HIGHLIGHTED TOPIC | Mechanisms and Modulators of Temperature Regulation

Aerobic fitness does not influence the biventricular response to whole body passive heat stress

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Aerobic fitness does not influence the biventricular response to whole body passive heat stress. J Appl Physiol 109: 1545–1551, 2010. First published August 19, 2010; doi:10.1152/japplphysiol.00769.2010.—We examined biventricular function during passive heat stress in endurance trained (ET) and untrained (UT) men to evaluate whether aerobic fitness alters the biventricular response to whole body passive heat stress. Whole body passive heat stress reduced biventricular end-diastolic (ET, −19.5 ± 24.0 ml; UT, −25.1 ± 23.8 ml) and end-systolic (ET, −15.9 ± 8.8 ml; UT, −17.6 ± 7.9 ml) volumes and left atrial volume (ET, −19.2 ± 11.6 ml; UT, −15.0 ± 12.7 ml) and significantly increased heart rate (ET, 29.3 ± 9.0 beats/min; UT, 31.7 ± 10.4 beats/min) and cardiac output (ET, 3.8 ± 2.2 l/min; UT, 3.2 ± 1.4 l/min) similarly, while biventricular stroke volume was unchanged. There were no between-group differences in any parameter. Heat stress increased (P < 0.05), as a percentage of baseline values, biventricular ejection fraction (ET, 3.4 ± 5.3%; UT, 4.4 ± 3.7%), annular systolic tissue velocities (ET, 32.5 ± 34.9%; UT, 44.0 ± 38.1%), and peak LV twist (ET, 51.6 ± 59.7%; UT, 59.7 ± 54.2%) and untwisting rates (ET, 45.5 ± 42.3%; UT, 51.8 ± 55.0%) similarly in both groups. Early LV diastolic tissue and blood velocities, volumetric flow rates, and strain rates (diastole) were unchanged with heat stress in both groups. The present findings indicate that aerobic fitness does not influence the biventricular response to passive heat stress.

Cardiac magnetic resonance imaging

Whole body passive heat stress poses a significant cardiovascular challenge. To facilitate heat exchange, cutaneous blood flow increases by as much as 7 l/min (29). The ability of the cardiovascular system to maintain arterial blood pressure is therefore dependent on its capacity to compensate for a large reduction in total peripheral resistance (i.e., which follows the increase in cutaneous vascular conductance). Augmented cardiac output, which can more than double during passive heating (31), in combination with increased sympathetic vasoconstriction of skeletal muscle (8, 23), and the renal and splanchnic (7, 21, 30, 32) vascular beds serve to maintain arterial blood pressure during heat stress. Rowell et al. (33) first demonstrated that the change in cardiac output is predominantly heart rate mediated; however, these investigators also found that stroke volume increased by as much as 11% during passive heating. This original finding has been confirmed with more recent investigations reporting maintenance or a small increase in stroke volume (20, 25, 37–38), in the face of significant reductions in filling time, filling pressures (37–38), and preload (i.e., left ventricular end-diastolic volume) (22, 37). The maintenance of ventricular filling and stroke volume, despite the reduced filling pressures, are likely the consequence of increased ventricular contractility (2, 7, 22) and the associated increase in ventricular suction (22).

Because of the predominant role of displaced central blood volume (unloading) with heat stress, we sought to determine if individuals with susceptibility to larger reductions in preload have distinct responses to heat stress. Long-term endurance training is associated with eccentric left ventricular (LV) re-modeling and increased LV compliance. As a result, endurance trained individuals experience larger decreases in ventricular volumes, compared with untrained individuals, when filling pressures are reduced (18). Consistent with these observations, endurance trained athletes have an increased risk of orthostatic intolerance and exhibit greater reductions in LV end-diastolic volume and stroke volume, compared with sedentary individuals, when exposed to orthostatic stress (18, 19). Passive heat stress also reduces central blood volume (7) and cardiac filling pressures (6, 33, 37) similar to the postural changes commonly associated with orthostatic stress. However, no study to date has investigated the volumetric or functional response of the ventricles to passive heat stress in endurance trained individuals. In sedentary individuals, end-diastolic volume is reduced by ~11% with heat stress (22); however, it remains unknown if the more compliant ventricle of an endurance athlete will exacerbate this response. It is also possible that other mechanisms may compromise LV filling in the endurance trained athlete. Specifically, compared with normally active controls, endurance athletes were recently found to have a greater reduction in early diastolic filling rates when exposed to mild orthostatic stress, associated with a reduction in peak LV twist and untwisting rates (13). Ventricular untwisting is associated with early diastolic LV pressure decay (11, 24) and with the subsequent trans-mitral pressure gradient that drives early diastolic filling (5, 17). During heat stress, when cardiac filling pressures are reduced, increased LV twist and untwisting rates

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1545
have been proposed to contribute to the preservation of early diastolic filling (22). The response of LV twist and untwisting rates to heat stress for endurance trained athletes remains unknown.

The purpose of this study was therefore to investigate the influence of aerobic fitness on the biventricular volumetric response to passive heat stress. We tested the hypothesis that biventricular end-diastolic volume would decrease to a greater extent in endurance trained subjects compared with untrained controls. Furthermore we sought to characterize the effect of aerobic fitness on early diastolic function in response to passive heat stress. We hypothesized that early diastolic function would be reduced in endurance trained athletes during heat stress, demonstrated by reductions in early diastolic filling velocities, filling rates, tissue velocities, and strain rates.

**METHODS**

Approval was obtained from the University of Alberta Health Research Ethics Board, and all subjects provided written informed consent.

**Subjects**

Twenty healthy men volunteered to participate in the present study (10 endurance trained male athletes, and 10 healthy normally active control subjects; Table 1). The endurance trained athletes (ET) were recruited based on the following criteria: 1) at least 4 years of consistent endurance training, 2) a minimum of 10 h training/wk, 3) cycling >250 km/wk or running >90 km/wk, and 4) a maximal oxygen consumption \((\dot{VO}_2\text{max})\) > 60 ml·kg\(^{-1}\)·min\(^{-1}\). The control subjects (untrained, UT) were active but not participating in systematic aerobic training (<5 h/wk). All subjects were free of cardiovascular, metabolic, or neurological diseases, and none were taking any medications.

**Experimental Protocol**

Day 1. Subjects completed an incremental exercise test on a cycle ergometer and anthropometric data were recorded.

![Table 1. Participant characteristics and training history](http://jap.physiology.org/)  

<table>
<thead>
<tr>
<th></th>
<th>Untrained</th>
<th>Endurance Trained</th>
<th>(P) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>24.6 ± 2.8</td>
<td>25.3 ± 2.7</td>
<td>0.574</td>
</tr>
<tr>
<td>Height, cm</td>
<td>179.0 ± 3.6</td>
<td>178.3 ± 9.7</td>
<td>0.840</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>77.3 ± 9.7</td>
<td>71.4 ± 8.4</td>
<td>0.162</td>
</tr>
<tr>
<td>Body surface area, m(^2)</td>
<td>1.96 ± 0.11</td>
<td>1.93 ± 0.16</td>
<td>0.297</td>
</tr>
<tr>
<td>(\dot{VO}_2\text{max}, \text{l/min})</td>
<td>3.6 ± 0.6</td>
<td>4.6 ± 0.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>(\dot{VO}_2\text{max}, \text{ml·kg}^{-1}·\text{min}^{-1})</td>
<td>46.3 ± 6.2</td>
<td>64.4 ± 3.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV internal diameter, cm</td>
<td>5.9 ± 0.4</td>
<td>6.2 ± 0.4</td>
<td>0.049</td>
</tr>
<tr>
<td>LV mass, g</td>
<td>102.9 ± 21.9</td>
<td>125.8 ± 14.4</td>
<td>0.013</td>
</tr>
<tr>
<td>High volume training experience, yr</td>
<td>—</td>
<td>7.2 ± 3.7</td>
<td>—</td>
</tr>
<tr>
<td>Aerobic training volume per week, hrs</td>
<td>1.6 ± 1.2</td>
<td>12.2 ± 1.9</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are presented as means ± SD; \(n\), number of subjects; \(\dot{VO}_2\), rate of oxygen consumption; LV, left ventricle.
Day 2. Subjects dressed in a three-piece high density tube-lined suit (Allan VanGaurd Technologies, Ottawa, ON, Canada) and rested on a cot in the supine position. The suit covered the entire body except for the hands, feet, and face. During the normothermic condition, 34°C water was circulated throughout the suit to establish a physiological baseline. Core body temperature was then elevated by circulating warm water throughout the suit for 60 min. After 45 min of passive heating (50°C), the water circulating throughout the suit was cooled to 47°C to attenuate the rate of rise in core body temperature (2). Core and skin temperature could not be monitored during MRI because of the presence of ferrous metal in the telemetry pill and dermal patches. Therefore, this test was used to establish the physiological response to passive heating on test day 3.

Day 3. At least 7 days after test day 2, subjects dressed in the tube-lined suit and repeated the experimental protocol performed on test day 2, while cardiac magnetic resonance imaging was performed.

Measurements

Anthropometric. Height and weight were measured by the same investigator, using a standard stadiometer and balance scale. Body surface area was calculated as previously described by Du Bois and Du Bois (12).

Incremental \( \dot{V}_{O_2\text{max}} \) test. The \( \dot{V}_{O_2\text{max}} \) was determined by an incremental protocol on a cycle ergometer (Monark 894E, Varberg, Sweden), for which the workload increased by 25–30 W every 2 min until ventilatory threshold, after which workload increased by the same increment every minute until volitional exhaustion. The untrained subjects began at 100 W, while the endurance trained subjects started between 200 to 250 W. Ventilatory threshold was defined by a systematic rise in the ventilatory equivalent for O\(_2\), without an increase in the ventilatory equivalent for CO\(_2\) (10). Expired gas samples were analyzed with a metabolic measurement cart (TrueOne 2400, ParvoMedics), calibrated according to manufacturer specifications prior to each test.

Body temperature. Core temperature was measured telemetrically with a Jonah ingestible capsule (VitalSense, Mini Mitter, Bend, OR). Mean skin temperature was calculated as previously described (26), with the use of four biomedical ceramic chip thermistors (MA 100, 10KO negative coefficient, Thermometrics, NJ) placed on the left side of the body and recorded using an 8-channel data logger (SmartReader 8 Plus, ACR Systems, Surrey, BC, Canada).

Heart rate and blood pressure. Beat-by-beat heart rate was quantified from R-wave detection from a single-lead ECG. Arterial blood pressure was obtained from an automatic blood pressure cuff.

Cardiac MRI. Short axis cine images covering the length of the right and left ventricle (RV and LV, respectively) were acquired to measure end-diastolic and end-systolic volumes. Image acquisition parameters were as follows: repetition time = 3.0 ms; echo time = 1.5 ms; flip angle = 78°; slice thickness = 8 mm with a 2-mm gap between slices; matrix = 256 × 192; field of view = 300 to 380 mm; 25 ms temporal resolution with 30 reconstructed phases over the cardiac cycle. Similarly, short axis cine images were acquired covering the length of the left atrium (LA) to assess LA volume. Short-axis through-plane phase-contrast cine images, prescribed at the level of the mitral valve leaflets, were used to measure the early (E) and late (A) diastolic filling velocities. Four-chamber view oriented phase-contrast blood velocity images, sensitive to in-plane velocities, were used to calculate the LV intraventricular pressure gradients (IVPGs) (36). Peak annular tissue velocities in the RV free wall, septum, and LV lateral wall were assessed by tracking the tissue motion at the

![Fig. 2. Right ventricular (RV) end-diastolic volume (A), ejection fraction (B), end-systolic volume (C), and stroke volume (D) in endurance trained (trained) and untrained individuals at baseline (solid bars) and during passive heat stress (shaded bars). Vertical markers indicate a significant main effect for thermal condition (2-way ANOVA) and horizontal markers indicate a significant main effect for fitness level (2-way ANOVA; \( P < 0.05 \)). Data reported as means ± SD.](http://jap.physiology.org/doi/fig/10.220.32.246)
annulus from a four-chamber cine image series (with 64 reconstructed phases). Myocardial tissue tagging was applied to five short axis images, evenly spaced from apex to base with 1.6 cm interslice spacing, to assess peak twist and peak untwisting rate, as well as circumferential and radial strain (systole) and strain rate (diastole). Myocardial tissue tags fade throughout the cardiac cycle; therefore tags were applied ~150 ms after the R-wave, to ensure sufficient tag contrast during diastole. All image acquisitions were cardiac gated based on the electrocardiogram and acquired during end-expiratory breath holds. The order of image acquisition was kept constant between baseline and heat stress, with image acquisition time between 15 and 20 min at baseline and ~10 min during whole body passive heat stress. During the heat stress protocol, image acquisition always began after 45 min.

**Data analysis.** RV, LV, and LA volumes were measured according to the method of discs as previously described (4, 22). LV mass was calculated by using the difference in LV volumes measured with endocardial and epicardial tracings at end diastole. Stroke volume, cardiac output, ejection fraction, LV end-systolic elastance (3), and LV end-systolic wall stress (27) were also calculated.

In-plane blood velocities (two perpendicular directions) were measured in the four-chamber view orientation for the estimation of intraventricular pressure gradients (36). Pressure differences between the base of the left ventricle and the left ventricular apex were calculated by integrating the pressure gradient field over space between these points, for each cardiac phase, similar to methods reported using color M-mode Doppler imaging (15).

Tagged myocardial images were analyzed using custom image morphing software, developed in house, as previously described (4, 13, 22). An image morphing algorithm was used to determine the spatial deformation field for the myocardium for each slice as a function of the cardiac phase, relative to a reference cardiac phase in diastasis. The deformation fields were used to calculate rotation and circumferential and radial strains. Global twist was calculated as the difference between the counter-clockwise rotation at the apex and the clockwise rotation at the base (viewed from the apex to the base). The rate of untwisting was calculated as the discrete time derivative of the twist vs. time curve.

**Data analysis.** All data are reported as means ± SD (9). Independent t-tests were used to determine differences between fitness levels at baseline. Two-way analysis of variance was used to test for the effect of fitness on cardiac parameters, comparing baseline with passive heat stress. To isolate differences between groups, Bonferroni post hoc tests were run. Main effects are illustrated with vertical (baseline vs. heat stress, independent of fitness) and horizontal (trained vs. untrained, independent of condition) bars on each figure. Significance was set a priori at an α = 0.05.

**RESULTS**

Subject characteristics are reported in Table 1. There was no between-group difference in body surface area; therefore, cardiac volumes are reported in absolute values. Endurance trained subjects had a higher VO₂max and larger LV diastolic diameters and LV mass compared with the untrained group (all P < 0.05). Baseline heart rate was lower in the endurance athletes compared with the normally active group (ET, 52 ± 5; UT, 63 ± 11 beats/min; P < 0.05). No between-group differences were found in baseline systolic blood pressure (ET, 120 ± 7 mmHg; UT, 121 ± 8 mmHg), diastolic blood pressure (ET, 69 ± 7 mmHg; UT, 70 ± 6 mmHg), or mean arterial blood pressure (ET, 86 ± 6 mmHg; UT, 87 ± 6 mmHg), right or left ventricular ejection fraction (Figs. 1 and 2), or cardiac output (ET, 7.5 ± 1.4 l/min; UT, 7.7 ± 2.0 l/min). Biventricular end-diastolic and end-systolic volumes and stroke volumes were greater in the endurance trained group compared with the untrained group (Figs. 1 and 2, P < 0.05). On average, core temperature was 37.0 ± 0.2°C and skin temperature was 33.3 ± 0.5°C at baseline, with no difference between the two groups.

After 45 min of passive heat stress, core temperature increased 0.63 ± 0.2°C above baseline, averaging 0.78 ± 0.2°C above baseline between minutes 45 and 60 (equivalent to the time of image acquisition). Likewise, mean skin temperature increased by 3.9 ± 0.6°C during passive heating (both P < 0.001). Both groups significantly increased heart rate (ET, 29 ± 9 beats/min; UT, 32 ± 10 beats/min; P < 0.05), while there was a significant decrease (P < 0.05) in biventricular end-diastolic volume (Figs. 1A and 2A) and LA volume (ET, −19.2 ± 11.6 ml; UT, −15.0 ± 12.7 ml), with no between-group differences found (fitness level × time). Stroke volume was unchanged by heat stress in both groups (Figs. 1D and 2D) as a result of a significant decrease in biventricular end-systolic volume (Figs. 1C and 2C) that matched the decrease in end-diastolic volume. Cardiac output was therefore significantly elevated in both groups above baseline (ET, 11.3 ± 2.5 l/min; UT, 10.9 ± 2.1 l/min).

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**Fig. 3.** Peak left ventricular twist (B) and untwisting rates (A) at baseline (solid bars) and during passive heat stress (shaded bars) in a group of endurance trained (trained) and untrained volunteers. Vertical markers indicate a significant main effect for thermal condition (P < 0.05). Data reported as means ± SD.
LV end-systolic wall stress decreased significantly with heat stress (ET, $-20.0 \pm 35.9 \text{mmHg/cm}^2$; UT, $-27.0 \pm 24.2 \text{mmHg/cm}^2$; $P = 0.613$), along with significant increases in biventricular ejection fraction (Figs. 2B and 3B), LV end-systolic elastance (ET, $0.40 \pm 0.27 \text{mmHg/ml}$; UT, $+0.51 \pm 0.23 \text{mmHg/ml}$; $P = 0.187$), LV twist (Fig. 3) and LV circumferential strain (Table 2). Systolic annular tissue velocities also increased with heat stress in both groups similarly (RV free wall: ET, $+2.7 \pm 3.2 \text{cm/s}$; septum: ET, $+1.0 \pm 1.7 \text{cm/s}$, UT, $+2.6 \pm 2.7 \text{cm/s}$; and LV lateral wall: ET, $+4.7 \pm 2.0 \text{cm/s}$, UT, $+4.4 \pm 2.4 \text{cm/s}$; $P < 0.05$), with no between-group differences.

LV untwisting rate increased to the same extent between groups (Fig. 3), with no change in early diastolic filling velocities (Table 2), volumetric filling rates (Table 2), or the LV intraventricular pressure gradient (Table 2). Early diastolic annular tissue velocities also remained unchanged with heat stress (Table 2), along with circumferential and radial strain rates (Table 2). Consistent with our previous report (22), and the work of others (2), late diastolic filling velocities, volumetric filling rates, and late diastolic annular tissue velocities significantly increased ($P < 0.05$) with heat stress, with no differences between groups (data not included).

**DISCUSSION**

This study tested the hypothesis that high levels of aerobic fitness would exacerbate the reduction in end-diastolic ventricular volumes in response to whole body passive heat stress. Endurance athletes were expected to have a larger reduction in preload and reduced early diastolic function in response to heat stress; however, contrary to our hypothesis, endurance trained and untrained hearts unloaded similarly (ET by $-19.5 \pm 24.0 \text{ml}$; UT by $-25.1 \pm 23.8 \text{ml}$; $P > 0.05$), reduced end-systolic volumes similarly (ET by $-15.9 \pm 8.8 \text{ml}$; UT by $-17.6 \pm 7.8 \text{ml}$; $P > 0.05$), had no significant change in filling parameters and had significant and similar increases in peak LV twist (ET by $5.5 \pm 5.5^\circ$; UT by $6.6 \pm 4.8^\circ$; $P > 0.05$) and LV untwisting rates (ET by $-56.4 \pm 54.0^\circ$/s; UT by $-63.5 \pm 50.2^\circ$/s) during heat stress. To our knowledge, this is the first study to evaluate the influence of aerobic training status on the biventricular response to whole body passive heat stress. These results indicate that despite mounting evidence that endurance trained and untrained hearts respond differently to volume unloading, there appears to be no difference in the response to passive heat stress.

Levine and colleagues (1, 18) demonstrated that endurance trained athletes, similar to those studied in the present investigation, have greater ventricular compliance as a result of chronic volume overload (i.e., eccentric LV hypertrophy) from years of endurance training. In their original investigation, this group demonstrated that increased ventricular compliance is detrimental during periods of reduced cardiac filling pressures and venous return [such as with lower body negative pressure (LBNP) or postural changes]. The more compliant ventricle is susceptible to greater volume reductions (i.e., end-diastolic volume and stroke volume) when cardiac filling pressures are reduced to the same extent. Passive heat stress significantly reduces cardiac filling pressures (7, 20, 25, 37, 38); therefore, it remains unknown why the endurance athletes in the present investigation did not experience a greater reduction in preload during heat stress compared with their untrained counterparts. These findings (i.e., the similarity of the groups) are in contrast to the well described divergent responses of these two populations to volume unloading with LBNP. Specifically, using an identical MRI protocol and similar samples of untrained and endurance trained individuals (i.e., similar $V_{\text{O2max}}$), Esch et al. (13) showed that LV end-diastolic volume is reduced by $26.6 \pm 8.7 \text{ml}$ in athletes as opposed to $17.3 \pm 8.6 \text{ml}$ in sedentary controls (13) with $-30 \text{mmHg}$ of LBNP. Thus, while LBNP and heat stress are similar in that central blood volume is reduced, the magnitude of the unloading with heat stress does not seem to depend on factors such as a ventricular compliance, as is the case with LBNP. The present results therefore suggest that, unlike LBNP, aerobic fitness does not influence the biventricular response to passive heat stress.

Previously, we demonstrated that increased LV recoil, associated with reduced end-systolic volumes and increased LV untwisting rates, are the likely mechanisms for preserving early diastolic filling during passive heat stress (22). Increased LV twist and reduced end-systolic volumes would both increase the storage of elastic energy that is released in early diastole, potentially enhancing ventricular suction and maintaining early filling despite the reduction in filling pressures. These mechanisms appear to be similar for endurance athletes and untrained controls. In particular, the rate of left ventricular untwisting, which is the predominant myocardial deformation during isovolu-

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**Table 2. The influence of aerobic fitness on select cardiac events during whole body passive heat stress**

<table>
<thead>
<tr>
<th></th>
<th>Endurance Trained</th>
<th>Untrained</th>
</tr>
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<tbody>
<tr>
<td><strong>Main Effect</strong></td>
<td></td>
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</tr>
<tr>
<td>Peak circumferential strain, %</td>
<td>17.5 ± 1.6</td>
<td>19.8 ± 2.0</td>
</tr>
<tr>
<td>Peak radial strain, %</td>
<td>-37.3 ± 8.2</td>
<td>-36.3 ± 6.0</td>
</tr>
<tr>
<td>Peak circumferential strain rate (diastole), %</td>
<td>1.5 ± 0.19</td>
<td>1.5 ± 0.31</td>
</tr>
<tr>
<td>Peak radial strain rate (diastole), %</td>
<td>2.4 ± 0.55</td>
<td>2.6 ± 1.1</td>
</tr>
<tr>
<td>Early filling velocity, cm/s</td>
<td>56.8 ± 9.0</td>
<td>57.3 ± 12.8</td>
</tr>
<tr>
<td>Early filling rate, ml/s</td>
<td>574.9 ± 97.6</td>
<td>535 ± 122.2</td>
</tr>
<tr>
<td>RV early tissue velocity, cm/s</td>
<td>9.5 ± 2.1</td>
<td>10.6 ± 3.6</td>
</tr>
<tr>
<td>Septal early tissue velocity, cm/s</td>
<td>7.5 ± 1.2</td>
<td>8.6 ± 1.8</td>
</tr>
<tr>
<td>LV early tissue velocity cm/s</td>
<td>8.8 ± 1.8</td>
<td>11.3 ± 2.1</td>
</tr>
<tr>
<td>Intraventricular pressure gradient, mmHg</td>
<td>1.3 ± 0.4</td>
<td>1.4 ± 0.5</td>
</tr>
</tbody>
</table>

Data reported as means ± SD. n = 20, 10 endurance trained and 10 normally active. *Significant main effect for time ($P < 0.05$). †Significant main effect for fitness level ($P < 0.05$).
mic relaxation, increased by 56.4 ± 56.0% in ET and 63.5 ± 50.2% in UT subjects with heat stress. Also, end-systolic volume was reduced similarly in both groups, by 16.9 ± 9.6% in ET and 21.3 ± 8.8% in UT. Invasive animal studies have indeed demonstrated a strong relationship between the rate of LV untwisting and the time constant of isovolumic pressure decay (tau) and with the LV intraventricular pressure gradient. Early diastolic filling is largely determined by the transmitral pressure gradient, driving blood into the LV after mitral valve opening (5, 17). Since passive heat stress reduces cardiac filling pressures and left atrial volume at the time of mitral valve opening, we reason that augmenting LV untwisting rate preserves the filling gradient by lowering LV filling pressure. The present results support our assumption, demonstrating a preservation of the early diastolic intraventricular LV pressure gradient and indexes of early diastolic filling in both untrained and endurance trained individuals (Table 3).

The relationship between LV untwisting and aerobic fitness was recently evaluated (13), and it was shown that changes in peak torsion, peak untwisting rate, and early filling rates with LBNP appear to be related to Vo2max. Esch et al. (13) found that endurance trained athletes (Vo2max > 60 ml·kg⁻¹·min⁻¹) had reduced untwisting rates with LBNP. The divergent responses of untrained individuals and endurance trained athletes to volume unloading were not observed with heat stress however. Both groups were found to maintain early diastolic filling velocities, filling rates, and intraventricular pressure gradients with passive heating, with similar increases in twist and untwisting rates and changes in end-systolic and end-diastolic volumes as well as similar increases in heart rate. Thus the effects of increased heart rate and enhanced systolic function (e.g., reduced end-systolic volume and increased twist), which are similar between groups, seem to dominate the changes in function observed with heat stress.

There are several limitations that must be considered when interpreting the present results. Cutaneous blood flow was not measured in the present investigation, thus it remains unknown if the endurance athletes had a similar cutaneous vasodilatory response compared with the normally active controls. Cross-sectional (16, 34) and longitudinal (28, 35) data support the idea that endurance training increases the skin blood flow response to increases in body temperature. While the mechanism is beyond the scope of this study, increased sensitivity of the hypothalamus, increased neurotransmitter release, and increased receptor sensitivity have all been suggested as potential mechanisms for this response. Importantly, if skin blood flow was higher in the endurance athletes, central blood volume and cardiac filling pressures may have decreased to a greater extent than the normally active group, thus exacerbating volume unloading. This did not appear to be the case. Furthermore, it is important to note that the above studies focused on reflex-induced dilation (in response to a change in core temperature) rather than local heating as performed in the present investigation. Aerobic exercise training is also known to result in hypervolemia (14). It is therefore possible that the similar response observed between groups may have been the result of a smaller decline in central blood volume and cardiac filling pressures in the trained group as a result of their elevated blood volume. However, we contend that any difference between the two groups between either the skin blood flow response or elevated blood volume would be minimized by their two opposing effects (i.e., hypervolemia would not result in an attenuated reduction in central blood volume and cardiac filling pressures, as the heightened cutaneous vasodilatory response would “absorb” this difference). Furthermore, left atrial volume at the time of mitral valve opening, a surrogate measure of filling pressure, decreased to a similar extent in both groups, reflective of a similar change in cardiac filling pressures between groups.

In conclusion, this study examined the influence of aerobic fitness on the biventricular response to whole body passive heat stress. Contrary to our hypothesis, both groups experienced a similar volume change (LV end-diastolic volume and LA volume), increased contractile function (indexed by ejection fraction, end-systolic elastance, and annular tissue velocities), and maintenance of early diastolic function (early filling velocities and filling rates, annular diastolic tissue velocities, and strain rates). These data suggest that aerobic training status does not influence the biventricular response to whole body passive heat stress.

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