Effect of exercise, heat stress and dehydration on myocardial performance

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Background Myocardial dysfunction is a well-documented outcome of extended periods of high cardiac output. Whether similar effects occur during firefighting, an occupation characterized by repeated periods of work compounded by dehydration and heat stress, is uncertain.

Aims To investigate the independent and combined effects of moderate heat stress and dehydration on indicators of myocardial performance following intermittent, submaximal treadmill exercise while wearing personal protective equipment (PPE).

Methods Twelve aerobically fit young men (age 21.5 ± 2.6 years; maximal oxygen uptake [VO2max] 60.3 ± 4.4 ml kg⁻¹ min⁻¹) performed intermittent treadmill walking exercise consisting of three 20 min bouts at an intensity of ~40% VO2max separated by two periods of rest in four different conditions in random order: (i) no heat stress-euhydrated, (ii) heat stress-euhydrated (heat stress created by wearing PPE), (iii) no heat stress-dehydrated and (iv) heat stress-dehydrated. We measured core temperature by a telemetric gastrointestinal pill. We determined cardiac variables by standard echocardiographic techniques immediately before and ~30 min after exercise.

Results We recorded no significant changes in markers of systolic (ejection fraction, shortening fraction, tissue Doppler-S) or diastolic (mitral peak E velocity, tissue Doppler-E' and E/E') function following exercise in any of the four conditions.

Conclusions In this model of exercise designed to mimic the work, heat stress and dehydration associated with firefighting activities, we observed no negative effects on myocardial inotropic or lusitropic function.

Key words Cardiac strain; diastolic function; firefighting; heat stress; systolic function.

Introduction

Experimental and observational studies have identified myocardial dysfunction as a consequence of prolonged periods of cardiac work. These reports describe depression of echocardiographic measures of both inotropic and/or lusitropic function in disparate conditions characterized by sustained levels of high cardiac output, including ultra-endurance athletic events [1], hyperthyroidism [2], pregnancy [3], obesity [4], anaemia [5] and arterio-venous malformations [6]. Myocardial dysfunction in some of these cases may be transient and reversible [7]; in others the disturbance of cardiac contractile and relaxation properties may be sufficiently severe as to manifest itself as congestive heart failure and contribute to disease morbidity and mortality [4].

Firefighting is an occupation characterized by repeated episodes of increased cardiac work, compounded by dehydration and augmented sympathetic-adrenal stimulation surrounding both heat and emotional stress [8]. These effects are compounded by the wearing of personal protective equipment (PPE), which increases the metabolic work demands on the cardiovascular system, augments thermal strain and accentuates dehydration via increased sweat loss [9,10]. Previous studies have documented the resultant tachycardia and diminished stroke volume during firefighting activity [11].

Fernhall et al. [12] studied echocardiographic measures of cardiac size and performance after repeated bouts of firefighting training exercises over a 3 h period. After firefighting, a significant decline was observed in left ventricular
dimension, shortening fraction and transmitral ventricular filling velocity. There was no alteration of systolic function or of ventricular septal diastolic function. However, a reduced longitudinal relaxation rate was detected at the lateral wall. The extent that these findings might reflect decreased filling volume from dehydration and/or local myocardial dysfunction as a result of firefighting activity was not clear.

We designed this study to examine echocardiographic measures of systolic and diastolic function in young aerobically fit men in a controlled laboratory setting in which the amount and nature of physical work, heat stress and level of dehydration simulated firefighting. Specifically, the goal of the study was to examine the independent and combined effects of moderate heat stress and dehydration on indicators of myocardial performance following intermittent, submaximal treadmill exercise while wearing PPE. We hypothesized that the combination of heat stress and dehydration would result in myocardial dysfunction similar to that reported with firefighting.

Methods

Twelve young, physically active men (mean age 21.5 ± 2.6 years) volunteered for exercise testing. All were in good health, non-obese and non-smokers. None were taking medications that would affect cardiovascular performance or heat tolerance. Participants provided written informed consent prior to the study procedures. The study was approved by the College's Institutional Review Board. All participants were considered to be non-heat acclimatized, as testing took place in upstate New York in the winter and early spring.

We employed a 2 × 2 (thermal stress status: heat stress [HS] or no heat stress [NoHS]; hydration status: euhydrated [EUH] or dehydrated [DEH]) factorial study design. Participants completed an alternating exercise/rest protocol during four different conditions: no heat stress-euhydrated (NoHS-EUH), heat stress-euhydrated (HS-EUH), no heat stress-dehydrated (NoHS-DEH) and heat stress-dehydrated (HS-DEH). Conditions were performed in random order. For each participant, experimental trials were conducted at the same time of day, separated by at least 48 h and completed within a 4-week period. All exercise sessions were conducted in a thermoneutral laboratory (21.2 ± 0.9°C; 29 ± 12% relative humidity).

Heat stress was induced by the wearing of structural firefighting PPE, consisting of turnout coat and trousers (G-Xtreme, Globe Manufacturing Co., LLC, Pittsfield, NH, USA), flash hood, helmet, gloves, boots and self-contained breathing apparatus (SCBA). During the NoHS trial, participants wore a weighted vest (sleeveless garment worn around the torso that allowed for the positioning of added weight near the wearer’s centre of gravity) and carried SCBA that were matched in weight to that of the PPE in the HS trial. Thus, the absolute workload for each participant was identical in the HS and NoHS trials. Additionally, during the NoHS trial, participants wore a cooling shirt (Cool Shirt, Shafer Enterprises, Stockbridge, GA, USA) that pumped cold water through tubing embedded in the shirt and were exposed to a fan to enhance evaporative cooling.

For both EUH conditions, during the 24 h prior to testing participants were provided with 30 ml of water per kg body mass. Euvhydration was confirmed by a urine specific gravity of <1.020 prior to testing [13]. During the exercise protocol, participants consumed sufficient water to offset sweat loss. For both DEH trials, participants were provided 15 ml of water per kg body mass during the 24 h period preceding exercise and received minimal water supplementation during the test (200 ml). Nude body mass was recorded before the 24 h pre-exercise period as well as immediately before and after exercise. Height was measured to the nearest 0.01 m using a stadiometer (Seca, Hanover, MD, USA), and body mass was measured with a physician’s scale (599KL, Health-O-Meter, Alsip, IL, USA) to the nearest 0.1 kg. Body composition was estimated by a total body dual-energy X-ray absorptiometry scan (iDXA, GE Lunar, Madison, WI, USA). Maximal oxygen uptake (VO2max) was determined during a graded treadmill exercise test using indirect calorimetry (TrueOne 2400 Metabolic Measurement System, ParvoMedics, Sandy, UT, USA).

Participants underwent a familiarization session before data collection. While wearing PPE and carrying SCBA, participants walked on a treadmill at a 5% grade and speed sufficient to elicit a heart rate (HR) of ~75% of maximal HR, which resulted in an oxygen uptake of ~40% VO2max. Body fluid loss was estimated from change in nude body weight to allow calculation of fluid replacement necessary to maintain a euhydrated state.

Approximately 8 h prior to the exercise trials, participants ingested a telemetric gastrointestinal pill (HQ Inc., Palmetto, FL, USA) to transmit Tc. Participants ingested a protein bar (390 calories; 12 g fat, 49 g carbohydrate, 30 g protein) 1 h before each test. Participants were instrumented with a HR monitor (Zephyr BioHarness BT2, Annapolis, MD, USA), and we recorded HR and Tc at baseline, throughout the exercise protocol and during recovery.

Each exercise trial included 100 min of alternating exercise and rest, with three 20 min bouts of treadmill walking separated by a 20 min rest period between exercise periods. Each participant performed the same workload during all trials, as established during the familiarization trial. During exercise, we measured HR and Tc continuously, and participants reported ratings of perceived exertion and thermal sensations at 5 min intervals. We measured oxygen uptake via indirect calorimetry between minutes 10 and 18 of the third 20 min exercise bout. During the 20 min rest periods, participants doffed gear and weighted vest and sat in front of a fan.
We obtained echocardiographic measures immediately before and ~30 min following the exercise protocol with participants in the supine left lateral position (SSD 5500 SV, Aloka, Tokyo, Japan). We recorded left ventricular dimensions during systole and diastole in triplicate using standard 2D-guided M-mode techniques and averaged for analysis [14]. We defined end diastolic dimension (LVED) as the distance from the trailing edge of the ventricular septum to the endocardial surface of the posterior wall at the level of the tips of the open mitral valve leaflets. We measured left ventricular end systolic dimension (LVES) as the shortest vertical distance from the trailing edge of the ventricular septum to the posterior wall in systole. We determined left ventricular end systolic (LVESV) and diastolic volumes (LVEDV) as described by Teicholz et al. [15]. We assessed left ventricular systolic function by (i) fractional shortening [(LVES – LVESV)/LVESV] × 100 and (ii) ventricular ejection fraction [(LVEDV − LVESV)/ LVEDV] × 100.

We recorded peak early (E) and late (A) diastolic inflow blood velocities at the open mitral leaflets by pulse Doppler echocardiography in the apical four-chamber view. Peak E velocity is a measure of the pressure gradient across the mitral valve, estimating the balance between upstream filling pressure and downstream pressure as a manifestation of ventricular diastolic (relaxation) properties. A decline in the ratio of early versus late diastolic flow (the latter indicated by peak A velocity, a marker of atrial upstream contractile contribution) is considered an indication of depressed ventricular diastolic function [16]. We calculated the average of three to five highest E and A velocities from offline measurements.

We recorded longitudinal myocardial velocities in both systole and diastole at the septal and lateral aspects of the mitral valve in a four-chamber view by pulse Doppler tissue interrogation (TDI-S and TDI-E′, respectively). We considered transducer alignment optimal when the ventricular septum was vertical. Again, we performed each offline analysis from the average of the 3–10 greatest velocities. We considered TDI-S and TDI-E′ as markers of ventricular systolic and diastolic function, while we recorded the ratio of E/E′ as an indicator of left ventricular filling pressure [17].

We calculated changes in HR and Tc during the intermittent exercise/rest protocol across the five periods of the study: Exercise Period 1, Rest Period 1, Exercise Period 2, Rest Period 2 and Exercise Period 3. We developed mixed models with random intercepts where thermal stress status (NoHS versus HS), hydration status (EUH versus DEH) and their interactions were tested as predictors of change in HR and Tc in each of the five periods (Post–Pre). We used a Markov Chain Monte Carlo method to impute missing values.

We developed mixed models with random intercepts for echocardiographic data analysed pre and post the intermittent exercise protocol, where we tested thermal stress status, hydration status and their interactions as predictors of change in each of the cardiovascular outcomes. Because we used multiple measures to assess the impact of heat stress and hydration on systolic and diastolic function, we used a family-wise error rate in each category (i.e. 0.05/N of outcomes within systolic and diastolic function measures). We created all models in SAS 9.3 Proc MIXED.

**Results**

Anthropometric measures and VO₂max and peak HR during the pre-study treadmill test are presented in Table 1. Results are expressed as mean ± SD in tables and mean ± SE in figures. All participants had high aerobic fitness (VO₂max range: 51.9–66.4 ml kg⁻¹ min⁻¹) [18]. The mean body mass index (BMI) of participants was in the normal range (18.5–24.9 kg m⁻²) [19]; five participants were overweight (BMI 25.0–29.9 kg m⁻²) but none were obese (BMI ≥ 30 kg m⁻²) based on BMI criteria [19]. Average body fat percentage was 15.0% (range: 11.3–23.5%); one participant had >22% body fat, which is considered unsatisfactory for health [18].

Fluid restriction (15 ml of water per kg body mass) in the 24 h preceding exercise resulted in 2.5 and 2.1% (NoHS-DEH and HS-DEH, respectively) body mass loss in the dehydration trials. The exercise protocol resulted in 0.4 and 1.2% body mass losses in the NoHS and HS dehydration trials, respectively. Thus, there was an overall reduction in body mass of ~3% in both dehydrated trials, but no significant alteration in body mass in the two EUH trials. Table 2 presents exercise responses during the third exercise bout. Oxygen uptake was higher (~5 ml kg⁻¹ min⁻¹) in the HS compared with the NoHS condition, and this was reflected by participants reporting greater perceived effort and higher thermal sensations. However, hydration status had no statistical effect on these measures.

Heat stress resulted in the highest HR during the exercise protocol, and this was accentuated by dehydration. As seen in Figure 1, HR increased during exercise and returned towards baseline during recovery for all four conditions. For each exercise bout, the change in HR was greater when participants were in the HS

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
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<tbody>
<tr>
<td>Age (years)</td>
<td>21.5 ± 2.6</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.78 ± 0.07</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>77.9 ± 11.2</td>
</tr>
<tr>
<td>BMI (kg m⁻²)</td>
<td>24.6 ± 2.8</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>15.0 ± 3.4</td>
</tr>
<tr>
<td>Maximal oxygen uptake (ml kg⁻¹ min⁻¹)</td>
<td>60.3 ± 4.4</td>
</tr>
<tr>
<td>Maximum HR (beats min⁻¹)</td>
<td>190 ± 6</td>
</tr>
</tbody>
</table>
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condition versus the NoHS condition. At the end of the third exercise bout, HR was 47 beats min\(^{-1}\) higher in the heat stress condition than when equal weight was carried in a weighted vest and a cooling shirt was worn.

As indicated in Figure 2, there were significant differences in change in \(T_c\) for the last two exercise bouts, but not for the other study periods. The increase in \(T_c\) during the second exercise period in the HS condition was more than double that in the NoHS condition (0.7 ± 0.0 versus 0.3 ± 0.1°C; \(P < 0.01\)). During the third exercise bout, the increase in \(T_c\) during the HS condition was three times that which occurred during the NoHS condition (0.6 ± 0.1 versus 0.2 ± 0.1°C; \(P < 0.01\)).

Echocardiographic measures pre- and post-exercise are presented in Table 3. Neither heat stress status nor hydration status were statistically significant predictors of change in any marker of systolic or diastolic function. However, analysis revealed a significant interaction between thermal stress and dehydration status for \(E'\) at the lateral wall.

**Discussion**

The main finding in this study was the absence of any observed negative effect on systolic or diastolic myocardial function 30 min after an intermittent exercise challenge combined with moderate heat stress, dehydration or a combination of both. Compared with pre-exercise values, global left ventricular contractility, assessed by single and 3D measurements of shortening fraction and ejection fraction, respectively, remained unchanged. Similarly, ventricular diastolic function remained stable, as indicated by similar mitral \(E:A\) ratios, transmitral pressure gradient (\(E\)) and left ventricular filling pressure (\(E'/E\)). Thus, despite recurrent increases in body temperature and increased cardiac work during exercise, as occurs during firefighting activity, we found no evidence of impaired myocardial function in this group of young, aerobically fit men.

This study also demonstrated that both heat stress and dehydration contribute to cardiac strain during exercise. However, increased core temperature (1.2°C) had a much larger effect than moderate dehydration (~3% body weight loss) on HR responses to exercise. This study matched HS and NoHS conditions for weight; therefore, the marked differences in increase in \(T_c\) (1.2 versus 0.6°C), peak HR (152 versus 105 beats min\(^{-1}\) and oxygen uptake (43% VO\(_{2max}\) versus 35% VO\(_{2max}\)) during the HS-EUH compared with the NoHS-EUH condition combined with the encapsulating properties and multiple layers of PPE.

<table>
<thead>
<tr>
<th>Variable</th>
<th>NoHS-EUH</th>
<th>NoHS-DEH</th>
<th>HS-EUH</th>
<th>HS-DEH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass change, P24 to pre-exercise (%) (n = 12)</td>
<td>−0.2 ± 0.8</td>
<td>−2.5 ± 0.9</td>
<td>−0.1 ± 0.7</td>
<td>−2.1 ± 0.7</td>
</tr>
<tr>
<td>Body mass change, pre- to post-exercise (%) (n = 12)</td>
<td>0.3 ± 0.6</td>
<td>−0.4 ± 0.6</td>
<td>−0.3 ± 0.6</td>
<td>−1.2 ± 0.3</td>
</tr>
<tr>
<td>Body mass change, P24 to post-exercise (%) (n = 12)</td>
<td>0.2 ± 0.8</td>
<td>−2.9 ± 0.6</td>
<td>−0.4 ± 0.6</td>
<td>−3.3 ± 0.6</td>
</tr>
<tr>
<td>Oxygen uptake (%VO(_{2max})) (n = 11)</td>
<td>34.6 ± 4.2</td>
<td>35.7 ± 3.7</td>
<td>42.7 ± 3.6</td>
<td>43.3 ± 4.0</td>
</tr>
<tr>
<td>Rating of perceived exertion (n = 9)</td>
<td>10.7 ± 1.6</td>
<td>10.6 ± 1.7</td>
<td>13.4 ± 1.7</td>
<td>13.3 ± 1.1</td>
</tr>
<tr>
<td>Thermal sensations (n = 9)</td>
<td>4.1 ± 0.3</td>
<td>4.1 ± 0.7</td>
<td>6.1 ± 0.5</td>
<td>6.3 ± 0.4</td>
</tr>
</tbody>
</table>

Values are mean ± SD. P24, body mass 24 h prior to trial. VO\(_{2max}\), maximal oxygen uptake. Rating of perceived exertion scale ranged from 6 to 20, with 20 corresponding to the highest level of exertion; thermal sensation scale ranged from 0 to 8, with 0.0 corresponding to ‘unbearably cold’ and 8.0 to ‘unbearably hot’.

*Statistical difference (\(P < 0.05\)) compared with EUH conditions.

**Figure 1.** HR responses (mean ± SE) during the alternating exercise/rest protocol (\(N = 12\)). NoHS, no heat stress (weighted vest/cooling shirt); EUH, euhydrated; HS, heat stress (PPE); DEH, dehydrated. *Significant (\(P < 0.05\)) main effect of thermal stress status.
The main strength of this study was the study design, which allowed for examination of both the independent and combined effects of heat stress and dehydration on cardiac performance under carefully controlled conditions. Firefighters routinely face the twin challenges of heat stress and dehydration on the fireground and the effects of these factors on cardiac strain are often interwined; however, the relative role each factor plays on cardiac performance is not well defined. Additional strengths include the use of an alternating work/recovery protocol to mimic the work/rehabilitation cycles experienced by a firefighter during a typical fireground operation.

The study also has limitations. Study participants were young, healthy, aerobically fit men, which limits the generalizability of the data. Exercise bouts were performed in a thermoneutral laboratory rather than under live fire conditions. However, the level of thermal strain attained and HR responses were within the range reported in previous studies of firefighting activity [8,12,20–23].

Previous echocardiographic investigations of normal cardiovascular responses to progressive exercise in normothermic conditions by healthy, euhydrated individuals have indicated that during such exercise, myocardial contractility and relaxation properties are both augmented as a mechanism for maintaining stable stroke volume as ventricular ejection and filling times are shortened by tachycardia [17]. Concomitantly, left ventricular filling volume (preload) remains stable while systolic dimension decreases. During such exercise, then, augmented myocardial oxygen uptake to effect increases in
ventricular contractility must be additive to the influence of tachycardia in establishing cardiac work.

Studies of the effects of passive heat stress (i.e. without the work of exercise) on cardiac dynamics have indicated tachycardia with decreases in ventricular preload and stroke volume, presumably related to a rise in sympathetic activity combined with a direct effect of heat on the sinus node [24]. In these study models, applied heat (by a water-perfused suit) caused an increase in ventricular inotropy but unchanged diastolic function.

In the exercise trials accompanied by heat stress induced by PPE in this study, oxygen uptake demands were accentuated compared with the non-heat stress trials, reflecting the recognized effect of increased temperature on metabolic rate. In the final analysis, however, we found no evidence of myocardial dysfunction, measured ~30 min following exercise, as a consequence of the augmented inotropic and lusitropic demands that occurred during the exercise, regardless of metabolic demand.

In the only previous echocardiographic investigation of cardiac performance changes with firefighting, Fernhall et al. [12] described equivocal evidence of myocardial dysfunction after a 3h training exercise, consisting of repeated bouts (~20 min each) of live fire evolutions. No changes were observed after the firefighting in longitudinal velocity of myocardial contraction by tissue Doppler imaging and no significant change was recorded in ventricular ejection fraction (although the mean value fell from 60.3 to 54.3%). However, left ventricular shortening fraction fell from 33.0 ± 6.3% to 28.6 ± 6.0%. With regard to diastolic function, mitral E velocity fell from 81 ± 14 to 71 ± 14 cm s⁻¹. Ventricular longitudinal diastolic relaxation velocity decreased significantly at the ventricular lateral wall but was unchanged at the septum. The authors concluded that the decreases in left ventricular function were likely produced by a combination of high levels of physical work, hyperthermia and dehydration associated with firefighting.

Our failure to demonstrate adverse effects on myocardial function in laboratory-simulated exercise combining heat stress and dehydration is in contrast to findings following repeated bouts of live firefighting activities [12]. Study design and participant demographics may explain different findings. The participants in this study were young, healthy, aerobically fit men. Fluid ingestion was highly regulated rather than ad libitum (as in the Fernhall study) and exercise was performed in thermoneutral conditions. Also, the absolute change noted in mitral E velocity (pre: 82 ± 12 cm s⁻¹; post: 72 ± 13 cm s⁻¹) in our HS-DEH trial is very similar to that reported by Fernhall et al. [12]. Thus, whether the lack of negative findings regarding effects on myocardial function would hold true in other populations could be clarified by future research. Compared with our participants, workers in occupations such as firefighting and underground mining who routinely experience heat stress and dehydration are generally older, have a higher BMI, are less fit and may have more cardiovascular disease risk factors [25–28]. Thus, extending this research to populations more representative of the impacted occupational groups is particularly relevant.

### Key points

- Repeated bouts of moderate-intensity exercise (40% VO₂max) in young, aerobically fit men wearing structural firefighting gear resulted in significant cardiac and thermal strain during exercise.
- Heat stress resulting in an increase (1.2°C) in core temperature had a much larger effect than moderate dehydration (~3% body mass loss) on heart rate responses to exercise.
- Following a short recovery period (~30 min), we found no evidence of myocardial dysfunction following any experimental condition in a group of young, aerobically fit men.

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### Conflicts of interest

None declared.

### References


