Left ventricular mechanics and arterial-ventricular coupling following high-intensity interval exercise

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Cote AT, Bredin SS, Phillips AA, Koehle MS, Glier MB, Devlin AM, Warburton DE. Left ventricular mechanics and arterial-ventricular coupling following high-intensity interval exercise. J Appl Physiol 115: 1705–1713, 2013. First published September 19, 2013; doi:10.1152/japplphysiol.00576.2013.—High-intensity exercise induces marked physiological stress affecting the secretion of catecholamines. Sustained elevations in catecholamines are thought to desensitize cardiac beta receptors and may be a possible mechanism in impaired cardiac function following strenuous exercise. In addition, attenuated arterial-ventricular coupling may identify vascular mechanisms in connection with postexercise attenuations in ventricular function. Thirty-nine normally active (NA) and endurance-trained (ET) men and women completed an echocardiographic evaluation of left ventricular function before and after an acute bout of high-intensity interval exercise (15 bouts of 1:2 min work:recovery cycling: 100% peak power output and 50 W, respectively). Following exercise, time to peak twist and peak untwisting velocity were delayed (P < 0.01) but did not differ by sex or training status. Interactions for sex and condition (rest vs. exercise) were found for longitudinal diastolic strain rate (men, 1.46 ± 0.19 to 1.28 ± 0.23 s−1 vs. women, 1.62 ± 0.25 to 1.63 ± 0.28 s−1; P < 0.01) and arterial elastance (men 2.20 ± 0.65 to 3.24 ± 1.02 mmHg·ml−1·m−2 vs. women 2.51 ± 0.61 to 2.93 ± 0.68 mmHg·ml−1·m−2; P = 0.04). No cardiac variables were found associated with catecholamine levels. The change in twist mechanics was associated with baseline aortic pulse-wave velocity (r2 = 0.27, P = 0.001). We conclude that males display greater reductions in contractility in response to high-intensity interval exercise, independent of catecholamine concentrations. Furthermore, a novel association of arterial stiffness and twist mechanics following high-intensity acute exercise illustrates the influence of vascular integrity on cardiac mechanics.

twist; strain; arterial stiffness; catecholamines

Prolonged exercise is well known to elicit transient reductions in ventricular function in healthy individuals (24, 28). Traditionally thought to be influenced by exercise duration (51), evidence also suggests that intensity of exercise influences the cardiac response to prolonged exercise (1). Specifically, a recent study reported impaired ventricular function following an acute bout of high-intensity interval exercise (41). Dimin-ished ventricular function may be due to the sympathetic nervous system response to strenuous exercise. Accumulating evidence supports a downregulation of β-adrenergic responsiveness as a meaningful contributor to the attenuated left ventricular (LV) function demonstrated with strenuous exercise (1, 17, 40).

Catecholamine levels, either sustained moderate elevations or acute marked elevations, have been proposed to mediate β-adrenergic sensitivity (1, 23). Catecholamines support the sympathetic system in modifying the blood flow during exercise, as well as increasing heart rate, contractile force and cardiac output by stimulation of the adrenergic β1-receptors in the myocardium (48). It is thought that elevated circulating catecholamines from strenuous exercise leads to decreased functional activity of cardiac β1-adrenergic receptors and thus to marked desensitization of the heart to inotropic β-adrenergic stimulation (22). In humans, exercise intensity is positively related to catecholamine levels (52) and has been shown recently to be negatively associated with β-adrenergic sensitivity (1).

Physiological adaptations with endurance training may convey divergent cardiac responses in response to high-intensity acute exercise. Recently, work from our research group found endurance-trained males experienced depressed systolic and diastolic function following high-intensity interval exercise, whereas this did not occur in normally active males (41). Plasma catecholamines have been shown to be significantly elevated in trained vs. untrained males during exercise (52) and thus may be a mechanism in postexercise attenuations in ventricular function. However, similar responses have not been shown in females (40, 52).

It has been demonstrated that LV performance is influenced by net arterial load and that cardiovascular performance is dependent on the interaction of both the ventricular and vascular systems (i.e., arterial-ventricular coupling) (18). Recently, Stohr et al. (45) found ventricular twist mechanics to be associated with augmentation index in healthy males, and that this relationship differed according to training status. At rest and during submaximal exercise, arterial-ventricular coupling has been shown to differ according to sex (26). Thus vascular mechanisms may also be responsible for postexercise attenuations in ventricular function and may be differentiated by sex and training status.
Accordingly, we sought to investigate postexercise depressions in ventricular function via a comprehensive assessment of cardiovascular function in endurance-trained and normally active males and females, following high-intensity interval exercise. Specifically, alterations in cardiac mechanics relative to catecholamines and vascular indexes were central to this investigation. We hypothesized that endurance-training and male sex would be distinguishing factors associated with susceptibility to an acute bout of high-intensity interval exercise as evidenced by greater reductions in left ventricular strain and twist mechanics, in the presence of greater catecholamine concentrations and diminished vascular indexes.

**METHODS**

Participants and ethical approval. We recruited 39 healthy, non-smoking men and women between the ages of 20–45 yr who met the inclusion criteria for normally-active (not engaged in any formal endurance training program) or endurance-trained (V\(_{\text{O}}\text{2max} \geq 55\) ml kg\(^{-1}\) min\(^{-1}\) and endurance training \(\geq 10\) h/wk for a minimum of 2 yr). Training volume was determined through questionnaire. Individuals were also prescreened via the Physical Activity Readiness Questionnaire for Everyone (PAR-Q\(^+\)) (49). Approval for this research was obtained via the Clinical Research Ethics Board at the University of British Columbia and conformed to the standards set by the latest revision of the Declaration of Helsinki. All participants provided written informed consent.

Experimental protocol. Participants visited the laboratory on two separate occasions. The first testing day involved prescreening and training questionnaire, as well as the acquisition of basic anthropometric data (height, body mass), baseline vascular measures (arterial compliance and pulse wave velocity), and the determination of maximal aerobic power (V\(_{\text{O}}\text{2max}\)). At the second visit, blood, urine, body mass, and blood pressure preceded echocardiographic assessments prior to and following a high-intensity interval exercise session. Measures of blood pressure and cardiac volumes were also measured 30 min postexercise to provide an assessment of cardiac function representing the recovery period. A minimum of 1 wk separated the two exercise visits. All participants were instructed to abstain from caffeine and alcohol for 12 h prior to each testing day. In addition, participants were asked to avoid strenuous exercise (i.e., interval training, hill climbs, weight training) in the 48-h time period prior to testing.

Anthropometrics, arterial stiffness, and compliance. Height and body mass were measured to the nearest 0.1 cm and 0.1 kg, respectively. Baseline vascular measures were assessed following 10 min of supine rest, and measured in the supine position. Participants were instrumented with an ECG and finger plethysmography secured to the middle finger (Finapres; Ohmeda, Englewood, CO). Automated blood pressure measurements were obtained by brachial occlusion (BpTRU 100, Coquitlam, Canada) for calibration purposes and collected in duplicate. In addition, pulse wave contours were collected at the carotid and femoral arteries using infrared photoelectric sensors (ADInstruments, Colorado Springs, CO). Continuous recordings were sent to a data acquisition system (PowerLab/16SP ML 795, ADInstruments) and displayed using Chart (version 7.0, ADInstruments). Pulse wave velocity was determined via postcollection analysis as previously described (34).

Arterial compliance was measured noninvasively via applanation tonometry with the HDI CR-2000 (Hypertension Diagnostics, Eagan, MN) for diastolic pulse contour analysis. This method of vascular assessment using waveform shape analysis is considered optimal for measuring systemic compliance (21) and is based on a modified Windkessel model that allows for the estimation of large (capacitive) artery and small (oscillatory) artery compliance. After stabilizing the wrist and maximizing signal strength, radial artery tonometry measurements were collected using the right wrist with the automated sphygmomanometer affixed to the upper left arm. Measurements were taken in duplicate with the average used for analysis.

\(V_{\text{O}}\text{2max}\). An incremental bike test to exhaustion (Velotron Dynafit Pro, RacerMate, Seattle, WA) was used for the assessment of \(V_{\text{O2max}}\) as previously described (41). Expired gases analyzed by a calibrated metabolic cart (Medisoft Ergocard, Belgium), heart rate (Polar, La-chine, Canada), and arterial hemoglobin via pulse oximeter (Masimo Radical, Irvine, CA), were measured throughout the test.

**Hydration assessment, blood sampling, and high-intensity interval exercise.** Urine specific gravity (Atago Pocket Refractometer, PAL-10S), was used for assessing hydration status prior to the high-intensity exercise session (33). A result of >1.02 (ratio of the density of urine to the density of water) served as the threshold of hydration status acceptable to commence testing (44).

Pre- and postexercise blood samples (6 ml each) were drawn from the medial cubital vein using a needle (23-gauge butterfly) and Vacutainer system to EDTA tube. The blood was centrifuged to remove plasma. Serum samples were stored at \(-80^\circ\text{C}\) until analysis. Epinephrine and norepinephrine concentrations were derived from immunoassay (TriCat Elisa RE59395, IBL Diagnostics, Hamburg, Germany).

Prior to the high-intensity interval exercise session, seated blood pressure was obtained after 5 min of quiet sitting, and repeated immediately following exercise (BpTRU 100, Coquitlam, Canada). A warm-up of 5 min of cycling followed by 2 min of inactive rest while seated on the bike, preceded the interval exercise: 15 one-min maximal work bouts (maximum workload attained at \(V_{\text{O2max}}\)) interspersed at the level of the real apex (12). All strain imaging was acquired by a trained clinical sonographer using a portable ultrasound unit (Vivid i, GE Healthcare) with simultaneous ECG and a 2.5-MHz phased-array transducer. Participants were positioned in the left lateral decubitus position for imaging. Apical two- and four-chamber views were acquired for the assessment of LV volumes and function, in accordance with the recommendations of the American Society of Echocardiography (20). Optimal parasternal short-axis views obtained at the base (mitral valve leaflets visible) and apex (distal to the papillary muscles at the level proximal to luminal obliteration) were acquired for the assessment of LV rotation, rotation rate, twist, circumferential and radial strain, and strain rate. Three consecutive cardiac cycles were recorded. Special care was taken to ensure the LV apical cross-section was as circular as possible, of clear image quality, and at the level of the real apex (12). All strain imaging was acquired at high frame rates (80–90 frames/s).

**Data analysis.** Ventricular volumes, diameters, and function were analyzed offline (EchopAC, GE Healthcare, v. 110.1.1). Left ventricular mass and volumes were determined using the linear method, and Simpson’s bi-plane method, respectively (20). Stroke volume and cardiac output were indexed for body surface area [stroke volume index (SVI) and cardiac index (CI)]. Ejection fraction (EF) was calculated as stroke volume as a percentage of end-diastolic volume (EDV). Fractional shortening (FS) was calculated from ventricular diameters using the parasternal long-axis window and expressed as a percent. Arterial elastance was calculated as \(E_a = \) end-diastolic pressure (ESP)/stroke volume, where ESP = 0.9 \(\times\) systolic blood pressure. Ventricular elastance was calculated via the formula \(E_{LV} = ESP/ESV\). Arterial-ventricular coupling was then determined as the ratio of arterial and ventricular elastance and indexed to BSA (\(E_a/\))

\(E_{LV}\)). One additional time point of postexercise arterial-ventricular coupling measure was calculated from blood pressure and volumes obtained 30 min postexercise. LV wall stress was determined noninvasively as calculated as 0.133 \(\times\) \(P \times R/2\) \(T \times (1 + T/2R)\), where \(P\)
is systolic blood pressure, $R$ is LV end-systolic diameter, and $T$ is LV systolic posterior wall thickness (38).

Stricker tracking analysis was used to assess all of the LV strain, rotation, and twist parameters. Twist and twist velocity were obtained by subtracting apical from basal rotation and rotation rates (30) and adjusted for inter- and intraindividual differences in heart rate; raw data were normalized to a percentage of systolic duration, with the onset of the QRS being 0% and aortic valve closure equivalent to 100%, while diastole was from 100% onward. Cubic spline interpolation was then used to normalize the data at 2% increments of systolic and diastolic duration throughout the cardiac cycle (8).

**Statistical analysis.** Data are presented as means ± SD. Baseline characteristics between sex and training status (“group”) were assessed using a two-way ANOVA. The effect of exercise on dependent variables were analyzed using a three-way ANOVA (sex × group × time). Pearson correlation coefficient was performed between the change in cardiac parameters and surrogates of loading. Linear regression was used to assess the effect of vascular measures and catecholamines on cardiac mechanics, adjusted for age and sex. The alpha level for statistical significance was set a priori at $P \leq 0.05$.

**RESULTS**

**Participant characteristics.** The endurance-trained (ET) group represented 20 cyclists (9 females). Their training frequency and volume were 5.2 ± 1.0 days and 12.2 ± 1.4 h of training per week, respectively. The normally active (NA) group consisted of 19 individuals (9 females) who performed moderate levels of physical activity, 3.9 ± 1.5 days/wk (i.e., yoga, resistance training, recreational level soccer). Participant characteristics including vascular assessments are presented in Table 1. Sex differences ($P < 0.05$) were evident for height, body mass, arterial compliance, LV mass, SV, and CI. The ET differed from NA participants with respect to $V_{O2\text{max}}$, heart rate, large artery compliance, LV mass, SV, and CI ($P < 0.05$).

**Hydration, catecholamines, and hemodynamic parameters.** Prior to the interval session, urine specific gravity was within the range considered to represent adequate hydration, averaging 1.012 ± 0.006. Body mass decreased 0.27 ± 0.47 kg following exercise, and this response was unaffected by sex or training status. The effect of exercise on the change in catecholamines is shown in Fig. 1. No effect of sex was found for the change in catecholamines with exercise. With respect to training status, epinephrine increased to a greater extent in ET than NA (3.38 ± 0.57 and 2.87 ± 0.54 nmol/L, respectively; $P = 0.016$). Norepinephrine increased with exercise ($P < 0.001$) and was not different between groups. However, LV mass was significantly correlated with the change in norepinephrine ($r = 0.38$, $P = 0.032$) with a stronger association found when LV mass was expressed according to body surface area ($r = 0.51$, $P = 0.003$).

There was an interaction of condition by group for exercise heart rate. In ET, resting heart rate was lower than NA, but increased to a greater extent during the interval exercise session, and then returned to lower values in recovery ($\Delta$HR, $\pm 106$ beats/min vs. 113 beats/min in NA and ET, respectively; $P = 0.012$). Mean arterial pressure was similarly affected (NA men: 80.6 ± 4.0 to 77.2 ± 9.2; NA women: 75.6 ± 7.5 to 72.8 ± 8.0; ET men: 81.1 ± 4.9 to 74.1 ± 8.0; ET women: 76.0 ± 11.0 to 74.7 ± 12.8 mmHg, baseline to postexercise, respectively; $P = 0.013$). ET displayed greater reductions in SVI than NA (ET, $-16.6$ ± 12.9%; NA, $-15.3$ ± 19.9%, $P = 0.024$). There was also an independent effect of sex for SVI as men experienced
greater reductions postexercise than women (men: 51.3 ± 12.9 to 39.7 ± 9.0 ml/m²; women: 41.8 ± 9.2 to 36.6 ± 7.1 ml/m², p = 0.028).

Cardiac function and arterial-ventricular coupling. Fractional shortening, wall stress, septal and lateral wall tissue velocities, and isovolumetric relaxation time were reduced postexercise (Table 2). An interaction was present only for early diastolic filling (E), whereby NA began with higher baseline early filling but postexercise displayed lower early diastolic filling than ET. Longitudinal, circumferential, and radial strain and strain rate, in addition to rotation and twist parameters (Table 3), were similar regardless of sex or training status, except for longitudinal diastolic strain rate. Diastolic longitudinal strain rate was reduced postexercise in men, but not women (P = 0.011; Fig. 2). Time to peak twist and time to peak untwisting rate were delayed postexercise (P < 0.000 and 0.002, respectively; Fig. 3). Baseline pulse wave velocity was significantly associated with twist augmentation following exercise (r² = 0.27, P = 0.001; Fig. 4), indicating that individuals with higher pulse wave velocity (greater arterial stiffness) were less likely to increase twist following exercise. Changes in apical rotation rate were also significantly associated with PWV (systolic: r = 0.449, P = 0.004; diastolic: r = −0.398, P = 0.012). Other baseline vascular measures were not associated with change in twist (LAC: r = −0.171, P = 0.299; SAC: r = −0.029, P = 0.863).

Arterial-ventricular coupling (EAI/ELVI; Fig. 5) was significantly different from baseline (P = 0.008) at 30 min postexercise, and an interaction between sex and condition was revealed for arterial elastance (P = 0.043) at this same time point. Independent of training status, the recovery arterial elastance component (postexercise 2) was similar to baseline values in the women, whereas this was significantly elevated in the males. Ventricular elastance was significantly elevated from baseline at the first postexercise assessment only (P = 0.02). No effect of training status (group) was found for any of the arterial-ventricular coupling measures. No correlations were found for changes in heart rate, strain rate, diastolic filling, pressures, or volumes. In addition, changes in catecholamines were not associated with any cardiac parameters.

DISCUSSION

The main findings of this investigation are 1) lower baseline arterial stiffness is associated with greater cardiac twist augmentation following exercise; 2) males demonstrate greater postexercise reductions in cardiac contractility, and increased arterial elastance than females following exercise; 3) the change in plasma catecholamines was not associated with alterations in cardiac function; and 4) training status did not distinguish the cardiovascular response to high-intensity interval exercise. Collectively, these results demonstrate independent effects of vascular structure and sex on cardiac mechanics following strenuous exercise in healthy adults.

Arterial stiffness and twist. We found a novel association between baseline arterial stiffness and change in twist mechanics following high-intensity exercise; individuals with lower arterial stiffness were better able to augment cardiac twist in the face of increased demands. It is well known that an elastic vascular system reduces cardiac demand (2) and this relationship may partially mediate the influence of afterload on LV twist mechanics (42). Increased large artery stiffness augments output impedance through increased propagation velocity of reflected pulse waveforms (29). Following this, afterload amplification due to increased aortic stiffness may not be present when blood pressure is measured at the brachial level, but would be occurring at the level of the heart (21). In support of the relationship between arterial stiffness and cardiac twist mechanics, Stohr and colleagues (45) demonstrated a correlation between cardiac twist and augmentation index (another marker of arterial stiffness) when pooling together values taken at rest and during submaximal exercise (45). Aortic distending pressure and sympathetic activity both increase during exercise and acutely stiffen both central and peripheral arterial segments.
Cardiac Mechanics Following High-Intensity Interval Exercise • Cote AT et al.

Table 3. Peak left ventricular mechanics at rest and during exercise

<table>
<thead>
<tr>
<th></th>
<th>NA</th>
<th>ET</th>
<th>NA</th>
<th>ET</th>
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</thead>
<tbody>
<tr>
<td>Basal rotation, °</td>
<td>−11.71 ± 21.27</td>
<td>−5.85 ± 2.20</td>
<td>−5.76 ± 1.68</td>
<td>−6.12 ± 2.71</td>
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<tr>
<td>Apical rotation, °</td>
<td>12.94 ± 5.16</td>
<td>15.01 ± 2.79</td>
<td>12.20 ± 3.93</td>
<td>11.69 ± 4.55</td>
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<tr>
<td>Twist, °</td>
<td>17.79 ± 6.15</td>
<td>19.82 ± 3.26</td>
<td>17.36 ± 4.94</td>
<td>16.65 ± 7.13</td>
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<td>Basal level</td>
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<tr>
<td>Radial strain, %</td>
<td>−18.95 ± 2.90</td>
<td>−16.79 ± 2.41</td>
<td>−18.51 ± 2.06</td>
<td>−16.47 ± 1.79</td>
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<tr>
<td>Apical rotation, °</td>
<td>40.95 ± 10.33</td>
<td>28.74 ± 12.60</td>
<td>56.82 ± 13.71</td>
<td>43.10 ± 19.25</td>
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<tr>
<td>Radial strain, %</td>
<td>10.37 ± 8.96</td>
<td>13.69 ± 9.98</td>
<td>10.96 ± 11.90</td>
<td>15.20 ± 9.52</td>
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<td>Circumferential strain, %</td>
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<tr>
<td>Basal level</td>
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<tr>
<td>Apical level</td>
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</tr>
<tr>
<td>Radial strain, %</td>
<td>−16.33 ± 5.29</td>
<td>−13.63 ± 3.21</td>
<td>−16.60 ± 4.25</td>
<td>−16.06 ± 4.40</td>
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<td>Basal level</td>
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<tr>
<td>Basal level</td>
<td>−68.54 ± 11.81</td>
<td>−65.83 ± 27.68</td>
<td>−53.43 ± 21.72</td>
<td>−62.75 ± 21.67</td>
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<td>Apical rotation, °</td>
<td>72.06 ± 17.85</td>
<td>94.52 ± 27.33</td>
<td>54.97 ± 16.43</td>
<td>68.02 ± 19.68</td>
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<td>Twist velocity, °</td>
<td>111.3 ± 31.88</td>
<td>121.77 ± 26.42</td>
<td>101.63 ± 21.39</td>
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<td>Strain rate, s−1</td>
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<tr>
<td>Longitudinal</td>
<td>−1.01 ± 0.17</td>
<td>−0.96 ± 0.16</td>
<td>−0.89 ± 0.11</td>
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<td>Radial basal</td>
<td>1.90 ± 0.84</td>
<td>1.91 ± 1.25</td>
<td>1.53 ± 0.29</td>
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<td>Radial apical*</td>
<td>1.03 ± 0.46</td>
<td>1.40 ± 0.65</td>
<td>1.10 ± 0.57</td>
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<td>−1.78 ± 0.54</td>
<td>−1.82 ± 0.68</td>
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<td>Basal level</td>
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<td>66.18 ± 12.60</td>
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<td>Apical rotation, °</td>
<td>−85.57 ± 30.71</td>
<td>−108.57 ± 30.40</td>
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<td>−139.86 ± 44.45</td>
<td>−128.03 ± 23.09</td>
<td>−108.71 ± 32.99</td>
<td>−120.22 ± 41.61</td>
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<tr>
<td>Strain rate, s−1</td>
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<tr>
<td>Longitudinal</td>
<td>1.58 ± 0.14</td>
<td>1.32 ± 0.29</td>
<td>1.36 ± 0.17</td>
<td>1.24 ± 0.17</td>
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<tr>
<td>Radial basal</td>
<td>−1.58 ± 0.39</td>
<td>−1.66 ± 0.51</td>
<td>−1.48 ± 0.44</td>
<td>−1.65 ± 0.41</td>
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<tr>
<td>Radial apical*</td>
<td>1.36 ± 0.70</td>
<td>1.96 ± 1.34</td>
<td>1.14 ± 0.53</td>
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<td>Circumferential basal</td>
<td>1.16 ± 0.53</td>
<td>1.08 ± 0.35</td>
<td>1.11 ± 0.29</td>
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<td>Circumferential apical</td>
<td>2.72 ± 0.96</td>
<td>2.32 ± 1.40</td>
<td>2.94 ± 1.08</td>
<td>3.15 ± 1.3</td>
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</table>

Values are means ± SD. *P < 0.05 vs. baseline; ‡P < 0.05 condition by sex interaction.

(35, 36). Although we did not measure PWV during exercise, it is possible that a stiffer resting central arterial segment translates increases in peripherally mediated afterload during exercise back to the heart and effectively inhibits increases in twist compared with those with a more compliant central arterial segment. In the absence of a follow-up measure of arterial stiffness, we are unable to present whether the relative differences between groups observed at baseline remained constant during and after exercise. Nonetheless, considering our findings with that of Stohr et al. (45), the importance of arterial function on LV mechanics is evident and requires further study.

Sex differences. This investigation is the first to report sex differences in cardiac mechanics following high-intensity interval exercise. Peak longitudinal strain rate has been shown to closely correlate with invasive markers of LV contractility, and is considered a robust measure of regional myocardial dysfunction (28). Ventricular dysfunction has been suggested to begin in the longitudinal plane while circumferential and radial compensations remain to preserve ejection fraction in individuals with cardiovascular risk factors (25). Thus the reduced longitudinal diastolic strain rate postexercise in males and not females suggests men experience greater reductions in contractility with strenuous exercise and is in agreement with our previous findings using graded dobutamine infusions following prolonged strenuous exercise (40).

Arterial-ventricular coupling provides insight into the interaction between the LV and the arterial system and is expressed as a ratio of arterial and ventricular elastance (Ea/Elv) to which a higher ratio indicates an optimal stroke work and energetic efficiency (3). The arterial elastance component was significantly higher in the men compared with the women, regardless of training status. This component is considered a measure of impedance (being influenced by static and pulsatile afterload, and by heart rate) (9). Regarding the constituents of the arterial elastance factor, men and women displayed similar alterations in end-systolic pressure; yet SVI decreased significantly greater in the males resulting in a higher Ea. A higher ratio of end-systolic volume to end-diastolic volume in the men can be interpreted to reflect higher afterload compared with the women. In healthy hearts, modest elevations of afterload can increase the rate of LV active relaxation (11) and thus may explain why the Ea1 Voldemort component, and as such the Ea/Ea1 Voldemort ratio, was not different between the sexes. The higher recovery Ea1 in the men is likely an effenter response to maintain blood pressure (16). As the Ea/Ea1 Voldemort ratio was within an ideal range, neither sex was limited in the ability to adequately transfer blood from the LV to the periphery (5).

With respect to the ventricular component, women across all ages have been shown to have a higher Ea1 Voldemort than age-matched males (37), suggesting this higher LV contractility may allow women to tolerate cardiovascular stress better than men (6). In the present study, women tended to have a higher Ea1 Voldemort (P = 0.07), suggesting enhanced contractility (9). Coupled with a higher EF for a given EDV (another indication of enhanced systolic function), these findings are indicative of enhanced systolic function in the women in both rest and exercise conditions. Collectively, the sex differences presented in this
investment provide evidence of enhanced cardiac function in females in response to high-intensity exercise, and to our knowledge has not been reported before.

**Exercise intensity and training status.** Alterations in systolic and diastolic parameters illustrate the effect of high-intensity interval exercise on the heart. Prior work assessing very short duration, supramaximal efforts such as the Wingate (39), and strenuous exercise of 6–8 min failed to show any LV functional decline (7, 27). Similar to other forms of strenuous exercise (24), we found reduced function in traditional systolic indexes such as fractional shortening and wall stress, in addition to strain and twist mechanics. The reductions in peak longitudinal strain and peak systolic tissue velocities are indicative of altered myocardial contractility (14, 46). Furthermore, the delayed twist mechanics occurring during a shortened isovolumetric relaxation phase likely impacted diastolic suction (31) as evidenced by reduced early diastolic filling. The reduced strain, strain rate, and delayed twist may reflect altered intrinsic myocardial relaxation properties as a result of fatigue-induced impaired metabolism within cardiomyocytes (10, 31). The present findings corroborate our previous work (41) and demonstrate that high-intensity interval exercise is a sufficient stimulus to cause impaired relaxation and contraction of the LV (32). However, in contrast to our previous work demonstrating cardiac fatigue in endurance-trained but not normally active individuals following the same protocol, the present sample of endurance-trained and normally active individuals performed similarly postexercise, with improved diastolic filling in ET. The greater elevations in epinephrine following exercise in ET align with previous work reporting higher catecholamine concentrations in trained individuals following high-intensity acute exercise (52). In addition, while we did not see a distinction in norepinephrine response between ET and NA, the association of change in norepinephrine to LV mass (absolute and relative) may be perceived as an indication of training status. Previous work has demonstrated that norepinephrine spillover is increased in athletes with LV hypertrophy compared with an inactive control group (43). Plasma catecholamines have been shown to be significantly elevated in trained males vs. untrained individuals and is thought to be due to a greater metabolic rate and substrate degradation, stimulating higher sympathoadrenal activation in trained individuals (15). However, in the present study, following high-intensity interval exercise, changes in catecholamines were not associated with any measures of cardiac function. Thus we must reject our hypothesis that ET males would demonstrate the

**Fig. 2.** Effect of exercise on diastolic longitudinal strain rate by individual. Diastolic longitudinal strain rate was reduced postexercise in men, but not women regardless of training status. *P < 0.05 vs. baseline.

**Fig. 3.** Time course of left ventricular twist and twist rates by sex. Time to peak twist (A) was delayed postexercise (*P < 0.0001) and was not different based on sex or training status. Time to peak twist rate (B) was not significantly different from baseline; however, time to peak untwist rate (B) was significantly delayed (*P = 0.002). Both peak twist rate and peak untwist rate were not different among groups.
greatest reductions in cardiac function, associated with higher catecholamine concentrations.

Limitations. In this study our objective was to assess cardiac function as close to the cessation of exercise as possible. We did not attempt to image the heart during exercise as obtaining quality images at such high work rates is exceptionally difficult. With the exception of ventricular-vascular coupling, we did not perform additional cardiac assessment in the hours following exercise, by which the persistence of altered ventricular function could be determined. Recently, Goodman et al. (13) demonstrated a preserved left ventricular function during prolonged strenuous exercise that became significantly reduced in the recovery period (13). However, in our previous investigation (41) two postexercise cardiac assessments revealed no additional depressions in ventricular function at follow-up. Furthermore, it may be seen as a limitation that the assessment of LV mass in this investigation was performed using the ASE formula. As this method does not take LV length into account, the area-length method or truncated ellipsoid formulas may be more appropriate in athletic populations.

Relevance. The findings of this research are relevant to advancing our understanding of the acute effects of high-intensity exercise on the heart. High-intensity interval training has gained popularity in nonathlete populations driven largely by accumulating evidence showing greater cardiovascular and metabolic benefits for both healthy and patient populations compared with traditional submaximal aerobic exercise (4, 19). However, there is also an acute increase in the risk for cardiac events during and following vigorous intensity activities compared with inactive periods (50).

Conclusions. In summary, we present novel evidence of an association between arterial stiffness and cardiac twist suggesting the importance of vascular integrity for the adequate cardiac compensation to the demands of strenuous work. Further, we surmise that endurance-trained and normally active individuals similarly experience exercise-induced reductions in cardiovascular function following high-intensity interval exercise, which are not associated with increases in plasma catecholamines. Greater reductions in cardiac contractility demonstrated in males may reflect differential intrinsic myocardial relaxation properties, the mechanisms of which require further research.

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