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Original Research

The Effects of Acute Anaerobic Exercise on the Cardiovascular and Metabolic Response to the Cold Pressor Test in Healthy Adult Males

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ABSTRACT

International Journal of Exercise Science 13(3): 1729-1740, 2020. Little is known about the physiological response to the cold pressor test (CPT) when in a clinically-induced state of autonomic nervous system (ANS) imbalance, despite its utility in various disease- and injury-states. To date, research in this area is limited to acute aerobic and isometric exercise, with a paucity of research investigating the effects of anaerobic exercise on the physiological response to the CPT. Therefore, the purpose of our study was to assess the effects of the Wingate anaerobic cycle test (WAT) on cardiovascular (CV) and metabolic recovery following the CPT in a group of healthy adult males. A pre-post intervention study was conducted, whereby 10 healthy adult males (age = 29 ± 4 years, height = 182 ± 7 cm, mass = 83 ± 9 kg) completed a baseline cold pressor test (CPT-only) and a follow-up cold pressor test preceded by a Wingate anaerobic exercise test (WAT+CPT). Recovery slopes for various CV and metabolic variables, including heart rate (HR), blood pressure (BP), and relative oxygen consumption ($\dot{V}O_2$) were analyzed using single-subject analysis, with celeration line slopes calculated for all participants in the CPT-only and WAT+CPT testing sessions. Celeration line slopes were compared between testing sessions using paired *t*-tests. No differences were identified for recovery slopes for HR (p = .295), diastolic BP (p = .300), and relative $\dot{V}O_2$ (p = .176) when comparing CPT-only and WAT+CPT testing sessions. Our results suggest that the CPT elicits a CV and metabolic response beyond that elicited solely by an acute bout of anaerobic exercise. As such, the CPT may be able to serve as a surrogate test for anaerobic exercise for individuals where high-intensity exercise may be contraindicated. Future research is warranted however, as the specific physiological mechanisms governing the observed responses have yet to be elucidated.

KEY WORDS: Autonomic nervous system, exercise physiology, physiological recovery

INTRODUCTION

Monitoring one's physiological response to a stressor can be useful in the assessment of an individual's health state. The cold pressor test (CPT) is one such stressor and involves the submersion of an individual's hand and wrist in near freezing water for a specified amount of time and tracking their physiological response. The CPT elicits cardiovascular (CV) stimulation,

as indicated by increases in heart rate (HR) (12, 21, 32, 33, 35), systolic blood pressure (SBP), and diastolic blood pressure (DBP) (3, 5, 12, 22, 32); a result of sympathetic nervous system (SNS) activation and parasympathetic nervous system (PNS) withdrawal (35). Previous studies have assessed the effects of the CPT on autonomic nervous system (ANS) control using biomarkers such as heart rate variability (HRV), however HR and blood pressure (BP) responses to the CPT appear to be more reliable than other CV variables (12, 36). These consistent physiological responses, along with the test's simplicity and inexpensiveness, make the CPT a prime candidate in disease and injury management (10, 13, 26).

To date, the CPT has been used to predict future CV events in individuals with various chronic CV conditions (26), however the clinical utility of the CPT may extend even further with preliminary evidence suggesting a blunted CV response to the CPT in individuals with acute concussion (13). While this blunted HR response is likely due to concussion-induced ANS dysregulation (11), there is a paucity of research examining how the physiological response to the CPT may be altered in healthy individuals when placed in clinically-induced states of ANS imbalance; making it difficult to contextualize CPT-related findings in these various disease/injury states. Given the athletic-nature of concussion-related injuries, it is imperative that physiological responses to the CPT following both acute and chronic bouts of exercise be profiled. Previous research in this area is generally limited to investigating the influence of acute bouts of aerobic (6, 27) or isometric (7, 24, 34) exercise on the physiological response to the CPT in healthy adult males, whereas the effects of anaerobic exercise remain under investigated. While one earlier study did examine the effects of an acute bout of anaerobic exercise on the CPT-related physiological response, the authors' primary focus was on renal vascular function and findings were limited with regard to recovery of various CV and metabolic variables (31).

Considering the gaps in knowledge and limitations from previous studies, the purpose of our study was to assess and compare CV and metabolic recovery following the CPT, both at rest and following the completion of an acute bout of anaerobic exercise in healthy males. Based on preliminary findings by Schlader et al. (31), we hypothesized that an acute bout of anaerobic exercise would attenuate CV and metabolic recovery following the CPT, as indicated by flatter recovery slopes for the variables of interest.

METHODS

Participants

A pre-post intervention case series was conducted, whereby ten healthy male participants were recruited by word of mouth to participate in this study. Participant demographics are presented in Table 1. Sample size was calculated a priori using an effect size of 1.23, $\alpha = .05$, $\beta = .20$ (4, 33, 35), resulting in a sample size of eight, which was rounded up to 10 participants. Inclusion criteria included: male; between the ages of 18-35 (inclusive); no previous diagnosis of hypertension; recreationally active. Exclusion criteria included: one or more contraindications to exercise as determined by the Physical Activity Readiness Questionnaire Plus; unable to understand instructions; unable to provide written consent. Participants were asked to refrain

from ingesting caffeine and alcohol, and to avoid participating in strenuous physical activity 12 hours prior to each testing session, and to avoid eating within four hours of testing.

All participants provided written informed consent. This study was approved by the local University Institutional Review Board. This research was carried out fully in accordance to the ethical standards of the International Journal of Exercise Science (23).

Table 1. Demographic variables.

	Mean ± SD	Range
Age, y	29 ± 4	(22-34)
Height, cm	182 ± 7	(170-191)
Body mass, kg	83 ± 9	(66-97)
BMI	25 ± 2	(22-27)
Tegner activity level	7.1 ± 1.6	(4-10)
Hand grip strength, kg		
Left	50 ± 6	(43-56)
Right	51 ± 9	(31-60)

Note: y = years; cm = centimeters; kg = kilograms.

Protocol

All participants reported to the research laboratory for two testing sessions between December 2018 and January 2019. The CPT-only session served as a control visit, whereby a baseline CPT was administered. The second session consisted of a Wingate anaerobic cycle test (WAT) followed immediately by a follow-up CPT (WAT+CPT). Time between successive testing sessions was 22.6 ± 18.9 days, with participants instructed to avoid changes in diet, training, and other lifestyle habits in order to mitigate the influence of confounding variables.

Upon arrival for their first testing session, participants had all study procedures explained to them by a member of the research team and provided written informed consent. Each participant completed a Physical Activity Readiness Questionnaire Plus to ensure they were free from any neurological, CV, or respiratory disorders which may put them at risk to participate in the study. Once consent was obtained and it was deemed safe for the participant to participate, a demographic questionnaire and a modified Tegner activity scale were completed, and height and weight were measured. Hand grip strength was also measured to ensure participants were within normative values (9), as hand grip strength has prognostic value related to health (15). Each participant was then seated in a comfortable position and all measurement equipment was connected. The equipment included: metabolic measurement system to analyze all expired air (TrueOne 2400 metabolic cart; Parvo Medics Inc., Sandy, UT), with the gas analyzer calibrated before each test with known gas concentrations (1.00% CO_2, 19.51% O_2) and flowmeter calibrated before each test using a 3 L calibration syringe; HR monitor (Polar H10; Polar Electro, Kempele, FI); automatic BP cuff (Omron BP710N; Omron Healthcare, Inc., Lake Forest, IL). Once all equipment was in place, participants rested quietly for five minutes to assess resting measurements. Following the collection of resting data, participants begin either the CPT or WAT, as outlined below.

The CPT began by placing the participant's hand in a tub of ice water (between 1-4°C) for two minutes while in a seated position, making sure water covered the entire hand up to the wrist, with the participant's fingers spread. Following two minutes of immersion, the participant's hand was removed from the tub of water and dried with a towel. Fifteen minutes of seated rest were then given to monitor physiologic recovery from the CPT.

The same equipment and procedures were used at follow-up visits, with the only modification being the addition of the pre-CPT WAT to increase SNS activity and decrease PNS activity (8). For this testing session, the participants completed a five-minute warm-up at a power output of 100 Watts on a cycle ergometer following five minutes of seated rest. The WAT began immediately after the warm-up period by having the participant pedal against a resistance equal to 7.5% of the participant's body weight for 30 seconds at maximal effort (14). Three minutes of active recovery peddling at 75 Watts began following the termination of the WAT, after which participants returned to a seated position and the follow-up CPT was initiated, as described previously.

Data was collected throughout the CPT (including recovery) and primary outcome measures included recovery slopes for HR, BP, and metabolic data (relative oxygen consumption, $\dot{V}O_2$; relative carbon dioxide production, $\dot{V}CO_2$; and relative minute ventilation, $\dot{V}_E$). Timepoints for HR and all metabolic data included: 30, 60, 90, 120, 420, 720 and 1,020 seconds. Timepoints for BP included 60, 120, 420, 720, and 1,020 seconds, due to the time required for automatic BP collection. Additionally, all participants were asked to rate the pain experienced during the CPT on a Likert scale ranging from 0 (no pain) to 10 (worst pain you have ever felt). The testing environment was kept as quiet as possible, with interactions between tester(s) and study participants kept at a minimum (e.g. only asking participants about pain, comfort, etc.). Participants were also asked to stay as still as possible for the duration of the CPT. A schematic for the experimental protocol can be found in Figure 1.

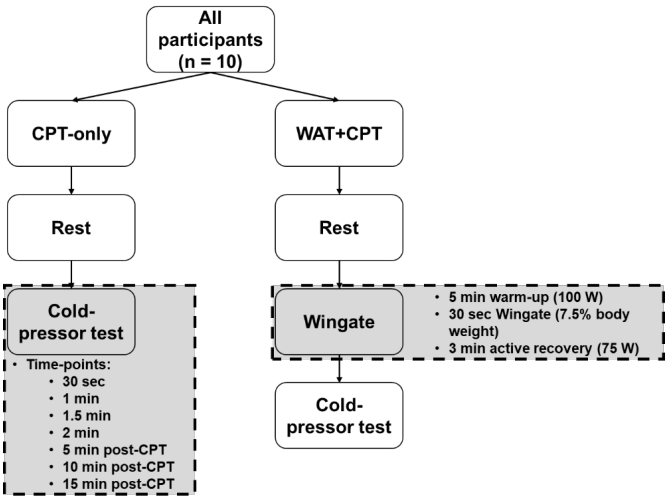

Figure 1. Schematic outline of experimental protocol.

Statistical Analysis

Results of descriptive statistics are presented as means ± standard deviation. Resting physiological variables (HR, BP, relative $\dot{V}O_2$, etc.) between CPT-only and WAT+CPT were

analyzed using paired t-tests. Physiological variables for the CPT-only session were compared at rest and at the first CPT time-point using paired t-tests. Physiological variables for the WAT+CPT session were compared at rest, post-WAG, and at the first CPT time-point using a one-way repeated measures ANOVA. To assess recovery of physiological variables in response to the CPT, single-subject design analyses were used. This was because completion of a WAT would result in an increased HR prior to the CPT in the WAT+CPT session, making comparisons to the CPT-only group difficult. Celeration line slopes were calculated for both CPT-only and WAT+CPT session data (25). In short, CPT data was first divided into lower and upper halves along the x-axis, after which the median score along the y-axis from each half was identified (P1). The median point along the x-axis was then found for both the lower- and upper-halves along the x-axis (P2). The intersection between points P1 and P2 for both lower- and upper-halves were then joined to form the celeration line. Slope was then calculated for each celeration line using the standard formula of:

$$Slope = \frac{(Y_2 - Y_1)}{(X_2 - X_1)}$$

Slopes for CPT-only and WAT+CPT were compared using paired t-tests. Where applicable, effect size (Cohen's *d*) was calculated. All analyses were completed using SPSS 24 (IBM Corp, Armonk, NY, USA), with an alpha level set at $p < .05$, and Bonferroni corrections used for any pairwise comparisons.

RESULTS

There were no differences noted in celeration line slopes for recovery HR, DBP, and relative $\dot{V}O_2$ between CPT-only and WAT+CPT (Table 2). Alternatively, WAT+CPT recovery line slopes were steeper for SBP, and relative $\dot{V}CO_2$ and $\dot{V}_E$ as compared to CPT-only, by factors of 1.8x, 4.0x and 2.9x, respectively.

No differences were noted in resting variables between CPT-only and WAT+CPT (Table 3). Several physiological variables were higher at the first CPT time-point compared to resting values for the CPT-only session, with HR, SBP, DBP, and relative $\dot{V}O_2$ increasing by 14%, 11%, 17%, and 23% respectively (Table 4). No differences were noted between rest and first CPT time-point values for relative $\dot{V}CO_2$ and $\dot{V}_E$ in the CPT-only session. All physiological variables, except for DBP, increased above resting levels in response to the WAT, while all variables were higher than resting levels at the first CPT time-point after the WAT (Table 5). No differences were noted for any physiological variables when comparing post-WAT to the first CPT time-point for the WAT+CPT session.

Table 2. Celeration line slopes for physiological variables.

	CPT-only (n = 10) Mean ± SD	WAT+CPT (n = 10) Mean ± SD	p	Effect size (95% CI)
Heart rate (beats per min)	-0.012 ± 0.008	-0.015 ± 0.010	.295	-0.4 (-1.3, 0.5)
Systolic blood pressure (mmHg)	-0.012 ± 0.012	-0.021 ± 0.015*	.016	-0.6 (-1.5, 0.3)
Diastolic blood pressure (mmHg)	-0.014 ± 0.012	-0.009 ± 0.010	.300	0.5 (-0.4, 1.4)
Relative $\dot{V}O_2$ (ml*kg^{-1}*min^{-1})	-0.003 ± 0.002	-0.004 ± 0.002	.176	-0.6 (-1.5, 0.3)
Relative $\dot{V}CO_2$ (ml*kg^{-1}*min^{-1})	-0.002 ± 0.003	-0.008 ± 0.002*	< .001	-1.5 (-2.5, -0.5)
Relative $\dot{V}_E$ (ml*kg^{-1}*min^{-1})	-0.091 ± 0.113	-0.260 ± 0.075*	.001	-1.3 (-2.3, -0.4)

Note: CPT = cold pressor test; WAT = Wingate anaerobic test; SD = standard deviation; 95% CI = 95% confidence interval; min = minutes; mm Hg = millimeters of mercury; ml = milliliters; kg = kilograms; $\dot{V}O_2$ = rate of oxygen consumption; $\dot{V}CO_2$ = rate of carbon dioxide production; $\dot{V}_E$ = minute ventilation. *indicates difference. Significance set at p < .05.

Table 3. Resting physiological values between testing sessions.

	CPT-only (n = 10) Mean ± SD	WAT+CPT (n = 10) Mean ± SD	p
Heart rate (beats per min)	64 ± 13	65 ± 12	.752
Systolic blood pressure (mmHg)	124 ± 10	125 ± 14	.681
Diastolic blood pressure (mmHg)	72 ± 11	72 ± 11	.866
Relative $\dot{V}O_2$ (ml*kg^{-1}*min^{-1})	4.0 ± 0.9	3.8 ± 0.8	.084
Relative $\dot{V}CO_2$ (ml*kg^{-1}*min^{-1})	3.1 ± 1.1	3.2 ± 0.5	.776
Relative $\dot{V}_E$ (ml*kg^{-1}*min^{-1})	116 ± 35	112 ± 17	.616

Note: CPT = cold pressor test; WAT = Wingate anaerobic test; SD = standard deviation; min = minutes; mm Hg = millimeters of mercury; ml = milliliters; kg = kilograms; $\dot{V}O_2$ = rate of oxygen consumption; $\dot{V}CO_2$ = rate of carbon dioxide production; $\dot{V}_E$ = minute ventilation. Significance set at p < .05.

Table 4. Physiological variables at rest and at the 1st CPT time-point during CPT-only session.

	Rest (n = 10) Mean ± SD	1st CPT time-point (n = 10) Mean ± SD	p
Heart rate (beats per min)	64 ± 13	73 ± 15*	.014
Systolic blood pressure (mmHg)	124 ± 10	138 ± 13*	.001
Diastolic blood pressure (mmHg)	72 ± 11	84 ± 12*	.002
Relative $\dot{V}O_2$ (ml*kg^{-1}*min^{-1})	4.0 ± 0.9	4.9 ± 1.6*	.033
Relative $\dot{V}CO_2$ (ml*kg^{-1}*min^{-1})	3.1 ± 1.1	3.9 ± 1.5	.201
Relative $\dot{V}_E$ (ml*kg^{-1}*min^{-1})	116 ± 35	151 ± 47	.101

Note: CPT = cold pressor test; SD = standard deviation; min = minutes; mm Hg = millimeters of mercury; ml = milliliters; kg = kilograms; $\dot{V}O_2$ = rate of oxygen consumption; $\dot{V}CO_2$ = rate of carbon dioxide production; $\dot{V}_E$ = minute ventilation. *indicates difference. Significance set at p < .05.

Table 5. Physiological variables at rest, post-WAT, and at 1st CPT time-point for WAT+CPT session.

	Rest (n = 10) Mean ± SD	Post-WAT (n = 10) Mean ± SD	1st CPT time-point (n = 10) Mean ± SD	p
Heart rate (beats per min)	65 ± 12	99 ± 22*	101 ± 21*	<.001
Systolic blood pressure (mmHg)	125 ± 14	153 ± 10*	147 ± 7*	<.001
Diastolic blood pressure (mmHg)	72 ± 11	75 ± 8	83 ± 9*	.008
Relative $\dot{V}O_2$ (ml*kg^{-1}*min^{-1})	3.8 ± 0.8	9.1 ± 2.5*	8.5 ± 1.3*	<.001
Relative $\dot{V}CO_2$ (ml*kg^{-1}*min^{-1})	3.2 ± 0.5	10 ± 2*	10 ± 2*	<.001
Relative $\dot{V}_E$ (ml*kg^{-1}*min^{-1})	112 ± 17	359 ± 95*	356 ± 82*	<.001

Note: CPT = cold pressor test; WAT = Wingate anaerobic test; SD = standard deviation; min = minutes; mm Hg = millimeters of mercury; ml = milliliters; kg = kilograms; $\dot{V}O_2$ = rate of oxygen consumption; $\dot{V}CO_2$ = rate of carbon dioxide production; $\dot{V}_E$ = minute ventilation. *indicates difference. Significance set at p < .05.

DISCUSSION

The main findings from our study were that HR and DBP recovery slopes following the CPT were not different when the CPT was preceded by an ANS-modifying stimulus (WAT), as compared to a non-modified CPT response, despite the fact that exercise increased CV parameters prior to the initiation of the CPT in the WAT+CPT condition. The recovery slope of relative $\dot{V}O_2$ was also unaffected in the WAT+CPT session, as compared to the CPT-only session. Taken together, these findings do not support our original hypothesis.

To date, acute aerobic and acute isometric exercise are the two primary exercise-induced states of ANS imbalance whose effects on the CV response to the CPT have been investigated. With respect to acute aerobic exercise, Ebbesen et al. reported a dampening effect of aerobic cycling exercise on CPT-mediated increases in CV variables in healthy adult males (6). More specifically, participants that performed exercise on a stationary bike at 50-55% of their $\dot{V}O_{2max}$ for one hour had a lower peak SBP in response to the CPT compared to those in the control group (6), while DBP response to the CPT was blunted in participants that exercised for either one or two hours at 50-55% of their $\dot{V}O_{2max}$ (6). Another study noted the CPT-mediated HR response was dampened when participants were exposed to 30 minutes of aerobic cycling exercise at 60% of their $\dot{V}O_{2max}$ (27). While there was no suppression of SBP in response to the CPT following aerobic exercise in this previous study, the authors speculate that there may be a dose-response relationship between aerobic exercise and CV response to the CPT (27). Specific reasons for the suppressed CV response to the CPT remain elusive, however it is speculated that vasoconstriction due to cold-exposure is mitigated following aerobic exercise, thereby reducing the CPT-mediated CV response following aerobic exercise (6).

The association between acute isometric exercise and the CPT is slightly different, with previous studies employing methodologies that have participants perform the CPT during acute isometric exercise. Peikert and Smolander reported that HR and DBP were higher when CPT and isometric knee extension were performed in tandem as compared to knee extension only (24), with similar findings noted in response to the combined execution of isometric hand-grip exercise and CPT (7, 34); all of which were found in samples of mostly healthy adult males. It

remains likely that a greater degree of vasoconstriction is the main cause of the increased response of variables to the CPT in the stacked protocols (24), however this has yet to be fully elucidated.

Although the effects discussed above are of importance, the effects of acute anaerobic exercise on the CV and metabolic response to the CPT remain sparse, with our searches yielding only a single study in this area (31). While the primary focus of this earlier study was on renal hemodynamics, the authors did note an altered CV response to the CPT when preceded by an acute bout of anaerobic exercise; namely lower changes in HR and mean arterial pressure from baseline to the first minute and second minute of the CPT, respectively. Therefore, we sought to investigate the effects of the WAT, a common maximal effort anaerobic exercise test, on CV and metabolic recovery following the CPT given the paucity of information in the literature. Our findings identified a typical CPT response in the CPT-only session, as noted by the increases in HR, SBP, and DBP (3, 5, 12, 16, 18, 22, 28, 32, 33, 35), but not in the WAT+CPT session. A likely explanation for this is the large increase in CV variables above resting levels induced by the WAT. In anticipation of this obstacle, celeration line slopes were calculated and compared for the CPT-only and WAT+CPT sessions. Our findings indicate no differences in recovery slopes for HR, DBP, and $\dot{V}O_2$, suggesting that the CPT continues to elicit a CV and metabolic response despite prior stimulation of the ANS via the WAT. This is not in agreement with our proposed hypothesis, and likely suggests a CPT-mediated increase in SNS activity and/or PNS withdrawal beyond that of the WAT alone. This is further evidenced by the increase in DBP above resting levels at the first CPT time-point in the WAT+CPT session but not at the post-WAT time-point. It is difficult to compare our findings to those of Schlader et al. due to differences in variables and analyses, however the dampened CV response to the CPT following the WAT reported previously (31) was not noted for a number of variables in our study. Alternatively, our findings are somewhat in agreement with the studies that combined isometric exercise and the CPT (7, 24, 34). Nevertheless, it has been suggested that an increase in pain may cause the increased CV response during the CPT (35), as pain also elicits an increase in HR (2) and BP (29) through ANS modulation (1). Recent research indicates that acute anaerobic exercise mitigates the sensation of pain (30), potentially reducing the CPT-mediated CV response following anaerobic exercise, however participants in the present study reported similar pain sensations in response to the CPT during the CPT-only (4.8 ± 1.6) and WAT+CPT (5.4 ± 1.7; p = .168) sessions. Thus, this lack of dampened pain response indicates that the similar recovery slopes for HR, DBP, and relative $\dot{V}O_2$ following the CPT in both testing sessions may be mediated by a similar pain-response. It is worth noting that the lowered pain threshold post-WAT noted previously was found using water that was substantially warmer than the present study (10°C vs. 1-4°C) (30), and is likely the reason why the pain-response was still a factor in our findings (20).

The present study is not without its limitations. The WAT and CPT were effective in increasing physiological measures indicative of increased CV activity, suggesting an increase in SNS activity and/or PNS withdrawal (8). More direct measures of ANS activity (e.g. HRV) would have benefitted the robustness of the present study, however early attempts at collecting this data using available equipment proved to be largely inconsistent. Furthermore, it has been

suggested that CPT-mediated changes in HR and BP are more reliable than changes in HRV (12, 36).

While our findings identified increases in HR, BP, and $\dot{V}O_2$ at the first CPT time-point compared to resting values, it appears as though there were some non-responders to the CPT, with non-response having been noted previously (3). Reasons for the lack of CV and/or metabolic response could be tied to individual differences in pain experienced during the CPT (2, 29, 35), however our study was not designed to investigate this further.

The CV response to the CPT has been shown to be consistent across multiple studies (3, 5, 12, 21, 22, 32, 33, 35), however the reliability of the CPT has been called into question due to the wide variety of water temperatures in various protocols. Indeed, a difference of only 2°C can result in different pain-scale ratings and can be mitigated by continuously circulating the water (20). While the present study did not circulate the water in the water bath, there were no differences in pain ratings between testing sessions, supporting the notion that water temperature was a non-factor in our results.

The WAT elicited an ANS stimulus that may have been too strong, potentially overpowering any contribution that the CPT may have had on any of the CV and/or metabolic variables. However, the recovery slope and DBP findings in the present study suggest that this was not the case, with the CPT appearing to evoke an ANS response in addition to that brought on by the WAT alone.

Participants were limited to males, and activity level data was collected using a modified Tegner activity scale. Thus, findings may not be generalizable to females, or to those of varying fitness levels, as direct measurement of $\dot{V}O_{2max}$ was not performed. It remains possible that fitness level may have played a role in our findings, as improved cardiorespiratory fitness has been shown to mitigate the hemodynamic response to the CPT (5).

Status of bladder filling/voiding was also not accounted for in the present study which may have an effect on the CV response to the CPT, as bladder control is regulated by the ANS (19). Given that a full bladder corresponds to an increase in SNS activity (19), it is likely that an individual's CV response to the CPT would be amplified if their bladder was full.

A conscious effort was made to keep time of day for testing as consistent as possible for all participants at all testing sessions as time of day can influence ANS balance (17), however individual schedules prevented consistency between participants.

In conclusion, our findings are the first to illustrate the effects of anaerobic exercise on cardiometabolic recovery following the CPT. The results from the present study demonstrate that recovery of HR, DBP, and $\dot{V}O_2$ following the CPT were not altered when preceded by an acute bout of anaerobic exercise. It therefore remains possible that the CPT may serve as a surrogate test for anaerobic exercise and may prove useful in the management of various conditions where high-intensity exercise is contraindicated. Although these findings are specific

to healthy individuals, there is now an opportunity to determine if recovery slopes for CV and metabolic variables differ between CPT-only and WAT+CPT conditions is various disease and injury states, namely those affecting the ANS.

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