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Echocardiography

Reduced Right Ventricular Myocardial Strain in the Elite Athlete May Not Be a Consequence of Myocardial Damage. "Cream Masquerades as Skimmed Milk"

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Background: Latest research shows that the lower resting values of right ventricular (RV) myocardial % strain may represent a physiologic change rather than subclinical myocardial damage. Therefore, we assessed load-independent changes to the RV as a consequence of high intensity training by measuring the Isovolumic acceleration (IVA) of the free wall of the RV in conjunction with NT pro-BNP measured by an electrochemiluminescence assay. Methods: Seventeen controls (mean age 27 $\pm$ 4), 24 soccer footballers (mean age 24 $\pm$ 4), and 18 elite rowers (mean age 22 $\pm$ 4) were studied. Left ventricular (LV) and RV % strain were measured using two-dimensional (2D) speckle based automated functional imaging (AFI) software. RV free wall IVA was measured using pulsed-wave tissue Doppler at the lateral tricuspid annulus. Standard 2D echo were used to measured RV parameters including the Tei index (systolic and diastolic function) and the total annular plane systolic excursion (TAPSE) of the RV annulus. NT pro-BNP was measured by an electrochemiluminescence assay. Results: The RV diameter was increased in the footballers and elite rowers compared with controls ($P < 0.001$). RV wall size was greater in the elite rowers compared with controls and footballers ($P = 0.002$). The peak IVA of the RV was higher in the rowers, compared with the footballers and to controls ($P < 0.001$). The mean LV and RV % myocardial strain were lower in the elite athletes and the footballers compared with controls ($P < 0.001$). There was no difference in RV Tei index, levels of BNP, and TAPSE across all subjects. Conclusions: This study showed a significant increase in IVA of the RV of athletes despite reduced myocardial % strain and normal levels in NT-proBNP. This suggests that the decrease in % strain is not a consequence of myocardial damage, but may represents a part of the physiological response to endurance exercise. Therefore, a reduced IVA in a remodeled RV could herald a pathological response. (Echocardiography 2013;30:929-935)

Key words: athlete, right ventricle, strain, isovolumic acceleration, sub-clinical dysfunction

Although there is compelling evidence for the cardiovascular benefits of regular physical exercise, recent studies have shown that transient myocardial damage may occur during intense training regimes and prolonged exercise, especially in amateur participants.[1–4] A link between chronic intense physical exercise and right ventricular (RV) myocardial dysfunction has been reported in previous studies.[1] The echocardiographic measures of strain and strain rate (SR) can be used to quantify myocardial deformation and deformation rate, respectively. Recently, Teske et al.[5] demonstrated that strain and SR of the RV free wall were reduced in highly trained athletes relative to nonathletes; particularly in basal segments. This finding raises concern given that mild RV dysfunction has been associated with ventricular arrhythmias and a propensity to sudden cardiac death in elite endurance athletes.[6–8]

It has been demonstrated that strain and SR are reliable surrogates of global function and myocardial contractility.[9,10] However, deformation measures are influenced by ventricular load and geometry making comparisons between athletes and nonathletes difficult.[11] Moreover, the interpretation that reduced SR may reflect diminished RV contractility in athletes is at odds with their enhanced cardiac output during

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strenuous exercise.[12] However, La Gerche[13] and colleagues showed that the RV functional reserve is the same in both elite athletes and nonathletic individuals despite reduced strain in the athletes at rest thus suggesting physiological changes rather than subclinical myocardial damage. On the other hand, isovolumic acceleration (IVA) proved a more reliable surrogate of RV contractility in the setting of altered ventricular load.[14,15]

If one can assume that RV myocardial function must be preserved, if not enhanced, in well-trained athletes to enable greater augmentation of cardiac output during exercise, then this would suggest that strain and SR are unreliable measures of RV function at rest. We hypothesized that, compared with nonathletes; RV IVA would be maintained or enhanced in athletes thus providing a more physiologically plausible summary of function. Accordingly, we compared echocardiographic deformation imaging and IVA in highly trained athletes (semiprofessional footballers and elite endurance first team rowers) and nonathletes.

Methods:

Study population:

This study was conducted in close conjunction with the Human Performance laboratory and anatomy laboratory at Trinity College, Dublin and the Limerick Institute of Technology where the athletes were recruited from a national database. The study population was all male, and comprised of 3 groups, elite rowers (n = 18), semiprofessional footballers (n = 24), and healthy nonathlete controls (n = 17). The athletes were actively involved in training regimes during the time course of the study and no athlete was accepted into the study if training had ceased for more than 24 hours prior to the study. At the time of echocardiographic examination, the subject's height, weight, heart rate, and blood pressures were recorded and blood was taken. This study was approved by the local hospital ethics committee and informed consent was obtained from all participants.

High endurance elite rowers' training regimes:

At the time of data acquisition, all rowers were involved in preparation phase training programs for the rowers involved 3–8 hours of 3–4 "on water" low intensity base aerobic conditioning sessions per week, training in rowing techniques, alternating with 2–3 hours per week of higher intensity aerobic work on land-based rowing ergometers in or around the lactate anaerobic threshold. In addition, most rowers participated in up to 3 resistance-training sessions per week to build and maintain core strength, usually done on the same days as the high intensity aerobic ergometer work. The rowers also performed some aerobic cross training such as running, cycling or swimming on active recovery days.

Semiprofessional footballers' training regimes:

The footballers (soccer) participated in 2 full-team sessions per week with a mid-week and weekend match with active recovery sessions following match days. Thus, training or games occur 6–7 days a week, with sessions lasting 60–90 minutes, typically starting with warm up drills and dynamic stretching exercises, proceeding to speed endurance and agility drills, and then sport-specific skills and/or competitive games followed by cool down, core strengthening and flexibility exercises. In addition, most performed their resistance or further aerobic-type conditioning programs depending on their own individual fitness requirements a further 2–3 times a week.

Conventional echocardiographic measurements:

Echocardiography was performed on all subjects using a commercially available ultrasound system (Vivid 7 Dimension, GE Healthcare, Horten, Norway). All images were digitally stored for offline analysis and all Doppler and tissue Doppler velocities were recorded at a sweep speed of 100 mm/sec. Standard two-dimensional (2D) images were acquired according to the recommendations of the American Society of Echocardiography.[16] All left ventricular (LV) and RV dimensions were corrected for body surface area (BSA). LV mass was determined from septal and posterior wall thickness and LV end-diastolic dimensions. Left atrial (LA) size was determined as LA diameter on M-mode from the parasternal long-axis view. LV ejection fraction was calculated using the modified Simpson's biplane method. LV diastolic dysfunction was defined using the criteria provided by the Canadian Consensus Guidelines using transmitral Doppler (TMD) with and without the Valsalva maneuver.[17,18] The peak early (E) and late (A) transmitral filling velocities, the E/A ratio, E deceleration time and the isovolumic relaxation time (IVRT) with and without Valsalva maneuver were measured using conventional Doppler ultrasound. Pulsed-wave tissue Doppler imaging (TDI) velocities were assessed at 4 conventional sites around the mitral annulus in the apical four and two-chamber views, and averaged to give myocardial peak systolic (Sm) and peak early (Em) diastolic velocities. We assessed LV diastolic stiffness using an index we have previously reported, as the ratio of (E/Em)/LV end-diastolic diameter.[19,20] Right ventricular (RV) size was determined by the mid-RV cavity diameter in the apical four-chamber view, and RV free wall thickness from the

apical and subcostal four-chamber views. All contemporary guidelines on RV measurement clearly state that the lateral tricuspid (free wall) annulus is used for the measurement of tricuspid annular plane systolic excursion (TAPSE) and RV velocities.[21] The TAPSE was measured by M-mode echocardiography at the lateral tricuspid annulus.[22] The RV myocardial performance index (Tei index) was derived from the tricuspid valve opening and closure times, and the RV ejection time.[23] Systolic pulmonary arterial pressures were calculated using the modified Bernoulli equation.[24]

Isovolumic acceleration:
Myocardial acceleration during the isovolumic phase of contraction is relatively load-independent and has been proposed for the assessment of RV contractile function.[14] A 2 mm pulsed-wave TDI sample volume was placed at the annulus and in basal and mid-ventricular segments in the apical four-chamber view and averaged**.** RV myocardial velocities during peak systole, early and late diastole were recorded. RV IVA (Fig. 1) was calculated as the difference between baseline and peak myocardial systolic velocities divided by the time interval from onset of the myocardial velocity during isovolumic contraction to the time at peak velocity of this wave.[25] The potential effect of respiration was minimized by averaging multiple consecutive beats.

Two-dimensional speckle tracking and automated functional imaging (AFI):
Strain is a dimensionless parameter representing deformation of a myocardial segment relative to its original dimensions within a systolic time frame (Fig. 2). With standard 2D echocardiographic gray-scale imaging, reflected ultrasound gives rise to speckles of bright signals within the myocardium. Frame-by-frame tracking of the movement of these natural acoustic markers throughout the cardiac cycle allows assessment of temporal displacement between neighboring markers and quantification of myocardial deformation. AFI, incorporated into the custom software used for strain analysis, is a cross-correlation semiautomated technology that recognizes and tracks the myocardial region of interest, defined with minimal user input.

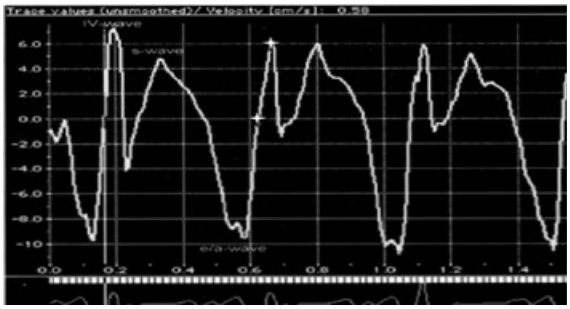

Figure 1. Right ventricular free wall isovolumic acceleration was measured using pulsed-wave tissue Doppler at the lateral tricuspid annulus. Difference between baseline and peak myocardial systolic velocities divided by the time interval from onset of the myocardial velocity during isovolumic contraction to the time at peak velocity of the wave.

We acquired 2D images of the LV and RV in the apical four and two-chamber views and examined longitudinal LV and RV strain, the lateral and septal annulus and the apex in each image. AFI software was then used to track the LV and RV myocardium after manually defining the endocardial border of the LV and RV. The software automatically divides the LV, RV free wall and interventricular septum into 3 equidistant segments. The average strain for each segment and a global longitudinal strain value was averaged from the 6 segmental strain values.[26–28] Every effort was made to ensure that the entire LV and RV were imaged with minimal foreshortening. Acquisition was made at frame rates at or close to the patient's own heart rate to facilitate optimal tracking of the myocardium during post processing. NT-proBNP was measured by an electrochemiluminescence assay using the automated assay of Roche Diagnostics (Elecsys proBNP, Roche, Indianapolis, IN, USA). The sensitivity of the test is 5 pg/mL. Intra- and inter-assay coefficients of variance at 175 pg/mL are 2.7% and 3.2%, respectively. There are no cross reactions with other hormones or pharmaceutical drugs. Normal $\leq$ 100 pg/mL.

Statistical methods:
Descriptive analyses are presented as means (standard deviations, [SD]) for continuous measures. Normality of data distribution was tested using a Kolomogorov–Smirnov test before applying parametric tests for data comparisons. Analysis of variance (ANOVA) was used to compare groups and Tukey's test for multiple comparisons was applied where there was found to be a significant difference between groups. With a sample size of approximately n = 17 in each group the study was powered at 80% and 5% significance level to detect a difference of 1 SD between any 2 groups. Inter-observer variability in M-mode measurements, Doppler parameters of diastolic function, LV and RV strain were assessed by 2 readers (Gerard King & Ibrahim Almuntaser) and measured by the interclass correlation co efficient (ICC). A P-value of <0.05 was considered statistically significant. All analyses were performed using SPSS (version 16, SPSS Inc, Chicago, IL, USA). The ICC was 0.969 which was highly significant, $P < 0.001$.

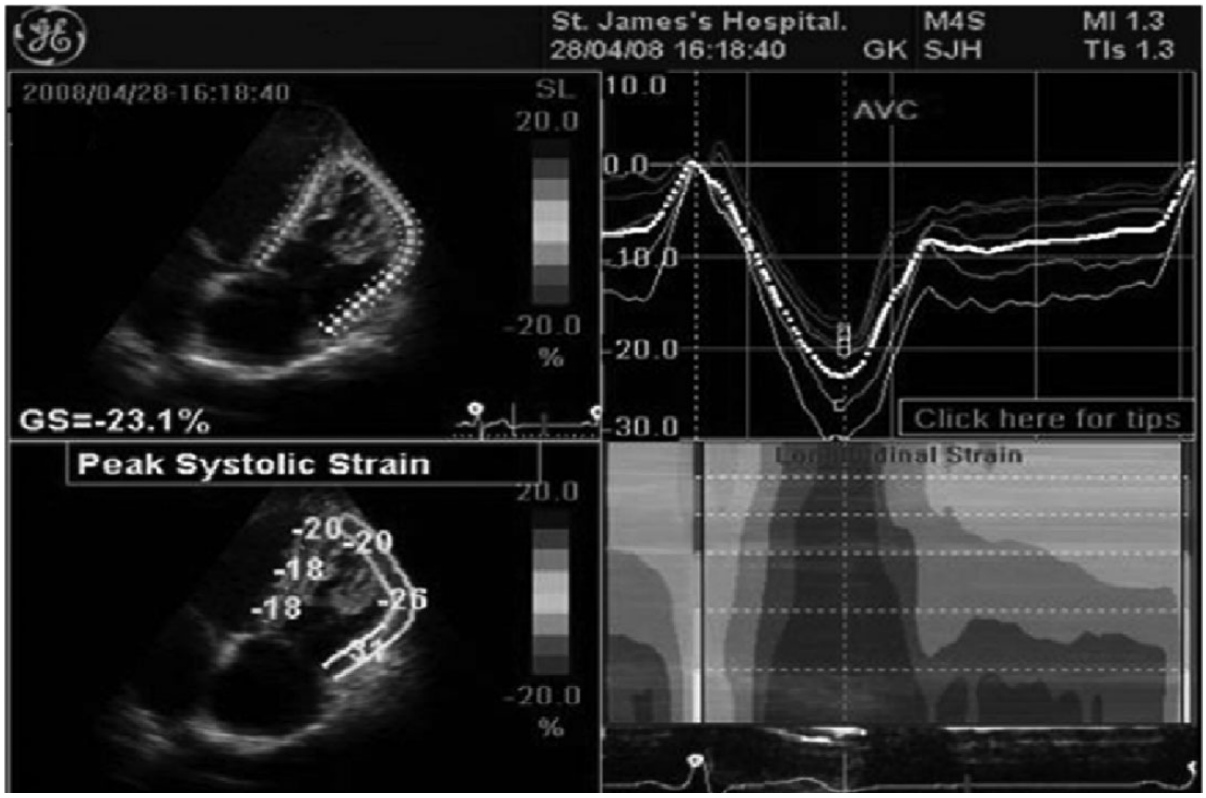

Figure 2. Right ventricular global longitudinal strain was measured using speckle based automated functional imaging software.

Results:

There were 17 controls, 24 footballers, and 18 elite rowers in the study. Demographic and clinical characteristics of the 3 groups (rowers, footballers, and controls) are presented in Table I and show there was a small significant difference in age between the 3 groups, with controls slightly older than rowers and footballers.

Table II summarizes LV measures comparing the 3 groups of rowers, footballers. The left ventricle was more dilated in rowers compared with controls but the differences between the groups was not statistically significant ($P = 0.336$). In addition, there was no significant difference in ejection fraction between the 3 groups. LV diastolic parameters demonstrate a reduced stiffness index in rowers compared with footballers and controls ($P = 0.007$ between groups). RV dimensions and functional measures are provided in Table III and greater differences between groups are apparent than for the LV. RV wall size was greater in the elite athletes compared with controls and footballers ($P = 0.002$). There was an increase in RV size with rowers, compared with footballers and controls ($P = 0.031$). The mean RV % strain was lower and RV mass higher in the elite rowers and the footballers compared with the control group ($P < 0.001$) while RV IVA was higher in the elite rowers, compared with the footballers and the controls ($P = 0.002$). The peak IVA of the RV was higher in the elite rowers, compared with the footballers and to controls ($P < 0.001$). There were no significant differences in myocardial performance index (Tei) or TAPSE. Interobserver variability, as assessed by interclass correlation coefficients, was strong for M-mode measurements ($r = 0.90$, $P < 0.0001$), Doppler parameters of diastolic function ($r = 0.96$ and $P < 0.0001$), IVA ($r = 0.96$, $P < 0.0001$), and biventricular strain ($r = 0.92$ and $P < 0.0001$).

Discussion:

As documented previously, we demonstrate that RV global strain is reduced in well-trained athletes particularly endurance athletes with dilation of the RV.[5] However, in our study we show that RV IVA is enhanced in the elite rowers compared with footballers and nonathletes. Differences in ventricular loading between rest and exercise may be greater for the RV than for the LV and may be greater still for athletes as compared with nonathletes.[29] This would suggest that resting measures provide limited insight into the work requirements of the RV during exercise. It should not necessarily be implied that reduced RV % global strain at rest also predicts reduced global

TABLE I

Demographic and Clinical Parameters of the Study Population: Mean (Standard Deviation)

Variable	Controls (n = 17)	Footballers (n = 24)	Rowers (n = 18)	ANOVA P-value
Age, years	27.06 (3.5)	24.3 (4.2)	22.38 (3.7)	0.114
Body mass index, kg/m^2	24.19 (3.9)	24.64 (1.8)	24.86 (1.6)	0.586
BSA, m^2	1.93 (0.2)	2.0 (0.2)	2.08 (0.13)	0.535
Heart rate, bpm*	71.71 (9.31)	61.29 (9.31)	61.72 (8.59)	0.002
QTc interval, msec*	380.5 (24.8)	405.2 (15.2)	399.0 (15.1)	0.001
Systolic blood pressure, mmHg*	128.4 (5.8)	132.9 (10.1)	120.8 (9.8)	<0.001
Diastolic blood pressure, mmHg*	79.7 (6.5)	79.3 (7.9)	73.5 (6.8)	NS

*In the table denotes statistically significant differences between the 3 groups after adjusting the following factors using linear regression analysis: age, BSA, and heart rate. In the case of heart rate the means are adjusted for age and BSA only. In the case of BSA the means are adjusted for heart rate and age only.
ANOVA = Analysis of variance; BSA = body surface area.

TABLE II

Left Heart Dimensions, and LV Systolic and Diastolic Function in the Study Population

					Multiple Comparison Tests		
Variable	Controls (n=17)	Footballers (n=24)	Rowers (n=18)	ANOVA P-value	P-value Control versus Footballer	P-value Control versus Rower	P-value Footballer versus Rower
LVEDD, mm	51.6 (4.6)	54.1 (3.8)	55.9 (5.2)	0.336			
LV mass/BSA indexed, g/m^2	99.2 (21.1)	111.3 (26.0)	113.1 (26.1)	0.172			
LA size, mm	34.6 (4.5)	33.6 (3.8)	34.3 (5.5)	0.479			
Ejection fraction, %	65.4 (5.6)	62.9 (4.4)	63.9 (7.3)	0.285			
Deceleration time, msec	193.1 (5.3)	180.8 (28.7)	152.6 (27.9)	0.001	NS	0.001	0.003
Transmitral E/A ratio	1.9 (0.5)	2.1 (0.5)	2.2 (0.4)	0.804			
IVRT, msec	81.5 (7.7)	76.4 (6.4)	72.4 (5.1)	0.012	NS	0.004	NS
Em velocity, cm/sec	17.5 (2.5)	18.2 (2.7)	18.6 (3.8)	NS			
LV stiffness index	1.1 (0.3)	1.0 (0.3)	0.8 (0.2)	0.007	0.004	NS	0.024

Values are mean (standard deviation), regression analysis was used to adjust for age, heart rate, BSA.
BSA = body surface area; NS = nonsignificance; LV = Left ventricular; LA = Left atrial; IVRT = isovolumic relaxation time.

% strain during exercise. Moreover, we demonstrate that athletes had greater RV dilation and it has been demonstrated that % RV global strain reduces with increasing ventricular size.[30] We would contend that % RV global strain should be used with caution as a surrogate of underlying myocardial contractility.

Vogel et al[14] suggested that IVA could be considered a surrogate of (dP/dt) max because both indices describe the rate of change of contractile force during isovolumic contraction. Similarly, Missant et al[14] demonstrated the superiority of IVA over deformation measures when RV contractility was assessed in the context of altered ventricular load. In a recent study,[31] IVA remained the same when peak systolic strain, measured at the basal segment of LV septum, increased significantly, and the conclusion was that IVA is unchanged following significant increases in preload in healthy subjects, and thus is a potentially useful measure of global LV contractility. Some authors have suggested that athlete's RV function is in fact superior to nonathletes during exercise when it

TABLE III

Right Ventricular 2D, Doppler, and Tissue Doppler Imaging Velocities in the Study Population

Variable	Controls (n = 17)	Footballers (n = 24)	Rowers (n = 18)	ANOVA P-value	Multiple comparison tests: P-value Control versus Footballer	P-value Control versus Rower	P-value Footballer versus Rower
RV diameter, mm	22.5 (6.9)	33.5 (7.7)	34.0 (6.8)	0.031	0.009	NS	NS
RV wall thickness, cm	0.4 (0.1)	0.4 (0.1)	0.5 (0.3)	0.014	0.011	NS	0.011
RV global longitudinal strain, %	25.4 (2.6)	21.8 (2.8)	20.1 (2.8)	<0.001	NS	<0.001	0.001
RV myocardial performance index	0.3 (0.1)	0.3 (0.1)	0.4 (0.2)	NS			
TAPSE, mm	25.9 (3.2)	25.8 (3.2)	26.5 (3.2)	NS			
Pulmonary arterial pressures, mmHg	21.1 (4.4)	23.4 (6.0)	24.1 (5.1)	NS			
IVA, m/sec^2	1.3 (0.5)	1.5 (0.3)	2.0 (0.4)	NS	NS	0.017	0.001
BNP	<100pg/mL	<100pg/mL	<100pg/mL	NS	NS	NS	NS

Values are mean (standard deviation), Regression analysis was used to adjust for age, heart rate, BSA, QTc interval, systolic blood pressure, diastolic blood pressure.
NS = nonsignificance; TAPSE = tricuspid annular plane systolic excursion; RV = right ventricular; IVA = Isovolumic acceleration.

is influenced positively by preload.[32] This would suggest that an increase in IVA is an indicator of a good resting RV response to intense exercise training, which is not sensitive to loading conditions but to its precontractile ability which is an overall indicator of its global ability or global reaction to subclinical changes that are transient rather than permanent. Therefore, an increase in IVA at rest may be a good indicator of a positive response by the RV cavity to exercise, independent of preload variations.

The prediction of "true" RV dysfunction is important in athletes and a noninvasive technique that allows for this distinction would be an important clinical tool in sports cardiology. Our study is limited by small numbers of both athletes and controls. The study was performed at rest; it would be interesting to assess these parameters at peak exercise and in recovery.

In conclusion, our study demonstrates that RV subclinical changes do occur after training in elite endurance rowers. Reduced global % strain occurs in this cohort, is more prevalent in the elite endurance athlete than in the semiprofessional footballers or controls and may reflect ventricular loading and geometry rather than underlying myocardial properties. There is also an increase in IVA of the free wall of the right ventricle in the elite endurance rower. We therefore hypothesize that reductions in IVA may be a more robust indicator of myocardial pathology, although this remains to be tested.

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