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Pre-Conditioning with Peristaltic External Pneumatic Compression Does Not Acutely Improve Repeated Wingate Performance nor Alter Blood Lactate Concentrations During Passive Recovery Compared to Sham

Running Head: Pre-conditioning with Peristaltic Pneumatic Compression and Repeated Anaerobic Exercise Performance

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ABSTRACT

Application of dynamic external pneumatic compression (EPC) during recovery from athletic activities has demonstrated favorable effects on flexibility, soreness, swelling and blood lactate (BLa) concentrations. However, the effects of “pre-conditioning” with a peristaltic pulse EPC device on subsequent performance and BLa concentrations have not been characterized. Herein, we demonstrate that pre-treatment for 30-min with EPC has no effect on subsequent supramaximal exercise performance or BLa concentrations during passive recovery.

Keywords: Intermittent Pneumatic Compression Devices; Athletic Performance; Lactic Acid; Devices; Exercise Therapy; Sports Medicine; Exercise; Regional Blood Flow; Nitric Oxide;
INTRODUCTION

Dynamic external pneumatic compression (EPC) use in athletic recovery paradigms has demonstrated favorable effects on flexibility (Sands et al. 2014b), muscle soreness (Sands et al. 2014a), and muscle swelling (Chleboun et al. 1995). Moreover, dynamic EPC has been shown to improve local contractile capacity in a model of acute recovery from a fatigue protocol (Wiener et al. 2001). We recently demonstrated that 30 minutes of dynamic EPC during recovery from repeated Wingate Anaerobic Tests (WAnT) significantly lowered blood lactate (BLa) concentrations compared to passive recovery (Martin et al. 2015b). We also recently found that vascular reactivity was improved systemically in the muscular conduit arteries and locally in the resistance arteries of healthy subjects following 1 hour of dynamic EPC (Martin et al. 2015a). Given the role of vascular reactivity and nitric oxide (NO) bioavailability in exercise induced hyperemia and metabolite clearance (Maiorana et al. 2012), it follows that “pre-conditioning” with EPC may also improve BLa clearance following an intense exercise effort. Therefore, the purpose of this study was to evaluate the effects of a single 30 min EPC bout on subsequent repeated bouts of supramaximal exercise and BLa concentrations during passive recovery.

MATERIALS AND METHODS

Subjects

Ten (n=10) Division I men’s college hockey players were recruited for participation in this crossover design study during their off-season. Subject characteristics were as follows (mean ± SD): age, 22.8 ± 1.1 years; body mass, 84.7 ± 3.9 kg; height, 181.4 ± 4.9 cm; and body fat, 12.7 ± 1.3%. This study was approved by the Quinnipiac University Institutional Review
Board and written informed consent was obtained from all participants prior to their participation.

Procedures

All participants reported for a total of 3 visits: (a) familiarization session, (b) testing session 1, and (c) testing session 2. Visits were separated by 3-7 days. At study entry, each subject was randomly assigned to 1 of 2 groups: EPC at testing session 1 and sham at session 2, or vice versa.

For all sessions, subjects reported to the Quinnipiac University Cardiovascular Laboratory in a rested, hydrated, post-prandial state ($\geq$ 2 hours) and had abstained from caffeine, alcohol and strenuous exercise for at least 48 hours. Participants were also instructed to maintain normal dietary habits throughout the study and replicate their 24-hour diet for subsequent visits. All testing was performed at the same time of day for each subject to control for diurnal variation.

Familiarization Session

At least 72-hours prior to the first testing session, all participants reported to laboratory to become familiarized with the WAnT and expectations for the protocol. At this time, the investigator explained and demonstrated the procedures and instructed the subject through a 30-second WAnT, 3 minutes of active rest (pedaling at 70 rpm against minimal resistance), and initiation of a second WAnT. All subjects were instructed, on the investigators verbal command, to maximally accelerate at the start of the WAnT and maintain a maximal effort throughout the 30-second test.

Testing Sessions
Upon reporting to the laboratory, adherence with dietary and physical activity guidelines was confirmed with each subject. Thereafter, height and body mass (weigh beam scale with height rod; Detecto 439, Webb City, MO USA), body fat (bioelectrical impedance analysis; OMRON HBF306C, Kyoto, Japan), and heart rate (HR; Polar FT1, Polar Electro, Kempele, Finland) were measured. In addition, resting BLa was evaluated via fingertip blood sample collected with a single-use contact-activated lancet. A Lactate Scout portable lactate analyzer (EKF Diagnostics, Cardiff, Wales, UK) was used for BLa measurement. Fingertip capillary blood sampling was performed following cleaning and drying of the site with sterile techniques, using an alcohol swab and gauze pad, respectively.

Following baseline measurements, subjects were treated with EPC or sham for 30 minutes. Thereafter, the cycle ergometer (894E Peak Test Cycle, Monark Exercise AB, Vansbro, Sweden) seat height and position were adjusted for the subject, BLa was measured again, and a 5-minute warm-up period began by pedaling against minimal resistance at ~70rpm. Following the warm-up, subjects were instructed to start the WAnT as described in the familiarization test and the brake weight resistance (7.5% of subject’s body mass) was applied automatically when the subject reached 100 rpm. Immediately following the completion of the first 30-second WAnT (WAnT1), brake weight resistance was removed and the subject was instructed to pedal at ~70 rpm until the second WAnT (WAnT2) commenced 3 minutes following WAnT1. The same procedure was employed following WAnT2 for transition to WAnT3. After completion of WAnT3, subjects were instructed to pedal at ~70 rpm for 1 minute followed by dismount from the bike and translocation to a treatment table for a 30 minute passive rest recovery period.
At exactly 5 minutes following WAnT3, BLa concentration was measured. Moreover, BLa concentration was evaluated 10 and 15 minutes thereafter (15, 30 minutes post-WAnT3).

HR was recorded prior to and immediately following each of the WAnTs, as well as at 5 minute intervals throughout the recovery period. Peak power, mean power, and the fatigue index were measured as parameters of anaerobic cycling performance and recorded through a PC interface. Peak power was calculated as the highest running average of 1 second (Watts), average power was calculated as the mean power of the entire 30-second test (Watts), and the fatigue index was calculated as the percentage change in power output between the first and last five seconds of the 30-second test.

*Treatments (EPC and Sham)*

A dynamic, sequential pulse EPC device (NormaTec, Newton Center, MA, USA) was used for treatments. Briefly, the EPC device consists of two separate “leg sleeves” which contain five circumferential inflatable chambers (arranged linearly along the limb) encompassing the leg from the feet to the hip/groin. The “leg sleeves” are connected to an automated pneumatic pump at which target inflation pressures for each zone and the duty cycle can be controlled. However, the unit is commercially marketed with pre-programmed defaults for the duty cycle and recommended inflation pressure settings. In this study, we chose to use the recovery protocol recommended by the manufacturer which consists of target inflation pressures of ~70mmHg for each chamber. The device and compression protocol has been described in detail previously (Martin et al. 2015b).

The sham treatment condition consisted of 30 minute application of the EPC “leg sleeves” and connection to the pneumatic pump, without actual compression. This protocol was employed to control for any thermogenic effect as heat loss from the legs is likely affected.
lower pressure treatment was not employed for the sham treatment given the potential for even very low pressures having an effect on the study outcomes.

**Statistical Analysis**

Subject HR and BLa concentrations at baseline between treatment conditions were evaluated using paired t-tests. Continuous primary outcome variables were analyzed by two-way repeated measures ANOVA and partial eta squared ($\eta^2_p$) values were calculated as estimates of effect size using Cohen’s guidelines of small, medium and large effects ($\eta^2_p$=0.01, 0.09 and 0.25, respectively) (Cohen 1992). When a significant main effect of time was observed, between time point differences, on average (i.e. collapsing across treatments), were evaluated using paired t-tests with Bonferroni correction for multiple comparisons. Bonferroni corrected p-values are presented in these instances. Paired t-tests and Cohen’s d effect sizes were also used to analyze treatment effects on the overall fatigue index and BLa concentration area under the curve (AUC) with d values of 0.2, 0.5, and 0.8 corresponding to small, moderate and large effect sizes, respectively. AUC analysis using the trapezoidal method was performed for BLa from 5 to 35 minutes of recovery. An alpha level of $P < 0.05$ was required for statistical significance. Data are presented as mean ± SD and [brackets] indicate 95% confidence intervals around the mean. All statistical analyses were performed using SPSS version 23.0 for Windows (SPSS, Chicago, IL, USA).

**RESULTS**

All subjects completed the entire study protocol without incident. No significant differences between trials were observed for HR (66 ± 11 bpm vs. 64 ± 12 bpm, for EPC and sham, respectively; $P=0.597$), or BLa concentrations at baseline (2.0 ± 0.9 mmol/L vs. 1.7 ± 0.8 mmol/L for EPC and sham, respectively; $P=0.529$).
Parameters of anaerobic cycling performance are shown in Figure 1 (panels A-C). A main effect of time ($P<0.001$, $\eta_p^2=0.884$), but no main effect of treatment ($P=0.309$, $\eta_p^2=0.114$) or time*treatment interaction ($P=0.928$, $\eta_p^2=0.008$) was observed for peak power with the consecutive WAnTs. Similarly, a main effect of time ($P<0.001$, $\eta_p^2=0.944$) but no main effect of treatment ($P=0.217$, $\eta_p^2=0.164$) or time*treatment interaction ($P=0.600$, $\eta_p^2=0.055$) was observed for average power. Between time point differences, on average, illustrating the main effect of time for peak power and average power are presented in Figures 1A and 1B, respectively ($P<0.002$ for all significant differences). No main effect of time ($P=0.093$, $\eta_p^2=0.469$), treatment ($P=0.889$, $\eta_p^2=0.052$) or time*treatment interaction ($P=0.908$, $\eta_p^2=0.063$) was observed for the fatigue index during consecutive WAnTs (Figure 1C). Moreover, the overall fatigue index (ratio of peak power during the first 5 seconds of WAnT1 and last 5 seconds of WAnT3; $57.6\pm9.4\%$ vs. $58.1\pm6.3\%$ for EPC and sham, respectively) was not different between treatments ($-0.51\pm9.06\% [-7.00, 5.97]$; $P=0.862$; $d=0.07$)

For HR measured immediately before and following the consecutive WAnTs, a main effect of time ($P<0.001; \eta_p^2=0.957$), but no main effect of treatment ($P=0.475; \eta_p^2=0.058$) or time*treatment interaction ($P=0.647; \eta_p^2=0.041$) was observed (Figure 1D). HR responses during 30 minutes of passive recovery demonstrated a main effect of time ($P<0.001; \eta_p^2=0.975$), but no main effect of treatment ($P=0.141; \eta_p^2=0.225$) or time*treatment interaction ($P=0.180; \eta_p^2=0.134$) (Figure 1E). Between time point differences, on average, illustrating the main effect of time are presented in figures 1D and 1E ($P<0.05$ for all).

BLa concentrations following EPC and sham were $1.99\pm0.71$ and $1.94\pm0.84$ mmol/L, respectively. No significant main effect of time ($P=0.639; \eta_p^2=0.026$), main effect of treatment ($P=0.440; \eta_p^2=0.068$) or time*treatment interaction ($P=0.743; \eta_p^2=0.013$) was observed for
treatment-mediated changes in BLa concentrations at rest (i.e. before WAnT1). For BLa concentrations during 30 minutes of recovery, a main effect of time ($P<0.001; \eta_p^2=0.918$), but no main effect of treatment ($P=0.364; \eta_p^2=0.092$) or time*treatment interaction ($P=0.271; \eta_p^2=0.279$) was observed. Between time point differences, on average, illustrating the main effect of time are presented in Figure 1F ($P\leq0.003$ for all). AUC for BLa concentrations during recovery (9.2 ± 1.6 vs. 9.7 ± 2.0 mmol/L/min for EPC and sham, respectively) were not different during recovery between groups (-0.4 ± 1.5 mmol/L/min [-1.48, 0.63]; $P=0.250$; $d=0.24$).

**DISCUSSION**

Given our prior data demonstrating that EPC increases vascular reactivity at rest (Martin et al. 2015a) and significantly reduces BLa concentrations following repeated WAnTs (Martin et al. 2015b), we hypothesized that EPC pre-conditioning may have potentiated greater metabolite clearance during multiple WAnTs. Notwithstanding, we report that pre-conditioning with EPC does not alter peak BLa concentrations observed following intense exercise, nor the subsequent decline in circulating BLa concentrations during passive recovery. Granted, given the present data set, we cannot determine if BLa production and/or clearance are affected by pre-conditioning with EPC, only that circulating concentrations do not differ between treatment conditions. Ultimately, while reducing BLa concentrations during recovery may be a desired effect, the fact that pre-conditioning with EPC does not alter peak BLa levels following intense exercise suggest that it does not mitigate the exertion-induced increase in BLa. This is a particularly important point when considering that skeletal muscle acidity has been suggested to play a role in preserving and/or improving muscle force (Allen et al. 2008).

Recent studies in athletes have shown nitrate supplementation to increase endurance performance by potentially increasing NO bioavailability and blood flow to working muscle.
(Cermak et al. 2012, Handzlik and Gleeson 2013). To this end, EPC has been shown to enhance blood flow and skeletal muscle NO production (Martin et al. 2015a, Kephart et al. 2015). In addition, multiple models of dynamic compression have been shown to acutely increase intramuscular gene expression patterns related to mitochondrial biogenesis and vascular biology (Sheldon et al. 2012, Kephart et al. 2015). However, herein we report that pre-conditioning with EPC does not acutely improve performance and/or mitigate fatigue during subsequent, repeated WAnTs tests.

Our null performance findings with EPC pre-conditioning may have arisen from multiple factors including: a) performance during repeated WAnTs may largely hinge upon the ATP/PCr bioenergy system and be less reliant on an enhancement in lower limb blood flow, and it is likely that pre-conditioning with EPC does not enhance the former metabolic pathway; b) while application of EPC enhances lower limb vascular reactivity and blood flow, these pre-conditioning effects may be largely negated at the onset of exercise during high intensity muscle contractions; and c) a relatively small sample size with highly variable performance measures resulting in low observed power (<0.25) to detect a treatment effect. Finally, it is important to note that we only investigated peripheral fatigue and the effect of EPC on central factors (e.g. descending motor drive), which may play a role in performance fatigue (Rattray et al. 2015), is largely unknown. Thus, although acute pre-conditioning with EPC does not appear to be effective for improving subsequent repeated supramaximal cycling efforts, future studies should continue to investigate the potential ergogenic effects of EPC in acute and chronic application models.

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CONFLICTS OF INTEREST

Partial support (~50%) for consumable costs associated with this study was provided by NormaTec USA (Newton Center, MA USA) through a contract awarded to the corresponding author (J.S.M.). However, the authors have no conflict of interest to disclose.
REFERENCES


FIGURE LEGENDS

Figure 1. Panels A-D: parameters of anaerobic cycling performance and heart rate (HR) during Wingate tests (WAnT). Following 30 minutes of treatment with external pneumatic compression (EPC), WAnTs were performed consecutively with 3 minutes of active rest between bouts. A) Peak anaerobic power (highest 1 second running average during 30-second WAnT) in Watts, B) average power (mean power for the entire 30-second WAnT) in Watts, C) the fatigue index (percent drop in power from first 5 seconds of the test to the last 5 seconds of the test), and D) HR immediately before or after performance of respective WAnTs. Panels E-F: HR and blood lactate concentrations (BLa) during passive recovery following WAnT3. When a significant main effect of time was observed, post hoc analysis for between time points comparisons was performed using Student’s paired t-tests with Bonferroni correction for multiple comparisons. Time points with different letter superscripts denotes significant between time point difference ($P<0.05$). Values are presented as mean ± SD.
Panels A-D: parameters of anaerobic cycling performance and heart rate (HR) during Wingate tests (WAnT). Following 30 minutes of treatment with external pneumatic compression (EPC), WAnTs were performed consecutively with 3 minutes of active rest between bouts. A) Peak anaerobic power (highest 1 second running average during 30-second WAnT) in Watts, B) average power (mean power for the entire 30-second WAnT) in Watts, C) the fatigue index (percent drop in power from first 5 seconds of the test to the last 5 seconds of the test), and D) HR immediately before or after performance of respective WAnTs. Panels E-F: HR and blood lactate concentrations (BLa) during passive recovery following WAnT3. When a significant main effect of time was observed, post hoc analysis for between time points comparisons was performed using Student’s paired t-tests with Bonferroni correction for multiple comparisons. Time points with different letter superscripts denotes significant between time point difference (P<0.05). Values are presented as mean ± SD.

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