Kinetics of Left Ventricular Strains and Torsion During Incremental Exercise in Healthy Subjects
The Key Role of Torsional Mechanics for Systolic-Diastolic Coupling

Grégory Doucende, MS; Iris Schuster, MD, PhD; Thomas Rupp, PhD; Aliona Startun, MD; Michel Dauzat, MD, PhD; Philippe Obert, PhD; Stéphane Nottin, PhD

Background —The dynamics of systolic and diastolic strains and torsional mechanics of the left ventricle (LV) and their relation to diastolic filling never have been evaluated at various exercise intensities.

Methods and Results —Speckle tracking echocardiography was performed in 20 healthy sedentary subjects at rest and during a progressive submaximal exercise test at 20%, 30%, and 40% of maximal aerobic power. LV twist increased progressively with exercise intensity (10.5±3.2 to 15.8±4.5°; P<0.001), whereas longitudinal strain remained unchanged after the first workload, underlining the key role of torsional reserve in systolic-diastolic coupling during exercise. The increase in diastolic untwisting (−88.7±34.2 to −182.9±53.5 deg·s⁻¹; P<0.01) was correlated to enhanced systolic twist (R=0.61; P<0.001), and its magnitude of increase was significantly higher compared to diastolic longitudinal and circumferential strain rates (119±64% versus 65±44% and 57±24%, respectively), emphasizing its contribution to diastolic filling. The timing of peak untwisting and the chronology of diastolic mechanical events were unchanged during effort. Untwisting was driven mainly by apical rotation and determined mitral opening and isovolumic relaxation time (R=0.47 and 0.61, respectively; P<0.001), whereas basal rotation and longitudinal and circumferential diastolic strain rates were major determinants of increased early diastolic filling (R=0.64, 0.79, and 0.81, respectively; P<0.001).

Conclusions —The use of speckle tracking echocardiography gives new insights into physiological adaptive LV mechanics during incremental exercise in healthy subjects, underlining the key role of torsional mechanics. It might be useful to better understand the mechanisms of diastolic dysfunction and exercise intolerance in various pathological conditions. (Circ Cardiovasc Imaging. 2010;3:586-594.)

Key Words: echocardiography • exercise • torsion mechanical

During diastole, left ventricle (LV) filling results from a complex interplay among numerous factors, such as myocardial relaxation, LV compliance, untwisting, and loading conditions. During systole, contraction of the cardiomyocytes induces both normal and shear strains, including LV torsion (ie, basal clockwise rotation and apical counterclockwise rotation) due to the helical orientation of myofibers.1–3 LV diastolic untwisting is a consequence of both an active relaxation of the myocardium and a release of the energy stored in compressed titin4 and elastic components of the interstitium.5 Importantly, untwisting occurs predominantly during isovolumic relaxation (IVR) and promotes LV suction by increasing LV intraventricular (ie, from apex to base) pressure gradients.6,7 Interestingly, LV untwisting is decreased or delayed in states associated with diastolic dysfunction, such as tachycardia-induced heart failure,8 dilated cardiomyopathy,9 and aortic stenosis,10 or following strenuous exercise.11 The evaluation of LV normal and shear strains and their timing during diastole is therefore of significant interest to assess diastolic dysfunction.

Clinical Perspective on p 594

Exercise stress echocardiography is a powerful tool to provide additional diagnostic and prognostic information in a variety of diseases12 and allows early detection of subtle myocardial dysfunction. During exercise, the increase in stroke volume is limited by diastolic filling13,14 because the increase in heart rate shortens the duration of diastole. Additionally, LV filling must be accomplished at relatively low filling pressures to avoid pulmonary vascular congestion. Early studies underlined the key role of untwisting in LV filling during exercise,15–17 but the time course of diastolic mechanical events, including both LV...
strains and torsional mechanics, has not been fully described. Moreover, no data are available regarding kinetics with increasing exercise intensity and timing of diastolic myocardial events during incremental exercise. Recently, speckle tracking echocardiography (STE) has provided the ability to quantify LV strains16 and torsion19 at rest and during exercise.15,16

In the present study, we aimed to describe dynamics and timing of LV myocardial mechanical events during an incremental exercise test. We used STE at rest and during an exercise conducted on a dedicated ergometer. We hypothesized that diastolic untwisting would increase progressively with exercise intensity, paralleling enhanced systolic twisting, and that amplitude and timing of LV diastolic mechanics would adapt to preserve or enhance LV filling when diastolic shortens during effort.

Methods

Study Population
We evaluated 20 young (mean age ± SD, 25 ± 9 years), healthy, sedentary men. None reported regular training habits or had any clinical or anamnestic evidence of cardiovascular disease or arterial hypertension. Subjects were excluded if resting echocardiography demonstrated an ejection fraction <50%, significant valvular disease, abnormal right ventricle function, or systolic arterial pulmonary pressure >35 mm Hg. This study was approved in Nimes from a part of the national ethics committee, and written informed consent was obtained from all subjects.

Experimental Protocol
Body height and mass were assessed. The maximal aerobic power was initially estimated via the Wasserman equation ([(body mass × (50.72−0.372×age))−350]/10.3) and corrected for the semisupine position (20% was removed from normal values).

The subjects were installed on a dedicated semisupine cycling ergometer (E-Bike). After a 15- to 20-minute resting period, each subject underwent an exercise test that included 3 workload stages of 6 minutes duration at 20% (W1), 30% (W2), and 40% (W3) of their maximal aerobic power and then stages of 1 minute from 50% to exhaustion by 10% increments. The pedaling rate was kept constant at 70 rpm for all subjects.

Two-dimensional and Doppler echocardiographic data were recorded at the end of the resting period and during the last 4 minutes of the W1, W2, and W3 stages. During the last 30 seconds of the test, we measured stroke volume. Gas exchanges were measured continuously by means of a cardiopulmonary exercise system (Ergocard). Systemic arterial blood pressure was measured during each stage of exercise in the left arm using manual sphygmomanometry and auscultation. Mean arterial pressure was calculated as 1/3(systolic pressure) + 2/3(diastolic pressure).

Echocardiographic Data Acquisition
Images were obtained with a commercially available system (Vivid 7) with a 3.5-MHz sector scanning electronic transducer. We recorded cine loops in parasagittal short-axis (basal, papillary muscle, and apical levels) and apical 4-chamber views. Two-dimensional gray-scale harmonic images were obtained at a rate of 65 to 90 frames per second, and color tissue velocity images were acquired at a rate of 120 to 140 frames per second. Images were acquired in cine loops triggered to the QRS complex and saved digitally for subframes per second, and color tissue velocity images were acquired at a rate of 65 to 90 frames per second. Systemic arterial blood pressure was measured during each stage of exercise in the left arm using manual sphygmomanometry and auscultation. Mean arterial pressure was calculated as 1/3(systolic pressure) + 2/3(diastolic pressure).

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Two-Dimensional and Tissue Doppler Echocardiography
M-mode measurements were obtained offline from the parasternal short-axis view recorded at the papillary muscle level. Pulsed Doppler LV inflow (E- and A-wave) recordings were performed in the apical 4-chamber view. Aortic blood flow velocity was recorded in the ascending aorta with a 2.0-MHz transducer (Pedof) placed at the suprasternal notch to assess stroke volume and cardiac output, as previously used in our laboratory.20,21 Systemic vascular resistance was estimated at each workload as mean arterial pressure divided by cardiac output. We measured the time delay in milliseconds from the onset of the ECG QRS interval to the onset of aortic blood flow (aortic opening [AO]), the peak of aortic blood flow, the end of aortic blood flow (aortic closure [AC]), the onset of early filling blood flow (mitral opening [MO]), the peak of early filling blood flow (peak-E), and the end of early filling blood flow. IVRT was calculated as MO−AC.

Tissue Doppler evaluation was performed offline from color cine loops recorded in the apical 4-chamber view. We assessed wall motion velocities at the mitral annulus level on the septal and lateral walls. The ratio of transmitial peak-E on peak early myocardial velocity of the lateral wall was used as an index of LV filling pressure.22

STE
Analysis of strain and torsion was conducted as previously described.11,13 After manually tracing the endocardial border on the end-systolic frame of the 2D sequence, the software automatically tracked myocardial motion. Whenever the software signaled poor tracking efficiency, the observer readjusted the endocardial trace line, the width of the region of interest, or both until a better tracking score could be obtained. Results were averaged on 3 to 5 cardiac cycles. LV longitudinal strain and strain rate (SR) were assessed using an apical 4-chamber view. Circumferential strain, SR, and LV rotation and rotational rate were assessed from short-axis views at basal and apical levels. 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 to improve LV apical rotation measurement.24

Two-dimensional strain data were processed with a specific toolbox (Scilab 4.1) developed in our laboratory. For temporal analysis, this software adjusted all strain variables for intersubject differences in heart rate and transducer frame rate acquisition. The time sequence was normalized to the percentage of systolic and diastolic duration (ie, AC represented 100% of systole, and end of cardiac cycle represented 100% of diastole) using interpolations. After normalization, the software averaged the data from 3 to 5 cardiac cycles and performed the detection of peak strain events and their timing (expressed as percentage of systolic duration). Net LV torsion was calculated as the instantaneous difference between LV apical and LV basal rotations. We calculated the following indexes of diastolic function during IVRT: untwisting angle (UT°/twist at AC−twist at end-IVRT); percentage of untwisting during IVRT [%UTIVRT=(UT°/twist at AC)×100]; and mean rate of untwisting during the IVRT (mean UTIVRT, deg · s⁻¹ = −UTIV°/IVRT). To assess the dynamics of global LV torsion and its relationship to radial displacement (reflecting volumetric changes of the LV) throughout the cardiac cycle, we constructed twist-radial displacement loops.11,13 Radial displacement data from 6 segments in basal and apical short-axis planes were averaged to obtained the mean value of radial displacement.

Statistical Analysis
All values in the text and tables are expressed as mean±SD and are shown as mean±SE in the figures. The statistical analysis was performed using specific software (Statview 5.0). For each cardiac variable, a repeated-measures ANOVA was performed with post hoc Bonferroni correction. Linear regressions were used to determine the relationships among IVRT, peak-E, and LV diastolic mechanical events. A multiple stepwise regression analysis was done to determine the mechanical events responsible for the increase in early filling during effort. Statistical significance for all analyses was assumed at P<0.05. Intraobserver reproducibility of STE previously was assessed in 12 subjects and was inferior at 8% for both strains and rotations.
Results

Height and body mass of the subjects were 177±5 cm and 72±8 kg, respectively. Resting echocardiographic and blood pressure data are shown in Table 1. At the end of exercise, the maximal values were as follows: aerobic power, 221±33 W; oxygen uptake, 34±5 mL · min⁻¹ · kg⁻¹; heart rate, 179±12 bpm; systolic pressure, 197±28 mm Hg; diastolic pressure, 99±7 mm Hg; stroke volume, 115.9±17.3 mL; cardiac output, 20.6±2.7 L · min⁻¹. The W1, W2, and W3 stages were performed at 33 W; oxygen uptake, 34±2%, and 38±3% of maximal aerobic power, respectively. Heart rate during each stage was 100±12, 110±12, and 121±12 bpm, respectively.

Kinetics of LV Hemodynamic Parameters

Kinetics of LV internal diameters and stroke volume are shown in Figure 1. LV end-diastolic diameter increased from rest to W1 and then slightly decreased from W1 to W3. LV end-systolic diameter progressively decreased from rest to W3. Stroke volume increased by 14±8% from rest to W1 and then remained constant until W3. Peak-E increased from rest to W2 (0.88±0.19 versus 1.23±0.17 m · s⁻¹; P=0.0001) then remained constant (1.23±0.17 versus 1.28±0.17 m · s⁻¹; NS), whereas peak mean early diastolic myocardial velocity increased only between rest and W1 (12.4±1.7 versus 14.6±1.3 cm · s⁻¹; P<0.0001) then plateaued until W3 (14.6±1.3 versus 14.7±1.9 cm · s⁻¹; NS). E/E* ratio, an index of LV filling pressure, increased from rest to W1 (6.5±1.1 versus 7.5±1.4 mm Hg; P<0.0001), and then remained constant (7.5±1.4 versus 8.1±1.5, NS).

Kinetics of LV Systolic Strains

Kinetics of LV longitudinal and circumferential (averaged from basal and apical levels) peak systolic strains and LV rotations are shown in Figure 2. LV longitudinal strain increased only from rest to W1, whereas LV circumferential strain and rotations progressively increased from rest to W3. As a result, LV torsion increased progressively from rest to W3 (10.5±3.2 to 15.8±4.5°; P<0.0001). Times to peak strains and torsion did not change during exercise excepted for LV apical rotations, which appeared earlier at W2 and W3 (Table 2).

Peak LV systolic SR and rotational and twisting rates progressively increased from rest to W3. Respective times to peak were not affected by exercise intensity except for LV peak basal rotational rate, which was significantly delayed during exercise. LV peak apical rotational rate showed no significant change from rest to exercise and occurred earlier than all other peak SRs (Table 3) independent of exercise intensity.

Kinetics of LV Diastolic Strains

During diastole, peak LV SR and rotational and untwisting rates increased progressively from rest to W3 (Table 3). A strong correlation was observed between peak LV torsion and peak LV untwisting rate (R=0.61; P<0.001). The magnitude of increase (expressed as percentage of resting conditions) was significantly higher for the LV untwisting rate (119±64%) than for changes of peak longitudinal and circumferential diastolic SRs (65±44% and 57±24%, respectively) (Figure 3). Whatever the exercise intensity, peak LV apical rotational and untwisting rates occurred closely to MO, whereas peak LV basal rotational rate and SRs were concomitant with transmitral peak-E (Figure 4). Peak untwisting rate (R=0.63; P<0.001) and peak LV diastolic apical rotational rate (R=0.48; P<0.001) were correlated with IVRT, whereas peak LV diastolic basal rotational rate and longitudinal and circumferential SRs were related to peak-E (R=0.64, 0.79, and 0.81, respectively; P<0.001). The stepwise regression analysis showed that these mechanical events mainly explained the increase in peak-E with exercise intensity (Table 4).

Mean UTIVRT progressively increased from rest to W3 (Table 5). The %UTIVRT decreased from rest to W1

![Figure 1](https://example.com/figure1.png)

**Figure 1.** Kinetics of LV end-diastolic and end-systolic diameters and stroke volume. EDD indicates end-diastolic diameter; ESD, end-systolic diameter. *Significant difference.

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**Table 1. Resting Echocardiographic and Blood Pressure Data**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate, bpm</td>
<td>66±9</td>
</tr>
<tr>
<td>Stroke volume, mL</td>
<td>99±14</td>
</tr>
<tr>
<td>Cardiac output, L · min⁻¹</td>
<td>6.4±1.4</td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>127±9</td>
</tr>
<tr>
<td>Diastolic</td>
<td>86±7</td>
</tr>
<tr>
<td>LV-EDV, mL</td>
<td>132±17</td>
</tr>
<tr>
<td>LV-EVS, mL</td>
<td>43±8</td>
</tr>
<tr>
<td>LVST, cm</td>
<td>0.9±0.1</td>
</tr>
<tr>
<td>PWT, cm</td>
<td>0.9±0.1</td>
</tr>
</tbody>
</table>

EDV indicates end-diastolic volume; ESV, end-systolic volume; IVST, inter-ventricular septum thickness; PWT, posterior wall thickness.
(45.3±22.5% versus 28.0±15.4%; \(P=0.0003\)) then remained unchanged during exercise (from 28.0±15.4% to 30.6±16.6%, NS). Peak untwisting rate occurred during the IVRT at rest and was delayed after MO during exercise when the IVRT shortened (Figure 4). UT° was stable from rest to W3 (between 3.5±1.6 and 4.1±2.1°; NS).

Twist-radial displacement loops (Figure 5) indicated that exercise intensity did not affect the relationship between radial displacement and torsion during the cardiac cycle. Systole was characterized by a roughly linear relationship between torsion and radial displacement and diastole by rapid untwisting before radial displacement.

**Discussion**

This study based on STE reports comprehensive kinetics of LV systolic and diastolic strains and torsion and their relationship to diastolic filling at different submaximal exercise intensities in healthy sedentary subjects. The major findings of the study were (1) that LV systolic twist increased progressively with exercise intensity, whereas systolic longitudinal strain remained unchanged after the first workload, underlining the key role of torsional reserve in systolic-diastolic coupling during exercise, and (2) that increased diastolic untwist, paralleling enhanced systolic twist, was driven mainly by early apical rotation and determined early mitral valve opening, whereas basal rotation and longitudinal and circumferential diastolic SRs were major determinants of increased early diastolic filling.

**Key Role of LV Torsional Reserve in Systolic-Diastolic Coupling During Exercise**

Very few data are available regarding the kinetics of LV strains during an incremental exercise test.\(^26\) Using STE, we observed that although longitudinal strains remained unchanged after the first workload of 20% of maximal aerobic power (Figure 1), LV circumferential strains, rotations, and torsion increased regularly with exercise intensity. The underlying mechanisms responsible for this heterogeneous response of LV strains are not well understood. LV torsion results from a complex arrangement of myocardial fibers within the LV wall.\(^27,28\) Contraction of the opposite helically oriented fibers creates LV torsion, which follows subepicardial layers because of their longer lever,\(^4\) and the progressive increase in LV torsion during effort reflected a higher contribution of subepicardial versus subendocardial layers.\(^29\) An explanation could be that subendocardial contractility could be blunted during effort because of its higher sensitivity to local ischemia.\(^30\)

Another explanation could be based on the regular decrease in LV end-diastolic diameters observed with exercise intensity (Figure 1). Indeed, a decrease in LV internal diameters improves the mechanical advantage of subepicardial layers\(^29,31\) that in turn could progressively enhance LV torsion during effort.

During the incremental exercise, the increase in LV untwisting rate was progressive and correlated to enhanced systolic torsion. The magnitude of increase was signifi-

**Table 2. Time to Peak of LV Strains, Rotations, and Torsion**

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>W1</th>
<th>W2</th>
<th>W3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Longitudinal strain</td>
<td>103±8</td>
<td>103±5</td>
<td>102±6</td>
<td>100±6</td>
</tr>
<tr>
<td>Circumferential strain</td>
<td>103±8</td>
<td>104±6</td>
<td>104±6</td>
<td>104±7</td>
</tr>
<tr>
<td>Basal rotation</td>
<td>108±20</td>
<td>109±13</td>
<td>109±16</td>
<td>111±17</td>
</tr>
<tr>
<td>Apical rotation</td>
<td>91±7</td>
<td>89±10</td>
<td>78±18*</td>
<td>72±18</td>
</tr>
<tr>
<td>Torsion</td>
<td>92±4</td>
<td>93±7</td>
<td>92±5</td>
<td>92±6</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD percentage of systolic duration. *Significant difference from previous workload.
fundamental mechanism to support diastolic filling with LV twist that is released during early diastole seems to be a with increasing heart rates. Thus, the storage of energy during become more important to LV filling as diastole shortens gest that the contribution of LV torsional mechanics might mechanics with increasing exercise intensities. Our results sug-

Table 3. Peak and Time-to-Peak SRs, Rotational Rates, and Untwisting Rate During Systole and Diastole

<table>
<thead>
<tr>
<th>Systole</th>
<th>Rest</th>
<th>W1</th>
<th>W2</th>
<th>W3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Longitudinal SR, s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>−0.96±0.14</td>
<td>−1.36±0.22*</td>
<td>−1.51±0.23*</td>
<td>−1.75±0.23*</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>44±11</td>
<td>43±7</td>
<td>44±7</td>
<td>39±8</td>
</tr>
<tr>
<td>Circumferential strain rate, s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>−1.18±0.15</td>
<td>−1.49±0.23*</td>
<td>−1.86±0.30*</td>
<td>−1.98±0.25*</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>46±5</td>
<td>47±7</td>
<td>46±6</td>
<td>47±9</td>
</tr>
<tr>
<td>Basal rotational rate, deg·s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>−72.0±16.5</td>
<td>−92.2±32.6*</td>
<td>−107.5±35.5*</td>
<td>−117.7±29.4</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>60±9</td>
<td>65±11*</td>
<td>66±10</td>
<td>68±11</td>
</tr>
<tr>
<td>Apical rotational rate, deg·s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>79.1±16.8</td>
<td>105.8±21.3*</td>
<td>117.5±23.6</td>
<td>133.1±23.7*</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>24±4</td>
<td>23±5</td>
<td>23±5</td>
<td>23±6</td>
</tr>
<tr>
<td>Twisting rate, deg·s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>68.3±15.6</td>
<td>97.1±31.9*</td>
<td>114.9±28.8*</td>
<td>124.1±35.3</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>56±11</td>
<td>52±18</td>
<td>55±17</td>
<td>53±18</td>
</tr>
<tr>
<td>Diastole</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Longitudinal strain rate, s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>1.24±0.34</td>
<td>1.77±0.36*</td>
<td>1.89±0.36</td>
<td>195±0.38</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>142±6</td>
<td>143±7</td>
<td>143±8</td>
<td>143±10</td>
</tr>
<tr>
<td>Circumferential strain rate, s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>1.00±0.14</td>
<td>1.42±0.21*</td>
<td>1.79±0.26*</td>
<td>1.87±0.27</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>145±6</td>
<td>145±8</td>
<td>146±11</td>
<td>146±12</td>
</tr>
<tr>
<td>Basal rotational rate, deg·s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>49.9±18.3</td>
<td>79.1±21.0*</td>
<td>90.8±29.5*</td>
<td>109.2±27.9*</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>135±16</td>
<td>141±18</td>
<td>144±19</td>
<td>148±21</td>
</tr>
<tr>
<td>Apical rotational rate, deg·s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>−78.5±31.3</td>
<td>−115.1±38.4*</td>
<td>−139.5±53.2*</td>
<td>−167.3±61.8*</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>114±10</td>
<td>119±9</td>
<td>119±6</td>
<td>114±6</td>
</tr>
<tr>
<td>Untwisting rate, deg·s⁻¹</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Peak</strong></td>
<td>−88.7±34.2</td>
<td>−119.3±42.8*</td>
<td>−157.1±53.0*</td>
<td>−182.9±53.5*</td>
</tr>
<tr>
<td><strong>TPP</strong></td>
<td>119±9</td>
<td>121±7</td>
<td>122±8</td>
<td>122±7</td>
</tr>
</tbody>
</table>

TPP indicates time-to-peak SR.

*Significant difference from previous workload.

stantly higher for untwisting rate than for other mechanical components (Figure 3 and Table 3), confirming its key role when diastolic time shortens. During diastole, whatever the exercise intensity, LV untwisting occurs earlier than LV normal strains, inducing an LV intraventricular pressure gradient that drives LV filling. The extent of untwisting increase was highly significant even between W2 and W3, suggesting that there is still an untwisting reserve when exercise intensities are higher.

This important torsional reserve probably plays a key role in systolic-diastolic coupling during exercise: A decreased end-diastolic volume due to a shortened filling period could induce enhanced systolic LV twist, which in turn results in increased diastolic untwisting to enable rapid filling and thus support stroke volume. Previous studies demonstrated the close functional relationship in normal subjects between systolic twisting and early diastolic untwisting, generating ventricular recoil and a negative intraventricular pressure gradient or suction. However, the present study is, to our knowledge, the first to report the kinetics of torsional mechanics with increasing exercise intensities. Our results suggest that the contribution of LV torsional mechanics might become more important to LV filling as diastole shortens with increasing heart rates. Thus, the storage of energy during LV twist that is released during early diastole seems to be a fundamental mechanism to support diastolic filling with increasing workloads.

**Unchanged Timing of Mechanical Diastolic Events**
The timing of diastolic mechanics are of major interest to better understand diastolic function, but studies in normal
subjects according to exercise intensity are lacking.\textsuperscript{16,17} Our results indicated that all time periods of the cardiac cycle were reduced proportionally with increasing heart rate (Figure 4) and that the chronology of different diastolic mechanical events was respected when time was expressed in percentage of systolic duration. The time-to-peak untwisting rate during effort was not different from resting values and was not affected by exercise intensity (Figure 4 and Table 3). Diastolic untwisting always occurred close to MO, preceding peak diastolic longitudinal and circumferential SRs, which occurred close to transmirtal peak-E (Figure 4) as evidenced by the similar profile of the torsion-radial displacement loops during diastole at rest and during exercise (Figure 5). The %UTIVRT decreased only from rest to W1 but was not further affected by exercise intensity (Figure 4 and Table 3). Diastolic untwisting always occurred close to MO, preceding peak diastolic longitudinal and circumferential SRs, which occurred close to transmirtal peak-E (Figure 4) as evidenced by the similar profile of the torsion-radial displacement loops during diastole at rest and during exercise (Figure 5). The %UTIVRT decreased only from rest to W1 but was not further affected by exercise intensity. Very few studies reported %UTIVRT during effort. Notomi et al\textsuperscript{17} obtained similar results at a heart rate of 112±10 bpm, whereas Esch et al\textsuperscript{16} did not find any difference between rest and effort in young healthy subjects. UT° was approximately 4° at rest and whatever the exercise intensity (Table 5), implying that a constant angle of untwist enabled a sufficient drop in LV pressure to MO. During effort, at end-systole, the atrioventricular pressure gradient is higher compared to resting values,\textsuperscript{32} suggesting that for a given elastic recoil (ie, for a given untwisting angle), the drop in LV pressure is higher during effort.

The Specific Roles of Apical and Basal Rotations

Previous studies documented that the LV apex rotates earlier than the LV base at rest,\textsuperscript{16,17,33} but information on timing of LV rotational events during exercise is missing. The results from the present study highlight that this chronology was respected during effort (Figure 4). Moreover, the difference between time-to-peak apical and basal rotation was increased when exercise intensity increased. Peak apical rotation occurred early in systole and time to peak shortened when exercise intensity increased (Table 2), whereas peak basal rotation occurred after AC, and time to peak remained constant during effort.

Consequently, during diastole, peak rotational rate occurred earlier at the apex, close to MO, whereas peak basal rotation occurred later, close to peak-E. At rest and during incremental exercise, the diastolic untwisting rate closely followed apical rotation, and their peaks occurred closely to MO. Strong correlations were found among peak untwisting, diastolic apical rotational rate, and IVRT, underlining their role for early MO when heart rate increases. Peak basal rotational rate appeared close to transmirtal peak-E (Tables 3 and 5). Strong correlations were found between peak-E and peak basal rotational rate, and stepwise regression analysis (Table 4) showed that 57% of peak-E was determined by 2 main factors: peak basal rotational rate (which was the strongest predictor) and longitudinal SR.

Previous studies also observed an apex-to-base dispersion in regional timing of LV strain events.\textsuperscript{34–36} The postsystolic strains of basal regions, partly due to a higher electrical-mechanical delay,\textsuperscript{37} have already been reported as an active mechanism enhancing the apex-to-base gradient of relaxation that facilitates rapid expansion of the LV cavity near the apex, causing a rapid decrease of LV pressure during IVRT.\textsuperscript{35} The results of the present study demonstrate that this mechanism also is observed during effort.

Clinical Implications

To our knowledge, this study is the first to report dynamics of LV torsion and strains during an incremental submaximal exercise test in healthy sedentary subjects. It gives new insights into physiological adaptive mechanisms of myocardial mechanics to preserve stoke volume when diastolic time shortens during effort, underlining the key role of torsional reserve in systolic-diastolic coupling with increasing workloads.

This potential of STE to assess the kinetics of LV diastolic mechanics and their interaction with LV filling during an incremental exercise is of major interest in the investigation of LV diastolic dysfunction in various pathological conditions.\textsuperscript{9,17} Previous studies documented altered torsional mechanics in aortic stenosis, hypertrophic cardiomyopathy, and dilated cardiomyopathy.\textsuperscript{11,19,23} but subtle dysfunction might become more apparent with exercise.
Few studies investigated LV torsion in patients at rest and at 1 single exercise load with a maximal heart rate $\leq 100$ bpm. In patients with heart failure with preserved ejection fraction, systolic LV twist did not increase normally with exercise, and the diastolic untwisting rate was significantly lower than in controls. In a study of 7 patients with hypertrophic cardiomyopathy, systolic torsion was higher in the patients than in the controls at rest but did not increase normally with exercise, whereas untwisting was delayed and nonenhanced.
The possibility of following the dynamics of these variables at different submaximal exercise intensities in patients should allow better understanding of the underlying mechanisms of exercise intolerance. Besides altered relaxation and increased ventricular stiffness, altered untwisting should be considered as 1 possible mechanism that contributes to exertional dyspnea.

Study Limitations
The use of STE limits the frame rate so that strain mechanics can be explored only at submaximal exercise intensities. However STE has greater feasibility and reproducibility than tissue Doppler imaging and allows timing of different mechanical events with sufficient accuracy for the explored exercise intensities and with a mean heart rate of 120 bpm at W3. Furthermore, stroke volume leveled off at W2 and remained unchanged between W2 and W3, so we can assume that major adaptive mechanisms during exercise took place during the 3 explored workloads. Previous studies indicated that exercise at 120 to 130 bpm was sufficient to detect diastolic dysfunction occurring during effort.12,17

The exact location of the basal and apical planes may be different from patient to patient. However, previous studies have validated the accuracy of STE versus tagged MRI,19 and care was taken to be at similar planes both at rest and during exercise.

Table 5. LV Diastolic Parameters

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Rest</th>
<th>W1</th>
<th>W2</th>
<th>W3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean UT_{vert} (deg · s⁻¹)</td>
<td>64.1±28.8</td>
<td>76.9±35.7</td>
<td>103.6±47.7*</td>
<td>110.7±47.6</td>
</tr>
<tr>
<td>UT°</td>
<td>3.9±1.5</td>
<td>3.5±1.6</td>
<td>4.1±2.1</td>
<td>3.8±1.9</td>
</tr>
<tr>
<td>%UT_{vert}</td>
<td>45.3±22.5</td>
<td>28.0±15.4</td>
<td>30.6±16.6</td>
<td>29.8±20.4</td>
</tr>
<tr>
<td>IVRT</td>
<td>66±18</td>
<td>46±13*</td>
<td>41±12</td>
<td>35±10</td>
</tr>
<tr>
<td>% systolic duration</td>
<td>20±6</td>
<td>17±5*</td>
<td>16±5</td>
<td>15±5</td>
</tr>
</tbody>
</table>

| Time to transmittal peak-E (ms)               | 480±33 | 394±35* | 364±33* | 342±35* |
| % systolic duration                           | 149±9   | 149±10  | 149±10  | 151±10  |

Data are presented as mean±SD.

*Significant difference from previous workload.

Discussion

Acknowledgments

We thank the Medical Sports Center of Nîmes city for support.

Disclosures

None.

References


**CLINICAL PERSPECTIVE**

Stress echocardiography is a powerful tool to provide additional diagnostic information in a variety of diseases and allows early detection of subtle myocardial dysfunction. Speckle tracking echocardiography has the potential to assess left ventricular (LV) strain and torsion during the cardiac cycle, but the kinetics of LV diastolic mechanics and their interaction with LV filling during incremental exercise have not been reported previously. This study shows that LV systolic twist and circumferential strain increased progressively with exercise intensity, whereas systolic longitudinal strain remained unchanged after the first imposed workload, underlining the key role of torsional reserve in systolic-diastolic coupling during exercise. During effort, the progressive increase in diastolic untwist, paralleling the increase in systolic twist, mainly was driven by early apical rotation and determined early mitral valve opening, whereas basal rotation and longitudinal diastolic strain rate were major determinants of increased early diastolic filling. The present study gives new insights into physiologic adaptive mechanisms of myocardial mechanics to preserve stroke volume when the length of diastole shortens during effort. By defining the normal response, these data also provide a reference with which the exercise response in pathologic states may be compared.
Kinetics of Left Ventricular Strains and Torsion During Incremental Exercise in Healthy Subjects: The Key Role of Torsional Mechanics for Systolic-Diastolic Coupling
Grégory Doucende, Iris Schuster, Thomas Rupp, Aliona Startun, Michel Dauzat, Philippe Obert and Stéphane Nottin

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