (LV) mass.[1-5] Conventional Doppler echocardiography is widely used to evaluate athlete's heart and to distinguish it from LV diseases.[6-10] Furthermore, pulsed-wave tissue Doppler[11-14] and two-dimensional strain echocardiographic (2DSE) analysis have recently been applied in the evaluation of either physiologic or pathologic LV hypertrophy.[15-17]

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The aim of the present study was to define the full range of LV velocities and deformation indexes in a large population of highly trained athletes, analyzing possible differences induced by different long-term training protocols.

METHODS

Study Population

From June 2007 to April 2009, 650 consecutive highly trained athletes were referred to the Sports Medicine Ambulatory Service of Monaldi Hospital (Naples, Italy) for cardiovascular preparticipation screening[18] and afterward to our echocardiographic laboratory for the purpose of the present study. All subjects underwent detailed histories, physical examinations, electrocardiography, chest radiography, and comprehensive transthoracic echocardiography, including standard Doppler, pulsed-wave tissue Doppler, and 2DSE studies. On the basis of their training protocols, the athletes were categorized into two groups: 370 endurance-trained athletes (ATE) and 280 power athletes (ATP). The study was approved by the local ethics committee.

Abbreviations

ATE = Endurance-trained athletes

ATP = Power athletes

CI = Confidence interval

Ds = End-systolic diameter

ESSc = Circumferential end-systolic stress

GLS = Global longitudinal strain

LV = Left ventricular

Ps = Posterior wall thickness in systole

SBP = Systolic blood pressure

2DSE = Two-dimensional strain echocardiographic

Exclusion criteria were coronary artery disease, arterial hypertension, valvular and congenital heart disease, bicuspid aortic valve, congestive heart failure, cardiomyopathies, diabetes mellitus, sinus tachycardia, use of anabolic steroids and of other drugs, and echocardiograms of inadequate quality.

Training Protocols

Because the specific nature of sports training has a major influence on cardiac structural adaptations, our athletes were selected on the basis of their training protocols. All the subjects had been trained intensively for 15 to 20 hours/week for >4 years.

The ATE group (long-distance and middle-distance swimming or running, soccer, and basketball) was submitted to intensive aerobic isotonic dynamic exercise at incremental workloads of 70% to 90% of maximal heart rate. In particular, they performed 3 hours/day of incremental long-distance swimming (7,000 m/day divided into series of 400–800 m) or 3 hours/day of long-distance running and only 2 hours/week of weightlifting at low workload.

On the other hand, the ATP group (weightlifting, martial arts, and windsurfing) underwent anaerobic isometric static exercise at incremental workloads of at 40% to 60% of maximal heart rate. In particular, their training protocol included both 2 hours/day of short-distance running and 3 hours/day of weightlifting at high workload.

Imaging Protocol

Standardized transthoracic echocardiographic, Doppler, pulsed-wave tissue Doppler, and 2DSE examinations were performed using commercially available equipment in all subjects (Vivid 7; GE Healthcare, Milwaukee, WI). Specific views included the parasternal long-axis and short-axis views (at the mitral valve and papillary muscle levels); apical four-chamber, two-chamber, and three-chamber views; and subcostal views, including respiratory motion of the inferior vena cava. Pulsed-wave and continuous-wave Doppler interrogation was performed on all four cardiac valves.

All studies were reviewed and analyzed offline by two independent observers blinded to the clinical characteristics of the study population. Specific measurements were made by the average of three to five cardiac cycles.

M-Mode and B-Mode Measurements

M-mode measurements (LV diastolic and systolic diameters, interventricular septal and posterior wall thickness, and left atrium) were performed in the parasternal long-axis view with the patient in the left lateral position. LV mass was calculated by the Penn convention[19] and indexed for height$^{2.7}$ (Cornell adjustment).[13,20] Relative diastolic wall thickness was determined as the ratio between the sum of septal and posterior wall thicknesses and LV end-diastolic diameter. Circumferential end-systolic stress (ESSc) was calculated as a measurement of LV afterload using a cylindrical model according to the following formula:

$$\mathrm{ESSc}\ (\mathrm{g/cm^2}) = \mathrm{SBP} \times 0.5\mathrm{Ds}^2 \left\{1 + \left[(0.5\mathrm{Ds} + \mathrm{Ps})^2/(0.5\mathrm{Ds} + 0.5\mathrm{Ps})^2\right]\right\} \Big/ (0.5\mathrm{Ds} + \mathrm{Ps})^2 - 0.5\mathrm{Ds}^2,$$

where SBP is systolic blood pressure, Ds is end-systolic diameter, and Ps is posterior wall thickness in systole.[21]

LV ejection fraction was calculated using the biplane Simpson's rule in the apical four-chamber and two-chamber views. Left atrial maximal volume was measured at the point of mitral valve opening using the biplane area-length method and corrected for body surface area (left atrial volume index).[22]

Color Doppler Analysis

Valvular regurgitation was quantified from color Doppler imaging and categorized as absent, minimal (within normal limits), mild, moderate, or severe, on the basis of the width of the vena contracta. Intermediate vena contracta values (3–7 mm) were confirmed by the proximal isovelocity surface area method.[23] Doppler-derived LV diastolic inflow was recorded in apical four-chamber view by placing the sample volume at the tip level. The following LV diastolic measurements were measured: E and A peak velocities (meters per second) and their ratio, E-wave deceleration time (milliseconds), isovolumic relaxation time (milliseconds, as the time interval occurring between the end of systolic output flow and the transmitral E-wave onset, by placing pulsed Doppler sample volume between outflow tract and mitral valve).

LV stroke volume was calculated as LV outflow tract area × outflow tract time-velocity integral.[24] Cardiac index was calculated as stroke volume × (heart rate/body surface area).

Tissue Doppler Analysis

By pulsed-wave tissue Doppler, interrogation of multiple myocardial segments was obtained. In the apical four-chamber view, a Doppler sample volume was placed in correspondence of both the LV basal septum and the basal lateral wall. The apical view was chosen to obtain quantitative assessment of regional wall motion almost simultaneous to Doppler LV inflow and outflow and to minimize the incidence angle between Doppler beam and LV longitudinal wall motion. Pulsed-wave tissue Doppler is characterized by a myocardial systolic wave (S_m) and two diastolic waves, early (E_m) and atrial (A_m). The peak velocity of S_m (meters per second), early (E_m) and atrial (A_m) peak velocities (meters per second), and the E_m/A_m ratio were determined as myocardial measurements[13,25] (Figures 1 and 2).

Two-Dimensional Segmental Longitudinal Strain and Global Longitudinal Strain (GLS)

The speckle-tracking technique has been simplified and integrated into echocardiographic instruments to generate a parametric image of myocardial strain throughout the left ventricle, called automated function imaging.[26,27] In our study, digital loops with three successive cardiac cycles were acquired from apical two-chamber, three-chamber, and four-chamber views. Analysis was performed directly on the echocardiographic system for each of the three apical views, with the operator manually identifying three points: two on the each side of the mitral valve and a third at the apex of the left ventricle. The two-dimensional loops from the routine echocardiographic examination are processed offline. The end-systolic frame is first

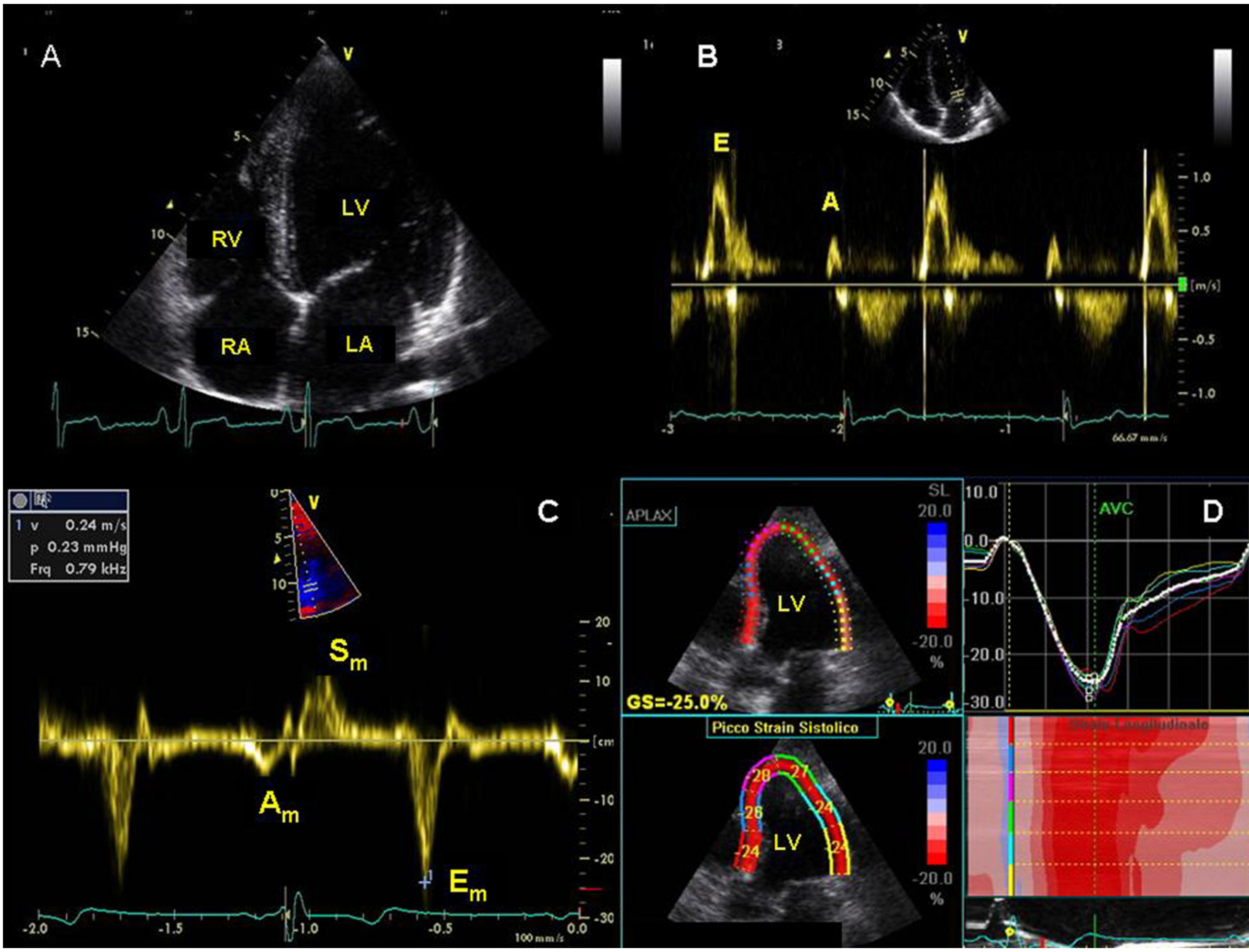

Figure 1 Echocardiographic evaluation of athlete's heart in an ATE. **(A)** Standard echocardiography, apical four-chamber view: left ventricular hypertrophy. **(B)** Transmitral flow pattern: high early diastolic wave. **(C)** Pulsed tissue Doppler pattern: increased early diastolic myocardial velocity. **(D)** Two-dimensional strain: normal myocardial longitudinal deformation.

defined in the apical long-axis (three-chamber) view, where the aortic valve is directly visible. Aortic valve closure time is marked. The time from R wave to aortic valve closure is then measured by the software.

As a result, the software detected the endocardium at end-systole, tracked myocardial motion during the entire cardiac cycle, and created U-shaped regions of interest that encompassed basal, middle, and apical segments of two opposite LV walls. Tracking quality was assessed by the operator and scored by the software. If the tracking was poor, the operator could repeat the imaging, readjusting the endocardial tracing or changing software parameters such as region of interest width and smoothing, until a better score was achieved. Inadequately tracked segments were automatically excluded from the analysis.

Longitudinal strains for each individual segment were measured and averaged for LV septal and lateral walls. In addition, the software calculated LV GLS by averaging local strains along the entire left ventricle (Figures 1 and 2).

Statistical Analysis

All analyses were performed using SPSS for Windows version 14.0 (SPSS, Inc., Chicago, IL). Variables are presented as mean ± SD. The *t* test for unpaired data was used to estimate differences between the two groups. Linear regression analyses and partial correlation tests using Pearson's method were done to assess univariate relations. To identify significant independent determinants of myocardial measurement in athletes, their individual associations with clinical relevant and echocardiographic variables were assessed using multivariate linear regression analysis. The following variables were included in the analysis: clinical data (age, gender, systolic blood pressure, and type and duration of sport training) and standard echocardiographic indexes (LV volumes and wall thicknesses, LV mass index, LV stroke volume, LV ejection fraction, and Doppler transmitral inflow measurements). These variables were selected according to their clinical relevance and potential impact on myocardial function. Variable selection was performed in the multivariate linear regression as an interactive stepwise backward elimination method, each time excluding the one variable with the highest *P* value according to Wald statistics.

Reproducibility of myocardial measurements was determined in all subjects by two independent observers blinded to previous measures. Intraobserver variability was examined using both Pearson's bivariate two-tailed correlations and Bland-Altman analysis. Relation coefficients, 95% confidence intervals (CIs), and percentage errors were reported.

RESULTS

Clinical Characteristics of the Study Population

Mean age was comparable between the two groups. In accordance with the effects of different training protocols, ATP at rest showed

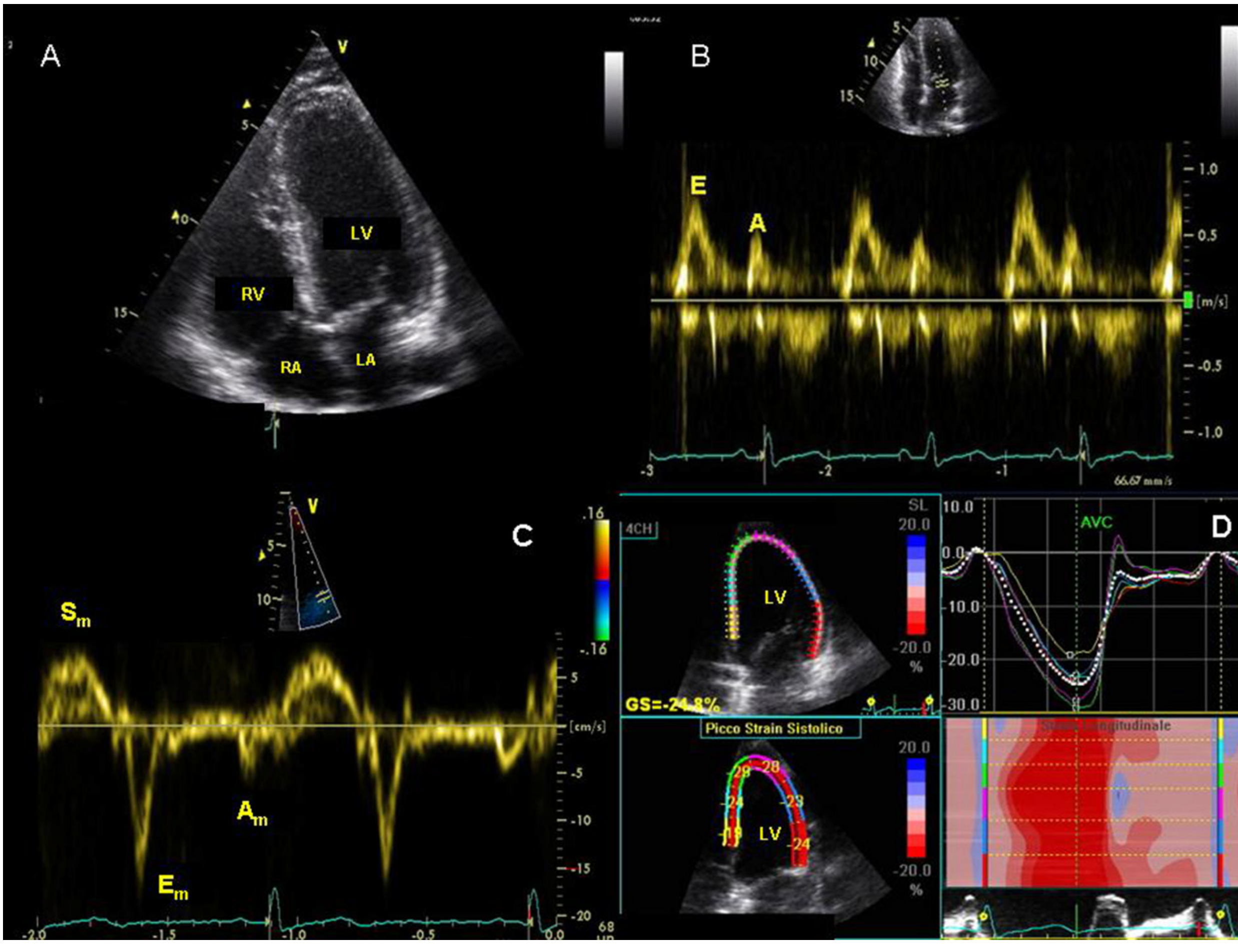

Figure 2 Echocardiographic evaluation of athlete's heart in an ATP. **(A)** Standard echocardiography, apical four-chamber view. **(B)** Transmitral flow pattern. **(C)** Pulsed tissue Doppler pattern. **(D)** Two-dimensional strain.

higher heart rates, body surface areas, and systolic blood pressures than ATE (Table 1).

Echocardiographic Features

M-Mode and B-Mode Measurements. ATP showed increased sum of wall thicknesses (septum + LV posterior wall), LV relative wall thickness, and ESSc, while LV end-diastolic volume was greater in ATE. LV mass index and LV ejection fraction did not significantly differ between the two groups, while left atrial volume index was increased in ATE (Table 2).

Color Doppler Analysis. The prevalence of valvular regurgitation was comparable between the two groups. Conversely, all transmitral Doppler indexes were higher in ATE, with increased E peak velocity, E/A ratio, E-wave deceleration time, and isovolumic relaxation time. Furthermore, LV stroke volume, cardiac index, and pulmonary artery systolic pressure were significantly increased in ATE (Table 3).

Tissue Doppler Analysis. Pulsed-wave tissue Doppler analysis showed higher early diastolic peak velocities and E_m/A_m ratios in ATE at both the septal and lateral wall levels, while systolic peak velocities were comparable in both groups. In the overall population, we found that 90% of the athletes had $E_m \geq 16$ cm/sec and $S_m \geq 10$ cm/sec, while 100% of athletes had E_m/A_m ratios ≥ 1 (Table 4).

Table 1 Study population characteristics

Variable	Overall (*n* = 650)	Endurance (*n* = 370)	Strength (*n* = 280)	*P*
Age (years)	28.5 ± 10.2	27.8 ± 10.1	29.2 ± 10.4	NS
Men	370	210	160	NS
BSA (m^2)	1.86 ± 0.6	1.84 ± 0.5	1.89. ± 0.6	<.01
HR (beats/m)	61.5 ± 9.6	51.1 ± 4.4	69.9 ± 9.8	<.001
SBP (mm Hg)	124.8 ± 7.8	119.8 ± 8.1	132.9 ± 7.1	<.001
DBP (mm Hg)	75.6 ± 4.5	72.7 ± 4.9	78.9 ± 4.0	NS

Data are expressed as mean ± SD or as numbers.
BSA, Body surface area; *DBP*, diastolic blood pressure; *HR*, heart rate; *SBP*, systolic blood pressure.

Two-Dimensional Strain Echocardiographic Analysis. Overall, LV speckle tracking was possible in 11,650 of 11,700 attempted segments from the 650 subjects with technically adequate images, with only 0.4% of segments eliminated (tracking variation scores > 2.5). Both regional and global myocardial deformation measurements were comparable between the two groups. In particular, 90% of athletes showed LV GLS ≤ −16% (Table 5).

Table 2 M-mode and B-mode analyses

Variable	Overall	Endurance	Strength	*P*
Septal wall thickness (mm)	10.6 ± 2.8	9.7 ± 3.1	11.3 ± 2.4	<.01
Posterior wall thickness (mm)	10.4 ± 2	9.3 ± 2.1	11.6 ± 1.6	<.01
Sum of wall thickness (mm)	20.5 ± 3.8	18.7 ± 4.4	22.3 ± 2.8	<.001
LV end-diastolic volume (ml)	135.8 ± 13.7	149.4 ± 13.9	119.2 ± 13.3	<.001
LV end-systolic volume (ml)	37.1 ± 9.4	40.3 ± 9.9	34.8 ± 8.1	NS
Relative diastolic wall thickness	0.41 ± 0.05	0.37 ± 0.04	0.45 ± 0.06	<.001
LV mass index ($g/m^{2.7}$)	63.6 ± 9.3	62.8 ± 9.9	64.5 ± 8.7	NS
LV ESSc (g/cm^2)	124.4 ± 18.4	90.3 ± 15.2	158.6 ± 19.5	<.001
LV ejection fraction (%)	68.9 ± 4.3	69.7 ± 4.7	67.1 ± 3.8	NS
Left atrial volume index (ml/m2)	27.8 ± 9.3	29.1 ± 9.1	26.4 ± 8.4	<.01

Data are expressed as mean ± SD.

Table 3 Color Doppler analysis

Variable	Overall	Endurance	Strength	*P*
Aortic valve regurgitation (%)	3.4	3.3	3.6	NS
Mitral valve regurgitation (%)	10.3	10.9	9.8	NS
Tricuspid valve regurgitation (%)	89.4	90.6	88.2	NS
Pulmonary valve regurgitation (%)	95.3	96.4	94.3	NS
Peak E velocity (m/s)	0.86 ± 0.15	0.94 ± 0.13	0.79 ± 0.16	<.01
Peak A velocity (m/s)	0.45 ± 0.23	0.45 ± 0.12	0.46 ± 0.14	NS
Peak E/A ratio	1.95 ± 0.7	2.2 ± 05	1.7 ± 0.8	<.001
E-wave deceleration time (ms)	162.5 ± 16.8	165.8 ± 16.3	159.5 ± 17.7	NS
Isovolumic relaxation time (ms)	74.5 ± 18.3	79.8 ± 20.7	69.3 ± 15.6	<.05
LV stroke volume (ml)	83.4 ± 5.9	98.4 ± 6.2	69.4 ± 3.3	<.001
LV cardiac index ($ml/min/m^2$)	2.6 ± 0.6	2.8 ± 0.7	2.4 ± 0.3	<.01

Data are expressed as mean ± SD.

Univariate Relations of Myocardial Indexes. By univariate analysis, in the overall population, pulsed-wave tissue Doppler E_m peak velocity was positively associated with LV end-diastolic volume ($r = 0.71$, $P < .0001$; Figure 3). In addition, LV GLS was inversely related to the sum of wall thicknesses ($r = -0.65$, $P < .0001$; Figure 4).

Multivariate Analysis. Stepwise forward multiple linear regression analyses were performed in the overall population to weigh the independent associations between LV myocardial parameters and LV standard measurements. By this model, after adjusting for potential determinants, the independent positive association between peak E_m velocity and LV end-diastolic volume ($\beta = 0.55$, $P < .001$) and the independent direct correlation of LV GLS with the sum of wall thicknesses ($\beta = -0.48$, $P < .005$) were confirmed.

Reproducibility of Myocardial Measurements

For intraobserver variability, Pearson's correlations were as follows: lateral wall E_m, $r = 0.87$ ($P < .00001$); septal wall E_m, $r = 0.87$ ($P < .00001$); and 2DSE LV GLS, $r = 0.85$ ($P < .00001$). On Bland-Altman analysis, lateral wall E_m had a 95% CI of ±1.8 and a percentage error of 3.2%, septal wall E_m had a 95% CI of ±1.2 and a percentage error of 3.3%), and 2DSE LV GLS had a 95% CI of ±1.5 and a percentage error of 3.4%.

For interobserver variability, Pearson's correlations were as follows: lateral wall E_m, $r = 0.86$ ($P < .00001$); septal wall E_m, $r = 0.85$ ($P < .00001$); and 2DSE LV GLS, $r = 0.84$ ($P < .00001$). On Bland-Altman analysis, lateral wall E_m had a 95% CI of ±1.9 and a percentage error of 3.4%), septal wall E_m had a 95% CI of ±1.6 and a percentage error of 3.5%), and 2DSE LV GLS had a 95% CI of ±1.8 and a percentage error of 3.6%.

DISCUSSION

Cardiac adaptations due to high-level training can vary in accordance with the type of sport. In particular, isotonic exercise associated with endurance sports is responsible for a chronic volume overload, with a predominant increase in LV mass and end-diastolic diameters (eccentric hypertrophy). On the other hand, isometric exercise, typical of strength disciplines, induces a prevalent increase in LV mass and wall thickness (concentric hypertrophy). However, athletes involved in high-level competitions can show combined cardiac features that are not easy to label and sometimes to distinguish from LV pathologies.[1-5]

Previous reports have documented that tissue Doppler and strain may represent useful additional ultrasound modalities for a comprehensive assessment of LV function in different clinical scenarios.[11-17] In particular, Baggish *et al.*[28] recently documented for the first time the relationship between competition level and myocardial parameters in a small population of 40 athletes practicing the same sport (20 elite, Olympic-caliber and 20 subelite university-level rowers). Compared with subelite rowers, elite rowers were found to have greater LV end-diastolic volume and mass and significantly more enhancement of late diastolic relaxation in both the left and right ventricles on color tissue Doppler analysis.

Table 4 Pulsed tissue Doppler assessment of basal IVS and basal LV lateral wall

Variable	Overall (*n* = 650)	Endurance (*n* = 370)	Strength (*n* = 280)	*P*
Basal IVS				
S_m peak (m/s)	0.13 ± 0.04 (0.08–0.18)	0.14 ± 0.03 (0.10–0.18)	0.12 ± 0.04 (0.08–0.15)	NS
E_m peak (m/s)	0.14 ± 0.03 (0.10–0.21)	0.16 ± 0.04 (0.12–0.21)	0.12 ± 0.03 (0.10–0.17)	<.001
A_m peak (m/s)	0.11 ± 0.03 (0.07–0.14)	0.12 ± 0.02 (0.09–0.14)	0.11 ± 0.03 (0.07–0.12)	NS
E_m/A_m ratio	1.2 ± 0.3 (1.05–1.6)	1.3 ± 0.3 (1.1–1.7)	1.05 ± 0.3 (1.05–1.4)	<.001
Basal LV lateral wall				
S_m peak (m/s)	0.15 ± 0.04 (0.09–0.20)	0.16 ± 0.02 (0.12–0.20)	0.15 ± 0.03 (0.09–0.18)	NS
E_m peak (m/s)	0.16 ± 0.05 (0.11–0.22)	0.19 ± 0.05 (0.14–0.22)	0.15 ± 0.03 (0.11–0.18)	<.001
A_m peak (m/s)	0.10 ± 0.03 (0.07–0.14)	0.10 ± 0.02 (0.08–0.14)	0.10 ± 0.03 (0.07–0.13)	NS
E_m/A_m ratio	1.62 ± 0.4 (1.2–2)	1.84 ± 0.5 (1.3–2)	1.53 ± 0.4 (1.2–1.8)	<.001

Data are expressed as mean ± SD (range).
IVS, Interventricular septum.

Table 5 Longitudinal 2DSE assessment of IVS, LV lateral wall, and LV GLS

Variable	Overall (*n* = 650)	Endurance (*n* = 370)	Strength (*n* = 280)	*P*
Mean 2DSE IVS peak (%)	−17.2 ± 3.3 (13 to 20)	−16.2 ± 3.2 (13 to 18)	−17.8 ± 4.8 (14 to 20)	NS
Mean 2DSE lateral wall peak (%)	−17.5 ± 3.6 (14 to 22)	−16.9 ± 3.4 (14 to 19)	−18.3 ± 3.5 (15 to 22)	NS
LV GLS (%)	−17.5 ± 3.5 (15 to 22)	−17.2 ± 3.1 (15 to 19)	−18.6 ± 3.7 (17 to 22)	NS

Data are expressed as mean ± SD (range).
IVS, Interventricular septum.

To date, no available LV velocities and deformation indexes have been validated in a large population of highly trained athletes practicing different kind of sport activities.

Uniqueness of the Present Study

This is the first study to report the full spectrum of pulsed-wave tissue Doppler velocities and 2DSE indexes, as well as the relative impact of anthropometric parameters and different long-term intensive training, among a large series of endurance and strength top-level athletes. Although conventional Doppler echocardiography has been widely used to evaluate athlete's heart and to distinguish it from LV pathologies,[5-10] it should be underlined that it allows only the assessment of global LV function. Conversely, pulsed-wave tissue Doppler extends Doppler application beyond the analysis of cardiac blood flow to the evaluation of myocardial wall motion. Velocities obtained from the analysis of mitral annular movement reflect the efficacy of LV contraction and relaxation and are less influenced by load conditions than standard Doppler parameters obtained by transmitral flow. In previous reports, we demonstrated the usefulness of pulsed-wave tissue Doppler in assessing the effects of endurance and strength training on myocardial regional function.[13,25] However, pulsed-wave tissue Doppler estimation, with its angle dependency, presents important limitations due to a poor alignment between the Doppler beam and the myocardial wall. In the present study, we also used 2DSE, a novel approach to quantify LV myocardial deformation within a scan plane that is inherently two dimensional and independent of interrogation angle as it tracks speckle patterns (acoustic markers) within serial B-mode sector scans.[26-29]

In our population of top-level athletes, most of athletes had $E_m \geq$ 16 cm/sec, $S_m \geq 10$ cm/sec, and GLS < −16%, confirming that in athlete's heart, cardiac morphologic variations induced by high-level training are always associated with normal systolic and diastolic parameters, closely associated with the enhancement of hemodynamic performance.

In particular, increased LV end-diastolic dimensions, caused by volume overload, are accompanied in athletes by an increase in early diastolic myocardial peak velocities. Because E_m peak velocity and the E_m/A_m ratio reflect the passive diastolic properties of the LV walls, their increase in athletes indicates a training-induced improvement in LV myocardial compliance.

In particular, isotonic aerobic sports requires prolonged elevation of cardiac output with reduced afterload and heart rate secondary to increased vagal tone. Reduced peripheral resistances typical of this kind of physical activity may determine venous overload, better right ventricular stroke volume, and consequent LV preload. Such LV volume overload might therefore induce greater LV end-diastolic diameter and earlier and better early diastolic stretching of myocardial fibers (i.e., higher E_m peak velocity), which in turn could be able to induce an enhanced LV stroke volume through a better use of the Frank-Starling mechanism.[30]

As for myocardial systolic function, by multiple linear regression models, LV GLS was independently correlated with the sum of LV wall thickness. Static anaerobic exercise is characterized by prolonged isometric contraction of single muscle fibers, with rapid increase of local metabolic requests without proportional increase of blood perfusion. In fact, enhanced sympathetic nervous system activity and external muscle compression of blood vessels induce only brief increases in cardiac output, with greatly increased heart rate and systemic peripheral resistance Therefore, LV parietal hypertrophy, mostly developed in ATP in response to pressure overload, seems to induce an enhancement of myocardial systolic function, able to better sustain the rapid increases of systolic blood pressure typical of static isometric exercise.[5]

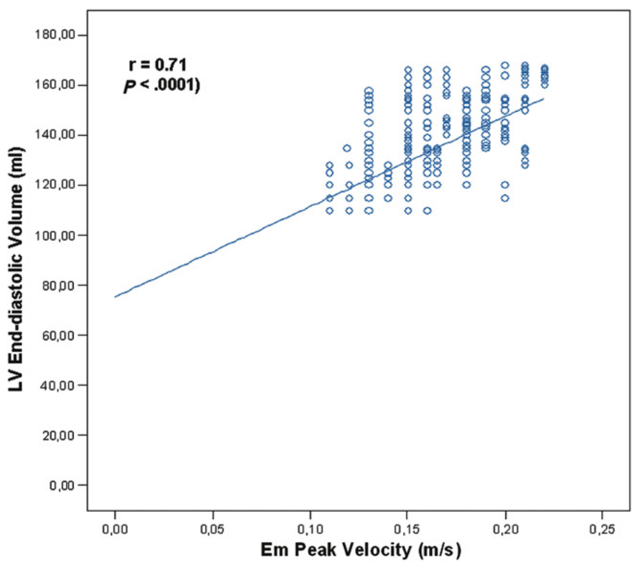

Figure 3 On univariate analysis, in the overall population, pulsed tissue Doppler E_m peak velocity was positively associated with LV end-diastolic volume.

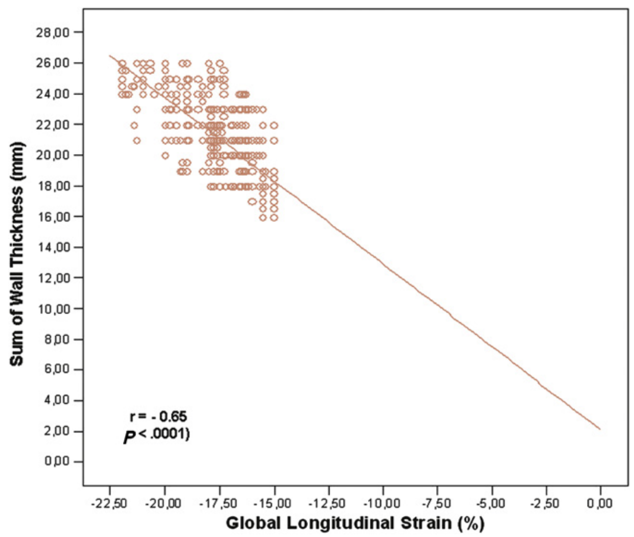

Figure 4 Scatterplots of LV global longitudinal strain with sum of LV wall thicknesses in the overall population of athletes.

Study Limitations

To put the results of the present investigation into a clinical perspective, a comparison of the values of myocardial parameters found in highly trained athletes versus normal values of sedentary individuals may be needed. However, the aim of the present study was only to explore for the first time the full spectrum of pulsed-wave tissue Doppler and 2DSE measurements and the impact of long-term intensive training in a large population of competitive athletes.

CONCLUSIONS

This study provides the full spectrum of systolic and diastolic myocardial velocities and deformation indexes obtained by pulsed-wave tissue Doppler and 2DSE analyses in a large population of competitive athletes practicing different sports. The majority of athletes had $E_m \geq 16$ cm/s, $S_m \geq 10$ cm/s, and GLS $< -16\%$. In particular, LV early diastolic myocardial function appears to be positively influenced by preload increase, while increased LV wall thickness seems to induce an enhancement of myocardial global systolic function. The combined use of conventional Doppler echocardiography, pulsed-wave tissue Doppler, and 2DSE may therefore better clarify the relative influence of the type of training on myocardial function and adaptations.

REFERENCES

1. Pelliccia A, Maron BJ. Outer limits of the athlete's heart: the effect of gender and relevance to the differential diagnosis with primary cardiac diseases. Cardiol Clin 1997;15:381-96.
2. Fagard RH. Athlete's heart: a meta-analysis of the echocardiographic experience. Int J Sport Med 1996;17:140-4.
3. Spirito P, Pelliccia A, Proschan MA, Granata M, Spataro A, Bellone P, et al. Morphology of the "athlete's heart" assessed by echocardiography in 947 elite athletes representing 27 sports. Am J Cardiol 1994;74:802-6.
4. Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. Ann Intern Med 1975;82:521-4.
5. D'Andrea A, Limongelli G, Caso P, Sarubbi B, Della Pietra A, Brancaccio P, et al. Association between left ventricular structure and cardiac performance during effort in two morphological form of athlete's heart. Int J Cardiol 2002;86:177-84.
6. Shapiro LM. Physiological left ventricular hypertrophy. Br Heart J 1984;52:130-5.
7. Maron BJ. Structural features of the athlete heart as defined by echocardiography. J Am Coll Cardiol 1986;7:190-203.
8. Douglas PS, O'Toole ML, Hiller WD, Reicheck N. Different effects of prolonged exercise on the right and left ventricles. J Am Coll Cardiol 1990;15:64-9.
9. Longhurst JC, Kelly AR, Gonyer WJ, Mitchell JH. Chronic training with static and dynamic exercise on heart volume, contractility and left ventricular dimensions. Circ Res 1981;48(suppl):I-171-8.
10. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med 1991;324:295-301.
11. Griffet V, Guérard S, Galoisy-Guibal L, Caignault J-R, Bernard F, Brion R. Valeur normale du pic protodiastolique Ea au Doppler tissulaire myocardique chez les athletes de haut niveau, à propos de cent sportifs. Arch Mal Coeur Vaiss 2007;100:809-15.
12. Vireneau D, Florescu N, Sclulthorpe N, Tweddel AC, Stephens MR, Fraser AG. Differentiation between pathologic and physiologic left ventricular hypertrophy by tissue Doppler assessment of long-axis function in patients with hypertrophic cardiomyopathy or systemic hypertension and in athletes. Am J Cardiol 2001;88:53-8.
13. Caso P, D'Andrea A, Galderisi M, Liccardo B, Severino S, De Simone L, et al. Pulsed Doppler tissue imaging in endurance athletes: relation between left ventricular preload and myocardial regional diastolic function. Am J Cardiol 2000;85:1131-6.
14. Cardim N, Oliveira AG, Longo S, Ferreira T, Pereira A, Reis RP, et al. Doppler tissue imaging: regional myocardial function in hypertrophic cardiomyopathy and in athlete's heart. J Am Soc Echocardiogr 2003;16:223-32.
15. Saghir M, Areces M, Makan M. Strain rate imaging differentiates hypertensive cardiac hypertrophy from physiologic cardiac hypertrophy (athlete's heart). J Am Soc Echocardiogr 2007;20:151-7.
16. Richand V, Lafitte S, Reant P, Serri K, Lafitte M, Brette S, et al. An ultrasound speckle tracking (two-dimensional strain) analysis of myocardial deformation in professional soccer players compared with healthy subjects and hypertrophic cardiomyopathy. Am J Cardiol 2007;100:128-32.
17. Poh KK, Ton-Nu TT, Neilan TG, Tournoux FB, Picard MH, Wood MJ. Myocardial adaptation and efficiency in response to intensive physical training in elite speedskaters. Int J Cardiol 2008;126:346-51.

18. Mellwig KP, van Buuren F, Gohlke-Baerwolf C, Bjørnstad H. Recommendations for the management of individuals with acquired valvular heart diseases who are involved in leisure-time physical activities or competitive sports. Eur J Cardiovasc Prev Rehabil 2008;15:95-103.
19. Devereux RB, Reicheck N. Echocardiographic determination of left ventricular mass: anatomic validation of the method. Circulation 1977;55: 613-8.
20. De Simone G, Daniels SR, Devereux RB, Meyer RA, Roman MJ, de Divitiis O, et al. Left ventricular mass and body size in normotensive children and adults: assessment of allometric relations and impact of overweight. J Am Coll Cardiol 1992;20:1251-60.
21. Gaasch WH, Zile MR, Hoshino PK, Apstein CS, Blaustein AS. Stress-shortening relations and myocardial blood flow in compensated and failing canine hearts with pressure-overload hypertrophy. Circulation 1989;79: 872-83.
22. Ujino K, Barnes ME, Cha SS, Langins AP, Bailey KR, Seward JB, et al. Two-dimensional echocardiographic methods for assessment of left atrial volume. Am J Cardiol 2006;98:1185-8.
23. Bonow RO, Carabello BA, Chatterjee K, de Leon AC Jr., Faxon DP, Freed MD, et al. 2006 Writing Committee Members; American College of Cardiology/American Heart Association Task Force. 2008 focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1998 Guidelines for the Management of Patients With Valvular Heart Disease): endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. Circulation 2008;118:e523-661.
24. Dubin J, Wallerson DC, Cody RJ, Devereux RB. Comparative accuracy of Doppler echocardiographic methods for clinical stroke volume determination. Am Heart J 1990;120:116-23.
25. D'Andrea A, Caso P, Severino S, Galderisi M, Sarubbi B, Limongelli G, et al. Effects of different training protocols on left ventricular myocardial function in competitive athletes: a Doppler tissue imaging study. Ital Heart J 2002;3:34-40.
26. Delgado V, Mollema SA, Ypenburg C, Tops LF, van der Wall EE, Schalij MJ, et al. Relation between global left ventricular longitudinal strain assessed with novel automated function imaging and biplane left ventricular ejection fraction in patients with coronary artery disease. J Am Soc Echocardiogr 2008;21:1244-50.
27. D'Andrea A, Caso P, Scarafile R, Riegler L, Salerno G, Castaldo F, et al. Effects of global longitudinal strain and total scar burden on response to cardiac resynchronization therapy in patients with ischaemic dilated cardiomyopathy. Eur J Heart Fail 2009;11:58-67.
28. Baggish AL, Yared K, Weiner RB, Wang F, Demes R, Picard MH, et al. Differences in cardiac parameters among elite rowers and subelite rowers. Med Sci Sports Exerc 2010;42:1215-20.
29. Amundsen BH, Helle-Valle T, Edvardsen T, Torp H, Crosby J, Lyseggen E, et al. Noninvasive myocardial strain measurement by speckle-tracking echocardiography: validation against sonomicrometry and tagged magnetic resonance imaging. J Am Coll Cardiol 2006;47:789-93.
30. Patterson SW, Starling EH. On the mechanical factors which determine the output of the ventricles. J Physiol 1914;48:357-79.