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The Impact of Endurance Exercise Training on Left Ventricular Torsion

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OBJECTIVES We sought to examine the effect of endurance exercise training (EET) on peak systolic left ventricular torsion (LVT) and peak early diastolic untwisting rate (UTR).

BACKGROUND Left ventricular (LV) structural adaptations to EET have been well characterized. LVT, a recognized marker of LV function in numerous cardiac diseases, has recently been investigated in the setting of exercise. However, longitudinal data characterizing the impact of sustained exercise training on LVT have not been reported.

METHODS A prospective, longitudinal study design examined the impact of a 90-day period of training on LV twist mechanics in university male rowers (n = 15, mean age 18.6 ± 0.5 years). Conventional LV structural measurements, LV apical and basal rotation, peak systolic LVT, and peak early diastolic UTR were measured by 2-dimensional and speckle tracking echocardiography before and after the EET study period.

RESULTS Participants experienced LV eccentric hypertrophy, characterized by increased LV end-diastolic volume (80.8 ± 8.7 ml/m^2 vs. 91.3 ± 8.0 ml/m^2, $p < 0.001$) and LV mass (101.3 ± 11.4 g/m^2 vs. 115.7 ± 12.6 g/m^2, $p = 0.001$). There was a significant increase in peak systolic apical rotation (8.9 ± 4.2° vs. 12.7 ± 3.9°, $p = 0.002$) but no change in basal rotation. This translated into a highly significant increase in peak systolic LVT after EET (14.1 ± 5.0° vs. 18.0 ± 3.6°, $p = 0.002$). The impact of EET on LV twist mechanics was not confined to ventricular systole, as peak early diastolic UTR (−110.6 ± 41.8°/s vs. −148.0 ± 29.8°/s, $p = 0.003$) and the percentage of untwisting that occurred by the end of isovolumic relaxation (31.2 ± 12.0% vs. 39.9 ± 14.9%, $p = 0.04$) increased.

CONCLUSIONS Participation in EET was associated with significant changes in LV twist mechanics characterized by increased apical rotation, LVT, and UTR. These findings suggest that LVT and UTR augmentation may be an important and previously unrecognized component of exercise-induced cardiac remodeling. (J Am Coll Cardiol Img 2010;3:1001–9) © 2010 by the American College of Cardiology Foundation

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Left ventricular (LV) structural adaptations to endurance exercise training (EET) have been well characterized. Initial cross-sectional data and more recent longitudinal studies demonstrate eccentric LV hypertrophy among trained individuals (1,2). LV functional adaptations to EET are not as well understood (3). Left ventricular torsion (LVT), an important marker of LV function in numerous cardiac diseases (4), has recently been investigated in the setting of exercise (5). The development of echocardiographic imaging techniques such as speckle tracking imaging (STI) (6) has made the study of LVT more feasible and accessible.

During systole, when viewed from the LV apex, the base rotates in an overall clockwise direction, and the apex rotates in a counterclockwise direction, resulting in LVT (4). In addition to the systolic phenomenon of LVT, the subsequent recoil and untwisting during early diastole is an important determinant of LV filling (7). Several recent studies have begun to evaluate LVT and untwisting rate (UTR) in the context of exercise. Specifically, LVT has been evaluated at rest in trained athletes and controls (8,9), during laboratory-based submaximal exercise (7,10,11), and after completion of endurance exercise events (12,13). To our knowledge, longitudinal data characterizing the impact of sustained exercise training on LVT has not been reported.

ABBREVIATIONS AND ACRONYMS

EET = endurance exercise training

IVRT = isovolumic relaxation time

LV = left ventricular

LVT = left ventricular torsion

STI = speckle tracking imaging

UTR = untwisting rate

We sought to determine the impact of EET on parameters of LV twisting. Specifically, we hypothesized that significant alterations in peak systolic LVT and peak early diastolic UTR would accompany the development of eccentric LV hypertrophy in a cohort of young, amateur male athletes. To address this hypothesis, we used STI with rigorous torsion-specific imaging standardization to evaluate LV twist parameters in competitive university rowers before and after a 90-day period of organized EET.

METHODS

Study population. Students participating in competitive athletics affiliated with the Harvard University Department of Athletics participated in this study. Written informed consent was obtained from all participants. The Harvard University institutional review board and the Partners Human Research Committee approved the protocol before study initiation.

Individuals were considered eligible if they were first-year university student athletes, ≥18 years old, and recruited members of the men's competitive rowing program. Height, weight, and resting vital signs were recorded at the time of enrollment. Training volume during the pre-study period, defined as the 8 weeks before baseline assessment, was collected. To further characterize exercise exposure during the pre-study period, data were collected about the performance of endurance and strength-building activities. Endurance activity was defined as running, cycling, swimming, rowing, or aerobic machine use at an effort sustainable for ≥20 min. Strength activity was defined as weight lifting, plyometric exercise, and sprint running drills. The study period began at the time of enrollment and lasted for 90 days. Daily data were recorded on the duration and the type of training activities performed during the study period. Subjects performed rowing training aimed to optimize performance at a 5-km distance that consisted of long duration open water and indoor ergometer sessions (1 to 3 h) at low stroke rates (20 to 24 strokes per min). Training volumes during the pre-study and study period were characterized by total number of hours/week and the hours/week dedicated to either endurance or strength activities. All participants were questioned confidentially about anabolic steroid use and were excluded if a history of use was elicited. Subjects were excluded from the final data analysis if they undertook any breaks in training of ≥3 days during the study period. Our population of first-year university male rowers represents a group of athletes with relatively limited prior training experience.

Cardiac structure and function. Transthoracic echocardiography was performed before and after the EET study period using a commercially available system (Vivid-I, GE Healthcare, Milwaukee, Wisconsin) with a 1.9- to 3.8-mHz phased-array transducer. Participants were imaged at rest ≥12 h after the most recent training session. Two-dimensional (2D), pulsed-Doppler, and color tissue Doppler imaging were performed from standard parasternal and apical transducer positions with 2D frame rates of 60 to 100 frames/s and tissue Doppler frame rates >100 frames/s. For each participant, apical and basal 2D short-axis images were obtained at the same frame rate to facilitate subsequent LVT analysis. All data were stored digitally, and off-line data analysis was performed (EchoPac, Version 7, GE Healthcare) by 2 cardiologists blinded to the study time point.

Cardiac structural measurements were made in accord with current guidelines (14). The LV ejection fraction was calculated using the modified Simpson's biplane technique. The LV length was measured in the apical 4-chamber view and was defined as the end-diastolic length from the mitral valve hinge point plane to the most distal endocardium at the LV apex. Resting heart rates were obtained from the final loop of each study. Aortic valve opening/closure and mitral valve opening were measured from pulsed-wave Doppler images. Isovolumic relaxation time (IVRT) was defined as the time from aortic valve closure to mitral valve opening. The left ventricular outflow tract (LVOT) diameter was measured from the parasternal long-axis view. Stroke volume was calculated as follows: cross-sectional $\text{area}_{\text{LVOT}} \times \text{VTI}_{\text{LVOT}}$, where cross-sectional area $= \Pi(\text{radius}_{\text{LVOT}})^2$ and VTI = velocity time integral. Cardiac output was calculated as the product of stroke volume and heart rate.

Longitudinal tissue velocities were measured offline from 2D color-coded tissue Doppler images and are reported as the average of 3 consecutive cardiac cycles. Longitudinal systolic strain measurements were made using speckle tracking analysis, and the mean from 6 myocardial segments was reported.

LV rotation, LV torsion, and untwisting. For the purpose of LV rotation assessment, short-axis imaging standardization within and across subjects was maximized using the following criteria. The basal level was defined as the highest basal imaging plane at which uniform full thickness myocardium was observed surrounding the mitral valve at end-systole (Fig. 1A). As the location of apical imaging acquisition has been shown to confer significant variability in the measurement of apical rotation (15), we carefully standardized apical imaging. Specifically, from multiple apical acquisitions distal to the papillary muscles, the apical level was chosen as the imaging plane with no visible

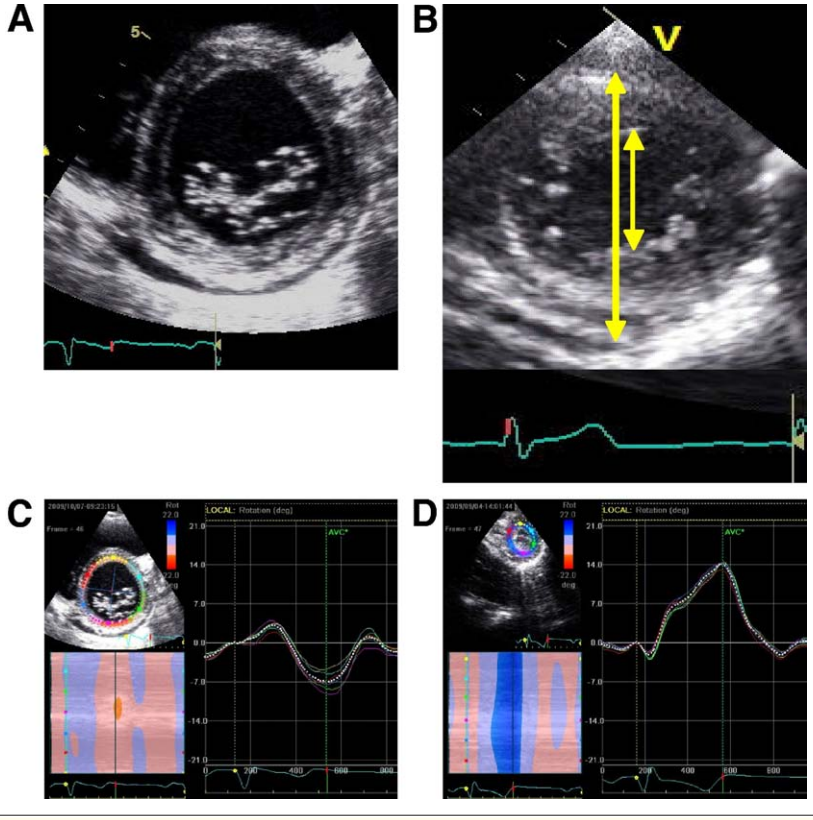

Figure 1. Representative Short-Axis Images and Rotation Curves

Strict imaging criteria were employed to standardize short-axis images. **(A)** An adequate basal image was defined by the presence of full-thickness myocardium surrounding the mitral valve at end systole. **(B)** The apical level was chosen as the imaging plane with no visible papillary muscles that most closely approximated an end-diastolic ratio of left ventricular (LV) cavity diameter **(short yellow arrow)** to total LV diameter **(long yellow arrow)** of 0.5. Speckle tracking analysis was used to generate **(C)** basal and **(D)** apical LV rotation profiles.

papillary muscles that most closely approximated an end-diastolic ratio of LV cavity diameter to total LV diameter of 0.5 (Fig. 1B).

Speckle tracking analysis was used to measure LV rotation, LVT, and UTR. The highest-quality digital 2D basal and apical images that met the above pre-specified criteria were selected, and the endocardium was traced. Within each subject, basal and apical images chosen for analysis had the same heart rate and were without sinus arrhythmia. A full-thickness myocardial region of interest was selected. The software automatically segmented the LV short axis into 6 segments and selected suitable speckles for tracking. The reliability of tracking was confirmed by the reliability parameter of the system (V = valid tracking; X = unacceptable tracking). When the software signaled poor tracking efficiency, the observer readjusted the endocardial trace line and/or region of interest width until an acceptable tracking score could be obtained. The LV rotation at the basal and apical short-axis planes was determined as the average angular displacement of 6 myocardial segments. Curves of basal and apical LV rotation (Fig. 1C and 1D), LVT, and UTR were automatically generated by the EchoPac Version 7 software.

All data were exported to a spreadsheet program (Excel, Microsoft Corporation, Redmond, Washington) for calculation of LVT and UTR. To adjust all parameters for intersubject differences in heart rate and imaging frame rate, the time sequence was normalized to the percentage of systolic ejection period duration (i.e., time 0% = the onset of the electrocardiographic QRS interval; time 100% = end-systole [aortic valve closure]). Peak systolic LVT was calculated as the maximum instantaneous difference between peak systolic apical and basal rotation. The timing of peak systolic LVT was determined as a percentage of systolic duration. Peak early diastolic UTR was defined as the peak untwisting velocity occurring in early diastole. The timing of peak early diastolic UTR was determined as a percentage of IVRT. The degree of untwisting that had occurred by the end of IVRT was also calculated. The LV rotation, LVT, and UTR are reported as absolute and LV length corrected values.

Measurement variability. The intraobserver and interobserver variability for LV twist parameters was examined. Intraobserver variability was performed by a single investigator by blinded assessment of the entire cohort on 2 separate occasions. Interobserver variability was assessed in a group of 10 randomly selected subjects by 2 investigators who were unaware of each other's measurements and of the study time point. Correlation coefficients for each measurement, derived from simple linear regression analysis, were used to quantify variability with the following results: intraobserver apical rotation (r^2 = 0.936), basal rotation (r^2 = 0.932), peak systolic LVT (r^2 = 0.948), and peak early diastolic UTR (r^2 = 0.942); interobserver apical rotation (r^2 = 0.979), basal rotation (r^2 = 0.948), peak systolic LVT (r^2 = 0.936), and peak early diastolic UTR (r^2 = 0.921).

Statistical analysis. Measurements are presented as mean ± SD. Student paired *t* test was used to compare baseline and post-training measurements after confirmation of normal distribution. Correlation analysis was performed using the Spearman and Pearson methods as appropriate for data distribution. A p value of <0.05 was considered significant.

RESULTS

Baseline and post-training clinical characteristics. We enrolled 15 male athletes, and all completed the training period. No participants were excluded because of steroid use or ≥3-day hiatus from training. Echocardiographic images suitable for complete analysis were obtained on all subjects. Baseline and post-training clinical characteristics are reported in Table 1.

Training regimens. During the 8 weeks before enrollment, subjects performed 8.5 ± 6.2 h/week of unsupervised training, which comprised 6.3 ± 5.3 h/week of endurance training and 2.2 ± 1.7 h/week of strength training. During the study period, subjects engaged in 13.6 ± 0.9 h/week of organized team training that was nearly exclusively dedicated to endurance activity (endurance = 12.6 ± 0.7 h/week; and strength = 1.0 ± 0.9 h/week). All participants engaged in organized training sessions ≥5 days/week throughout the entire study period.

Table 1. Baseline and Post-Training Clinical Characteristics

Clinical Parameter	Baseline	Post-Training	p Value
Age, yrs	18.6 ± 0.5	N/A	N/A
Height, cm	188.1 ± 6.7	N/A	N/A
Weight, kg	85.7 ± 8.9	86.8 ± 9.1	0.73
Body surface area, m^2	2.1 ± 0.1	2.1 ± 0.2	0.92
Heart rate, beats/min	60.5 ± 7.2	54.5 ± 5.7	0.03
Systolic blood pressure, mm Hg	113 ± 10	112 ± 11	0.78
Diastolic blood pressure, mm Hg	58 ± 6	53 ± 7	0.008

N/A = not applicable.

Cardiac structure. Baseline and post-training echocardiographic structural measurements are detailed in Table 2. Eccentric LV hypertrophy developed in participants. Specifically, there was an increase in LV end-diastolic volume and LV mass, with no significant change in relative wall thickness. The LV length increased after the EET period.

Cardiac function: conventional, strain, and tissue Doppler parameters. Baseline and post-training measures of cardiac systolic and diastolic function are detailed in Table 3. Stroke volume was significantly higher after the study period. Despite the increase in stroke volume, there was no change in resting cardiac output due to the concomitant fall in heart rate. Longitudinal strain and peak systolic basal lateral LV tissue velocity (S_m) significantly increased. Numerous parameters of diastolic function improved.

LV rotation, LV torsion, and untwisting. Absolute and LV length corrected twist data are summarized in Table 4, and composite rotation, LVT, and untwisting curves are shown in Figures 2 and 3. After the EET study period, there was a significant increase in peak systolic apical rotation but no change in basal rotation. This translated into a highly significant increase in peak systolic LVT after EET. There was no correlation between heart rate and LVT at either study time point, and there was a nonsignificant negative correlation between change in heart rate and change in LVT. Peak early diastolic UTR increased significantly after the study period. The time to peak UTR (% IVRT) did not change significantly with EET; however, there was a significant increase in the percentage of untwisting that occurred by end IVRT (Fig. 4). Finally, there was a significant correlation between the increase in peak systolic LVT and the increase in early diastolic UTR ($r^2 = 0.61$, $p < 0.001$).

Table 2. Comparison of Baseline and Post-Training LV Structural Parameters

LV Structural Parameter	Baseline	Post-Training	p Value
LVEDV/BSA, ml/m^2	80.8 ± 8.7	91.3 ± 8.0	<0.001
LVESV/BSA, ml/m^2	32.5 ± 5.1	36.1 ± 6.0	0.004
IVS/BSA, mm/m^2	4.6 ± 0.4	4.3 ± 0.6	0.15
PWT/BSA, mm/m^2	4.6 ± 0.6	4.5 ± 0.5	0.50
Relative wall thickness	0.35 ± 0.04	0.34 ± 0.04	0.08
LV mass/BSA, g/m^2	101.3 ± 11.4	115.7 ± 12.6	0.001
LV length, cm	8.9 ± 0.6	9.2 ± 0.6	0.01

BSA = body surface area; IVS = interventricular septum; LV = left ventricular; LVEDV = left ventricular end-diastolic volume; LVESV = left ventricular end-systolic volume; PWT = posterior wall thickness.

Prior studies examining LVT in the context of exercise can be categorized into 3 basic study designs. First, case-control comparisons of resting LVT have reported that professional soccer players (8) and elite cyclists (9) have significantly lower LVT than sedentary controls. Second, LVT and UTR have been shown to increase during laboratory-based submaximal exercise in healthy persons (7,10,11). Of note, subjects with pathologic LV hypertrophy and cardiac transplant recipients did not experience LVT/UTR augmentation during similar exercise (7,11). Finally, LV twist parameters have been assessed before and after completion of competitive athletic events (12,13). Chan-Dewar et al. (13) reported no significant change in LVT in amateur male runners after completing a marathon, whereas Nottin et al. (12) observed that LVT/UTR

Table 3. Comparison of Baseline and Post-Training LV Functional Parameters

	Baseline	Post-Training	p Value
Systolic function			
Stroke volume, ml	92.6 ± 18.9	104.3 ± 23.4	0.002
Cardiac output, l/min	5.6 ± 1.2	5.7 ± 1.5	0.71
LV ejection fraction, %	59.6 ± 3.8	59.7 ± 3.1	0.88
Systolic ejection period, ms	382.5 ± 24.3	379.8 ± 24.5	0.78
LV peak lateral S_m, cm/s	7.7 ± 1.5	8.8 ± 1.3	0.01
LV peak septal S_m, cm/s	6.5 ± 1.0	6.9 ± 1.1	0.29
LV longitudinal strain, %	−16.8 ± 2.1	−18.3 ± 2.8	0.02
Diastolic function			
Transmitral E-wave, cm/s	0.94 ± 0.2	0.99 ± 0.2	0.18
Transmitral A-wave, cm/s	0.37 ± 0.1	0.32 ± 0.1	0.06
E/A ratio	2.7 ± 0.7	3.2 ± 1.1	0.08
Isovolumic relaxation time, ms	61.9 ± 9.1	67.5 ± 10.8	0.08
Deceleration time, ms	181.1 ± 12.8	157.4 ± 16.4	<0.001
E_m basal septum, cm/s	−10.6 ± 1.9	−12.0 ± 1.8	0.003
A_m basal septum, cm/s	−3.5 ± 1.0	−3.1 ± 1.0	0.24
E_m basal lateral LV, cm/s	−12.5 ± 1.8	−14.5 ± 1.7	<0.001
A_m basal lateral LV, cm/s	−2.7 ± 1.0	−2.4 ± 0.7	0.34

A_m = late diastolic peak tissue velocity; E_m = early diastolic peak tissue velocity; LV = left ventricular; S_m = systolic peak tissue velocity.

DISCUSSION

We present novel, longitudinal data describing the impact of EET on LV twist mechanics in young male athletes during a 90-day period of endurance training. In this setting, the development of eccentric LV hypertrophy was associated with increases in LV apical rotation and peak systolic LVT. EET also led to significant increases in peak early diastolic UTR and the amount of untwisting that occurred during IVRT. To our knowledge, this study is the first longitudinal study evaluating changes in LV twist mechanics before and after a period of exercise training.

Table 4. Comparison of Baseline and Post-Training LV Twist Parameters

	Baseline	Post-Training	p Value
Systolic parameter			
Peak apical rotation, °	8.9 ± 4.2	12.7 ± 3.9	0.002
Normalized* peak apical rotation, °/cm	1.0 ± 0.5	1.4 ± 0.5	0.004
Peak basal rotation, °	−5.4 ± 1.8	−6.0 ± 2.2	0.49
Normalized* peak basal rotation, °/cm	−0.6 ± 0.2	−0.7 ± 0.2	0.62
Peak LVT, °	14.1 ± 5.0	18.0 ± 3.6	0.002
Normalized* peak LVT, °/cm	1.6 ± 0.6	2.0 ± 0.4	0.007
Time to peak LVT, % systole	96.1 ± 7.4	95.2 ± 7.3	0.76
Diastolic parameter			
Peak apical untwisting rate, °/s	−73.9 ± 23.9	−101.9 ± 36.9	0.01
Normalized* peak apical untwisting rate, (°/s)/cm	−8.4 ± 3.1	−11.3 ± 4.6	0.02
Peak basal untwisting rate, °/s	61.3 ± 12.8	59.1 ± 20.2	0.74
Normalized* peak basal untwisting rate, (°/s)/cm	6.9 ± 1.5	6.4 ± 2.0	0.49
Peak UTR, °/s	−110.6 ± 41.8	−148.0 ± 29.8	0.003
Normalized* peak UTR, (°/s)/cm	−12.6 ± 5.1	−16.2 ± 3.7	0.007
Time to peak UTR, % IVRT	78.9 ± 36.4	91.3 ± 31.6	0.23
% of untwisting at IVRT end, i.e., MVO	31.2 ± 12.0	39.9 ± 14.9	0.04

*Normalized for left ventricular (LV) length measured in the apical 4-chamber view.
IVRT = isovolumic relaxation time; LV = left ventricular; LVT = left ventricular torsion; MVO = mitral valve opening; UTR = untwisting rate.

were decreased and delayed after completion of an "Ironman" distance triathlon. Explanations for the discordant nature of these reports are unclear, but likely relate to subject age, prior athletic training, event type/duration, and measurement technique variability.

The present study examines LVT and exercise in a fourth and novel fashion. Specifically, we employed a longitudinal study design in which LVT-specific STI was used to examine LVT and UTR before and after a 90-day period of EET. Our data suggest that increases in LVT and UTR may represent a previously unrecognized and important component of the myocardial remodeling that occurs due to EET. In addition to the increases in LVT and UTR, physiologically relevant changes including augmentation of stroke volume and diastolic untwisting during IVRT were observed.

The finding that EET leads to increases in LVT and UTR may appear to contradict prior cross-sectional reports in athletes (8,9). The seemingly discordant nature of our data may be explained by several factors, including study population, study design, and measurement technique. First, differences in age and athletic background between our study population and prior cross-sectional studies deserve emphasis. Specifically, the present study consisted of incoming collegiate freshman (mean age of 18.6 ± 0.5 years) versus prior reports involving somewhat older subjects (8,9). More importantly, our study population consisted of inexperienced athletes with relatively more potential for cardiac remodeling than previously examined elite competitors (8,9). Second, prior cross-sectional data cannot accurately address the impact of training on LV twist mechanics. It is possible that previous studies report measurements that were taken at times of relative detraining, and thus reflect the regression that may occur during periods of reduced exercise. The third, and possibly the most significant, factor accounting for the differences between the current study and the previous cross-sectional data relates to measurement technique. As previously shown (15), the position of apical image sampling is a major determinant of the absolute values of calculated LVT. The majority of previous

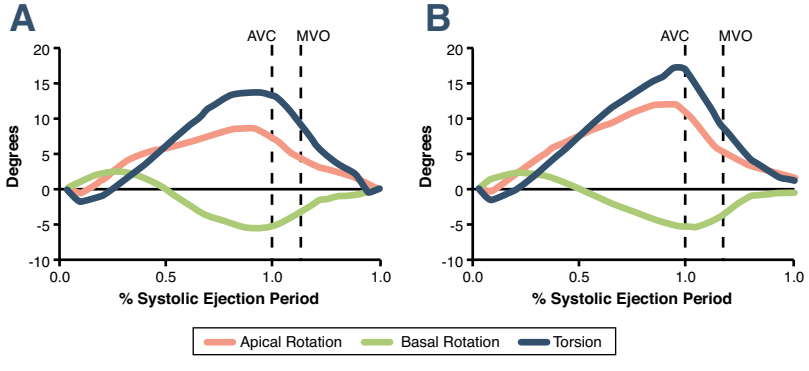

Figure 2. Composite Rotation and Torsion Curves

Apical rotation **(pink lines)**, basal rotation **(green lines)**, and left ventricular torsion **(blue lines)** profile curves **(A)** before and **(B)** after the exercise training period. An increase in left ventricular torsion was observed after exercise training, resulting from an increase in apical rotation. AVC = aortic valve closure; MVO = mitral valve opening.

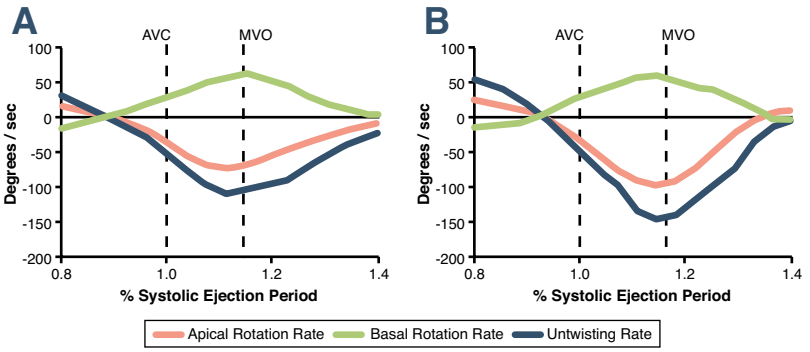

Figure 3. Composite Untwisting Curves

Apical rotation rate **(pink lines)**, basal rotation rate **(green lines)**, and untwisting rate **(blue lines)** curves **(A)** before and **(B)** after the exercise training period. An increase in apical rotation (untwisting) rate produced an increase in peak early diastolic untwisting rate. Abbreviations as in Figure 2.

LVT studies have defined apical imaging position simply as the location "below the papillary muscles" and that may predispose to measurement variability. Therefore, we utilized a rigorous apical standardization technique that led to both high reproducibility and absolute values that are consistent with other recent investigations (16).

Although mechanisms responsible for our findings remain speculative, factors including LV geometry, loading conditions, and cellular composition deserve mention. Length of the LV, a factor that should be accounted for when assessing LVT (16), increased in our participants after EET. However, changes in LV twist parameters remained significant after correction for LV length, demonstrating the limited contribution of this factor. In contrast, the contribution of loading conditions, specifically LV preload, may partially explain the observed results. It is know that EET results in significant blood volume expansion (17), and elegant prior animal work suggests that LV twist mechanics are preload dependent (18). As such, vascular volume expansion may contribute to the enhanced LVT and UTR that accompanied EET. Finally, changes in cellular protein expression may in part explain our observations. Titin, a myocardial cellular filament, plays an integral role in the storage and release of potential elastic energy during systole and diastole (19). Multiple splice pathways in the titin gene give rise to isoforms with different spring compositions (20), and variable titin isoform expression has been documented in several human cardiac diseases (21,22). Although cardiac titin isoform shifts have not been studied in the setting of EET, it is possible that this phenomenon may contribute to the observed changes in LV twist mechanics.

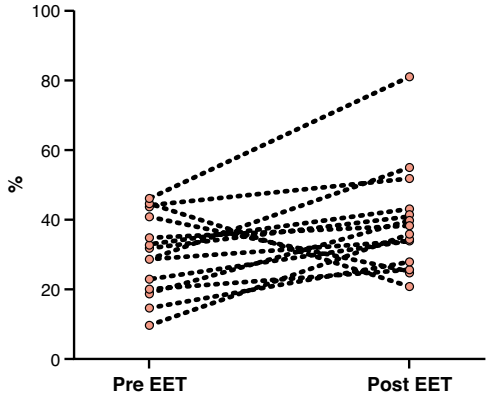

Figure 4. The Percentage of Untwisting by the End of the IVRT

There was a significant increase in the percentage (%) of untwisting that occurred by the end of the isovolumic relaxation time (IVRT) (31.2 ± 12.0% vs. 39.9 ± 14.9%, p = 0.04). This percentage increased in 13 of 15 subjects. EET = endurance exercise training.

There continues to be debate about whether the cardiac remodeling associated with athletic training is beneficial or maladaptive (3). Reduced LVT in the context of dilated cardiomyopathy (23) and after myocardial infarction (24) has established a potential association between reduced LVT and myocardial pathology. However, studies of pathologic LV hypertrophy have identified higher resting LVT in disease states such as aortic stenosis (25) and hypertrophic cardiomyopathy (7), raising the possibility that increased resting LVT actually rep-

resents a pathologic state. The current uncertainty regarding the significance of high resting LVT indicates that using LV twist parameters to differentiate physiologic and pathologic LV remodeling, although a worthy goal, is not feasible at the present time. It is likely that examination of LVT "reserve" (i.e., the ability to increase LVT during exercise) and analysis of exercise capacity/performance metrics in studies of LV twist mechanics in athletes may provide further insight.

There are several implications from our findings. First, the observation that LVT and UTR can augment with EET suggests that measurement of LV twist parameters has potential use as a noninvasive method for assessing the cardiac response to exercise. Further study in patients undergoing prescribed exercise for cardiac rehabilitation may be useful to address this issue. Second, the significant correlation between systolic twisting and diastolic untwisting coupled with the finding of increased untwisting during IVRT provides important insight into the physiology of the cardiac response to exercise. Finally, the use of torsion-specific STI protocols will help facilitate standardization of measurements and comparison of results from studies of LV twist.

Study limitations. The lack of data quantifying participant exercise capacity before and after the EET study period prevents us from reaching conclusions about the relationship between exercise capacity and LV twist mechanics. This represents an important area of on-going work. Second, this study included young, healthy, male subjects participating in a single form (rowing training) of EET. Therefore, our results may not be applicable to other populations participating in other forms of training.

CONCLUSIONS

We employed a prospective, longitudinal study design to determine whether EET produces changes in LV twist mechanics. We observed increases in peak systolic LVT and peak early diastolic UTR after 90 days of EET that remained significant after correction for LV length. These findings suggest that LVT and UTR augmentation may be an important and previously unrecognized component of exercise-induced cardiac remodeling.

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