The Role of Three-Dimensional Echocardiography in the Assessment of Right Ventricular Dysfunction after a Half Marathon: Comparison with Cardiac Magnetic Resonance Imaging

Sacha R. Oomah, BSc, Negareh Mousavi, MD, FRCPC, Navdeep Bhullar, BSc, Kanwal Kumar, MD, Jonathan R. Walker, MSc, Matthew Lytwyn, BSc, Jane Colish, BSc, Anthony Wassef, MD, Iain D. C. Kirkpatrick, MD, FRCPC, Sat Sharma, MD, and Davinder S. Jassal, MD, FRCPC,
Winnipeg, Manitoba, Canada

Background: Although marathon running is associated with transient right ventricular (RV) systolic dysfunction as detected by two-dimensional transthoracic echocardiography, quantitative assessment of the right ventricle is difficult because of its complex geometry. Little is known about the use of real-time three-dimensional echocardiography (RT3DE) in the detection of cardiac dysfunction after a half marathon. The aim of this study was to assess the extent of RV dysfunction after the completion of a half marathon using cardiac biomarkers, RT3DE, and cardiac magnetic resonance imaging (CMR).

Methods: A prospective study was performed in 15 individuals in 2009 participating in the Manitoba Half Marathon. Cardiac biomarkers (myoglobin, creatine kinase–MB and cardiac troponin T) were assessed and RT3DE and CMR were performed 1 week before, immediately after, and 1 week after the race.

Results: At baseline, cardiac biomarkers and ventricular function were within normal limits. Immediately following the half marathon, all patients demonstrated elevated cardiac troponin T levels, with a median value of 0.37 ng/mL. RV ejection fraction, as assessed by RT3DE, decreased from 59 ± 4% at baseline to 45 ± 5% immediately following the race ($P < .05$). On CMR, RV end-diastolic volume increased after the half marathon, and the RV ejection fraction was reduced, at 47 ± 5% compared with 60 ± 2% at baseline ($P < .05$). There were strong linear correlations between RV ejection fraction assessed by RT3DE and CMR at baseline and after the half marathon ($r = 0.69$ and $r = 0.87$, $P < .01$, respectively).

Conclusions: Compared with CMR, RT3DE is a feasible and reproducible method of assessing transient RV dysfunction in athletes completing a half marathon. (J Am Soc Echocardiogr 2011;24:207-13.)

Keywords: Real-time three-dimensional echocardiography, Cardiac magnetic resonance imaging, Half marathon, Endurance sport, Cardiac biomarkers

Marathon running has become increasingly more popular over the past decade, for both amateur and elite athletes. Research into the short-term consequences of endurance exercise on cardiac function, especially the right ventricle, has recently increased. Previous studies have found that the elevation of cardiac biomarkers in marathon runners, in particular myoglobin, creatine kinase (CK), and cardiac troponin T (cTnT), correlate with transient changes in right ventricular (RV) function as assessed by echocardiography.[1-14] Although multiple studies have demonstrated two-dimensional echocardiographic evidence of transient RV systolic and diastolic abnormalities after endurance sports, quantitative assessment of the right ventricle is difficult because of its complex geometry.[14-19]

The recent introduction of real-time three-dimensional echocardiography (RT3DE) has shown to be a feasible and reliable method of assessing RV ejection fraction (RVEF). A number of studies have compared RT3DE to cardiac magnetic resonance imaging (CMR) in the assessment of RV volumes and RVEF, demonstrating a high correlation with excellent intraobserver and interobserver reliability.[19-24] As a result, RT3DE has proven to be a cost-effective, a less invasive, and an accurate means of assessing RV function in a variety of cardiac disorders. Little is known, however, about the utility of RT3DE in the noninvasive assessment of the right ventricle after a marathon.

From the Institute of Cardiovascular Sciences, St. Boniface Research Centre (S.R.O., N.M., N.B., J.R.W., M.L., J.C., D.S.J.), the Section of Cardiac Surgery, Department of Cardiac Sciences (K.K.), the Section of Respiratory Medicine and Critical Care (S.S.) and Section of Cardiology (D.S.J.), Department of Internal Medicine (A.W.), and the Department of Radiology (I.D.C.K., D.S.J.), University of Manitoba, Winnipeg, Manitoba, Canada.

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Reprint requests: Davinder S. Jassal, MD, FACC, FRCPC, Section of Cardiology, Department of Internal Medicine, Room Y3010, 409 Tache Avenue, St. Boniface General Hospital, Winnipeg, MB R2H 2A6, Canada (E-mail: *djassal@sbgh.mb.ca*).

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Abbreviations

CK = Creatine kinase

CMR = Cardiac magnetic resonance imaging

cTnI = Cardiac troponin I

cTnT = Cardiac troponin T

FAC = Fractional area change

LV = Left ventricular

RT3DE = Real-time three-dimensional echocardiography

RV = Right ventricular

RVEF = Right ventricular ejection fraction

TAPSE = Tricuspid annular plane systolic excursion

TTE = Transthoracic echocardiographic

Recently, CMR has been used to validate RV systolic dysfunction following a full marathon.[25-27] Similar to previous echocardiographic studies,[14-19] CMR demonstrated transient RV systolic dysfunction immediately after a marathon that recovered within weeks.[25-27] The absence of delayed enhancement of the myocardium using CMR also revealed no evidence of permanent injury due to the strenuous exercise of full marathon running.[25] Whether transient RV systolic dysfunction occurs in the setting of a shorter distance such as a half marathon, however, remains ill defined.

The objectives of the current study were twofold: (1) to assess the extent and severity of changes in RV function following the completion of a half marathon, using serial cardiac biomarkers, RT3DE, and CMR, and (2) to determine the accuracy of RT3DE for determining RV dysfunction after a half marathon compared with CMR.

Table 1 Patient clinical characteristics (n = 15)

Characteristic	Baseline	After the race
Age (y)	22–39	
Gender		
Male	7 (47%)	
Female	8 (53%)	
Weight (kg)	70 ± 11	70 ± 11
Heart rate (beats/min)	66 ± 13	97 ± 11
SBP (mm Hg)	132 ± 13	118 ± 13
DBP (mm Hg)	74 ± 6	68 ± 4

Data are expressed as range, as number (percentage), or as mean ± SD.
DBP, Diastolic blood pressure; *SBP*, systolic blood pressure.

Table 2 Summary of serial cardiac biomarkers for total population (n = 15)

Characteristic	Baseline	After the race	1 week after the race
Myoglobin (mg/L)	25 (18–82)	698 (552–2,100)*	65 (44–88)
CK (U/L)	120 (117–190)	625 (441–1,922)*	210 (156–462)
cTnT (ug/L)	<0.01	0.37 (0.26–0.74)*	<0.01

Data are expressed as median (interquartile range).
*P < .05, after the race vs baseline.

METHODS

Study Population

A prospective study involving 15 healthy, nonelite volunteers participating in the 2009 Manitoba Half Marathon was performed. Subjects aged 18 to 40 years who completed the race were included. Patients with histories of coronary artery disease, hypertension, smoking, elevated lipids, diabetes, and/or contraindication to undergo CMR were excluded.

Cardiac Biomarkers

Myoglobin, CK, and cTnT were evaluated at three separate time points: (1) 1 week before the race, (2) immediately after the race, (3) and 1 week after the race. Myoglobin and CK levels were determined using a Roche Elecsys and a Roche 917 analyzer, respectively (Roche Diagnostics GmbH, Mannheim, Germany). Quantitative determination of cTnT levels was performed using a third-generation Roche Elecsys assay.

Echocardiography

All subjects underwent baseline transthoracic echocardiographic (TTE) imaging 1 week before the race, immediately following the race, and 1 week after race completion. Following completion of the half marathon, each patient was immediately transferred to the study hospital for performance of the TTE exam. The transfer time from the half marathon site to the study hospital was 10 min. All TTE studies were performed immediately upon arrival at the study hospital. All patients underwent TTE imaging using a GE Vivid 7 (GE Healthcare, Milwaukee, WI) at each time point. Left ventricular (LV) and left atrial cavity dimensions indexed to body surface area were determined from two-dimensional images in accordance with the guidelines of the American Society of Echocardiography.[28] RV cavity dimensions, RV fractional area change (FAC), and tricuspid annular plane systolic excursion (TAPSE) were also determined. Continuous-wave Doppler was used to measure the peak velocity across the tricuspid valve, and the maximal pulmonary arterial systolic pressure was estimated using the simplified Bernoulli equation.

Real-time three-dimensional TTE imaging was performed using a dedicated broadband, wide-angle, matrix-array transducer to acquire the entire RV cavity within the pyramidal scan volume. Acquisition of full-volume data sets was triggered to the R wave of every cardiac cycle to allow for an acquisition time of four heartbeats during an adequate breath hold. The subvolumes were automatically stitched to a sequence of full three-dimensional volumes covering the entire right ventricle and stored digitally for offline analysis using TomTec software (TomTec Imaging Systems, Unterschleissheim, Germany), as previously described.[29]

CMR

CMR was performed on all study participants at baseline and within 24 hours of completion of the half marathon using a 1.5-T scanner (Avanto; Siemens Medical Solutions, Erlangen, Germany). Specifically, following acquisition of the TTE images after the half marathon, each participant underwent CMR on an hourly basis, until all 15 studies were completed. Cine balanced steady-state free-precession short-axis images then encompassed the entire left ventricle from the base to the apex (stack of 10 sequential short-axis slices; repetition time, 64 ms; echo time, 1 ms; flip angle, 80°; slice thickness, 8 mm; interslice gap, 1.6 mm; matrix size, 192 × 132) to obtain the LV ejection fraction. To evaluate for myocardial edema, dark-blood T2-weighted turbo spin-echo short-axis images were obtained (repetition time, 1800–2100 ms; echo time, 74 ms; slice thickness, 8 mm; interslice gap, 4 mm; matrix size, 256 × 175). Late gadolinium enhancement images were obtained after 10 min of 0.2 mmol/kg injection

Table 3 Echocardiographic data in the patient population (n = 15)

Echocardiographic parameter	Baseline	After the race	Follow-up	*P*
LV parameters (2D TTE)				
LVEDD (mm)	51 ± 5	50 ± 4	51 ± 3	.94
LVESD (mm)	32 ± 6	32 ± 5	33 ± 4	.76
LVEDV (mL)	115 ± 17	112 ± 15	108 ± 28	.65
LVESV (mL)	39 ± 12	41 ± 23	40 ± 14	.54
IVS (mm)	9 ± 1	8 ± 1	9 ± 2	.65
PWT (mm)	9 ± 1	8 ± 1	9 ± 2	.49
LVEF (%)	64 ± 3	62 ± 6	63 ± 4	.63
LV mass/BSA (g/m^2)	101 ± 12	102 ± 22	98 ± 18	.52
LA parameters (2D TTE)				
LA diameter (mm)	37 ± 3	37 ± 2	36 ± 5	.64
LA volume (mL)	43 ± 12	45 ± 15	41 ± 11	.42
RA and RV parameters (2D TTE)				
RA volume (mL)	41 ± 12	57 ± 14*	38 ± 12	<.05
RVEDD (mm)	31 ± 2	41 ± 2*	30 ± 3	<.05
RV diastolic area (mm^2)	13 ± 4	17 ± 2*	12 ± 3	<.05
RV systolic area (mm^2)	7 ± 2	11 ± 4*	6 ± 2	<.05
RV FAC (%)	41 ± 2	33 ± 4*	41 ± 5	<.05
TAPSE (mm)	2.7 ± 0.4	1.7 ± 0.3*	2.6 ± 0.4	<.05
RV parameters (RT3DE)				
RVEDV (mL)	125 ± 26	186 ± 24*	125 ± 23	<.05
RVESV (mL)	50 ± 18	100 ± 21*	52 ± 19	<.05
RVEF (%)	59 ± 4	45 ± 5*	58 ± 4	<.05

Data are expressed as mean ± SD.
BSA, Body surface area; *IVS*, interventricular septum; *LA*, left atrial; *LVEDD*, LV end-diastolic diameter; *LVEDV*, LV end-diastolic volume; *LVEF*, LV ejection fraction; *LVESD*, LV end-systolic diameter; *LVESV*, LV end-systolic volume; *PWT*, posterior wall thickness; *RA*, right atrial; *RVEDD*, RV end-diastolic diameter in parasternal long-axis view; *RVEDV*, RV end-diastolic volume; *RVESV*, RV end-systolic volume; *TTE*, transthoracic echocardiography; *2D*, two-dimensional.
*P < .05, after the race vs baseline.

of gadolinium diethylenetriamine pentaacetic acid (Magnevist; Schering AG, Berlin, Germany) using a T1-weighted inversion recovery–prepared multislice true fast imaging with steady-state precession sequence with magnitude and phase-sensitive reconstruction. Images were acquired sequentially in the short axis, followed by horizontal and vertical long-axis images (repetition time, 700 ms; echo time, 1.0 ms; flip angle, 40°; slice thickness, 8 mm; interslice gap, 1.6 mm; matrix size, 192 × 144). Quantitative analysis was performed using dedicated computer software (CMR^{42} release 3.1.2; Circle Cardiovascular Imaging, Calgary, AB, Canada).

Statistical Analysis

Data are summarized as mean ± SD, number (percentage), or median (interquartile range). Paired Student's t tests were used to compare continuous variables. Chi-square and Fisher's exact tests were applied to compare categorical variables. One-way analysis of variance (nonparametric with Dunn testing) was used to compare baseline, immediate, and 1-week postrace cardiac biomarker and echocardiographic values. Linear regression analysis and Bland-Altman plots were used to compare RV volumes and RVEF between the various imaging modalities. The Bland-Altman method is a plot of the differences of the data on a chart with mean difference ± 1.96 × standard deviation of the differences. The 95% agreement limits are ±1.96 × standard deviation of the differences. Agreement between intraobserver and interobserver variability of the LV volumes and LV ejection fraction between the imaging modalities was computed from the absolute differences between repeated measurements using the Mann-Whitney U test. P values < .05 were considered statistically significant. SAS version 8.01 (SAS Institute Inc., Cary, NC) was used to perform the analysis.

RESULTS

Of the 3,953 amateur athletes (1,971 men; mean age, 35 ± 13 years) who completed the 2009 Manitoba Half Marathon, the mean finishing time was 137 ± 26 min. Our study population of 15 participants (seven men; mean age, 32 ± 6 years) completed the half marathon with an average finishing time of 130 ± 24 min. The start time of the Manitoba Half Marathon was 7 AM, and each study participant underwent TTE imaging 10 min after crossing the finish line. The first CMR was scheduled at 9 AM, and subsequent exams were performed on an hourly basis until all 15 participants had completed the study. The mean heart rates at the time of the TTE and CMR exams after the race were 97 ± 11 and 71 ± 12 beats/min, respectively. The patients were mildly trained, with a mean training distance of 19 ± 11 mi/week and a mean training time of 5 ± 3 hours/week for the 14 ± 6 weeks before the half marathon. Of the 15 subjects, only three had previously participated in a marathon. All 15 participants drank fluids freely during the race. The weights of the subjects did not change significantly after the half marathon (Table 1).

Myoglobin, CK, and cTnT levels were all normal at baseline (Table 2). After completion of the half marathon, myoglobin and CK levels increased significantly compared with baseline (Table 2). Although

Table 4 CMR data in the patient population after the marathon (n = 15)

CMR parameter	Before the marathon	After the marathon
LV parameters		
LVEDD (mm)	51 ± 4	50 ± 5
LVESD (mm)	30 ± 9	30 ± 4
LVEDV (mL)	157 ± 22	161 ± 18
LVESV (mL)	51 ± 11	55 ± 13
LVEDV/BSA (mL/m^2)	83 ± 10	87 ± 8
LVESV/BSA (mL/m^2)	26 ± 12	27 ± 15
IVS (mm)	9 ± 1	9 ± 1
PWT (mm)	9 ± 1	9 ± 2
LVEF (%)	66 ± 5	68 ± 3
LV mass (g)	218 ± 25	212 ± 16
LV mass/BSA (g/m^2)	123 ± 14	117 ± 11
LA parameters		
LA diameter (mm)	36 ± 3	37 ± 5
LA volume (mL)	42 ± 13	41 ± 16
LA volume/BSA (mL/m^2)	27 ± 6	25 ± 4
RA and RV parameters		
RA volume (mL)	40 ± 9	56 ± 12*
RVEDD (cm)	32 ± 6	43 ± 6*
RVEDV (mL)	135 ± 24	195 ± 21*
RVEF (%)	60 ± 2	47 ± 5*
RV mass (g)	66 ± 3	65 ± 4
RV mass/BSA (g/m^2)	33 ± 3	34 ± 3

Data are expressed as mean ± SD.
BSA, Body surface area; *IVS*, interventricular septum; *LA*, left atrial; *LVEDD*, LV end-diastolic diameter; *LVEDV*, LV end-diastolic volume; *LVEF*, LV ejection fraction; *LVESD*, LV end-systolic diameter; *LVESV*, LV end-systolic volume; *PWT*, posterior wall thickness; *RA*, right atrial; *RVEDD*, RV end-diastolic diameter in parasternal long-axis view; *RVEDV*, RV end-diastolic volume; *RVESV*, RV end-systolic volume.
*P < .05, after the race vs baseline.

Table 5 Intraobserver and interobserver variability in RV volumes and RVEF using RT3DE and CMR

	Intraobserver		Interobserver	
Variable	Absolute	%	Absolute	%
RVEDV (mL)				
RT3DE	8.2 ± 3.7	7.1 ± 3.9	9.5 ± 3.5	6.8 ± 3.2
CMR	5.2 ± 1.4	4.1 ± 1.5	6.3 ± 2.4	4.8 ± 1.7
RVESV (mL)				
RT3DE	7.2 ± 3.2	5.8 ± 3.2	9.4 ± 2.4	7.4 ± 2.8
CMR	5.0 ± 1.6	4.2 ± 1.8	6.1 ± 2.1	5.2 ± 1.5
RVEF (%)				
RT3DE	8.0 ± 2.7	6.4 ± 2.3	9.4 ± 3.4	7.0 ± 2.8
CMR	5.1 ± 1.4	4.1 ± 1.6	6.2 ± 2.0	4.9 ± 3.0

Absolute values are expressed as population mean ± SD of absolute differences between repeated measurements. Percentage values are expressed as population mean ± SD of absolute differences of repeated measurements normalized by the average of the two repeated measurements.
RVEDV, RV end-diastolic volume; *RVESV*, RV end-systolic volume.

myoglobin and CK are not cardiac specific, they do indicate the strenuous nature of a half marathon. Immediately after completing the half marathon, all patients demonstrated elevated cTnT levels, with a median value of 0.37 ng/mL (interquartile range, 0.22–0.68 ng/mL). Myoglobin, CK, and cTnT levels had normalized at 1-week follow-up in all patients.

LV dimensions, volumes, and ejection fractions remained unchanged from baseline to after the half marathon (Table 3). However, right atrial volume increased from 41 ± 12 mL at baseline to 57 ± 14 mL after the race (P < .05; Table 3). Compared with baseline values, RV FAC by two-dimensional TTE imaging decreased significantly (41 ± 2% vs 33 ± 4%, P < .05). TAPSE was significantly decreased compared with baseline values (2.7 ± 0.4 vs 1.7 ± 0.4 cm, P < .05). Both RV FAC and TAPSE values returned to normal 1 week after the half marathon. There was an increase in pulmonary arterial systolic pressure, from 14 ± 4 mm Hg at baseline to 44 ± 3 mm Hg immediately after the half marathon (P < .05).

Similarly, RV volumes increased and systolic function decreased following the half-marathon using RT3DE (Table 3). RV end-diastolic volume increased from 125 ± 26 mL at baseline to 186 ± 24 mL after the half marathon. RV end-systolic volume increased from 50 ± 18 mL at baseline to 100 ± 21 mL after the half marathon. Finally, the RVEF by RT3DE decreased from 59 ± 4% at baseline to 45 ± 5% immediately following the race (P < .05; Table 3). At 1 week follow-up, RV volumes and RVEF by RT3DE had returned to baseline values.

RV volumes and function were abnormal on CMR after the marathon, which paralleled our echocardiographic findings. RV end-diastolic volume increased after the marathon (135 ± 24 vs 195 ± 21 mL, P < .05), and the RVEF was reduced, at 47 ± 5% compared with 60 ± 2% at baseline (P < .05) (Table 4). As shown in Figure 1, there were strong linear correlations between RVEF as assessed by RT3DE and CMR at baseline and after the half marathon (r = 0.69 and r = 0.87, P < .01, respectively). There was no evidence of myocardial edema on T2 imaging or delayed enhancement of the LV myocardium, even though all patients demonstrated elevated cTnT levels at the time of CMR.

Table 5 demonstrates the results for intraobserver and interobserver variability of RV volumes and RVEF derived from both imaging techniques, revealing high reproducibility with RT3DE.

DISCUSSION

The present study confirmed a significant increase in the release of cardiac biomarkers of injury after the completion of a half marathon. Our study is the first to demonstrate RV systolic dysfunction in the half marathon setting using real-time three-dimensional TTE imaging. Finally, this study demonstrated an absence of delayed enhancement on CMR, indicating that the increase in cTnT following a half marathon does not correspond to true myocardial necrosis.

Cardiac biomarkers, including myoglobin and CK, have been shown to be elevated in half marathon participants immediately after the races, with values returning to baseline over the course of 24 hours.[30,31] Lippi *et al.*[30] demonstrated elevations of CK, myoglobin, aspartate aminotransferase, and lactate dehydrogenase at 3 and 6 hours after a half marathon in 15 trained athletes, without any elevation of cTnT. In a second study by Lippi *et al.*,[31] 17 endurance-trained male athletes who completed a half marathon were found to have elevated N-terminal pro–B-type natriuretic peptide levels but no elevations of cTnT. Both studies by Lippi *et al.* adjusted the elevation in cTnT/cardiac ratio troponin I (cTnI) to the percentage change in

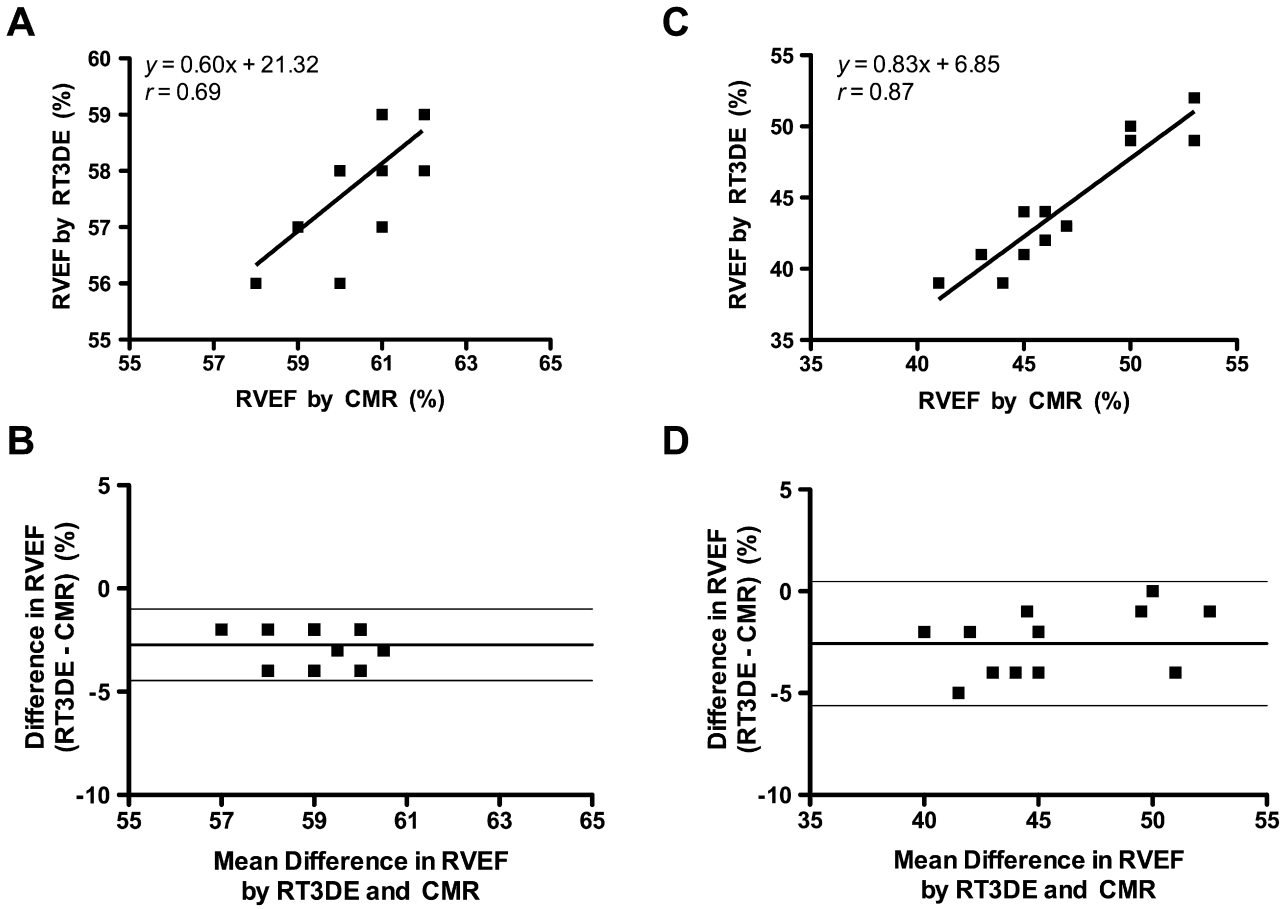

Figure 1 Linear regression and Bland-Altman plots comparing RVEF by RT3DE versus CMR at baseline **(A,B)** and immediately after the half marathon **(C,D)**.

plasma volume, calculated on the basis of the difference between pre-marathon and postmarathon hematocrit and hemoglobin.[30,31] On the contrary, Vidotto *et al.*[32] found elevations in cTnI and N-terminal pro–B-type natriuretic peptide 2 hours after a race in three of 25 trained athletes participating in the Wachau Half Marathon. Similarly, Tian *et al.*[33] found elevations in cTnT and cTnI above myocardial injury cutoffs in six of 10 less trained subjects 4 hours after the completion of a half marathon. Our study supports these previous findings[32,33] that cTnT is in fact elevated after a half marathon (median, 0.37 ng/mL) and returns to baseline over the course of a week. However, our study and the studies by Tian *et al.* and Vidotto *et al.* did not adjust cTnT/cTnI for percentage change in plasma volume, which may reasonably explain the elevation of these biomarkers.

Multiple studies have demonstrated transient RV systolic dysfunction in response to marathon running using two-dimensional TTE imaging.[1-14] Evidence of increased RV dimensions and decreased RV FAC and TAPSE have been demonstrated after various distances of endurance exercise, from marathons to ultramarathons.[25,26] To date, no studies have examined RV dysfunction in a half marathon setting using biomarkers and echocardiography. Our study demonstrated that RV systolic dysfunction occurs subsequent to running a half marathon, with a transient reductions in TAPSE and RV FAC that return to baseline at 1 week.

The transient dysfunction seen in our study was most likely due to exercise-induced pulmonary hypertension, with a 70% increase in pulmonary artery pressure due to increased RV afterload seen during a marathon.[34] Recently, evidence of bronchial epithelial inflammation due to excessive repeated shear stress with a corresponding increase in the inflammatory cytokine interleukin-8 and bronchial epithelial apoptosis was demonstrated after a half marathon.[35] A similar state of pulmonary inflammation was seen in athletes who had completed a full marathon.[36] Furthermore, the increase in pulmonary artery pressure may be due to decreased exhaled nitric oxide levels seen after intensive exercise, resulting in decreased pulmonary vasodilation.[37] The increase in pulmonary artery pressure after exercise overwhelms the capacity of the right ventricle, resulting in transient RV systolic dysfunction.[34]

RT3DE has been shown to be a feasible and reliable method of assessing RVEF noninvasively. A number of studies have compared RT3DE with CMR in the assessment of RV volumes and RVEF, demonstrating a high correlation with excellent intraobserver and interobserver reliability.[38-44] As a result, RT3DE has proven to be a cost-effective, a less invasive, and an accurate means of assessing RV function in a variety of cardiac disorders. Our current study demonstrated the utility of RT3DE in assessing RV systolic dysfunction after a half marathon. There was a strong linear correlation between RVEF as assessed by RT3DE and CMR after the half marathon ($r = 0.93$; Figure 1). Because of the greater time demands and costs associated with CMR, RT3DE may provide a reliable alternative in assessing RV dysfunction in athletes.[21]

Although RT3DE is comparable with CMR in the volumetric assessment of cardiac chambers, its ability to detect myocardial infarct scars is still in development.[45] The utility of gadolinium delayed

enhanced CMR (CMR) for detecting myocardial necrosis has been well established, with detection limits of 1 g of myocardium.[46-48] This corresponds to >0.1 ng/mL of cTnT.[49,50] Two recent studies have evaluated athletes (aged 18–45 years) participating in a full marathon.[25,34] Although there was evidence of cTnT release following the race, there was no evidence of delayed enhancement of the left ventricle to suggest true myocardial necrosis.[25,34] Our study demonstrated no evidence of myocardial necrosis by delayed enhanced CMR, supporting the studies by Mousavi *et al.*[25] and Trivax *et al.*[34] Our study indicates that the elevation of cTnT in half marathon runners is due to an increase in myocyte permeability rather than true necrosis.[25]

Limitations of this study included a small sample size with a heterogeneous population. Access to the use of multiple CMR units would have allowed for comparison of TTE and CMR values in a more temporal fashion after the race. Furthermore, some participants may have had transient microscopic structural damage following the half marathon that may not have been detected immediately following the marathon using T2 and delayed enhanced CMR. Future studies will examine the long-term consequences of half marathon running in a larger cohort of patients from all ages.

CONCLUSIONS

Compared with CMR, RT3DE is a feasible and reproducible method of assessing transient RV dysfunction in athletes completing a half marathon.

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REFERENCES

1. Jassal DS, Moffat D, Krahn J, Ahmadie R, Fang T, Eschun G, et al. Cardiac injury markers in non-elite marathon runners. Int J Sports Med 2009;30:75-9.
2. Siegel AJ, Januzzi J, Sluss P, Lee-Lewandrowski E, Wood M, Shirey T, et al. Cardiac biomarkers, electrolytes, and other analytes in collapsed marathon runners: implications for the evaluation of runners following competition. Am J Clin Pathol 2008;129:948-51.
3. Frassl W, Kowoll R, Katz N, Speth M, Stangl A, Brechtel L, et al. Cardiac markers (BNP, NT-pro-BNP, troponin I, troponin T, in female amateur runners before and up until three days after a marathon. Clin Lab 2008;54:81-7.
4. Tolkin L, Goldstein B, Rott D. Elevation of cardiac troponin T after running is not limited to marathon runners. Cardiology 2009;112:188-90.
5. Douglas PS, O'Toole ML, Hiller WD, Hackney K, Reichek N. Cardiac fatigue after prolonged exercise. Circulation 1987;76:1206-13.
6. Siegel AJ, Sholar M, Yang J, Dhanak E, Lewandrowski KB. Elevated serum cardiac markers in asymptomatic marathon runners after competition: is the myocardium stunned? Cardiology 1997;88:487-91.
7. Saenz AJ, Lee-Lewandrowski E, Wood MJ, Neilan TG, Siegel AJ, Januzzi JL, et al. Measurement of a plasma stroke biomarker panel and cardiac troponin T in marathon runners before and after the 2005 Boston marathon. Am J Clin Pathol 2006;126:185-9.
8. Leers MPG, Schepers R, Baumgarten R. Effects of a long-distance run on cardiac markers in healthy athletes. Clin Chem Lab Med 2006;44:999-1003.
9. Neumayr G, Pfister R, Mitterbauer G, Eibl G, Hoertnagl H. Effect of competitive marathon cycling on plasma N-terminal pro-brain natriuretic peptide and cardiac troponin T in healthy recreational cyclists. Am J Cardiol 2005;96:732-5.
10. Shave R, George KP, Atkinson G, Hart E, Middleton N, Whyte G, et al. Exercise-induced cardiac troponin T release: a meta-analysis. Med Sci Sports Exerc 2007;39:2099-106.
11. Scharhag J, Herrmann M, Urhausen A, Haschke M, Herrmann W, Kindermann W. Independent elevations of N-terminal pro-brain natriuretic peptide and cardiac troponins in endurance athletes after prolonged strenuous exercise. Am Heart J 2005;150:1128-34.
12. Urhausen A, Scharhag J, Herrmann M, Kindermann W. Clinical significance of increased cardiac troponins T and I in participants of ultra-endurance events. Am J Cardiol 2004;94:696-8.
13. Neumayr G, Gaenzer H, Pfister R, Sturm W, Schwarzacher SP, Eibl G, et al. Plasma levels of cardiac troponin I after prolonged strenuous endurance exercise. Am J Cardiol 2001;87:369-71.
14. Shave RE, Dawson E, Whyte G, George K, Ball D, Gaze DC, et al. Evidence of exercise-induced cardiac dysfunction and elevated cTnT in separate cohorts competing in an ultra-endurance mountain marathon race. Int J Sports Med 2002;23:489-94.
15. Perrault H, Péronnet F, Lebeau R, Nadeau RA. Echocardiographic assessment of left ventricular performance before and after marathon running. Am Heart J 1986;112:1026-31.
16. Neilan TG, Januzzi JL, Lee-Lewandrowski E, Ton-Nu TT, Yoerger DM, Jassal DS, et al. Myocardial injury and ventricular dysfunction related to training levels among nonelite participants in the Boston Marathon. Circulation 2006;114:2325-33.
17. Knebel F, Schimke I, Schroeckh S, Peters H, Eddicks S, Schattke S, et al. Myocardial function in older male amateur marathon runners: assessment by tissue Doppler echocardiography, speckle tracking, and cardiac biomarkers. J Am Soc Echocardiogr 2009;22:803-9.
18. Oxborough D, Shave R, Middleton N, Whyte G, Forster J, George K. The impact of marathon running upon ventricular function as assessed by 2D, Doppler, and tissue-Doppler echocardiography. Echocardiography 2006;23:635-41.
19. Neilan TG, Yoerger DM, Douglas PS, Marshall JE, Halpern EF, Lawlor D, et al. Persistent and reversible cardiac dysfunction among amateur marathon runners. Eur Heart J 2006;27:1079-84.
20. Gopal AS, Chukwu EO, Iwuchukwu CJ, Katz AS, Toole RS, Schapiro W. Normal values of right ventricular size and function by real-time 3-dimensional echocardiography: comparison with cardiac magnetic resonance imaging. J Am Soc Echocardiogr 2006;20:445-55.
21. van der Zwaan HB, Helbing WA, McGhie JS, Geleijnse ML, Luijnenburg SE, Roos-Hesselink JW, et al. Clinical value of real-time three-dimensional echocardiography for right ventricular quantification in congenital heart disease: validation with cardiac magnetic resonance imaging. J Am Soc Echocardiogr 2010;23:134-40.
22. Grewal J, Majdalany D, Syed I, Pellikka P, Warnes CA. Three-dimensional echocardiographic assessment of right ventricular volume and function in adult patients with congenital heart disease: comparison with magnetic resonance imaging. J Am Soc Echocardiogr 2010;23:127-33.
23. Leibundgut G, Rohner A, Grize L, Bernheim A, Kessel-Schaefer A, Bremerich J, et al. Dynamic assessment of right ventricular volumes and function by real-time three-dimensional echocardiography: a comparison with magnetic resonance imaging in 100 adult patients. J Am Soc Echocardiogr 2010;23:116-25.
24. Tamborini G, Marsan NA, Gripari P, Maffessanti F, Brusoni D, Muratori M, et al. Reference values for right ventricular volumes and ejection fraction with real-time three-dimensional echocardiography: evaluation in a large series of normal subjects. J Am Soc Echocardiogr 2010;23:109-15.
25. Mousavi N, Czarnecki A, Kumar K, Fallah-Rad N, Lytwyn M, Han SY, et al. Relation of biomarkers and cardiac magnetic resonance imaging after marathon running. Am J Cardiol 2009;103:1467-72.
26. Nassenstein K, Breuckmann F, Lehmann N, Schmermund A, Hunold P, Broecker-Preuss M, et al. Left ventricular volumes and mass in marathon runners and their association with cardiovascular risk factors. Int J Cardiovasc Imaging 2009;25:71-9.

27. Breuckmann F, Möhlenkamp S, Nassenstein K, Lehmann N, Ladd S, Schmermund A, et al. Myocardial late gadolinium enhancement: prevalence, pattern, and prognostic relevance in marathon runners. Radiology 2009;251:50-7.
28. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18: 1440-63.
29. Khoo NS, Young A, Occleshaw C, Cowan B, Zeng IS, Gentles TL. Assessments of right ventricular volume and function using three-dimensional echocardiography in older children and adults with congenital heart disease: comparison with cardiac magnetic resonance imaging. J Am Soc Echocardiogr 2009;22:1279-88.
30. Lippi G, Schena F, Salvagno GL, Montagnana M, Gelati M, Tarperi C, et al. Acute variation of biochemical markers of muscle damage following a 21-km, half-marathon run. Scand J Clin Lab Invest 2008;68:667-72.
31. Lippi G, Schena F, Salvagno GL, Montagnana M, Gelati M, Tarperi C, et al. Influence of a half-marathon run on NT-proBNP and troponin T. Clin Lab 2008;54:251-4.
32. Vidotto C, Tschan H, Atamaniuk J, Pokan R, Bachl N, Müller MM. Responses of N-terminal pro-brain natriuretic peptide (NT-proBNP) and cardiac troponin I (cTnI) to competitive endurance exercise in recreational athletes. Int J Sports Med 2005;26:645-50.
33. Tian Y, Nie J, Tong TK, Cao J, Gao Q, Man J, et al. Changes in serum cardiac troponins following a 21-km run in junior male runners. J Sports Med Phys Fitness 2006;46:481-8.
34. Trivax JE, Franklin BA, Goldstein JA, Chinnaiyan KM, Gallagher MJ, deJong AT, et al. Acute cardiac effects of marathon running. J Appl Physiol 2010;108:1148-53.
35. Chimenti L, Morici G, Paterno A, Santagata R, Bonanno A, Profita M, et al. Bronchial epithelial damage after a half-marathon in nonasthmatic amateur runners. Am J Physiol Lung Cell Mol Physiol 2010;298:L857-62.
36. Bonsignore MR, Morici G, Riccobono L, Insalaco G, Bonanno A, Profita M, et al. Airway inflammation in nonasthmatic amateur runners. Am J Physiol Lung Cell Mol Physiol 2001;281:L668-76.
37. Uyan AP, Uyan C, Ozyurek H. Assessment of right ventricular diastolic filling parameters by Doppler echocardiography. Pediatr Int 2003;45:263-7.
38. Bicudo LS, Tsutsui JM, Shiozaki A, Rochitte CE, Arteaga E, Mady C, et al. Value of real time three-dimensional echocardiography in patients with hypertrophic cardiomyopathy: comparison with two-dimensional echocardiography and magnetic resonance imaging. Echocardiography 2008;25:717-26.
39. Lang RM, Mor-Avi V, Sugeng L, Nieman PS, Sahn DJ. Three-dimensional echocardiography: the benefits of the additional dimension. J Am Coll Cardiol 2006;48:2053-69.
40. Bellenger NG, Burgess MI, Ray SG, Lahiri A, Coats AJ, Cleland JG, et al. Comparison of left ventricular ejection fraction and volumes in heart failure by echocardiography, radionuclide ventriculography and cardiovascular magnetic resonance; are they interchangeable? Eur Heart J 2000;21: 1387-96.
41. Bu L, Munns S, Zhang H, Disterhoft M, Dixon M, Stolpen A, et al. Rapid full volume data acquisition by real-time 3-dimensional echocardiography for assessment of left ventricular indexes in children: a validation study compared with magnetic resonance imaging. J Am Soc Echocardiogr 2005;18:299-305.
42. Gutiérrez-Chico JL, Zamorano JL, Pérez de Isla L, Orejas M, Almería C, Rodrigo JL, et al. Comparison of left ventricular volumes and ejection fractions measured by three-dimensional echocardiography versus by two-dimensional echocardiography and cardiac magnetic resonance in patients with various cardiomyopathies. Am J Cardiol 2005;95:809-13.
43. Kühl HP, Schreckenberg M, Rulands D, Katoh M, Schäfer W, Schummers G, et al. High-resolution transthoracic real-time three-dimensional echocardiography: quantitation of cardiac volumes and function using semi-automatic border detection and comparison with cardiac magnetic resonance imaging. J Am Coll Cardiol 2004;43:2083-90.
44. Walker J, Bhullar N, Fallah-Rad N, Lytwyn M, Golian M, Fang T, et al. Role of three-dimensional echocardiography in breast cancer: comparison with two-dimensional echocardiography, multiple-gated acquisition scans, and cardiac magnetic resonance imaging. J Clin Oncol 2010;28:3429-36.
45. Montant P, Chenot F, Goffinet C, Poncelet A, Vancraeynest D, Pasquet A, et al. Determination of infarct size and transmurality by contrast-enhanced 3D-echocardiography. Circ Cardiovasc Imaging. In press.
46. Pattynama PM, Lamb HJ, Van der Velde EA, Van der Geest RJ, Van der Wall EE, De Roos A. Reproducibility of MRI-derived measurements of right ventricular volumes and myocardial mass. Magn Reson Imaging 1995;13:53-63.
47. Cutrone JA, Georgiou D, Khan S, Fischer H, Belardinelli R, Laks MM, et al. Comparison of electron beam computed tomography scanning and magnetic resonance imaging quantification of right ventricular mass: validation with autopsy weights. Acad Radiol 1996;3:395-400.
48. Lorenz CH, Walker ES, Morgan VL, Klein SS, Graham TP Jr. Normal human right and left ventricular mass, systolic function, and gender differences by cine magnetic resonance imaging. J Cardiovasc Magn Reson 1999;1:7-21.
49. Perseghin G, De Cobelli F, Esposito A, Lattuada G, Terruzzi I, La Torre A, et al. Effect of the sporting discipline on the right and left ventricular morphology and function of elite male track runners: a magnetic resonance imaging and phosphorus 31 spectroscopy study. Am Heart J 2007;154: 937-42.
50. Nassenstein K, Breuckmann F, Lehmann N, Schmermund A, Hunold P, Broecker-Preuss M, et al. Left ventricular volumes and mass in marathon runners and their association with cardiovascular risk factors. Radiology 2008;249:829-35.