Depressed Systolic Function after a Prolonged and Strenuous Exercise

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ABSTRACT

VITIELLO, D., J. CASSIRAME, A. MENETRIER, T. RUPP, I. SCHUSTER, C. REBOUL, P. OBERT, N. TORDI, and S. NOTTIN. Depressed Systolic Function after a Prolonged and Strenuous Exercise. *Med. Sci. Sports Exerc.*, Vol. 45, No. 11, pp. 2072–2079, 2013. **Introduction**: Prolonged and strenuous exercise (PSE) induces transient left ventricular (LV) dysfunction. Although a consensus exists regarding the decrease in diastolic function, the existence of a decrease in systolic function by a PSE remains controversial, probably due to the transient tachycardia and changes in loading conditions observed upon the completion of exercise. Therefore, the objective was to evaluate LV systolic function before and after a PSE using two-dimensional speckle tracking echocardiography not only at rest but also during incremental tests to adjust heart rates (HR). **Methods and Results**: Sixteen healthy young men (23 ± 3 yr old) performed a 3-h period of intensity-controlled upright cycling. LV strain (*S*), systolic strain rate (SR), rotation, and systolic rotational rate were evaluated by two-dimensional speckle tracking echocardiography before and after a 3-h period of PSE at rest and during incremental tests. Posttest evaluation was performed once the HR had returned to the pretest value. Under resting conditions, parameters of systolic function were either unchanged or increased after the PSE. However, during the incremental test, all LV systolic SR and apical rotational rates were decreased after PSE (radial SR at workload 3 (W3): 2.21 ± 0.12.s^{-1} vs 1.87 ± 0.10.s^{-1}, $P < 0.01$ and apical rotational rate at W3: 128 ± 28 deg·s^{-1} vs 105 ± 26 deg·s^{-1}, $P < 0.05$). Regression analyses between LV systolic SR and HR showed lower *y*-intercepts without differences in slopes, suggesting a decrease of both global and regional systolic functions irrespective of HR after the PSE. **Conclusion**: Our findings based on LV *S* and SR data during incremental tests demonstrate that the 3-h period of PSE induces LV systolic dysfunction. **Key Words:** STRESS ECHOCARDIOGRAPHY, 2-D STRAIN ANALYSIS, PROLONGED EXERCISE, SYSTOLIC DYSFUNCTION

Although regular exercise has a protective effect on the cardiovascular system (25,33), it was well established that a prolonged and strenuous exercise (PSE), such as the marathon or the long-duration triathlon, induces transient left ventricular (LV) dysfunction (6,22,27,34). Furthermore, in some subjects, the LV dysfunction is also associated with the release of biomarkers of cardiac damage markers into the plasma (3,12,20).

The effect of PSE on cardiac function depends on both duration and intensity (18,26,27). After a marathon, diastolic function is generally depressed due, in part, to a decrease in LV intrinsic relaxation properties (6,9). However, the effect of this particular type of exercise on systolic function remains controversial, with studies reporting that the ejection fraction (EF) is either depressed (15) or unchanged (5,6,14). Using tissue Doppler imaging, George et al. (11) did not observed significant changes in longitudinal systolic tissue velocities. Recently, two-dimensional speckle tracking echocardiography (2DSE) has been used to evaluate LV strain (*S*) and strain rate (SR). LV *S* and SR from all planes (i.e., longitudinal, radial, and circumferential) were unchanged (6,10). However, the effect of running a marathon on LV systolic function remains to be elucidated.

All the aforementioned studies evaluated systolic function within 1 h of the completing the exercise, a period characterized by a persistent tachycardia and altered heart loading (6,10,15). In a recent study conducted in rats, we observed a depressed LV intrinsic myocardial contractility after a 4-h running period when loading conditions and HR were normalized using the Langendorff isolated and perfused heart model (32). In this context, we can hypothesize that the persistent tachycardia occurring immediately after the completion of a marathon could mask a subtle decrease in LV systolic function.

Accordingly, the main objective of the present study was to evaluate the effect of a PSE on LV systolic function using a specific methodology to overcome the influence of transient

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tachycardia commonly observed after a prolonged exercise. The PSE consisted of a 3-h period of pedaling on a cycle ergometer. Before and after the PSE, LV function was assessed not only at rest but also during incremental tests. Posttest evaluations were performed under similar HR as those reached during pretest evaluations. Both at rest and during incremental tests, LV systolic function was assessed using conventional tissue Doppler and also 2DSE, a new echocardiographic tool that enables the evaluation of LV *S* and SR providing the capacity to detect subtle LV systolic dysfunction (1,16). We hypothesize that the PSE induces LV systolic dysfunction associated with depressed LV *S* and SR.

METHODS

Subjects. Sixteen active young healthy males between ages 19 and 29 yr participated in the study. Subjects were recruited at the Université de Besançon, France. The subjects were free from cardiac diseases or arterial hypertension and were currently not taking any medications. All subjects were instructed to avoid intense activity, alcohol, and caffeine for a period of 48 h before the initiation of the first incremental test. The protocol was approved by the local ethics committee and all subjects provided a written informed consent.

Study design. The study design is depicted in Figure 1. After body height and weight assessments, maximal aerobic power was estimated via the Wasserman equation for the subject's age and body mass. After a 15-min resting period, each subject underwent a first incremental test on a dedicated cycle ergometer in a semisupine position (30° from the horizontal) and in a partial left decubitus position (E-Bike ergometer; GE Healthcare, Horten, Norway). The incremental test included three workloads of 6 min each at 20%, 30%, and 40% of their maximal aerobic power (W1, W2, and W3, respectively). The pedaling rate was kept constant at 70–80 rpm for all subjects. Then, each subject underwent the PSE consisting of a 3-h period of controlled exercise on a standard cycle ergometer in an upright position. After the PSE, subjects were allowed to recover for a period of 30 min, then a second incremental test was performed, during which each workload was adjusted to reach similar HR as the one attained during the first incremental test. For both pre- and post-PSE tests, cine loops were recorded at rest and during the last 4 min of each workload. Tissue Doppler and 2DSE offline analyses were conducted to assess less load-dependent indexes of LV contractility (8,17,30).

Prolonged strenuous exercise protocol. All the subjects performed the PSE consisting of a 3-h period of cycling at HR > 130 beats per minute on a standard cycle ergometer. Cardiovascular changes during the PSE were evaluated after 5 min, 1 h, 2 h, and 3 h of pedaling from the time–velocity integral of the ascending aorta flow using a Pedof probe (2 MHz; Esaote, Naples, Italy) positioned at the suprasternal notch (Mylab™30 echocardiograph; Esaote). The outline contour of the velocity curve over time (VTI) was traced manually (MyLab™ Desk software; Esaote). The end of each VTI was considered as the observed closure of the aortic valve. Values for VTI were averaged from three to five curves, with the highest values demonstrating crisp spectral envelopes. HR, stroke volume (SV), and cardiac output ($\dot{Q}$) were assessed, and systemic vascular resistances (SVR) were calculated as mean arterial pressure divided by $\dot{Q}$. Systolic and diastolic blood pressures were assessed using manual sphygmomanometry. HR was continuously monitored using Suunto Memory Belts® (Suunto; Vantaa, Finland). All participants were allowed to eat and drink *ad libitum* during the PSE.

Acquisition of echocardiographic data at rest and during incremental tests before and after the PSE. Images were obtained at rest and during the incremental tests using the Vivid 7 Dimension® ultrasound apparatus (GE Healthcare). Cine loops were recorded from apical four-chamber and parasternal short-axis views. Proper care was taken to ensure that the basal short-axis plane contained the mitral valve and that the apical plane was acquired with the probe in a caudal position (31). Two-dimensional gray-scale harmonic images were obtained at a rate of 65–90 frames

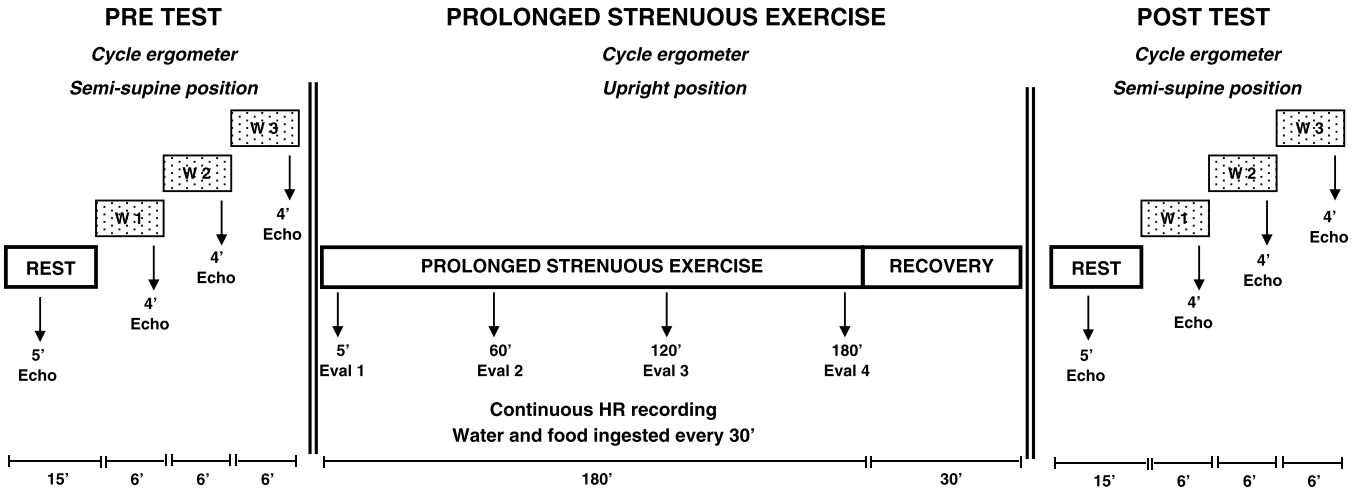

FIGURE 1—Study design. W, workload; Echo, echocardiographic acquisitions.

per second, and color tissue velocity images were acquired from an apical four-chamber view at a rate of 120–140 frames per second. Images were acquired in cine loop format triggered to the QRS complex and saved digitally for subsequent offline analyses with the dedicated software (EchoPac 6.0; GE Healthcare).

Conventional and tissue Doppler analyses. Peak early (E) and atrial (A) filling velocities were assessed from apical four-chamber views. The LV end-diastolic (EDD) and end-systolic (ESD) diameters were measured offline in short-axis loops recorded at the papillary muscle level, and the LV fractional shortening (FS) was calculated as FS = (EDD − ESD) / EDD × 100. We assessed wall motion velocities during early filling (E'), late filling (A') and ejection (S') from apical four-chamber view at the level of the mitral annulus and averaged the values for septal and lateral walls (21).

2DSE analyses. EchoPac™ software was used to assess regional LV function as previously described (7,23). After the manual tracing of the endocardial border on the end-systolic frame of the 2-D sequence, the software automatically tracked myocardial motions. If a poor tracking efficiency was signaled by the software, the observer readjusted the endocardial trace line and/or the region of interest width until a better tracking score could be obtained. Results were averages of three to five cardiac cycles. LV longitudinal S and systolic SR were assessed using an apical four-chamber view. Circumferential and radial systolic SR, both indices of LV intrinsic contractility (8), were averaged from short-axis views at the basal and apical levels. 2DSE data were then processed with a specific toolbox (Scilab 4.1; Consortium Scilab, INRIA-ENPC, Paris, France) developed in our laboratory. For temporal analyses, this software adjusted all strain variables for intersubject differences in HR and transducer frame rate acquisition. The time sequence was normalized to the percentage of systolic and diastolic duration (i.e., AVC representing 100% of systole and end of cardiac cycle representing 100% of diastole) using interpolations. After normalization, the software averaged each data set from three to five cardiac cycles and performed the detection of peak strain and their timing (expressed in percentage of systolic duration). Frame by frame, the evaluation of the LV volumes was automatically performed using the dedicated toolbox. Briefly, coordinates of mid-wall points were exported by the Echopac software. End-diastolic wall thickness and instantaneous transversal S were used to calculate coordinates of endocardial points from an apical four-chamber view. LV volumes were then calculated using the monoplane Simpson's method. Ejection fraction was calculated from end-diastolic and end-systolic volumes.

Statistical analyses. Statistical analyses were performed using Statview 5.0 (SAS Institute Inc., Cary, NC). Pre- and post-PSE values from conventional and 2DSE were analyzed using a two-way repeated-measures ANOVA. *Post hoc* tests were used when appropriate. Regression analyses between S', LV systolic longitudinal SR, and HR were also performed. Data were expressed as mean ± SD and statistical significance was assumed if $P < 0.05$. The authors had full access to the data and take full responsibility for the integrity of the data. All authors have read the manuscript and agreed with its content.

RESULTS

Cardiovascular parameters assessed during the PSE. During the PSE, subjects drank a mean of 420 ± 25 mL of water every 30 min. The body mass was not affected by the PSE (71.7 ± 7.8 vs 71.4 ± 7.5 kg, NS). During the PSE, HR increased (5 vs 180 min: 136 ± 22 vs 149 ± 14 beats per minute, $P < 0.001$), and the SV decreased progressively (5 vs 180 min: 87 ± 11 vs 80 ± 11 mL, $P < 0.001$).

LV function during resting condition. Conventional resting echocardiographic data before and after the PSE are presented in Table 1. After the PSE, systolic and diastolic arterial pressures and SVR were decreased. All diastolic parameters (i.e., LV end-diastolic diameter, E wave, and peak E' velocity from tissue Doppler evaluation) were lower after the PSE as compared with pretest evaluations. SV was lower but $\dot{Q}$ was higher due to tachycardia. In addition, EF and FS were also lower post-PSE. However, S' from TDI evaluation remained unchanged. LV S and rotations are presented in Table 2. LV S were significantly lower after the PSE, except for the radial S, which was not found to be statistically significant (Table 2). LV systolic SR tended to be higher after the PSE, although when compared with pretest evaluations, it reached statistical significance for the LV longitudinal component only. LV basal rotation increased while apical rotation remained unchanged. LV peak systolic rotational rate decreased at the apical level after the PSE and remained constant at the basal level.

LV function during incremental tests. Echocardiographic data obtained during incremental tests performed before and after the PSE are presented in Table 2 and in Figures 2 and 3. For each workload of the incremental test, no differences were observed on HR between pre- and post-PSE tests (97 ± 14 vs 100 ± 13 beats per minute at W1, 109 ± 13 vs 108 ± 14 beats per minute at W2, and 120 ± 14 vs 119 ± 14 beats per minute at W3, NS). Similar results were observed on SVR [5.8 ± 0.8 vs 5.9 ± 0.9 arbitrary unit (A.U.) at W1, 5.4 ± 0.9 vs 5.3 ± 0.8 arbitrary unit (A.U.) at W2, and 4.9 ± 0.8 vs 4.9 ± 0.8 A.U. at W3, NS], an overall index of afterload. Mechanical power, adjusted for each subject during posttest, was significantly lower when compared with the equivalent workload during the pretest (49 ± 5 vs 12 ± 14 W at W1, 75 ± 9 vs 29 ± 18 W at W2, and 100 ± 13 vs 52 ± 19 W at W3, $P < 0.001$).

The EF and FS were decreased during the W3 of the incremental test after the PSE (Table 2). Except for LV radial S, all LV S were decreased during the W3 after the PSE. Kinetics of peak S', systolic SR, and rotational rates are presented in Figure 2. Their increase during incremental exercise was lower after the PSE, and consequently, peak S' and peak systolic SR were lower during the W3 after the PSE. An example of an individual decrease in LV peak systolic longitudinal

TABLE 1. LV conventional echocardiographic data assessed at rest and during workload 3 of the incremental tests before and after PSE.

	Rest		Incremental Tests (W3)	
	Before PSE	After PSE	Before PSE	After PSE
Morphological parameters				
LV end-diastolic diameter (mm)	53.4 ± 4.8	51.6 ± 4.6***	53.9 ± 4.7	51.7 ± 4.9***
LV end-systolic diameter (mm)	34.7 ± 4.1	34.4 ± 3.6	31.0 ± 5.2†††	32.0 ± 4.6*,†††
LV end-diastolic volume (mL)	83.8 ± 14.6	78.4 ± 13.9***	85.2 ± 14.6	78.5 ± 14.7***
LV end-systolic volume (mL)	35.5 ± 8.2	28.7 ± 8.9***	34.7 ± 7.1	30.7 ± 8.4***
Global diastolic function				
Peak *E* velocity ($cm \cdot s^{-1}$)	78.4 ± 14.7	69.8 ± 13.8***	124 ± 21†††	107 ± 22***,†††
Peak *A* velocity ($cm \cdot s^{-1}$)	40.1 ± 7.4	48.6 ± 7.1	103 ± 28†††	97.9 ± 28.4†††
Peak *E/A* ratio	2.12 ± 0.59	1.53 ± 0.51***	1.26 ± 0.15††	1.03 ± 0.03***,††
Peak *E/E′* ratio	8.3 ± 1.5	7.7 ± 1.8*	9.7 ± 2.3†††	8.7 ± 2.1*,†††
Global systolic function				
Ejection fraction (%)	59.2 ± 7.4	55.1 ± 8.2**	67.7 ± 6.9†††	64.3 ± 8.2**,†††
Fractional shortening (%)	35.1 ± 3.7	33.5 ± 2.5***	42.7 ± 6.4†††	37.9 ± 4.9***,†††
Heart rate ($beat \cdot min^{-1}$)	64 ± 13	81 ± 15***	120 ± 14†††	119 ± 14†††
Stroke volume (mL)	111 ± 21	101 ± 21***	133 ± 30†††	120 ± 27***,†††
Cardiac output ($L \cdot min^{-1}$)	6.7 ± 1.5	7.9 ± 1.1**	15.3 ± 2.6†††	14.0 ± 2.4***,†††
AVC (ms)	359 ± 44	349 ± 39	239 ± 39	249 ± 33
Arterial pressures				
Systolic (mm Hg)	122 ± 7	116 ± 7**	148 ± 9†††	133 ± 10***,†††
Diastolic (mm Hg)	70 ± 7	67 ± 6*	71 ± 7	68 ± 5*
Systemic vascular resistances (AU)	9.9 ± 2.2	7.9 ± 1.1**	4.9 ± 0.8†††	4.9 ± 0.8†††
TDI assessments				
Peak *E′* ($cm \cdot s^{-1}$)	11.2 ± 1.3	10.4 ± 1.8*	15.7 ± 1.8†††	15.3 ± 2.3*,†††
Peak *A′* ($cm \cdot s^{-1}$)	4.9 ± 2.2	5.3 ± 1.2	10.5 ± 2.8	9.3 ± 1.6
Peak *S′* ($cm \cdot s^{-1}$)	8.6 ± 1.2	8.4 ± 0.9	13.1 ± 0.7†††	11.1 ± 1.7**,†††

Values are expressed as mean ± SD.
Significant differences with pretest values: $^{*}P < 0.05$, $^{**}P < 0.01$, and $^{***}P < 0.001$.
Significant differences with resting values: $^{††}P < 0.01$ and $^{†††}P < 0.001$.
AU, arbitrary unit; LV, left ventricle; IVRT, isovolumic relaxation time; AVC, aortic valve closure.

SR during the W3 is presented in Figure 3. Kinetics of LV basal and apical rotational rates were similar during both incremental tests. At the apical level, rotational rate was lower at rest and remained stable during the incremental test compared with values observed before the PSE.

Regression analyses between S', LV systolic longitudinal SR, and HR obtained at rest and during incremental exercise pre- and post-PSE are presented in Figure 4. For both parameters, no differences in slopes were observed. However, significantly lower *y*-intercepts during post-PSE incremental test were observed for both parameters, indicating that LV peak S' from tissue Doppler and LV systolic longitudinal SR decreased after the PSE independently of HR.

DISCUSSION

The main objective of the present study was to evaluate whether a 3-h period of a PSE could induce an LV systolic dysfunction. One of the key aspects of our study was that we overcame the influence of transient tachycardia commonly

TABLE 2. LV strains, SR, rotations, and rotational rates assessed at rest and during workload 3 of submaximal exercise before and after PSE.

	Rest		Submaximal Exercise (W3)	
	Before PSE	After PSE	Before PSE	After PSE
LV strains				
Longitudinal				
Strain (%)	−16.1 ± 2.2	−15.0 ± 2.7**	−18.7 ± 2.1†††	−17.3 ± 3.1**,†††
Systolic SR (s^{-1})	−0.89 ± 0.15	−0.98 ± 0.10*	−1.52 ± 0.22†††	−1.42 ± 0.27*,†††
Radial				
Strain (%)	32.8 ± 5.3	25.9 ± 7.3	37.1 ± 13.8	34.3 ± 7.1†
Systolic SR (s^{-1})	1.27 ± 0.26	1.42 ± 0.28	2.21 ± 0.12†††	1.87 ± 0.10**,†††
Circumferential				
Strain (%)	−22.5 ± 3.7	−20.6 ± 3.9***	−29.4 ± 4.8†††	−25.6 ± 4.8***,†††
Systolic SR (s^{-1})	−1.34 ± 0.27	−1.53 ± 0.29	−2.54 ± 0.12†††	−2.30 ± 0.14*,†††
LV rotations				
Basal level				
Rotation (°)	−3.8 ± 1.4	−5.1 ± 1.5**	−7.2 ± 1.9††	−6.4 ± 2.1††
Systolic rotational rate ($°\cdot s^{-1}$)	−58 ± 18	−58 ± 36	−92 ± 27†††	−93 ± 27†††
Apical level				
Rotation (°)	6.6 ± 1.9	5.9 ± 1.7	9.3 ± 1.8††	7.4 ± 1.0***,††
Systolic rotational rate ($°\cdot s^{-1}$)	87 ± 11	71 ± 15*	128 ± 28†††	105 ± 26*,†††

Values are expressed as mean ± SD. Strains data are presented as mean ± SD percentage of myocardial state change between the protodiastole state and the end-diastolic state.
Significant differences with pretest values: $^{*}P < 0.05$, $^{**}P < 0.01$, and $^{***}P < 0.001$
Significant differences with resting values: $^{††}P < 0.01$ and $^{†††}P < 0.001$.
LV, left ventricle.

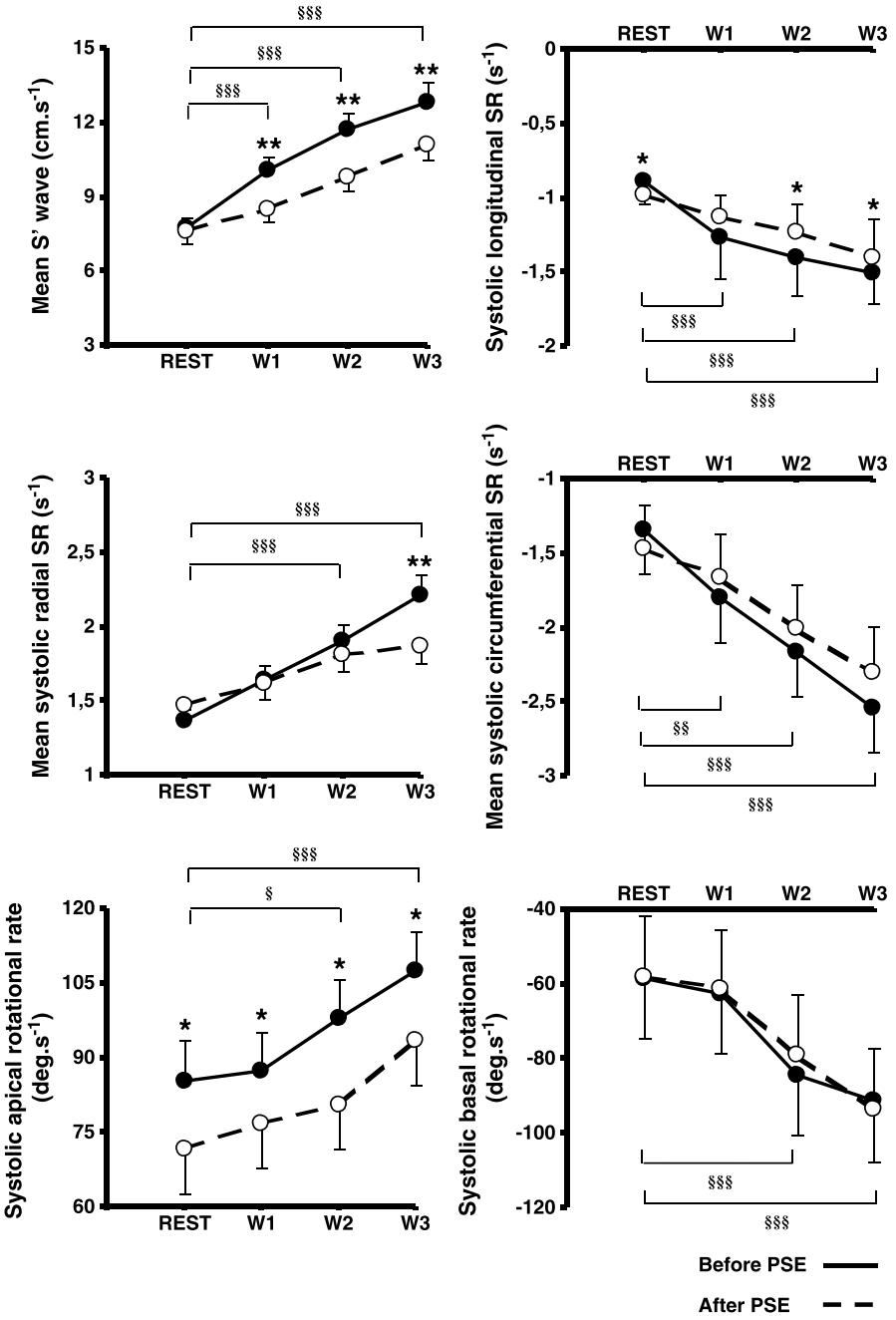

FIGURE 2—Kinetics of LV systolic function parameters measured during the incremental tests before and after the prolonged strenuous exercise. W, workload. Data corresponding to the pretest are represented with the continuous black line and those corresponding to the posttest kinetics with scattered line. Significant differences with pretest values: *$P < 0.05$ and **$P < 0.01$; and between workloads: $^{§§}P < 0.01$ and $^{§§§}P < 0.001$.

observed immediately after a PSE. Indeed, we assessed LV function not only at rest but also during incremental tests performed before and after the PSE at similar HR. Our results, based on conventional tissue Doppler imaging and 2DSE strongly support the existence of LV systolic dysfunction after a PSE of moderate duration.

Controversial decrease in LV systolic function after the PSE at rest. It is well documented that a PSE of several hours induced a transient diastolic dysfunction (2,6). Our results confirmed these data because, after 3 h of PSE, all diastolic parameters from conventional tissue Doppler and 2DSE decreased. However, the literature regarding a transient LV systolic dysfunction after such exercise remains controversial (2,5,6,14,15). In our study, global systolic function (i.e., SV and EF) was lower after the PSE, a result similar to those obtained in previous studies after a marathon (2,15,22). Of note, EF and LV strain depend on numerous factors and did not reflect LV intrinsic myocardial contractility. In the heart, the myocardium contractility can be defined as the maximal velocity of myocardial fiber shortening (4), and the gold standard for *in vivo* evaluation of intrinsic myocardial contractility in humans is dP/dt_{max}. However,

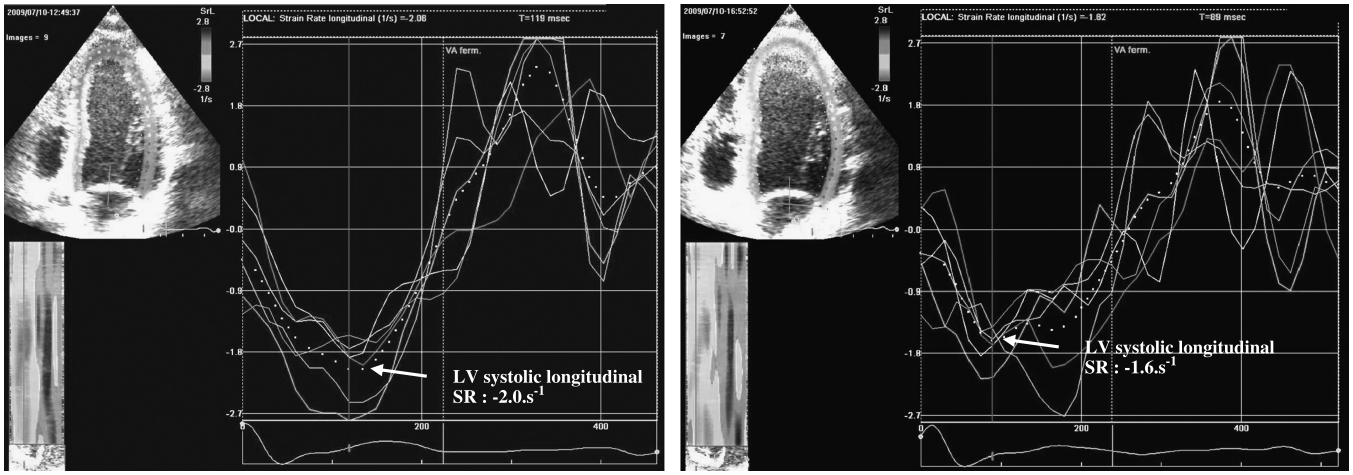

FIGURE 3—Exemplar color traces for LV SR displayed across the cardiac cycle before (left) and after (right) the PSE in a subject with major reduction in mean LV peak systolic basal longitudinal strain rates during workload 3 of the incremental test. HR, heart rate; SR, strain rate.

the use of invasive techniques is precluded in healthy subjects. In the present study, S' wave velocities from tissue Doppler evaluation and LV peak longitudinal systolic SR from 2DSE were considered as the most appropriate indexes of LV systolic function (30), despite the controversy over their load dependence (28). Contrary to global evaluation of systolic function, we observed that peak S' and LV peak systolic SR remained unchanged after the PSE. Similar results were recently observed after a marathon (5) or after a 2-h period of cycling (13). Using 2DSE, we also measured LV radial, circumferential SR and LV apical rotation, three well-accepted parameters to assess LV intrinsic contractility (8,17). Interestingly, all these parameters were unchanged or slightly improved after the PSE, suggesting that LV contractility seems to be stable after the PSE. On the basis of the stability of the LV function data under resting condition, the fact that the LV systolic function is depressed after a 3-h period of PSE remains controversial.

Cardiac evaluation during the incremental tests: evidence of LV systolic dysfunction after a PSE. Previous studies systematically evaluated systolic function within 1 h upon completion of a marathon, a period characterized by a persistent tachycardia (5,10,15). In the present study, we wanted to assess whether the elevated HR observed immediately after the end of the PSE could mask a subtle decrease in LV systolic function. To overcome these limitations, LV function was assessed, under conditions involving a defined workload performed before and after the PSE, at similar HR.

When assessed at similar HR, SV, EF, and also LV longitudinal and circumferential S remained decreased after the PSE. Interestingly, the kinetics of S' velocity (tissue Doppler imaging) and SR (2DSE) from rest to exercise were altered after the PSE. The increase in S' was blunted after PSE, and consequently, it was depressed at the W3 as compared with the values observed before PSE. Similar results were observed on LV systolic radial and circumferential SR. In a recent study conducted in animals, Ferferieva et al. (8) reported that radial and circumferential SR were robust measures of LV intrinsic contractility, being less influenced by cardiac load and structure than LV strains. LV apical rotation represents another noninvasive index of global LV contractility, which is less dependent on cardiac loading conditions (17).

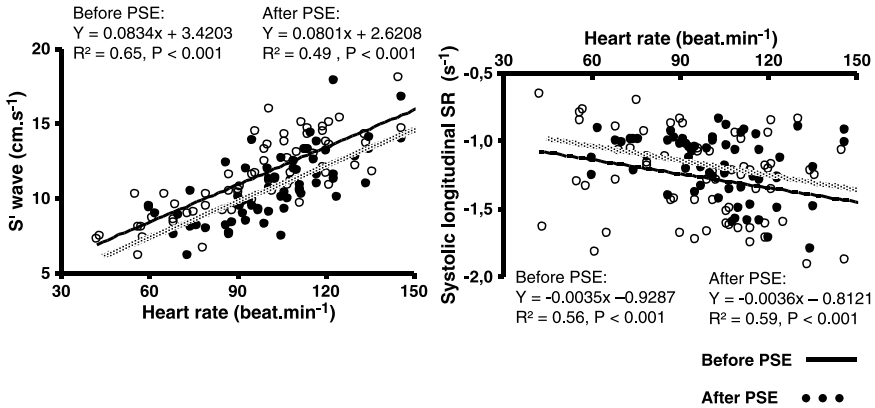

FIGURE 4—Regression analyses between S', LV systolic longitudinal SR, and HR. R^2, coefficient of determination; y, y-intercept. White circles, pretest; black circles, posttest.

Whereas LV apical rotation was higher during resting condition after PSE, its increase during tests was also blunted, and the values had a trend to be lower at the W3 despite the absence of statistical significance. Together, these data observed during exercise strongly support that a 3-h period of PSE induce a decrease in LV systolic function, which is probably associated with a decrease in LV intrinsic myocardial contractility. Nevertheless, despite that SVR (an overall index of LV afterload) were unchanged during W1, W2, and W3 workloads as compared with pretest values, loading conditions of the heart were probably different between pretest and posttest (as evidenced by differences in LV end-diastolic volume and systolic and diastolic blood pressures). Therefore, the interpretation of blunted SR and rotations during exercise must be interpreted with caution.

Regression analyses between S', LV systolic longitudinal SR, and HR demonstrated decreased y-intercepts without significant difference between the slopes. These findings suggest that regional systolic function was depressed regardless of HR, that is, not only during incremental tests but also at rest. These results highlight that the absence of LV systolic dysfunction observed in previous studies after a marathon (2,5,6,14,15) is probably due to the influence of transient tachycardia observed immediately after the completion of a PSE. During the recovery period, the concentration of the circulating catecholamines remained elevated, thus contributing to increase not only the HR but also the LV contractility by their positive inotropic effects. The human myocardial dysfunction observed in the present study confirms the same observations made in studies using an isolated and perfused rat heart model (32). The underlying mechanisms remain to be identified but could include ß-adrenergic receptor desensitization (15), increased oxidative stress (19,29,32), and reduced Ca^{2+} transients (24).

CONCLUSIONS

Our specific methodology based on echocardiographic evaluations during incremental tests indicates that a PSE induces a transient decrease in LV systolic function. Regional assessments of systolic function based on tissue Doppler and 2DSE indicate that the LV systolic dysfunction could be associated with depressed myocardial contractility. Of note, *in vivo* evaluation of LV contractility using these echocardiographic tools remains partially dependent on loading conditions, which were probably altered after PSE. The long-term consequences of such transient myocardial abnormalities after repetitive prolonged exercise are unknown and represent an important issue for future investigations.

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The authors declare no conflict of interest.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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