Hand cold pressor test induces thermogenesis in upper thoracic regions as measured by skin surface infrared thermography.

Short title: Cold pressor test induced thermogenesis in upper thorax

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Abstract

Background
Cold exposure may cause health problems and impaired productivity in outdoor or cold-temperature workers. The cold pressor test (CPT) is a laboratory procedure that measures cardiovascular and thermoregulatory responses to acute cold exposure such as metabolic activity in brown adipose tissue. How the body responds to acute cold exposure of a hand is not completely understood. We tested the hypothesis that the upper thorax produces heat during a single hand-CPT, which restores warmth to the cold-expose appendage.

Objectives
The objective was to measure skin temperature changes in the upper thoracic regions and the cold-exposed appendage during a CPT. The secondary objective was to determine if cardiovascular or psychological responses during CPT accounted for skin temperature changes.

Methods
50 healthy participants immersed their right hand up to wrist level in 4 °C water for three minutes. Surface skin temperatures were imaged by infrared thermography at baseline, during CPT, and in the recovery phase. Sublingual oral temperature and water bath temperature were recorded throughout the test. Cardiovascular responses were monitored by continuous finger pulse-wave plethysmography. Peak pain and peak stress were reported by the participants on a Likert scale.
Results

CPT increased the systolic blood pressure (+22 mmHg, p < 0.001), diastolic blood pressure (+15 mmHg, p < 0.001) and heart rate (+7 beats per minute, p = 0.024). During CPT, skin temperature increased on thoracic regions including mediastinal (+0.5°C, p < 0.021), sternal (+0.5°C, p < 0.002), right supraclavicular (+0.3°C, p < 0.042) and left supraclavicular (+0.3°C, p < 0.016) regions. During CPT, the hand was cooler on ventral (-14.6°C, p < 0.001) and dorsal (-15.2°C, p < 0.001) sides, and warmed up during recovery. The ventral forearm, dorsal forearm, antecubital fossa, and adjacent medial epicondyle region were significantly cooler throughout the recovery time. The oral temperature did not change during CPT. There were no correlations between the change in mediastinal skin temperature and the sex of the participant, or changes in cardiovascular parameters, peak pain, or peak stress values.

Conclusions

Localized hand cooling caused a rapid warming of the thorax, dissipation of cold in the forearm, and rewarming of the hand during recovery. Thermoregulation was not dependant on pain, stress, sex, or cardiovascular changes between participants. By understanding thermoregulation, better approaches can be developed to mitigate the negative impacts of cold exposure.
Introduction

Extreme temperatures can affect populations with high rates of morbidity such as diabetes and cardiovascular disease (1). Workers exposed to cold were more likely to display increased airway symptoms such as wheeze and cough (2), and long term cold-related circulatory complaints, fatigue, and performance degradation (3). Working in a cold environment was also related to increased reporting of chronic pain (4). Gaining a better understanding of how the body reacts to sudden cold exposure will help manage the implications that extreme climate conditions could have on health. The cold pressor test (CPT) has been used as an indication of blood pressure fluctuations as it relates to meteorological factors such as outdoor temperature (5). Such procedures are done on hands, fingers or feet submerged in a water bath at noxious temperatures (~0-7°C). CPT has been used to study the autonomic nervous system, thermoregulation and hypertension (11,17,18). When a limb is exposed to cold, a series of reactions occur that serve to preserve the function of the limb and maintain a stable core temperature. The first response occurs prior to cold exposure; the awareness of impending cold produces a small but significant rise in heart rate and blood pressure referred to as the alerting response (6). Localized, acute cold exposure rapidly increases blood viscosity (7) which reduces blood flow (8,9) and perfusion of oxygen into the tissue (10). During CPT, there is a profound increase in sympathetic nerve activity with concomitant parasympathetic withdrawal (11). Temperature sensing receptors in the skin send impulses to the vasomotor center of the brainstem which relays signals to sympathetic nerve pathways. This central neural reflex induces local vasodilation in the cold-affected region and initiates shivering or non-shivering thermogenesis (12). Moreover, it causes vasoconstriction of vascular beds of the kidney (13) and mesenteric (14) circulatory systems. Vasoconstriction of internal blood vessels causes a rise in systolic
blood pressure, diastolic blood pressure, systemic vascular resistance, and mean arterial blood
pressure (15). Together, these changes help to maintain circulation and temperature control in the
cold-affected tissues.

During acute cold exposure, non-shivering thermogenesis occurs in brown adipose tissue. In humans, brown adipose tissue is located subcutaneously at the supraclavicular fossa between anterior neck muscles, axilla, under the clavicles, surrounding the heart, aorta, major arteries, and in pericardial mediastinal fat (16). Acute cold exposure induced brown adipose tissue metabolic activity in thoracic regions, particularly in the right and left supraclavicular fossae (19,20). The overall amounts of brown adipose tissue in the adult human are considered to be rather small, and appear to contribute modestly to the basal metabolic rate (21). However, it was shown using thermal imaging that CPT increased skin temperature of the supraclavicular fossa increases by 0.3–0.7 °C, indicating that there was localized thermogenesis around the brachial arteries which leads to the arms (16,22). This would be essential for thermoregulation because limbs cannot produce significant amounts heat; limbs rely on circulatory control to maintain temperature homeostasis (23,24). One form of circulatory control is counter-current heat exchange, a process whereby venous blood exchanges heat with warm arterial blood thereby attenuating heat loss in the cold-affected limb. The functional anatomy for counter current heat exchange was shown in the legs of wading birds (25,26), however, it has not been studied extensively in humans. Theoretical models predicted that a small but significant amount of heat transfer occurs in the human limb due to counter-current heat exchange in the forearm (24,27).

Infrared thermography is a non-invasive method to assess skin temperature changes. Digital thermal imaging has been recently used to assess febrile temperature (28), bodily maps of emotions (29), and to infer brown adipose activity (16). In the present study, we tested the
hypothesis that acute hand cooling induces a rapid temperature increase in the upper thorax. We obtained evidence for circulatory control of arm temperature during CPT using infrared thermography and cardiovascular monitoring.

Materials and methods

Participants

This study was approved by the Concordia University human research ethics committee which follows the Declaration of Helsinki principles. Informed signed consent was obtained from 60 healthy participants of age 18 and older. Their health status was determined by self-reporting according to standardized questions throughout the screening phone-interview. To control for menstrual cycle phase, female participants were scheduled to participate during their follicular phase (11). Participants were asked to abstain from caffeine, alcohol, and exercise twelve hours prior to participation, and to not eat at least two hours before the study. Relational ties between participant and experimenter, for example family, partner, or spouse, were avoided given the reported association between empathy and stress-test results (30). Height and weight were obtained in the consultation room, and were used to calculate body mass index using the formula body mass (kg) divided by body height (m) squared. Brachial blood pressure and heart rate were measured in a consultation room after the participant had been sitting for at least 15 minutes (Accutorr Plus V, Medaval, Dublin, Ireland). Participants were excluded from the study if their systolic blood pressure was greater than 140 mmHg or less than 90 mmHg, heart rate was greater than 100 beats per minute or less than 50 beats per minute, or a body mass index greater than 27 kg/m². If these conditions were met, they were directed to see a physician. Of the 60 participants who consented to the study, data from 10 participants were removed because four
participants had low blood pressure prior to the test and did not undergo CPT, five participants felt faint or fainted during the CPT and researchers stopped the test, and one participant withdrew their hand from the water mid test. Finally, 50 participants completed the CPT.

**CPT procedure**

After consultation, participants moved to the testing room (Table 1). The CPT apparatus was not present when the participant entered the testing room to prevent an alerting response. The chair had a reclining function used in the event of fainting. Their left arm was rested on a chest-height table while the cardiovascular monitor was affixed (Nexfin®; BMEYE, Amsterdam, The Netherlands). The Nexfin is a finger plethysmograph which monitors beat-to-beat arterial blood volume fluctuations with waveform analysis using a finger pressure cuff attached to the contralateral (left) middle finger and calibrated with a heart-level sensor (31). Once the cardiovascular signal stabilized, the baseline was recorded for 15 minutes. In a separate room, the cold bath was prepared. The cold apparatus was a modified picnic cooler with freezer packs and a thermometer affixed to the interior walls of the cooler with industrial grade Velcro. Cold water and crushed ice was added until the bath reached a stable temperature. After the baseline recordings were complete, the cold bath was placed on a platform next to the right side of the chair. At least two minutes elapsed prior to hand immersion. This was considered the alerting response time and it was removed from analysis. At minute 17 the hand immersion commenced. They submerged their right hand to wrist level for 3 minutes in a passive open position; they were instructed to not make a fist. Throughout the baseline CPT and recovery, oral temperature was measured sublingually with a digital oral thermometer. Participants self-reported pain and stress throughout the baseline CPT on a visual analogue scale. A Likert scale was used to
determine pain and stress where 0 equalled no pain or stress at all, and 10 equalled the worst possible imaginable pain/stress.

**Table 1 Summary of Study Procedure**

<table>
<thead>
<tr>
<th>Location</th>
<th>Timeframe</th>
<th>Participants' Self-Reporting</th>
<th>Measurements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consultation Room</td>
<td>45 min before testing</td>
<td>Age, sex</td>
<td>blood pressure and heart rate measures by Accutorr, height, weight</td>
</tr>
<tr>
<td>Testing Room</td>
<td>15 min before testing</td>
<td>-</td>
<td>attach Nexfin, allow recordings to stabilize</td>
</tr>
<tr>
<td>Baseline 0-15 min</td>
<td></td>
<td>Pain at 1.5, 10, 15 min</td>
<td>continuous heart rate and blood pressure by Nexfin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stress at 1.5, 10, 15 min</td>
<td>Sublingual temperature 5, 10, 15 min</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thermograms 5, 10, 15 min</td>
</tr>
<tr>
<td>Water bath into room, alerting response 15-17 min</td>
<td>-</td>
<td>data excluded analysis</td>
<td></td>
</tr>
<tr>
<td>CPT 17-20 min</td>
<td></td>
<td>Pain at 17.5, 19, 20 min</td>
<td>continuous heart rate, blood pressure by Nexfin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stress at 17.5, 19, 20 min</td>
<td>water temperature 18, 19.5 min</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sublingual temperature 17.5, 19 min</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thermograms 17.5, 19, 20 min</td>
</tr>
<tr>
<td>Recovery 20-30 min</td>
<td></td>
<td>Pain at 25, 30 min</td>
<td>continuous heart rate, blood pressure by Nexfin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Stress at 25, 30 min</td>
<td>Sublingual temperature 25, 30 min</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thermograms 25, 30 min</td>
</tr>
</tbody>
</table>
Thermograms

The FLIR C2 portable infrared camera was used to take thermograms and simultaneous visible-light images (FLIR Systems Inc. Wilsonville, OR, USA). Prior to the test, anatomical landmarks were located and marked on the participant with an erasable pen. To find the jugular notch of the sternum at the top of the midline body structure of the thorax, we followed the clavicle towards the center and a point was placed on the clavicular notch felt at the dip. For the left and right supraclavicular fossae, we placed a point in the angle between the clavicle and the sternocleidomastoid tendon. The thorax image had four regions of interest: middle thorax (mediastinal), upper thorax (sternal, below the jugular notch/manubrium), left supraclavicular fossa and right supraclavicular fossa (between the sternal head of the sternocleidomastoid and the clavicle). For the dorsal hand we marked the intersection between the third extensor digitorum tendon and the purlicue (the fold between the thumb and index finger). For the dorsal forearm we marked halfway between the wrist line and antecubital fossa. For the ventral hand (palmar) we marked the intersection between the purlicue and the center of palm. For the ventral forearm we marked halfway between the wrist line and antecubital fossa. For medial epicondyle, with the participant's hand in supinated anatomical position (palm up), we marked the medial bony protuberance at the elbow. The regions for the arm in the ventral position (in standard anatomical position) included the antecubital fossa (synonymous with cubital fossa), the adjacent medial epicondyle, forearm (midway between wrist and elbow), and the center of the palm. The dorsal arm regions included the forearm midway between wrist and elbow, and the center dorsum of the hand. Thermograms were taken at a 1 meter distance from the participant at 5 min baseline, 10 min baseline, 15 min baseline, 0.5 min immersion, 2 min immersion, immediately
after hand removed from water, 5 min recovery, 10 min recovery (Table 1). The testing room did not contain other infrared light sources. The left arm was not imaged.

Data analysis

Thermogram files were renamed with randomized codes to ensure that the analysis was done blinded. Two researchers who were unaware of the file codes, independently analyzed the thermograms using FLIR Tools software. Rectangles of 16 x 12 mm were placed on the thoracic regions of interest. For the thermograms of the cold exposed arm, circles diameter 8 mm were placed on the regions of interest. The mean temperature for each region was recorded. The placement of the regions of interest were made using anatomical landmarks visible on the digital picture overlay. For each participant, skin temperature and cardiovascular readings were averaged into three phases: baseline (t1, t2, t3), during CPT (t3.5, t4, t5), and recovery (t6, t7). Thermograms were excluded from analysis if clothing obstructed the clavicle or mediastinal region, the elbow region or thorax was partially out of frame, or the perpendicular axis of the elbow was on an oblique angle. The number of thermograms analyzed were 50 for right supraclavicular, 49 for left supraclavicular, 50 for sternal, 50 for mediastinal, 49 for ventral hand, 48 for dorsal hand, 45 for ventral forearm, 45 for antecubital fossa, 45 for medial epicondyle region, 43 for and dorsal forearm. The null hypothesis was tested with an analysis of variance (ANOVA, p<0.05) and Tukey's test or Bonferroni tests. Software used was SPSS Statistics 24.0 (IBM, New York, USA). Pearson's test was used for correlation analysis.
Results

Cardiovascular Response to CPT

Fifty healthy participants completed a three minute CPT by immersing their right hand up to wrist level into ice-cold water. Of the participants who completed the CPT, 29 were female, 21 male, with an average age of 23.9 ± 5.4 years (range 20-43), weight of 68.7 ± 14 kg, and height of 1.70 ± 0.1 m. The participants self-reported their ethnicity as 31 White, six Middle Eastern, five Black, three Asian, two East Asian, one Latin, and two of mixed ethnicity. The average water temperature measured was 2.8 +/- 0.7 °C (N=50). The participants' systolic blood pressure, diastolic blood pressure, and heart rate increased during CPT and returned towards baseline in the recovery time, while the oral sublingual temperature remained constant throughout the test (Table 2).

Table 2 Cardiovascular, oral, and skin temperature measurements in 50 healthy participants undergoing CPT.

<table>
<thead>
<tr>
<th>Variable</th>
<th>BL</th>
<th>BL vs. CPT</th>
<th>CPT</th>
<th>CPT vs. REC</th>
<th>REC</th>
<th>BL vs. REC</th>
</tr>
</thead>
<tbody>
<tr>
<td>systolic BP mmHg</td>
<td>130 ± 18</td>
<td>***</td>
<td>152 ± 21</td>
<td>0.008</td>
<td>139 ± 19</td>
<td></td>
</tr>
<tr>
<td>diastolic BP mmHg</td>
<td>82 ± 12</td>
<td>***</td>
<td>97 ± 15</td>
<td>0.002</td>
<td>87 ± 13</td>
<td></td>
</tr>
<tr>
<td>heart rate bpm</td>
<td>70 ± 10</td>
<td>0.024</td>
<td>77 ± 14</td>
<td>0.003</td>
<td>68 ± 11</td>
<td></td>
</tr>
<tr>
<td>Oral temp. °C</td>
<td>36.6 ± 0.2</td>
<td>0.024</td>
<td>36.5 ± 0.2</td>
<td>0.003</td>
<td>36.6 ± 0.2</td>
<td></td>
</tr>
<tr>
<td>R Supraclavicular °C</td>
<td>35.4 ± 0.7</td>
<td>0.042</td>
<td>35.7 ± 0.6</td>
<td>0.003</td>
<td>35.6 ± 0.8</td>
<td></td>
</tr>
<tr>
<td>L Supraclavicular °C</td>
<td>35.1 ± 0.7</td>
<td>0.016</td>
<td>35.5 ± 0.6</td>
<td>0.003</td>
<td>35.4 ± 0.6</td>
<td></td>
</tr>
<tr>
<td>Mediastinal °C</td>
<td>34.0 ± 0.8</td>
<td>0.021</td>
<td>34.5 ± 0.8</td>
<td>0.003</td>
<td>34.3 ± 0.9</td>
<td></td>
</tr>
</tbody>
</table>
Cardiovascular and temperature recording during baseline (BL), CPT and recovery (REC). SD is shown. BP = blood pressure. Univariate ANOVA was performed with post hoc Tukey's test for multiple comparisons. Where the differences are significant, P values are shown in columns for baseline as compared to CPT, CPT compared to recovery, and baseline as compared to recovery. The exact p values are indicated, or ***p<0.001.

<table>
<thead>
<tr>
<th>Region</th>
<th>BL</th>
<th>CPT</th>
<th>REC</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sternal °C</td>
<td>33.9 ± 0.6</td>
<td>34.4 ± 0.7</td>
<td>34.3 ± 0.7</td>
<td>0.017</td>
</tr>
<tr>
<td>Ventral Hand (Palm) °C</td>
<td>31.8 ± 2.4</td>
<td>17.2 ± 2.7</td>
<td>26.2 ± 2.5</td>
<td>***</td>
</tr>
<tr>
<td>Dorsal Hand °C</td>
<td>30.4 ± 2.1</td>
<td>15.2 ± 2.2</td>
<td>21.9 ± 1.8</td>
<td>***</td>
</tr>
<tr>
<td>Ventral Forearm °C</td>
<td>32.7 ± 0.9</td>
<td>31.8 ± 1.4</td>
<td>31.1 ± 1.0</td>
<td>***</td>
</tr>
<tr>
<td>Dorsal Forearm °C</td>
<td>31.6 ± 0.9</td>
<td>31.5 ± 0.1</td>
<td>30.5 ± 1.2</td>
<td>***</td>
</tr>
<tr>
<td>Antecubital Fossa °C</td>
<td>33.7 ± 0.8</td>
<td>33.9 ± 0.8</td>
<td>33.0 ± 1.0</td>
<td>***</td>
</tr>
<tr>
<td>Medial Epicondyle °C</td>
<td>32.3 ± 0.9</td>
<td>31.9 ± 1.1</td>
<td>31.4 ± 1.6</td>
<td>0.002</td>
</tr>
</tbody>
</table>

**Skin temperature changes**

Skin temperatures were measured on the anterior thorax, cold exposed hand, and arm before, during, and after the CPT. We observed skin temperature increases on the thoracic regions during CPT and in the recovery phase (Table 2, Fig 1 a-f). The right and left supraclavicular regions were significantly increased during CPT as compared to baseline. The temperature changes observed on the right and left supraclavicular regions were correlated with each other (Pearson's correlation of 0.827). The mediastinal and sternal regions were significantly increased during CPT, and the sternal region remained elevated during recovery. With respect to the cold exposed hand, we observed ventral and dorsal skin temperature decreases during CPT which
partially rewarmed during recovery (Table 2, Fig 2a-f). The ventral forearm was cooler immediately after CPT and continued to cool during recovery. The dorsal forearm was cooler during recovery time as compared to CPT or baseline. The antecubital fossa skin temperature was cooler during recovery compared to baseline, and the adjacent medial epicondyle region was cooler during recovery, albeit to a lesser extent than observed for the antecubital fossa (Table 2).

**Fig 1.** Surface skin temperatures on the anterior thorax. Representative surface skin temperatures of a female participant (top row) and male participant (bottom row) are shown for baseline (a, d), CPT (b, e) and recovery (c, f).

**Fig 2.** Surface skin temperatures on hand and arm. Surface skin temperatures were measured on the hand and arm in ventral (top row) and dorsal (bottom row) positions. The pictures represent baseline (a, d), CPT (b, e) and recovery (c, f). The thermograms for the CPT was taken immediately after withdrawal of the hand from the ice bath at 3 minutes.

To account for possible psychophysiological variables, the participants were asked to report their pain and stress. The peak pain and peak stress values correlated to each other, however, they did not correlate significantly to the change in mediastinal skin temperature (Table 3). Cardiovascular changes in heart rate and systolic blood pressure did not correlate...
significantly to the change in mediastinal skin temperature (Table 4). When sex was analyzed as a confounding variable, there were no significant difference in the change such as mediastinal skin temperature during CPT compared between female and male participants ($p = 0.94$ unpaired t test). Thus, the rapid skin temperature changes did not appear to be influenced by self-reported pain, stress, cardiovascular measures, or sex. In summary, a three minute hand CPT induced rapid warming of the thorax, a sharp drop in temperature of the hand with subsequent rewarming, and a dissipation of cold throughout the forearm during recovery.

**Table 3 Pearson correlations between change in psychological variables with change in mediastinal skin temperature during CPT.**

<table>
<thead>
<tr>
<th>Pearson correlations</th>
<th>peak pain</th>
<th>peak stress</th>
<th>$\Delta$ mediastinal skin temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>peak pain</td>
<td>1</td>
<td>0.628 **</td>
<td>-0.120</td>
</tr>
<tr>
<td>peak stress</td>
<td>0.628 **</td>
<td>1</td>
<td>0.084</td>
</tr>
<tr>
<td>$\Delta$ mediastinal skin temperature</td>
<td>-0.120</td>
<td>0.084</td>
<td>1</td>
</tr>
</tbody>
</table>

** p < 0.01 significance level, $\Delta$ = change between CPT and baseline

**Table 4 Pearson correlations between change in cardiovascular variables with change in mediastinal skin temperature during CPT.**

<table>
<thead>
<tr>
<th>Pearson correlations</th>
<th>$\Delta$ heart rate</th>
<th>$\Delta$ systolic blood pressure</th>
<th>$\Delta$ mediastinal skin temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Delta$ heart rate</td>
<td>1</td>
<td>0.249</td>
<td>0.001</td>
</tr>
<tr>
<td>Δ systolic blood pressure</td>
<td>0.249</td>
<td>1</td>
<td>0.209</td>
</tr>
<tr>
<td>Δ Mediastinal Skin Temperature</td>
<td>0.001</td>
<td>0.209</td>
<td>1</td>
</tr>
</tbody>
</table>

No significant correlations were observed. $\Delta$ = change between CPT and baseline

**Discussion**

Acute cold exposure causes a rapid cardiovascular and thermal response that protects the exposed limb from hypothermia and maintains a constant core temperature. Our raw data is found in a supplemental data file (S1 File). We observed significant increases in systolic blood pressure, diastolic blood pressure, and heart rate which is the expected result of CPT (32). Furthermore, we observed the expected temperature increases the thoracic regions including right and left supraclavicular fossa. These results are similar to those reported by Sacks and Symonds 2013, who collected thermograms from a small sample of people undergoing CPT (16). Their analysis focused on skin temperature changes in the supraclavicular regions where brown adipose tissue is known to exist. Brown adipose tissue can induce thermogenesis in these supraclavicular regions due to increased metabolic activity as shown with positron emission scanning of metabolic tracers (33). We also detected skin temperature increases on the central mediastinal and upper sternal areas of the thoracic midline. Mediastinal skin temperature on the thorax may be influenced by dilation of epicardia coronary arteries in response to an increased myocardial metabolic demand during CPT. This would increase blood flow, and therefore increase skin temperature in the upper thoracic region (34). Another explanation is activity of deep muscles in the cervico-thoracic region that can produce heat by non-shivering
Thermogenesis (20). Thermogenesis is also potentially caused by brown or beige adipose tissue in the thorax. Beige adipose is a unique blend of brown adipose tissue and white adipose tissue that can have metabolic activity (35,36). One or all of these mechanisms could have contributed to the skin temperature increases we observed on the thorax during exposure of the hand to acute cold.

The sublingual temperature did not change during CPT or in recovery, which is consistent with other findings; short-term cold exposure of a limb did not alter body core temperature (37). Core temperature does not change during acute localized hand-cooling, as was shown previously using an intestinal thermometer during 3 minute hand-CPT in a normothermic ambient environment (38). It takes over 10-15 minutes of pressor response for core temperatures to show a difference which is longer than our three minute exposure protocol (39). As expected, the temperature of the cold-exposed hand decreased substantially, and partially rewarmed during the 10 minute recovery. In the cold-exposed forearm, which was not submerged in the ice bath, we observed modest skin temperature decreases immediately after CPT and during the recovery time. That finding indicates that venous return is carrying cool blood away from the cold-exposed hand. As warm arterial blood has the potential to exchange heat with the cooler venous return, the forearm may represent a form of counter-current heat exchange that maintains core temperature and slowly rewarms the hand during acute cold exposure.

We investigated the possible link between thermogenesis and the psychological factors of pain and stress. Psychological factors can influence cardiovascular changes during CPT, for example, we recently reported a positive correlation with the amount of pain reported by the participants and their change in heart rate (40). Other researchers showed that the emotion of anxiety raised thoracic skin temperature using psychological testing and body temperature

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mapping (29). In the present study, we did not observe correlations between self-reported peak pain or peak stress with the change in mediastinal skin temperature. Furthermore, we did not find correlations between cardiovascular changes and the change in mediastinal skin temperature. Together, these data suggest that thermogenesis during CPT is a rapid neural or hormonal reflex that is unaffected by pain, stress, sex, or cardiovascular factors.

The overarching purpose of this study was to learn more about the effects of acute cold exposure on human physiology. The technical limitations of our study included the use of an industrial grade thermal camera which has less resolution and precision than clinical grade thermal cameras. Some thermograms had to be excluded due to slightly incorrect positioning of the arm. Having a fixed camera and giving the participants better instructions on arm positioning would correct these limitations. One of the advantages of using a portable thermal camera is its low cost and flexibility. A portable thermal camera could be used for field work aimed at capturing the thermal responses in people working in a cold environment. Our study validates the use of thermal imaging as a non-invasive, cost-effective and flexible method to study thermogenesis. By understanding the fundamental mechanisms of action that the body uses to maintain thermoregulation, we can better guide new approaches to prevent cold exposure health issues.

Acknowledgments
References


Figure 2