eISSN 2255-0569

ORIGINAL

Myocardial function in long distance runners: Assessment by echocardiography, tissue Doppler and speckle tracking

Función y deformación miocárdica en corredores de fondo. Evaluación mediante ecocardiografía y speckle-tracking.

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Recibido: 25 – IX – 2013
Aceptado: 3 – XII – 2013

doi: 10.3306/MEDICINABALEAR.29.01.25

Resumen

Introducción y objetivos: El ejercicio físico provoca respuestas adaptativas y cambios morfológico-funcionales en el corazón del atleta. El objetivo del presente estudio es caracterizar dichos cambios mediante ecocardiografía convencional y las nuevas técnicas de deformación miocárdica mediante speckle tracking.

Material y métodos: Se estudiaron prospectivamente a 68 varones, 38 corredores de fondo (42±8 años), y 30 controles (40±7 años). Se les realizó estudio ecocardiográfico convencional modo M, 2D y doppler así como parámetros de deformación miocárdica mediante speckle tracking a nivel de eje corto basal (B), medio (M), apical (Ap) y planos apicales 4 y 2 cámaras de ventrículo izquierdo (VI) y ventrículo derecho (VD). Se midió strain (S) y strain rate (SR) longitudinal, radial y circunferencial así como la rotación (Rot) y el untwisting (UT) a nivel basal y apical.

Resultados: Todos los sujetos presentaron función cardíaca dentro de la normalidad. Se observaron mayor fracción de eyección (FE) así como mayores volúmenes ventriculares en el grupo de atletas. No se hallaron diferencias en los parámetros del anillo Mitral. Los niveles de S longitudinal y circunferencial así como la torsión (Tor) de VI fue mayor en el grupo de los deportistas (p=0.02, p=0.005 y p=0.03 respectivamente). Se observó menor S radial en el grupo de atletas. El tamaño, el TAPSE y la deformación longitudinal del VD presentaron diferencias estadísticamente significativas (p=0.01, 0.002 y 0.006 respectivamente).

Conclusiones: Los niveles de deformación miocárdica valorados mediante strain longitudinal y circunferencial son mayores en los atletas así como la torsión ventricular. El mayor grado de deformación longitudinal, circunferencial y la mayor torsión puede representar una respuesta adaptativa al ejercicio.

Palabras clave: Ecocardiografía. Ejercicio. Strain. Torsión ventricular. Deformación ventricular

Abstract

Background: Regular intense physical exercise causes cardiovascular adaptations as well as functional and morphological changes in the athlete's heart. The aim of the present study is to characterize those changes using conventional echocardiography and new speckle tracking techniques to study myocardial deformation.

Methods: 68 men were studied. 38 long distance runners (42±8 years), and 30 sedentary controls (40±7 years). Standard transthoracic echocardiography was performed: M mode, 2D, tissue and colour Doppler. Deformation parameters were studied by speckle tracking at short axis view basal (B), medium (M) and apical (Ap) levels and apical 4 and 2 chamber view of the left ventricle (LV) and right ventricle (RV). Longitudinal, radial and circumferential strain (S) and strain rate (SR) were measured as well as rotation (Rot) and untwisting (UT) at basal and apical levels.

Results: Left ventricular ejection fraction and volumes were significantly higher in the athlete group. There were no differences in conventional diastolic parameters. Longitudinal and circumferential left ventricular strain and torsion were higher in the athlete group (p=0.02, p=0.005 and p=0.03 respectively). In addition, radial strain was lower in the athlete group. Right ventricular diameters, tricuspid annular plane systolic excursion (TAPSE) and longitudinal deformation were significantly higher in the athlete group (p=0.01, 0.002 y 0.006 respectively).

Conclusion: Myocardial deformation parameters, studied by longitudinal and circumferential strain, as well as ventricular torsion are greater in athletes as compared to sedentary individuals. This increased myocardial longitudinal and circumferential deformation and ventricular torsion might represent an adaptive response to regular intense physical exercise.

Keywords: Echocardiography. Exercise. Strain. Ventricular torsion. Myocardial deformation

Introduction

Regular intense physical exercise is associated with a series of changes in myocardial morphology and function which have been described as the “athlete’s heart”[1-5]. These changes include an increase in left ventricular (LV) chamber size, wall thickness, ventricular mass and stroke volume. This remodelling reflects myocardial adaptation to adverse hemodynamic conditions during intense physical exercise.

Longitudinal, circumferential and radial deformation studies, as well as myocardial torsion and untwisting by speckle tracking techniques allow a different approach to understanding systolic and diastolic myocardial physiology [6-10]. The rotational movement in myocardial torsion and untwisting is caused by the helical disposition of the myocardial fibers already described by Torrent Guasp[11-12]. This movement constitutes an important aspect of ventricular biomechanics, which allow the high pressures generated to be homogeneously distributed along the myocardial wall [11-15].

Deformation parameters can be expected to be different in athlete and sedentary myocardium due to their relationship with both systolic and diastolic heart function and structure.

This study’s primary aim is to compare the athlete’s heart with that of sedentary individuals using a classic echocardiographic[16] approach as well as new myocardial deformation and torsion study techniques. A secondary aim is to describe the biomechanical changes of the heart associated with regular intense exercise.

Methods

Two groups were studied: the athlete group and a control group of sedentary individuals. The athlete group was comprised of healthy, male long distance runners with a training program of more than 40km per week for at least the last three years, between 20 and 55 years of age. Exclusion criteria were cardiovascular or metabolic disorders, cardiovascular risk factors or any pharmacological treatment. The control group consisted of healthy, sedentary individuals of similar age. Sedentary was defined as a type of lifestyle with no or very irregular physical activity that includes a majority of time sitting or resting.

Study inclusion was performed by clinical history, physical examination, blood pressure measurement and a 12 lead-ECG. A code number was randomly assigned to each participant. Echocardiograms were performed and later interpreted off-line without knowledge to the interpreter as to which study group the subject belonged. Thereby, the study was blind to the investigator interpreting the echocardiograms. Thirty-eight athletes and thirty sedentary controls were included.

Standard transthoracic echocardiography was performed according to the guidelines of the American Society of Echocardiography and the European Society of Echocardiography[16], on Vivid 7 Dimension GE Medical Systems, Milwaukee, Wisconsin. The images were digitally stored and analyzed off-line (EchoPac PC, GE Vingmed). The filters were set to exclude high-frequency signals and the Nyquist limit was adjusted to a range of 10 to 15 cm/s. Gain and sample volume were minimized to obtain clear tissue signal. TDI Doppler was used to measure the early diastolic velocity (E’) of the mitral annulus at septal and lateral level.

The frame rate for TDI measurements was >100/s. For 2 dimensional (2D) and 2D strain analysis, we used frame rates of 70/s to 90/s. Transmitral pw-Doppler inflow was measured to obtain E peak, A peak, and E/A ratio. The E deceleration time (DT) was derived from the transmitral pw-Doppler inflow at the tips of the mitral leaflets. E’ peak and A’ peak were measured at the lateral and septal mitral anulus.

TDI measurements were assessed in the apical 4-chamber view. 2D strain variables were measured in the parasternal short-axis at basal, medium and apical levels and apical 4-chamber and 2-chamber views. For 2D and TDI measurements, 3 beats were stored and analyzed.

As a marker of right heart function, tricuspid annular plane systolic excursion (TAPSE) was measured.

Measurement of myocardial deformation parameters (strain imaging) by speckle tracking was done off-line using the EchoPac Dimension 06 at B, M and Ap level in the short axis view as well as from apical 4 and 2 chambers view. Longitudinal, radial and circunferential strain and strain rate were analyzed as well as rotation and untwisting. The initial yellow point was adjusted to the beginning of the QRS. Torsion was defined as the net difference between Rot Ap and Rot B (**Table I**). In the apical four-chamber view, the endocardium of the right ventricle (RV) was manually drawn in end-systole thereafter the endocardial borders were automatically tracked throughout the cardiac cycle. Once approved by the reading analyst, the software displayed longitudinal strain and strain rate for the respective segments and the global longitudinal strain and strain rate. (**Figure 3**).

Statistical analysis

All numeric variables were expressed as mean ± Standard Deviation (SD). Statistical and power analyses were performed using IBM SPSS 15 Statistics software. The comparisons among athletes and controls were performed

Table I: Myocardial deformation parameters analyzed.

	Basal	Medium	Apical
Short axis	Rot, T-Rot, UT, T-UT, S cir, SR cir, S rd, SR rd	S rd, SR syst. rd, SR dias rd.	Rot, T-Rot, UT, T-UT, S cir, SR cir, S rd, SR rd

Rot: Rotation, T-Rot: Rotation time, UT: Untwisting, T-UT: Time to untwisting, S cir: Circunferential strain, SRcir: Circunferential strain rate, S rd: Radial strain, SR rd: Radial strain rate.

	4C	2C	RV
Apical	S long, SR syst. Long, SR dias. Long.	S long, SR syst. Long, SR dias. Long.	S long, SR syst. Long, SR dias. Long.

S long: Longitudinal strain, SR syst. Long: Longitudinal systolic strain rate, SR dias. Long: Longitudinal diastolic strain rate.

Table II: Baseline characteristics of study participants.

	Athletes n=38	Controls n=30	p
Age	**42±8**	**40±7**	**ns**
BSA m^2	1.8±0.14	1.97±0.14	0.0001
BPs mmHg	128±12	129±10	ns
BPd mmHg	77±9	79±8	ns
ST mm	8.9±2.2	8,6±2,0	ns
PWT mm	8.4±1.4	8.5±1.7	ns
LV mass g	199±39	172±48	0.01
LVEDD mm	53±5	51±5	ns
LVESD mm	33±6	33±4	ns
EF (Teicholz)	67±8	65±7	ns
HR bpm	52±9	68±10	0.0001
Weight (Kg)	67.8±8	79.4±10	0.0001
ILV mass g/m^2	110±21	87±20	0.0001
LVOT mm	22±1.6	22±1.6	ns
TAPSE mm	23±3	21±3	0.0015
RVTD mm	37±7	33±6	0.01
LLVD mm	87±9	82±11	0.056
EF (S)	64±7	60±7	0.02
LVSVI ml/m^2	30±9	23±7	0.0005
LVDVI ml/m^2	82.7±17	57.0±13	0.0001
LAVIml/m^2	34.5±9	21.4±7	0.0001

BSA: Body surface area, BPs: Systolic blood presure, BPd: Diastolic blood presure, ST: Septal thickness, PWT: Posterior wall thickness, LVEDD: Left ventricular end diastolic dimension, LVESD: Left ventricular end systolic dimension, EF(s): Ejection fraction (Simpson's). LAVI: Left atrial volume index, LVDVI: Left ventricle diastolic volume index, LVSVI: Left ventricle systolic volume index, LLVD: Longitudinal left ventricular dimension. ILV mass: Indexed left ventricular mass, LVOT: Left ventricle outflow tract, TAPSE: tricuspid annular plane systolic excursion, RVTD: Right ventricle transverse dimension.

using Student's unpaired t-test. The correlation analysis was performed using Pearson's coefficient. Multivariate analysis was conducted by logistic regression method.

Results

No differences were found in general baseline characteristics (age, blood pressure) between the two groups. No differences were found in left ventricular diameter and myocardial thickness but indexed left atrial and ventricular volumes and ventricular mass were greater in athletes. Ejection fraction calculated by Simpson's method was higher in athletes. All were statistically significant (p <0,05). (**Table II**).

Doppler hemodynamic parameters are represented in **Table III**. No statistically significant differences were found except in heart rate and derivated parameters such as E wave, E/A and LV outflow tract TVI (time velocity integral).

Myocardial deformation parameter analysis revealed that longitudinal strain from 4 and 2 chamber view was higher in the athlete's group (21.2±3.1% vs 19.6±2.7%), as well as circumferential strain at B level (16.6±5.1% vs 13.1±4.8% p=0.005). In contrast, radial strain rate at M level was lower in the athlete group (1.64±0.43s vs 1.98±0.69s p=0.01).

Basal rotation was higher in athletes (6.0±2.8º vs 4.3±2.3º p=0.007) and UT B was higher in athletes without reaching statistical significance. (70± 22 vs 52± 18 g/s p=0.06). At apical level no differences were found. Myocardial torsion was greater in the athlete group at the expense of Rot B (19.7±7.2 ° vs 15.8±7.3° p=0.03). (**Table IV**)

RV diameters were larger in the athlete group (37±7mm vs 33±6mm, p=0.01). Statistically significant differences were found in right heart function parameters. TAPSE was higher in the athlete's group (23±3mm vs 21±3mm, p=0.002) as well as longitudinal strain (24.0±3.4% vs 21.3±5.4%, p=0.006). No differences were found in RV systolic and diastolic strain rate. (**Table V**)

Ventricular torsion analysis showed a positive correlation with ejection fraction, circunferential strain at basal (R: 0,41, p=0.01) and apical level (R: 0,77, p=0.0001) as well as circunferential strain rate at apical level (R: 0,83 , p=0.0001).

Multivariate logistic regression analysis showed indexed volumes, circumferential basal strain and body surface area as independent variables (**Table VI**).

Discussion

The results of the present study evidence morphological and functional differences in the athlete's heart. Morphological adaptations have been described previously: higher ventricular and atrial volumes and ventricular mass in athlete's heart as well as greater ejection fraction measured by Simpson's method[17-20]. The main findings of the present study are the differences in deformation parameters, greater in longitudinal and circumferential strain but lower in radial strain rate in the athlete group as compared with sedentary controls. These deformation parameter changes in the athlete's heart could represent an increased ventricular efficiency as well as increased myocardial contractile reserve. Contrary to other findings[19], myocardial torsion/untwisting was greater in the athlete's heart, which is congruent with the higher circumferential strain and reinforces the idea of a greater intrinsic contractility and greater global myocardial functionality of the athlete's heart. The lower radial strain found at rest might be explained by the need for less transverse deformation to reach adequate levels of cardiovascular function in the athlete's heart. The trained heart will have this reserve in radial deformation that can be utilized during exercise. The athlete's heart will have the possibility to improve and optimize LV function during exercise by incrementing radial deformation. Differences in apical rotation were probably not statistically significant due to a methodological-technical difficulty during the echocardiographic study to identify the true apex in the parasternal short axis view. Analyzing deformation parameters in short axis views that might have been medially displaced and falsely interpreted as true apex could have influenced the rotation results. LV Torsion was measurable in every subject but we estimate that in about 15% of the studies we weren't able to reach the true apex. Contrary to what was expected when analyzing diastolic function parameters, no differences were found in mitral annulus velocity, which suggests a similar grade of relaxation at rest, although different results might be found during exercise. On the other hand, longitudinal diastolic strain rate was higher among athletes, representing higher velocities to baseline length recovery indicative of better relaxation. In the multivariate analysis, circumferential strain at basal level is an independent variable not related to athletes' higher left ventricular volume index. Left ventricular Torsion has a relation with basal circumferential strain. Both were higher in athletes and it is possible that are not related to the higher cardiac cavities volumes.

Figure 1: Left ventricle longitudinal strain and strain rate from apical 4 chamber view.

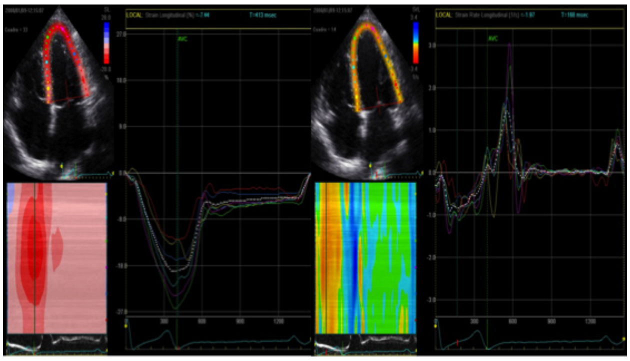
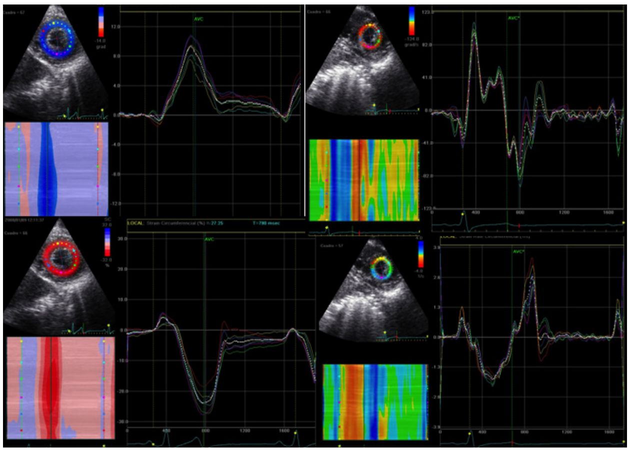
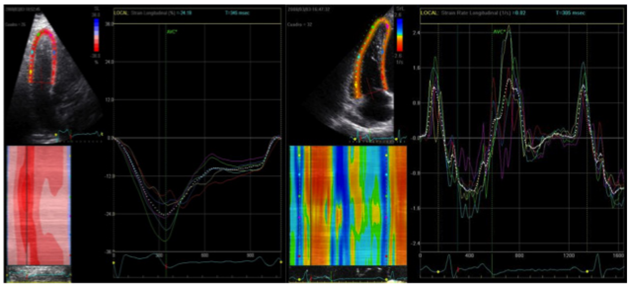


Figure 2: Left ventricle apical and basal rotation. Basal and apical circunferential strain and strain rate.



This study shows that physical exercise not only affects the left ventricle. Statistically significant differences were

Figure 3: Right ventricle longitudinal strain and strain rate from apical 4 chamber view.



found in right heart size, function parameters (TAPSE) and RV deformation parameters in the athlete group. Physical exercise also affects the right heart producing increased size, systolic function and longitudinal deformation, which contribute to the athlete's heart better efficiency at rest and probably during exercise[20].

Table III: Results hemodynamic/Doppler parameters.

	Athletes	Controls	p
E cm/s	74±13	65±13	0.006
A cm/s	48±12	49±11	ns
E/A	1.6±0.4	1.4±0.3	0.01
DT ms	168±37	185±49	ns
IVRT ms	102±24	96±11	ns
LVOT TVI cm	23.8±3	18.9±3	0.0001
C.I. L/min/m^2	2.6±0.6	2.4±0.5	ns
Eam cm/s	9.7±2.0	9.5±2.0	ns
Eal cm/s	14.2±2.7	13.2±2.9	ns
Eap cm/s	11.9±2.2	11.3±2.3	ns
VpE cm/s	84±51	72±48	ns
PVs cm/s	57±12	54±13	ns
PVd cm/s	50±9	46±10	ns
S/D vp	1.2±0.3	1.2±0.3	ns
PVa cm/s	25±6	26±5	ns
E/Eap	6.3±1.3	5.9±1.3	ns

DT: Deceleration time. IVTR: Isovolumic relaxation time. LVOT TVI: Left ventricular outflow tract time velocity integral. C.I.: Cardiac index. Eap: Mean value of medial and lateral mitral annulus velocity. VpE: Mitral inflow propagation velocity. Eam: Medial mitral annulus veocity. Eal: Lateral mitral annulus velocity. PVs: Pulmonary vein systolic velocity. PVd: Pulmonary vein diastolic velocity. PVa: Pulmonary vein atrial flow reversal velocity.

Table IV: Myocardial deformation parameters.

	Athletes	Controls	p
B Rot º	6.0±2.8	4.3±2.3	0.007
A Rot º	13.7±7.3	11.5±6,4	ns
B UT º/s	70±22	52±18	0.06
A UT º/s	88±43	89±46	ns
B cir S %	16.6±5.1	13.1±4.8	0.005
TOR º	19.7±7.2	15.8±7.3	0.03
SR sys M /s	1.64±0.43	1.98±0.69	0.015
Long S 4c %	20.5±3.1	19.1±2.6	0.05
SR dias lon4c /s	1.49±0.25	1.32±0.32	0.02
Long S 2c %	21.9±3.1	20.1±2.8	0.02
SR dias long2c /s	1.53±0.27	1.33±0.32	0.006
RV LS %	24.0±3.4	21.3±5.4	0.01

BRot: Basal rotation, ARot: Apical rotation, BUT: Basal untwisting , AUT: Apical untwisting, BcirS: Basal circunferential strain, TOR: Torsion, SR sys M: Medial systolic strain rate, LongS 4c: Longitudinal strain four chambers view, SR dias lon4c: Longitudinal strain rate four chambers view, Long S 2c: Longitudinal strain two chambers view, SR dias long 2c: Longitudinal diastolic strain rate two chambers view, RV LS: Right ventricle longitudinal strain.

Table V: Right ventricle study.

	RVTD	TAPSE	RVLS	RVsysSR	RVdiasSR
Athletes	37 ±7 mm	23±3mm	24.0±3.4%	1.20±0.25 /s	1.33±0.2 /s
Control	33 ±6 mm	21±3mm	21.3±5.4%	1.17±0.25 /s	1.25±0.3 /s
	P=0.01	P=0.002	P=0.006	ns	ns

RVTD: Right ventricle transverse dimension. RVLS: Right ventricular longitudinal strain. RVsysSR: Right ventricular longitudinal systolic strain rate. RVdiasSR: Right ventricular longitudinal diastolic strain rate.

Table V: Right ventricle study.

	RVTD	TAPSE	RVLS	RVsysSR	RVdiasSR
Athletes	37 ±7 mm	23±3mm	24.0±3.4%	1.20±0.25 /s	1.33±0.2 /s
Control	33 ±6 mm	21±3mm	21.3±5.4%	1.17±0.25 /s	1.25±0.3 /s
	P=0.01	P=0.002	P=0.006	ns	ns

RVTD: Right ventricle transverse dimension. RVLS: Right ventricular longitudinal strain. RVsysSR: Right ventricular longitudinal systolic strain rate. RVdiasSR: Right ventricular longitudinal diastolic strain rate.

Table VI: Multivariate analysis.

		'p'	OR	CI 95,0% for OR	
				Inferior	**Superior**
	IN_LVIDd	0.047	1.095	1.001	1.198
	St_circ_B	0.054	1.169	0.998	1.370
	LAVI	0.024	1.153	1.019	1.305
	BSA	0.040	0.002	0.000	0.762

IN_LVIDd: Indexed left ventricular end-diastolic internal diameter. St_circ_B: Circunferential strain at basal level. LAVI: Left atrial volume index. BSA:Body surface area.

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