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Research article

Similar Recovery of Maximal Cycling Performance after Ischemic Preconditioning, Neuromuscular Electrical Stimulation or Active Recovery in Endurance Athletes

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Abstract
This study investigated the efficacy of ischemic preconditioning (IPC) on the recovery of maximal aerobic performance and physiological responses compared with commonly used techniques. Nine endurance athletes performed two 5-km cycling time trials (TT) interspersed by 45 minutes of recovery that included either IPC, active recovery (AR) or neuromuscular electrical stimulation (NMES) in a randomized crossover design. Performance, blood markers, arterial O_2 saturation (S_pO_2), heart rate (HR), near-infrared spectroscopy-derived muscle oxygenation parameters and perceptual measures were recorded throughout TTs and recovery. Differences were analyzed using repeated-measures ANOVAs and Cohen's effect size (ES). The decrement in chronometric performance from TT1 to TT2 was similar between recovery modalities (IPC: -6.1 sec, AR: -7.9 sec, NMES: -5.4 sec, $p = 0.84$, ES 0.05). The modalities induced similar increases in blood volume before the start of TT2 (IPC: 13.3%, AR: 14.6%, NMES: 15.0%, $p = 0.79$, ES 0.06) and similar changes in lactate concentration and pH. There were negligible differences between conditions in bicarbonate concentration, base excess of blood and total concentration of carbon dioxide, and no difference in S_pO_2, HR and muscle O_2 extraction during exercise (all $p > 0.05$). We interpreted these findings to suggest that IPC is as effective as AR and NMES to enhance muscle blood volume, metabolic by-products clearance and maximal endurance performance. IPC could therefore complement the athlete's toolbox to promote recovery.

Key words: Blood flow restriction, endurance, lactate, muscle oxygenation, NIRS.

Introduction

Strategies for adequate acute recovery can make the difference between failure and success in many sports situations. This is particularly relevant when athletes have to perform maximal efforts interspersed with short recovery times (<1 h) that limit a complete return to homeostasis (e.g. track cycling or cross-country sprint skiing events) (Barnett, 2006). The inability to maintain subsequent performance is multifactorial (Knicker et al., 2011), but blood flow appears determinant during recovery by optimizing oxygen (O_2) and nutrient delivery and clearing away metabolic by-products from active muscles (Borne et al., 2017; Malone et al., 2014b). Active recovery (AR) is highly used by athletes (Ortiz et al., 2019; Van Hooren and Peake, 2018) and has been reported to improve subsequent performance compared to passive rest (Connolly et al., 2003; Greenwood et al., 2008; Weltman et al., 1977). For example, performing AR at 30% of maximal aerobic power (MAP) between two maximal aerobic efforts reduced blood lactate and led to greater performance in trained cyclists, compared to passive recovery and neuromuscular electrical stimulation (NMES) of quadriceps muscle (Malone et al., 2014a). While AR can readily maintain both the arterial inflow to and venous return from fatigued muscles, two main determinants of a successful recovery strategy, some authors have sugges-ted that the use of a passive approach with similar effects on blood flow could limit intramuscular glycogen store depletion and thereby further improve the overall reco-very process (Barnett, 2006; Monedero and Donne, 2000). The quest to optimize recovery strategies has led to investigations of varied passive modalities that could reproduce the benefits of AR on limb blow flow with the benefits of limiting exercise (Borne et al., 2017; MacRae et al., 2011; Malone et al., 2014a).

Among these recovery interventions, NMES represents an efficient alternative, particularly when the stimulations are executed on the calf muscles, which have been termed the "peripheral venous heart" (Borne et al., 2017; Izumi et al., 2010). For example, both NMES of the calf muscles for 15 min and AR, but not passive recovery, were found beneficial to speed up the return of pH, blood lactate and bicarbonate concentrations to initial values and to maintain running shuttle performance (Bieuzen et al., 2014). Furthermore, NMES performed for ~25-30 min increased calf arterial inflow, measured by plethysmography, and performance recovery between 30-s supramaximal efforts (Borne et al., 2017) and 1000-m kayak time trials (TT) (Borne et al., 2015). The NMES modality can be mixed with other strategies, such as hydration and nutritional intake, and represents an alternative during competitive events where a proper AR protocol is unfeasible, for example due to space or equipment availability. However, NMES suffers from some pitfalls (Barnett, 2006; Borne et al., 2015; Malone et al., 2014b). This recovery modality requires specific equipment that is not always available to large teams, requires drying of the skin, and stimulating electrodes must be worn beneath a skinsuit to be in direct contact with the athlete's skin, which is not practical in some situations. Thus, there is a scope to investigate the efficacy of other non-invasive, affordable and simple modalities for inclusion in the athlete's toolbox for enhanced recovery in order to face varied situations.

Ischemic preconditioning (IPC) is a strong candidate in this respect. This manoeuver involves repeated episodes of muscle ischemia administered via compression of a pressure cuff wrapped proximally around a limb, fol-

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lowed by rapid reperfusion. IPC can acutely improve performance shortly after the manoeuver, particularly during maximal aerobic exercise where the oxidative system is fully taxed (Bailey et al., 2012; Paradis-Deschênes et al., 2018; Salvador et al., 2016). Though the precise mechanisms of action are still under investigation, performance enhancement has been associated with improvements in local vasodilation, blood flow and, ultimately, O_2 uptake (Bailey et al., 2012; Enko et al., 2011; Kilding et al., 2018; Paradis-Deschênes et al., 2016). Clinical studies have also reported slowing acidosis, reduced lactate production, as well as lesser adenosine triphosphate and glycogen depletion during, or after prolonged ischemia preceded by IPC (Andreas et al., 2011; Salvador et al., 2016). Taken together, these results suggest a potential impact of IPC on recovery processes, but this has received very little experimental attention. Four cycles of intermittent bilateral cuff inflation performed before successive 50-m swimming sprints led to better performance 2 h (1.0%) later, compared to a SHAM procedure (Lisboa et al., 2017). In contrast, when IPC was performed after a simulated rugby match, it did not improve performance during an agility T-test and vertical jumps, compared to passive rest (Garcia et al., 2017). To the best of our knowledge, no study has documented this potency during endurance exercise, which could benefit the most from IPC.

This study therefore aimed to investigate the potential of IPC to enhance the recovery of performance and specific physiological responses, including blood markers, arterial O_2 saturation (S_pO_2), heart rate (HR), muscle oxygenation parameters (i.e., blood volume, O_2 extraction, tissue saturation index) during two simulated 5-km TT separated by a short (< 1 hr) resting period. A major limitation of IPC studies is the difficulty to blind participants to the experimental procedure, which involves high pressures, and which often leads to placebo effect. We therefore used a cross-over design to evaluate IPC against two other modalities that also enhance blood flow and performance and that are commonly used by trained athletes, namely active recovery and NMES. We hypothesized that the three modalities would impact muscle blood volume, but that IPC would further increase O_2 uptake and performance during a subsequent maximal effort.

Methods

Ethics approval

The study was approved by the Ethics Committee of the University, and adhered to the principles established in the Declaration of Helsinki. Participants provided written informed consent after being explained the experimental procedures, associated risks and potential benefits.

Participants

Thirteen trained male road cyclists, runners and triathletes volunteered. Two dropped out due to external commitments or injuries unrelated to the study protocol, 2 were excluded due to their maximal O_2 consumption (VO_2max) being < 50.0 $mL \cdot kg^{-1} \cdot min^{-1}$ as an initial selection criterion, and 9 completed the study (mean±standard error, age 26.4 ± 1.6 years; body mass 75.5 ± 3.5 kg; body height 1.80 ± 0.02 m; body fat 9.6 ± 1.4 %; VO_2max 61.9 ± 3.2 $mL \cdot kg^{-1} \cdot min^{-1}$; MAP 397 ± 11 W). Subjects trained on average 8.0 ± 0.6 h/week in an endurance sport at the time of the study and had at least 2 years of training history in their respective sport. A minimal cycling experience was required for runners. All participants were non-smokers, free of health problems and injuries, and did not use any medication or any other tobacco/nicotine products. None of them had previously used IPC or NMES.

Study design

Participants visited the laboratory for two preliminary visits (for a maximal incremental step test and familiarization of the recovery interventions, and for a5-km TT practice) and then for three experimental trials conducted in a randomized crossover design. During all experimental sessions, participants performed two TTs interspersed by one of the three recovery modalities, and measurements were made before, during and after the two TTs. The timeline for every experimental session was as follows**:** 5-min supine rest (near-infrared spectroscopy (NIRS) baseline: BS_{TT1}), 10-min standardized self-paced warm-up, 2-min rest seating on bike, 5-km TT (TT1), 2-min rest seating on bike (recovery measurements every 30 sec), blood samples (Post-TT1), 3-min cool down, 30-min recovery intervention (IPC, AR, NMES), 15-min rest (including blood samples, BS_{TT2}, transition and equipment adjustments), 3-min standardized self-paced re-activation, 2-min rest seating on bike, 5-km TT (TT2), 2-min rest seating on bike (recovery measurements), blood samples (Post-TT2), 3-min cool down, 5 min supine rest and arterial occlusion (AO, see *Near-infrared spectroscopy*). All sessions were performed at the same time of the day to avoid potentially confounding circadian rhythm effects and were separated by a minimum of 3 days to avoid residual fatigue and a maximum of 7 days. Temperature (22.1 ± 0.1°C) and humidity (31.5 ± .2%) were kept constant in the laboratory. Prior to each testing day, participants were asked to record and replicate their dietary intake and physical activity respectively for 24 h and 72 h before testing, vigorous exercise was avoided for 48 h and alcohol and caffeine were refrained from for 24 h. The handlebars and seat settings of each device (Excalibur Sport, Velotron Elite) were individualized and replicated throughout the study.

Experimental protocol

Preliminary testing: During the first visit, resting HR and blood pressure (inclusion criteria: <100 beats per minute, <140/90 mmHg) were recorded in a seated position and baseline characteristics (body height, body mass and body fat) were measured. The percentage body fat was measured by bioelectrical impedance (Tanita TBF-310; Tanita Corp. of America Inc., Arlington). Thigh skinfold thickness (mean 6.4 ± 0.6 mm) and thigh circumference (mean 57.5 ± 1.5 cm) were also measured as there are known factors to respectively influence the penetration of near-infrared light through muscle tissue (see *Near-infrared spectroscopy*) (McCully and Hamaoka, 2000) and the occlusion of arterial blood flow (Loenneke et al., 2012). The percentage body fat was measured by bioelectrical impedance (Tanita TBF-310; Tanita Corp. of America Inc., Arlington Heights,

IL). The participant was then familiarized, for 5 to 10 min per modality, to IPC by progressively inflating blood pressure cuffs to 220 mmHg (1 cycle per lower limb), and to NMES by increasing transcutaneous electrical impulses until visible contraction of the calf muscles. After the familiarization, the participant was positioned on an electromagnetically-braked cycle ergometer (Excalibur Sport, Lode, the Netherlands) for a 2-min baseline in a seated position and a 5 min warm-up at 100 W before the maximal incremental step test (30 W per min until volitional exhaustion) to assess the VO_2max and the MAP. Expired gases were analyzed breath-by-breath throughout the test (Breezesuite, MedGraphics Corp., Minnesota, Saint Paul, USA).

The second preliminary visit started with a 10-min self-paced warm-up during which power output, speed and gear were continuously noted by the experimenter and strictly reproduced thereafter. After a 2-min bike seated rest, the participant performed a 5-km TT**.**

5-km ergometer time trial: The 5-km TTs were executed on a computer-controlled electrically braked cycle ergometer (Velotron Elite, RacerMate, Seattle, WA, USA). The 10-min warm-up before TT1 and the 3-min re-activation phase before TT2 were self-paced and standardized for each athlete, so that power output, speed and gear from the first session were strictly reproduced in every experimental session. This ensured minimum effect of warm-up on primary variables of investigation between trials. Participants were instructed to complete the 5-km distance as quickly as possible and to remain seated, with the distance travelled as the only available information to them. They were strongly verbally encouraged during all exercise protocols and were warned to give a maximal effort in the first TT1 without pacing. The 3-min cool down was performed at a load individually adjusted from the results of their maximal incremental step test (30% MAP) after each TT.

Recovery modalities: The delay between both TTs was timed and reproduced at every session (IPC: 54 ± 1 min; NMES: 54 ± 1 min; AR: 53 ± 1 min). Recovery interventions were specifically applied to replicate as much as possible the procedures commonly employed by athletes in the field and in research studies and were matched for total resting duration (30 min). They were as follows:

1) IPC: In a supine position, non-elastic nylon blood pressure cuffs (WelchAllyn, Skaneateles Falls, NY, USA, width: 21 cm) were positioned around each upper thigh under the gluteal line and rapidly inflated to 220 mmHg for 5 min to prevent arterial inflow. This was repeated three times per limb, alternately, with each compression episode separated by 5 min of reperfusion (cuff release). This protocol has previously been shown to completely occlude vascular arterial inflow (Sabino-Carvalho et al., 2016), alter physiological responses and enhance acute endurance performance (Bailey et al., 2012; Paradis-Deschênes et al., 2018). The intervention lasted 30 min.

2) AR: The participant remained seated on the bike after the 3-min cool down and cycled at 30% MAP for an additional 10 min in order to simulate a practical AR phase (i.e., often <15 min) that athletes would typically perform in the field when two maximal efforts are repeated close to each other (for review see (Van Hooren and Peake, 2018)). Then, they remained seated quietly for 20 min on a bed adjacent to the bike to complete the 30-min period.

3) NMES: In a prone position, two self-adhesive electrodes (Veinopack, Ad Rem Technology, Paris, France, surface 8 x 13 cm) were placed on the medio-central part of both calves and connected to an electrical stimulator (Veinoplus Sport, Ad Rem Technology, Paris, France). The stimulation voltage ranged from 9 to 18 Vpeak (corresponding level selected by the participants in the current study: 27 ± 2) and was adjusted manually depending on participant tolerance (i.e., comfortable sensation) and the investigator (minimal threshold: visible contraction of the calf muscles). The specific stimulation modulation pattern of the Veinoplus Sport automatically changed every 5 min and resulted in 60 to 90 calf muscles contractions per minute for 30 minutes. Although NMES has been applied for varied periods of time, this stimulation pattern has been previously described and successfully used on the calf muscles to optimize venous return and performance in varied recovery studies using maximal endurance exercise including cycling (Bieuzen et al., 2014; Borne et al., 2017; Borne et al., 2015; Izumi et al., 2010).

Instrumentation and measurements analysis

Power output: **T**he power output was continuously recorded from the Velotron ergometer and was averaged over a period of 10 sec leading up to every 250 m.

Near-infrared spectroscopy: A portable spatially resolved, dual wavelength NIRS apparatus (PortaMon, Artinis Medical Systems BV, The Netherlands) was installed on the distal part of the right vastus lateralis muscle belly (approximately 15 cm above the proximal border of the patella), parallel to muscle fibres, to quantify changes in the absorption of near-infrared light by oxy-hemoglobin (HbO_2) and deoxy-hemoglobin (HHb). The skinfold thickness (6.4 ± 0.6 mm) was measured at the site of the application of the NIRS using a Harpenden skinfold caliper (British Indicators Ltd, West Sussex, Great Britain) during the first session, and was less than half the distance between the emitter and the detector (i.e., 20 mm). This thickness allows adequate penetration of near-infrared light into muscle tissue for valid measurements (McCully and Hamaoka, 2000). The device was packed in transparent plastic wrap to protect it from sweat and fixed with tape. Black bandages were used to cover the device from interfering background light. The position of the apparatus on the thigh was marked with an indelible pen for repositioning during the subsequent visit. The pressure cuff used to induce IPC was positioned above the NIRS device and did not affect the placement of the device.

A modified form of the Beer-Lambert law, using two continuous wavelengths (760 and 850 nm) and a differential optical path length factor of 4.95 was used to calculate micromolar changes in tissue [HbO_2], [HHb] and total hemoglobin ([THb] = [HbO_2] + [HHb]). Changes in tissue saturation index (TSI = [HbO_2]/[THb]) were also used as an index of tissue oxygenation since it reflects the dynamic balance between O_2 supply and consumption in the

tissue microcirculation (Ferrari et al., 2004; van Beekvelt et al., 2001). This parameter is independent of near-infrared photon path length in tissue. Before the first TT, one minute of baseline values was analyzed once the signal was stabilized (BS_{TT1}). Muscle oxygenation changes (Δ[HHb], Δ[THb] and ΔTSI) during exercise and recovery were then normalized to this resting baseline in order to exclude the effects of TT1 and recovery on subsequent muscle oxygenation changes. Δ[HHb] was expressed in percentage of the average of the maximal amplitude calculated during the AO (Δ[HHb], %AO). The AO was performed after each experimental session by inflating the cuff on the right thigh at 220 mmHg for ~3–5 min to obtain a physiological calibration of the NIRS signals. The cuff pressure was released after reaching a plateau in the [HHb] and TSI signal. Δ[HHb] was taken as an index of muscle O_2 extraction (van Beekvelt et al., 2002), Δ[THb] as a change in regional blood volume (van Beekvelt et al., 2001), and the difference between BS_{TT1} and BS_{TT2} (ΔBS) as an index of change in muscle oxygenation parameters induced by the recovery modalities. NIRS data were acquired continuously at 10 Hz and were averaged over 10 sec for every 250 m of the TT, and every 30 sec for a 2-min period immediately after exercise. A 10th order zero-lag low-pass Butterworth filter was applied to smooth NIRS signal (Paradis-Deschênes et al., 2018).

Arterial O_2 saturation and heart rate: S_pO_2 and HR, measured from an adhesive forehead sensor secured with a headband connected a pulse oximeter (Nellcor Bedside, Nellcor Inc. Hayward, CA), were recorded every 250 m during TT and every 30 sec for a 2-min period immediately after exercise. This technique has been shown to be in good agreement with hemoglobin O_2 saturation based on arterial blood analysis over the 70-100% range (Romer et al., 2007). The S_pO_2 measured at the forehead is also highly correlated with the O_2 saturation measured by direct arterial blood measurements ($R^2 = 0.90$, $P < 0.0001$) and has significantly lower bias and greater precision for S_pO_2 (0.3 ± 1.5%) and HR (1.8 ± 5.5%) than finger probes in athletes (Yamaya et al., 2002).

Blood sampling: Blood samples (92-µL) were drawn from fingertips using disposable lancets (Safety-Lancet Neonatal, Sarstedt, Germany) at rest at the first experimental session (baseline), and at three additional time points during others testing sessions: 2 min after each TT and immediately after the 30-min period of each recovery modality. Samples were collected into a capillary tube (Epoc ® Care-fill™, Siemens Healthinners, Germany) and immediately transferred into the sample well of a test card (Epoc ® Test Card, Siemens Healthinners, Germany) for analysis with the Epoc® device (Epoc ® Blood Analysis System, Siemens Healthinners, Germany). This device was used to measure pH, carbon dioxide and O_2 partial pressure, concentrations of sodium, potassium, ionized calcium, chloride, glucose, lactate, and hematocrit. Moreover, concentrations of hemoglobin, bicarbonate, total carbon dioxide, arterial blood O_2 saturation, base excess of extracellular fluid, and base excess of blood were calculated in device. Prior to data collection, the analyzer was calibrated according to the manufacturer's specifications (i.e., thermal quality calibration with a buffered aqueous solution).

Perceptual measures: The rate of perceived exertion (RPE) was recorded every 500 m of the TT using the Borg 10-point scale to assess subjective perceived exertion. Recovery intervention was evaluated by two questions immediately after each recovery modality ("How do you rate the efficacy of this recovery intervention?" and 'How did you like this recovery intervention?") by means of a 10-point Likert scale, ranging from 1 (not at all) to 10 (very, very much) (Bieuzen et al., 2014).

Statistical analysis

We evaluated the magnitudes of difference for performance and physiological variables within condition (recovery vs. rest; TT2 vs. TT1). We also evaluated the percentage difference between IPC, AR and NMES for the TT1, the recovery period and the changes between trials (TT2 vs. TT1, ΔTT). Practical significance was evaluated using Cohen's effect sizes (ES) ± 90% confidence limits, and compared to the smallest worthwhile change that was calculated as the standardized mean difference of 0.2 between-subject standard deviations (Batterham and Hopkins, 2006; Hopkins et al., 2009). Standardized effects were classified as small (0.2-0.49), moderate (0.5-0.79) or large (≥0.8) (Hopkins et al., 2009). The effect of IPC was deemed "unclear" if chances of having better/greater and poorer/lower changes in performance and physiological variables were both >5% (Batterham and Hopkins, 2006; Hopkins et al., 2009). All variables were log-transformed before analysis (Hopkins et al., 2009), except for ΔTSI during recovery, [BE(ecf)], and [BE(b)]. Furthermore, the differences in performance and physiological responses across the two time trials and between the three recovery modalities were also analyzed using repeated-measures ANOVA tests (three levels: IPC, NMES and AR). Tukey's HSD post-hoc analyses were used to locate differences among pairs of means when ANOVAs revealed significant *F*-ratio for main and interactive effects. The level of significance was set at $P < .05$. Raw data are reported as mean± SE for clarity.

Results

Performance parameters

Average completion times and individual percentage differences in the two TTs are displayed in Figures 1 and 2, respectively. The calculated smallest worthwhile change for TT time in IPC, NMES and AR equated to 4.4 sec, 5.5 sec and 4.7 sec, respectively. When every recovery modality was examined separately, TT2 was clearly slower than TT1 after IPC (6.1 sec, ES 0.21) and AR (7.9 sec, ES 0.28), whereas the TT1-TT2 change was less than the smallest worthwhile change after NMES (5.4 sec, ES 0.19). Of note is that TT1 was the slowest in NMES (clear difference vs. AR: 2.5 sec, ES 0.21). However, when comparing performance changes between modalities, there was no difference between conditions (IPC vs. AR: ES 0.07, p = 0.84; IPC vs. NMES: ES -0.02, p = 0.97; AR vs. NMES: ES -0.09, p = 0.72).

The power output profiles during the TTs are displayed in Figure 3. There were clear differences within every condition with a lower power output in TT2

compared to TT1. The power output was higher in IPC and AR conditions, compared to NMES, during the first half of the TT1, but mirroring the chronometric performance, there was no difference between conditions for performance changes (IPC vs. AR: ES -0.03, p = 0.95; IPC vs. NMES: ES 0.01, p = 0.99; AR vs. NMES: ES 0.04, p = 0.93.

Physiological responses

Muscle oxygenation values at baseline and changes following TTs and recovery are displayed in Table 1. Figure 4 shows a schematic representation of these values over the course of the trials and recovery. Overall, all modalities increased local blood volume during the recovery period between the TTs, compared to resting baseline, and remained higher during the second TT with no differences between conditions (IPC vs. AR: ES 0.07, p = 0.81; IPC vs. NMES: ES 0.09, p = 0.61; AR vs. NMES: ES 0.02, p = 0.94). Muscle O_2 extraction did not change and there were no differences between conditions.

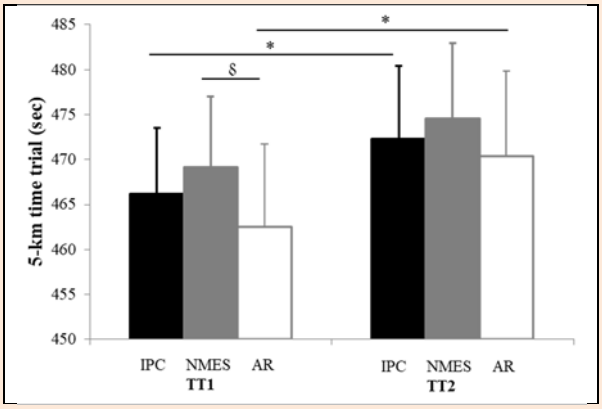

Figure 1. Average completion times during the 5-km TTs interspersed by IPC, AR or NMES. (*) denotes clear differences within conditions when comparing TT2 to TT1 (IPC: 1.3%, ES 0.21; AR: 1.7%, ES 0.28). (§) denotes a clear difference between AR and NMES at TT1 (1.4%, ES 0.23, 0.06;0.40). Values are mean ± SE.

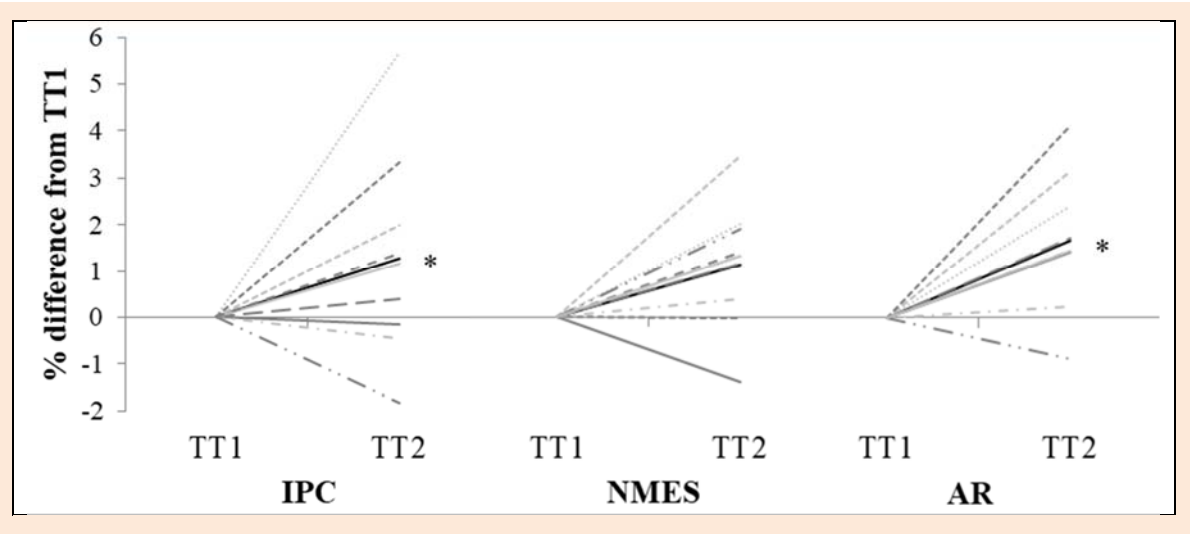

Figure 2. Average (black line) and individual (grey lines) percentage differences between 5-km time trials interspersed by IPC, AR or NMES. (*) denotes a clear difference within conditions at TT2 compared to TT1.

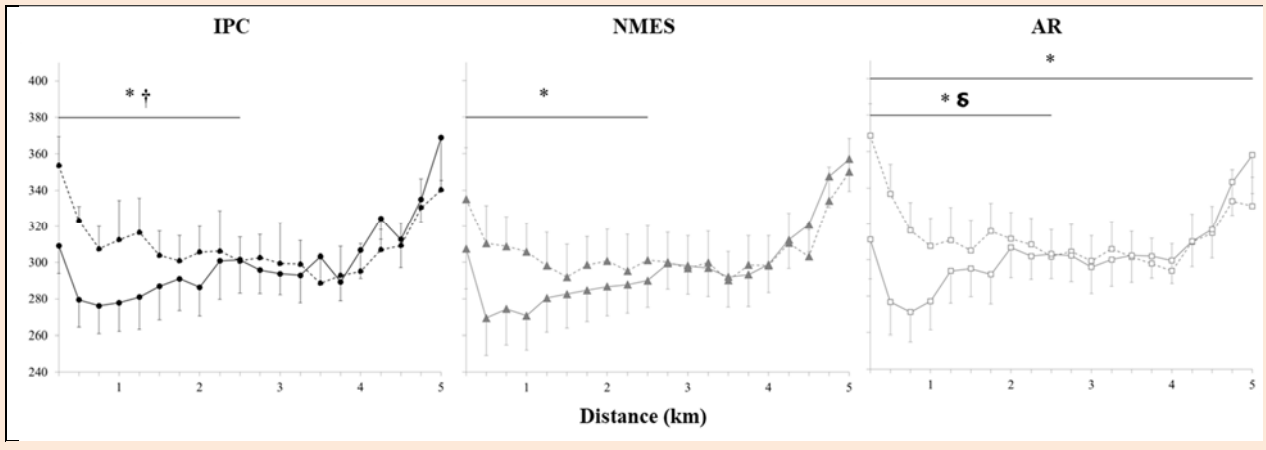

Figure 3. Power output profile during the 5-km time trials (TT1, dotted line; TT2, solid line) interspersed by IPC, AR or NMES (*) denotes clear differences within conditions at TT2 for the first half (IPC: ↓8.2%, ES -0.52 ± 0.27; AR: ↓8.2%, ES -0.52 ± 0.21; NMES: ↓7.2%, ES -0.46 ± 0.28), and the entire TT (AR: ↓3.5%, ES -0.22 ± 0.14), compared to TT1. Clear differences between IPC and NMES (-3.3%, ES -0.20 ± 0.32, †) and AR and NMES (4.7%, ES -0.29 ± 0.32, §) at TT1 are denoted. Values are mean ± SE.

Table 1. Muscle oxygenation at baseline, and changes during time trials and recovery. Values are means (±SE).

	IPC			NMES			AR		
	[HHb], µm	[THb], µm	TSI, %	[HHb], µm	[THb], µm	TSI, %	[HHb], µm	[THb], µm	TSI, %
BS_{TT1}	*29.5 (1.4)‡*	78.7 (3.2)	70.4 (2.3)	30.4 (1.5)	79.4 (3.7)	74.5 (3.3)	31.1 (1.6)	79.7 (4.2)	68.4 (2.5)
BS_{TT2}	30.2 (1.4)	89.2 (3.7)	78.1 (1.9)	30.5 (1.6)	91.4 (4.3)	76.5 (1.2)	30.4 (1.5)	91.1 (4.4)	77.3 (0.9)
	Muscle oxygenation changes during exercise and recovery (Δ)								
	%AO	µm	%	%AO	µm	%	%AO	µm	%
TT1	*101 (3)‡*	10.3 (1.4)	23.4 (2.5)†	96 (7)	9.4 (1.8)	27.1 (2.3)	96 (5)	9.0 (1.7)	24.3 (3.0)
R30	63.9 (5.4)	18.4 (2.4)	2.3 (3.4)†	65.4 (7.0)	18.3 (2.3)	8.7 (4.0)	59.0 (7.2)	17.0 (2.4)	3.4 (3.7)
R60	**38.6 (4.1)‡**	20.1 (2.4)	-5.7 (3.0)	37.3 (5.2)†	21.4 (1.4)	-1.0 (3.1)	29.9 (5.6)	18.2 (2.5)	-5.9 (3.0)
R90	***30.3 (3.9)†‡***	21.1 (2.1)	-7.9 (2.5)†	23.8 (5.4)	19.5 (2.1)	-2.2 (2.7)	19.8 (2.9)	19.5 (1.7)	-7.8 (2.6)
R120	**26.5 (4.0)†‡**	21.4 (2.3)	-7.6 (2.7)†	19.7 (5.6)	19.7 (2.4)	-3.3 (2.5)	14.3 (3.3)	19.5 (1.9)	-8.8 (2.5)
TT2	103 (4)	13.3 (1.6)	22.2 (3.3)	102 (9)	12.9 (2.3)	25.7 (2.8)	98 (4)	13.7 (1.8)	20.9 (2.8)
R30	67.1 (5.5)	20.9 (2.7)	3.2 (3.7)	66.9 (8.4)	22.9 (3.1)	7.1 (3.2)	59.5 (5.2)	21.4 (2.4)	1.8 (3.4)
R60	41.9 (4.8)	25.3 (2.8)	-5.8 (2.8)	35.3 (5.5)	24.5 (2.2)	-1.2 (2.9)	31.4 (5.1)	23.7 (1.6)	-7.0 (2.4)
R90	30.3 (3.7)	24.4 (2.4)	-8.2 (2.7)	28.1 (5.9)	26.0 (2.2)	-2.9 (2.7)	22.9 (3.6)	23.9 (1.8)	-8.6 (2.6)
R120	25.1 (3.6)	23.3 (1.9)	-8.6 (2.4)	22.9 (5.2)	24.4 (1.7)	-3.0 (2.6)	20.1 (4.8)	24.2 (1.2)	-8.5 (2.6)
	% difference within conditions (TT2 compared to TT1)								
ΔBS	*2.1%‡*	13.3%**	**11.1%*****†**	0.1%	15.0%***	3.3%	-2.0%	14.6%***	13.6%***
ΔTT	2.0%	33.8%*	*-9.0%*‡*	5.5%*	55.9%**	-6.7%	3.4%	45.6%*	-16.7%*
ΔR30	4.8%	20.2%*	*34.4%†*	1.8%	17.0%	-20.2%	3.6%	22.3%*	-59.5%
ΔR60	*9.1%†*	*31.2%**†*	-2.3%	-11.2%*	11.9%*	-23.5%	8.7%	68.0%	-17.5%
ΔR90	*9.5%†*	*15.5%*†*	-3.3%	12.0%	38.4%**	-28.0%	14.9%*	23.3%*	-10.3%
ΔR120	**-15.3%*†**	*13.1%*†*	-12.6%	21.3%*	31.6%**	9.5%	15.0%	27.8%*	2.9%

Within-condition clear differences for changes (Δ: TT2 compared to TT1) are denoted as small (0.20-0.49, *), moderate (0.50-0.79, **), or large (≥0.80, ***). Between-condition clear differences for the TT1 and recovery period and for changes are denoted for IPC vs. NMES (†) or IPC vs. AR (‡). For small, moderate or large effects, symbols are presented in *italics*, **bold**, or underlined, respectively. Abbreviations : AR, active recovery; BS, baseline preceding TT ($BS_{TT1,}$ BS_{TT2}); ES, effect size; [HHb], deoxy-hemoglobin; IPC, ischemic preconditioning; NMES, neuromuscular electrical stimulation; R(30 to 120), values after 30 to 120 sec of recovery; [THb], total haemoglobin; TSI, tissue saturation index; TT, time trial.

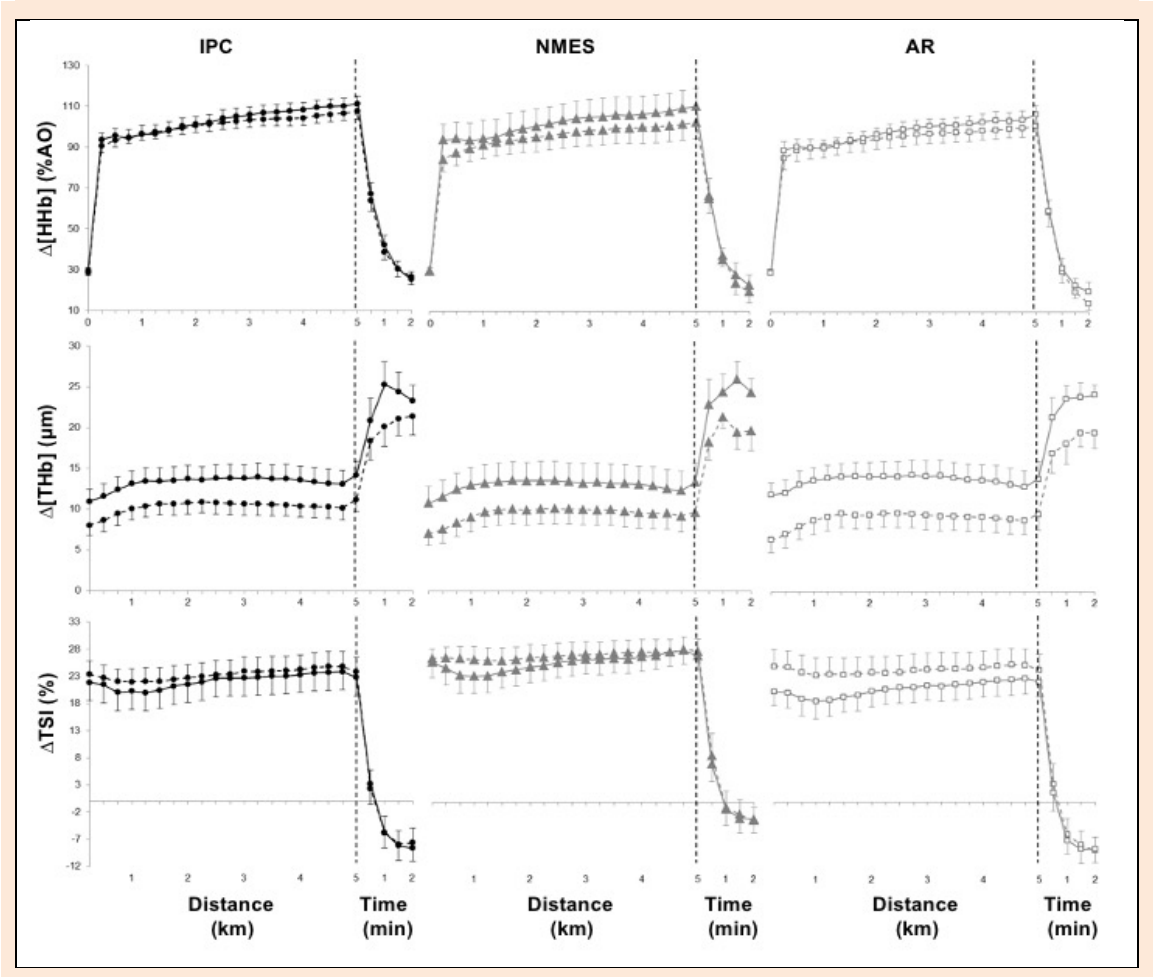

Figure 4. Average Δ[HHb], Δ[THb] and ΔTSI during the 5-km time trials and recovery (TT1, dotted line; TT2, solid line) interspersed by IPC, AR or NMES. Values are mean ± SE.

Table 2. Blood parameters at baseline and after time trials and recovery.

		BS	Post-TT1	Post-recovery	Post-TT2	ΔTT2 vs. ΔTT1
			Mean (SE)		%D within conditions	
pH	**IPC**	7.46 (0.02)	**7.22 (0.01)***** †	**7.44 (0.01)**‡	*7.26 (0.02)**** ‡‡	0.4%*
	NMES		7.28 (0.02)***	7.44 (0.01)	7.31 (0.02)***	0.7%***
	AR		7.24 (0.02)***	7.48 (0.02)*	7.29 (0.02)***	0.7%***
PCO_2 mmol·L^{-1}	**IPC**	34.7 (2.1)	*29.3 (1.7)**** †	33.5 (1.6)	**29.9 (1.2)***** †	*2.9%*‡
	NMES		27.6 (1.8)***	35.8 (1.1)*	27.1 (2.5)***	-4.0%
	AR		28.0 (2.2)***	31.7 (3.1)**	29.8 (1.8)***	7.0%*
PO_2 mmol·L^{-1}	**IPC**	86.8 (7.6)	95.5 (3.8)***	*70.3 (4.7)*** ‡	*99.8 (2.5)**** ‡	4.2%*
	NMES		100.4 (4.0)	66.8 (4.2)***	100.5 (7.3)***	-0.2%
	AR		95.5 (4.5) **	81.0 (7.2)	94.5 (4.2)	-1.0%
[Na^+] mmol·L^{-1}	**IPC**	147 (1)	144 (1)	144 (1)**	**142 (1)***** †	-0.9%*
	NMES		144 (1)*	144 (1)**	144 (1)**	0.0%
	AR		144 (1)**	146 (2)	143 (1)**	-0.1%
[K^+] mmol·L^{-1}	**IPC**	6.98 (0.50)	5.53 (0.41)**	6.26 (0.69)	5.08 (0.39)***	-6.9%
	NMES		5.95 (0.52)*	6.04 (0.70)	6.44 (1.01)	7.2%
	AR		5.80 (0.64)	6.30 (0.67)	5.73 (0.73)*	-2.6%
[$Ca2^+$] mmol·L^{-1}	**IPC**	1.22 (0.02)	1.26 (0.02)	1.19 (0.02)	1.22 (0.01)	-2.3%
	NMES		1.22 (0.03)	1.20 (0.02)	1.23 (0.02)	0.6%
	AR		1.24 (0.02)	1.19 (0.02)***	1.23 (0.02)	-0.6%
[Cl^-] mmol·L^{-1}	**IPC**	113 (2)	112 (1)	112 (2)	110 (1)	-2.5%*
	NMES		113 (1)	110 (1)	113 (3)	-1.2%
	AR		112 (2)	113 (2)	113 (3)	-0.7%
[Glc] mmol·L^{-1}	**IPC**	5.96 (0.30)	*7.14 (0.29)**** ‡	**5.78 (0.31)**†‡	**6.18 (0.36)**†	-10.4%**
	NMES		6.42 (0.48)	6.30 (0.35)	5.60 (0.32)	-15.3%***
	AR		6.94 (0.33)***	6.81 (0.36)**	5.97 (0.43)	-14.6%***
[Lact] mmol·L^{-1}	**IPC**	3.50 (0.50)	14.6 (0.5)***	*4.8 (0.4)**** †	12.5 (0.6)***	-11.8%*
	NMES		14.3 (0.7)***	4.1 (0.5)***	12.0 (0.8)***	-16.3%**
	AR		14.6 (0.6)***	4.1 (0.5)***	12.4 (1.0)***	-16.6%**
Hct %	**IPC**	48.1 (1.3)	**48.9 (0.8)**†	44.0 (1.3)***	**49.0 (1.0)**†	-0.4%
	NMES		49.8 (1.3)	44.6 (1.2)***	51.6 (1.6)**	3.5%*
	AR		49.3 (1.0)	44.9 (1.2)***	49.1 (0.6)	-0.2%
[HCO_3-] mmol·L^{-1}	**IPC**	24.3 (0.8)	12.0 (0.8)***	*22.7 (0.6)** †	13.3 (0.9)***	*9.9%*** ‡
	NMES		12.9 (1.0)***	24.2 (0.3)	13.6 (1.3)***	8.6%*
	AR		11.9 (0.6)***	22.8 (1.1)**	14.3 (0.9)***	19.2%***
[$cTCO_2$] mmol·L^{-1}	**IPC**	25.3 (0.9)	12.9 (0.8)***	*23.8 (0.6)** †	14.2 (1.0)***	*9.6%*** ‡
	NMES		13.7 (1.0)***	25.3 (0.4)	14.4 (1.4)***	7.7%
	AR		12.8 (0.7)***	23.8 (1.2)**	15.2 (0.9)***	18.2%***
[$Be_{(ecf)}$] mmol·L^{-1}	**IPC**	0.4 (0.8)	-15.6 (0.9)***	**-1.4 (0.5)*** †	*-13.9 (1.2)**** †‡	*-10.6%*** ‡
	NMES		-14.0 (1.2)***	0.1 (0.4)*	-12.7 (1.5)***	-14.6%**
	AR		-15.4 (0.6)***	-0.8 (0.7)**	-12.3 (1.1)***	-21.3%***
[$Be_{(b)}$] mmol·L^{-1}	**IPC**	1.0 (0.6)	-14.1 (0.8)***	**-0.6 (0.4)**** †	***-12.5 (1.2)****** †‡	*-11.0%*** ‡
	NMES		-12.2 (1.1)***	0.4 (0.4)*	-10.7 (1.3)***	-18.0%***
	AR		-13.7 (0.6)***	0.2 (0.4)*	-10.7 (1.1)***	-21.8%***
cSO_2 %	**IPC**	96.2 (0.7)	95.7 (0.5)	93.6 (1.3)**	*96.5 (0.4)** ‡	0.8%*
	NMES		96.8 (0.5)	92.9 (1.2)***	96.8 (0.5)	-0.2%
	AR		95.8 (0.7)	95.0 (2.0)	96.2 (0.7)	0.4%
[cHgb] g·dL^{-1}	**IPC**	16.4 (0.4)	*16.7 (0.3)*†	14.9 (0.4)***	**16.7 (0.3)**†	-0.9%
	NMES		17.0 (0.5)	15.1 (0.4)***	17.5 (0.6)**	3.1%
	AR		16.8 (0.3)	15.3 (0.4)***	16.8 (0.2)	-0.4%

Within-condition clear differences at time point (post-TT1, post-recovery, post-TT2), compared to baseline, and for changes (Δ: TT2 compared to TT1) are denoted as small (0.20-0.49, *), moderate (0.50-0.79, **), or large (≥0.80, ***). Between-condition clear differences at time point, compared to baseline, and between changes are denoted for IPC vs. NMES (†) or IPC vs. AR (‡). For small, moderate or large effects, symbols are presented in *italics*, **bold**, or underlined, respectively. Abbreviations: AR, active recovery; $Be_{(b)}$, $Be_{(ecf)}$, base excess of blood and extracellular fluid; BS, baseline; Ca^{2+}, ionized calcium; HCO_3^-, bicarbonate; cHgb, haemoglobin; Cl^-, chloride; cSO_2, arterial blood O_2 saturation; $cTCO_2$, total CO_2; %D, percentage difference; glc, glucose; Hct, hematocrit; IPC, ischemic preconditioning; K^+, potassium; lact, lactate; NMES, neuromuscular electrical stimulation; PCO_2 and PO_2, CO_2 and O_2 partial pressure.

Mean values of blood parameters at rest and immediately after TTs and recovery are displayed in Table 2. There were some clear differences between changes in IPC and AR. Specifically, differences between TT2 and TT1 were lower after IPC compared to AR, but not significant, for carbon dioxide partial pressure (ES 0.28 ± 0.38, p = 0.85), total carbon dioxide (ES 0.42 ± 0.52, p = 0.25), and concentrations of bicarbonates (ES 0.45 ± 0.56, p = 0.81) and base excess of blood (ES 0.36 ± 0.56, p = 0.43) and extracellular fluid (ES 0.38 ± 0.48, p = 0.28). However, no

Table 3. Heart rate and arterial O_2 saturation following time trials and during recovery. Values are means (±SE).

	IPC		NMES		AR	
	Heart rate and arterial O_2 saturation values					
	HR (bpm)	S_pO_2 (%)	HR (bpm)	S_pO_2 (%)	HR (bpm)	S_pO_2 (%)
TT1	175 (3)	96.8 (0.5)	175 (4)	96.9 (0.5)	176 (3)	96.5 (0.6)
R30	164 (3)	*96.9 (0.5) b*	163 (5)	96.7 (0.5)	165 (5)	96.4 (0.5)
R60	142 (4)	97.8 (0.5)	142 (5)	97.6 (0.4)	140 (4)	97.7 (0.5)
R90	124 (4)	98.1 (0.4)	124 (5)	98.2 (0.4)	126 (4)	97.9 (0.5)
R120	117 (5)	*98.2 (0.4) b*	115 (4)	98.2 (0.4)	119 (5)	97.9 (0.6)
TT2	171 (4)	97.8 (0.4)	172 (4)	97.8 (0.4)	171 (3)	97.6 (0.6)
R30	166 (5)	97.0 (0.5)	167 (4)	97.4 (0.5)	167 (5)	97.8 (0.5)
R60	144 (5)	98.0 (0.5)	143 (3)	98.6 (0.4)	147 (4)	98.3 (0.4)
R90	125 (5)	98.2 (0.5)	125 (4)	98.3 (0.4)	127 (5)	98.0 (0.4)
R120	118 (6)	98.4 (0.4)	111 (7)	98.4 (0.4)	118 (6)	98.4 (0.4)
	% difference within conditions (TT2 compared to TT1)					
ΔTT	-2.7% *	1.0% **	-1.7% *	0.9% *	-3.0% *	1.1% **
ΔR30	0.8%	***0.1% a*b**	2.6% *	0.8% **	1.1%	1.2% **
ΔR60	*1.3% b*	**0.2% a**	1.3%	1.0% **	4.6% *	0.5% *
ΔR90	0.5%	0.1%	0.9%	0.1%	0.8%	0.3%
ΔR120	0.4%	0.2% *a*	-4.4%	0.2%	0.6%	0.6% *

Within-condition clear differences for changes (Δ: TT2 compared to TT1) are denoted as small (*), moderate (**), or large (***). Between-condition clear differences for the first TT and recovery period and for changes are denoted for IPC vs. NMES (a) or IPC vs. AR (b). For small (0.20-0.49), moderate (0.50-0.79) or large (≥0.80) effects, symbols are presented in *italics*, **bold**, or underlined, respectively. Abbreviations: AR, active recovery; bpm, beat per minute; ES, effect size; HR, heart rate; IPC, ischemic preconditioning; NMES, neuromuscular electrical stimulation; R(30, 60, 90, 120), values after 30, 60, 90 or 120 sec of recovery; S_pO_2, arterial O_2 saturation; TT, time trial.

approach appears to provide a systematically greater benefit in the recovery of these measures overall, all appearing to allow for a rapid clearance of metabolic by-products. In all conditions, values remained within their normal clinical range at all times.

Table 3 shows changes to HR and S_pO_2 values during TTs and recovery. Overall, all modalities decreased HR and increased S_pO_2 during the second TT, compared to TT1, with no differences between conditions.

Perceptual measures

Mean RPE was not different within conditions (TT2 compared to TT1) and there was no difference between the three recovery modalities. There were, however, clear differences between conditions for the perception of efficacy (IPC: 8.3 ± 0.4; NMES: 7.6 ± 0.6; AR: 6.8 ± 0.8) and appreciation of the technique (IPC: 6.6 ± 0.5; NMES: 6.9 ± 0.8; AR: 7.9 ± 0.5), with clearly higher scores for IPC (compared to AR: ES 0.74 ± 0.79; NMES ES 0.31 ±0.26) and AR (IPC: ES 0.65 ± 0.60; NMES: ES 0.59 ± 0.81), respectively.

Discussion

This study examined the effects of IPC administered during recovery on subsequent endurance performance and associated physiological responses compared to two other recovery modalities commonly used by athletes in the sporting field (active recovery and neuromuscular electrical stimulation). The main practical finding was that the performance decrement in a second 5-km cycling time trial repeated after less than an hour of recovery was similar in the three modalities. IPC did not outperform AR nor NMES in preserving endurance performance. The increase in muscle blood volume and metabolic by-products clearance after recovery, as well as the physiological responses (i.e. muscle O_2 extraction, HR and S_pO_2) during the second 5-km TT were also similar in the three modalities.

IPC has been reported to improve acute performance shortly after the procedure in various contexts especially when the oxidative system contributes greatly to the energy provision (Bailey et al., 2012; Paradis-Deschênes et al., 2018; Salvador et al., 2016). However, studies where IPC is applied during recovery are scarce, do not include prolonged efforts and present conflicting results. For example, IPC performed after a simulated rugby match did not improve performance on an agility T-test and vertical jumps, compared to passive rest (Garcia et al., 2017). On the other hand, when compared to SHAM protocols, IPC induced beneficial effects on 50-m swimming sprints 2 and 8 h later (Lisboa et al., 2017) and on power production and sprint performance immediately and 24 h after IPC (Beaven et al., 2012), emphasizing the potential of this technique for recovery and the need for further investigations. Within the current cross-over randomised design, the performance decrement from one TT to the other was similar between the three recovery modalities (IPC: -6.1 sec; NMES: -5.1 sec; AR: -7.9 sec, p=0.84, ES 0.05), indicating that none of the tested strategies was more efficient than the others in these trained endurance athletes. The decrease in power output occurred mostly in the first half of the TT (IPC: -24 W; NMES: -21 W; AR: -25 W) and was not different between modalities despite participants' perception of efficacy and preference for IPC and AR, respectively. Corresponding to this reduce work rate, participants displayed higher S_pO_2 and lower HR and changes in blood markers after the second TT. NMES was the only condition with no clear within-group difference between TT1 and TT2 (ES 0.19). However, it is important to mention that TT1 was also the slowest in this condition (clearly different from AR for completion time and from AR and IPC for power output developed in the first half of the TT), likely leading to this statistical artifact.

One of the main pitfalls of IPC research is the difficulty to blind participants to the high cuff pressure exerted

on the limb. Thus, to avoid a potential placebo effect derived from the intervention and to reduce the cumbersomeness of the protocol, we opted to not include a SHAM condition, with a cuff inflated at ~20 mmHg as done in many IPC studies, or a fourth "true" control condition with passive rest between the two TTs. Instead, we chose to compare IPC to two other modalities that are commonly employed by athletes in training and competitive settings and whose recovery benefits have been robustly demonstrated and, like IPC, are also mainly derived from blood flow improvement (Barnett, 2006; Bieuzen et al., 2014; Borne et al., 2017; Greenwood et al., 2008; Malone et al., 2014b; Monedero and Donne, 2000; Weltman et al., 1977). Although it is difficult to ascertain whether IPC would really improve subsequent performance, the present design compares its potential efficacy against proven ergogenic methods, which were ultimately used as "practical controls". In fact, athletes do not use passive rest to accelerate the recovery of physiological responses and performance in real sport settings (Van Hooren and Peake, 2018), which makes the conclusion of the current study more relevant and applicable. Therefore, we interpreted these findings to suggest that IPC was at least as efficient in maintaining maximal endurance performance as the two other recovery modalities, which adds to the current literature on the efficacy of this technique for aerobic exercise when the choice of a particular modality is limited by space, equipment availability or other contextual reasons.

The similar effects of the three recovery modalities on performance could be partly explained by their equivalent effect on the increase of local blood volume (↑ΔBS, ~14%), suggesting an enhancement of muscle perfusion immediately before the second TT. IPC has been reported to improve vasodilation and blood flow at rest (Enko et al., 2011; Kraemer et al., 2011; Paradis-Deschênes et al., 2016), and NMES and AR are typically used in recovery for their "muscle pump effect" on the vascular system resulting from low-frequency stimulation and voluntary muscle contractions, respectively (Bangsbo et al., 1994; Borne et al., 2017; Grunovas et al., 2007; Layec et al., 2008). For example, Borne et al. reported a greater increase in calf arterial inflow after NMES (~243%), compared to passive rest (~66%), and this was positively correlated with performance (Borne et al., 2017). This NMES-derived hyper-perfusion response has also been demonstrated to reduce the muscle blood flow limitations before exercise or the spatial heterogeneities within the active muscles during exercise (Layec et al., 2008). Thus, despite the fact that NIRS does not offer a robust assessment of blood flow since it does not detect change in blood velocity (DeLorey et al., 2003), the increase in local blood volume in the present study is in accordance with previous studies and suggests enhanced perfusion following all three modalities.

The higher local blood volume in the quadriceps muscle following all three modalities did not transfer into higher muscle O_2 extraction (estimated non-invasively through [HHb] changes), which is in agreement with some, but not all, studies. Indeed, IPC accelerated muscle deoxygenation dynamics and enhanced performance during whole-body cycling (Kido et al., 2015), and increased muscle perfusion and O_2 uptake in strength-trained athletes, albeit during maximal contractions (Paradis-Deschênes et al., 2016). However, IPC applied in hypoxia before 2 repeated 5-km cycling TTs, compared to SHAM, did not improve the TT performed immediately after the intervention, but prevented the performance decrement 2 h later, probably through greater O_2 extraction (da Mota et al., 2019). This is in accordance with the delayed positive effect of IPC observed on sprint swimming performance at sea level (Lisboa et al., 2017). Thus, one may argue that there may exist a minimum time delay for the IPC-derived effects (e.g., improved tissue perfusion and metabolism) to come into play and enhance performance. The timing of the IPC procedure before a subsequent exercise and the delayed physiological effects should be investigated in future studies. It could also be argued that the subsequent TT in this study could have been limited by factors other than O_2 delivery and consumption (Amann, 2011; Knicker et al., 2011).

Maintaining blood flow after exercise is also paramount to remove metabolic by-products (such as hydrogen ions, inorganic phosphate) produced during high-intensity exercise from the contraction sites and to convert lactate back to glucose (Ament and Verkerke, 2009; Borne et al., 2017; Malone et al., 2014b; Neric et al., 2009). The ergogenic effects of AR and NMES in that regards have already been reported (Bieuzen et al., 2014; Borne et al., 2017) and clinical studies on IPC have also reported slowing of acidosis and reduced lactate production (Andreas et al., 2011; Salvador et al., 2016). However, experimental effects may be more easily detected when compared to a control condition with passive rest. In the present study, IPC was compared to modalities that were reported to be ergogenic on several occasions and, hence, displayed neither beneficial nor detrimental effects on blood markers during recovery and subsequent exercise. All TTs induced metabolic acidosis, and not surprisingly, a decrease in base excess, with no differential impact of the recovery modality. Moreover, despite some marginal differences between modalities after 30 min of recovery, values for pH (7.44 to 7.48) and the concentrations of lactate (4.1 to 4.8 $mmol \cdot L^{-1}$), bicarbonates (22.7 to 24.2 $mmol \cdot L^{-1}$) as well as the base excess of blood (-0.6 to 0.4 $mmol \cdot L^{-1}$) were within the normal clinical range. Studies including more frequent blood samples during recovery are warranted to evaluate potential differences between modalities in the time course of recovery. These results combined with the fact that the performance decrement was not different between conditions, suggested that the three modalities allowed an equivalent return to resting baseline conditions.

Conclusion

This study demonstrated that IPC enhanced muscle blood volume and metabolic by-product clearance and maintained muscle oxygenation and performance during a second 5-km time trial in endurance-trained athletes to the same extent as active recovery and neuromuscular

electrical stimulation. Thus, IPC may represent an affordable and easy technique for athletes when the choice of a recovery strategy is limited by practical and/or meteorological considerations.

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Key points

- The impact of IPC on the recovery of maximal endurance performance and physiological responses to exercise is unknown.
- IPC appears as effective as active recovery and neuromuscular electrical stimulation to enhance muscle blood volume and metabolic by-products clearance.
- The performance decrement in a second 5-km cycling time trial repeated after less than an hour of recovery is similar in the three recovery modalities.
- Ischemic preconditioning may complement the athlete's toolbox to promote recovery in particular settings.

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