

The Effect of Ischemic Preconditioning on Repeated Sprint Cycling Performance

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ABSTRACT

PATTERSON, S. D., N. E. BEZODIS, M. GLAISTER, and J. R. PATTISON. The Effect of Ischemic Preconditioning on Repeated Sprint Cycling Performance. *Med. Sci. Sports Exerc.*, Vol. 47, No. 8, pp. 1652–1658, 2015. **Purpose:** Ischemic preconditioning enhances exercise performance. We tested the hypothesis that ischemic preconditioning would improve intermittent exercise in the form of a repeated sprint test during cycling ergometry. **Methods:** In a single-blind, crossover study, 14 recreationally active men (mean \pm SD age, 22.9 ± 3.7 yr; height, 1.80 ± 0.07 m; and mass, 77.3 ± 9.2 kg) performed twelve 6-s sprints after four 5-min periods of bilateral limb occlusion at 220 mm Hg (ischemic preconditioning) or 20 mm Hg (placebo). **Results:** Ischemic preconditioning resulted in a $2.4\% \pm 2.2\%$, $2.6\% \pm 2.7\%$, and $3.7\% \pm 2.4\%$ substantial increase in peak power for sprints 1, 2, and 3, respectively, relative to placebo, with no further changes between trials observed for any other sprint. Similar findings were observed in the first three sprints for mean power output after ischemic preconditioning ($2.8\% \pm 2.5\%$, $2.6\% \pm 2.5\%$, and $3.4\% \pm 2.1\%$, for sprints 1, 2, and 3, respectively), relative to placebo. Fatigue index was not substantially different between trials. At rest, tissue saturation index was not different between the trials. During the ischemic preconditioning/placebo stimulus, there was a $-19.7\% \pm 3.6\%$ decrease in tissue saturation index in the ischemic preconditioning trial, relative to placebo. During exercise, there was a $5.4\% \pm 4.8\%$ greater maintenance of tissue saturation index in the ischemic preconditioning trial, relative to placebo. There were no substantial differences between trials for blood lactate, electromyography (EMG) median frequency, oxygen uptake, or rating of perceived exertion (RPE) at any time points. **Conclusion:** Ischemic preconditioning improved peak and mean power output during the early stages of repeated sprint cycling and may be beneficial for sprint sports. **Key Words:** ISCHEMIA, OCCLUSION, POWER OUTPUT, MULTIPLE SPRINT, FATIGUE

Ischemia-reperfusion injury underpins the damage caused by either disease and/or deliberately imposed interruption of blood supply to tissues. However, since 1986, brief and repeated bouts of ischemia/reperfusion, known as ischemic preconditioning, have been demonstrated to protect many organs, including the myocardium (32), liver (35), and skeletal muscle (21) from the damage caused by a subsequent prolonged ischemic event. In addition to the clinical use of ischemic preconditioning, this technique has also been applied immediately before exercise to improve performance. Across a range of various exercise modes, performance has been enhanced by 1%–8% (3,12,13,24,25), which makes it potentially beneficial for athletic events where such small margins are the difference between winning or losing.

Research to date has primarily focussed on events of an endurance nature and has identified improvements in peak oxygen uptake ($\dot{V}O_{2\max}$; 13), power output at $\dot{V}O_{2\max}$ (12), running time trial performance (3), 1000-m rowing

performance (25), and time to task failure (7) after ischemic preconditioning. Relatively little research has focused on performance during shorter durations, and the findings are conflicting. For example, an improvement in 100-m swimming performance was observed in elite national level swimmers (24), but no effect of ischemic preconditioning was demonstrated on single 30-m running sprint performance (19) or cycling exercise at $130\% \dot{V}O_{2\max}$ (12).

Repeated sprint exercise provides a model to investigate transitions from high to low metabolic work, a common feature of many team sports. The major energy demands of repeated sprint exercise are derived from phosphocreatine (PCr) and anaerobic glycolysis (18), and recent work suggests a strong relationship between PCr resynthesis and recovery of repeated sprint performance (31). Alternatively, there is an increased reliance on aerobic energy production during the latter stages of repeated intense exercise as evidenced by a larger reduction in anaerobic energy production than performance (18,30) and increased muscle oxygen uptake (6). Furthermore, reducing (5) or enhancing (4) oxygen availability during repeated exercise impaired or enhanced performance, respectively, which suggests that the aerobic system plays an important role, possibly through faster PCr resynthesis.

Ischemic preconditioning may improve aerobic metabolism as evidenced by increased $\dot{V}O_{2\max}$ (13), accelerated $\dot{V}O_2$ kinetics (34), and improved oxygenation of skeletal muscle (38); and it may therefore reduce the performance

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related decline in power output associated with repeated sprint exercise. Second, in ischemic reperfusion injury models, ischemic preconditioning enhances PCr resynthesis after ischemia (1,29) and thus may enhance the ability to recover between sprints. Therefore, the aim of this study was to investigate the effect of ischemic preconditioning on repeated sprint cycling performance. Given the apparent ability of ischemic preconditioning to improve aerobic metabolism and promote PCr resynthesis, it was hypothesized that it would improve repeated sprint cycling performance by reducing the rate of fatigue.

METHODS

Participants

In a randomized, single-blind, crossover study, 14 healthy males (mean \pm SD age, 22.9 \pm 3.7 yr; height, 1.80 \pm 0.07 m; mass, 77.3 \pm 9.2 kg) recreationally active in repeated sprint sports such as field hockey, soccer, and rugby volunteered to participate. Participants were naive to the effect of ischemic preconditioning on exercise performance and were not informed about the rationale of the study. They were fully informed of all procedures and associated risks before completing a training history questionnaire and providing written informed consent. Participants reported they had actively been involved in sport for an average of 12 yr, with time spent training each week reported as 6.7 \pm 2.3 h. Approval for the study's procedures was granted by St Mary's University Ethics Committee, which conformed to the Declaration of Helsinki.

Experimental Overview

All participants reported to the laboratory for four exercise trials. In the initial trial, data were obtained on individual anthropometric characteristics such as body mass, height, and four skinfolds (subscapular, biceps brachii, triceps brachii, and iliac crest). During this trial, the participants were familiarized with the repeated sprint cycling protocol, consisting of twelve 6-s cycle sprints with 30 s of passive recovery between each sprint. Trial 2 was a repeat of the first, to further familiarize the participants with the exercise protocol. Trials 3 and 4 were experimental trials that consisted of either ischemic preconditioning or placebo treatment before the exercise protocol. The experimental trials were performed in a counterbalanced manner, separated by 5–7 days to ensure no possible carryover of acute ischemic preconditioning (28). During both trials, respiratory gas exchange, electromyography (EMG) of the vastus lateralis (VL), and near-infrared spectroscopy (NIRS) of the VL were recorded. The participants indicated their rating of perceived exertion (RPE, 6–20; Borg scale), and blood was taken from the earlobe at rest and after sprints 4, 8, and 12 before being subsequently analyzed for lactate. The participants performed all of their trials at the same time of day (\pm 1 h), and laboratory conditions were controlled at

approximately 20°C and 38% relative humidity during all trials. The participants were instructed to maintain their normal diet, to refrain from any form of intense physical activity and caffeine for the 24 h period before testing, and not to eat for 3 h before each trial.

Experimental Measures

Ischemic preconditioning. In trials 3 and 4, exercise was preceded by ischemic preconditioning or placebo, performed in a supine position using bilateral occlusion (3,13). In the ischemic preconditioning trial, automatic occlusion cuffs (14.5-cm width; Delfi Medical Innovations, Vancouver, Canada) were positioned proximally around the thigh and inflated to 220 mm Hg for 5 min followed by 5 min of reperfusion. This procedure was repeated four times (3). The placebo trial was identical to the ischemic preconditioning trial except that the cuffs were inflated to 20 mm Hg. The time delay between the cuff removal and the beginning of the warm up for the exercise test was 30 min, as ischemic preconditioning has been demonstrated to improve exercise performance within 45 min of the final cuff inflation (3).

Repeated sprint cycling. The exercise protocol consisted of twelve 6-s sprints with resistance set at a torque factor of 1.0 N·m·kg⁻¹ on a cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands), with individual participant cycling position being established during visit 1 and then replicated on each subsequent visit. The participants performed a standardized warm-up, consisting of 3 min of cycling at 120 W, followed by two maximal 6-s sprints, with 1 min between efforts followed by 5 min of passive rest. Toe clips were used to secure the feet to the pedals, and strong verbal encouragement was provided throughout each trial. The participants performed each sprint with the pedals in the same starting position and were instructed to sprint as fast as possible, maintaining maximal effort until asked to stop. Each sprint was initiated by illuminating a series of 20 light-emitting diodes (LEDs), which were synchronized with the EMG recording. During the 30-s rest period after each sprint, the participants remained seated on the ergometer. Mean and peak power outputs were calculated for each condition. The percentage decrement score (S_{dec}) for all 12 sprints was calculated as the percent difference between total and ideal peak power outputs, where total power represents the sum of peak power values from all sprints (S_n , where $n = 1:12$) and ideal power represents the number of sprints multiplied by the highest peak power (S_{best}) achieved (20).

$$S_{dec}(\%) = \left[1 - \frac{S_1 + S_2 + \dots + S_{12}}{12S_{best}} \right] \times 100$$

Cardiorespiratory measures. Respiratory gas exchange was measured during the entire exercise protocol through breath-by-breath analysis using an open spirometric system (Oxycon Pro, Jaeger, Hoechburg, Germany). The gas analyzer was calibrated before each trial using oxygen and carbon dioxide gases of known concentrations

(Cryoservice, Worcester, UK), and the turbine volume transducer was calibrated using a 3-L precision syringe (Hans Rudolph Inc, Shawnee, KS USA). During the trials, the participants breathed room air through a facemask (Hans Rudolph, Kansas City, MO, USA) that was secured in place by a head-cap assembly (Hans Rudolph, Kansas City, MO, USA). Respiratory gas exchange data were subsequently averaged on a 1-s basis and then averaged for the overall exercise protocol, so that the total time of analysis was 432 s ($12 \times (6\text{-s sprint} + \text{the following } 30\text{-s recovery periods})$).

Muscle EMG. The EMG activity of the VL muscle of the right leg was recorded at 1000 Hz using a data acquisition system (Biopac MP150, Biopac Systems Inc, CA, USA). Before placement of the electrodes, the overlying skin was prepared. The hair was shaved and the skin thoroughly cleaned with alcohol to reduce skin electrode interference. Pregelled disposable hypoallergenic 1-cm snap electrodes (Performance Plus, Vermed, VT, USA) were fixed two thirds of the distance along a line from the anterior spina illaca superior to the lateral side of the patella (17). Electrode centers were placed 2.0 cm apart, parallel to the direction of muscle fibers, with a reference electrode located above a prepared site on the shaft of the tibia. The EMG electrode placement was marked on the skin by an indelible pen to ensure similar placement of electrodes between experimental trials. Electromyographic recording was initiated by a digital trigger coincident with the start of each 6-s sprint. The start of each sprint was identified from the square wave pulse provided by the synchronization trigger, and the subsequent 6 s of data were used for the analysis of each individual sprint. The raw EMG data were band pass filtered to remove the signal outside the 20- to 500-Hz range. To investigate the difference in VL EMG frequency between the two conditions, the filtered EMG data from each sprint were transformed in to the frequency domain using a fast Fourier transformation, and the median frequency (MDF) of the resulting power spectrum density was calculated. The MDF values from each of the 12 sprints were then analyzed using linear regression, and the gradient of this line was extracted as a representation of the change in frequency (fatigue) across the 12 sprints (33).

NIRS measurements. During the experimental trials, muscle oxygenation of the left VL was continuously monitored using portable NIRS apparatus, which is a wireless spatially resolved dual-wavelength spectrometer (Portamon, Artinis Medical Systems, BV, The Netherlands). Changes in tissue saturation index (TSI, expressed as %) were measured using two wavelengths (750 and 850 nm), using an arbitrary value for the differential pathlength of 3.83 (10). During rest and before the preconditioning procedure, a measure of TSI was taken. During the preconditioning and placebo procedures, TSI was averaged over the duration of each 5-min period of ischemic preconditioning and the value used was for the portion of time the cuff was inflated only (4×5 min of pressure). During the repeated

sprint cycling protocol, TSI was calculated as an average across all the sprints and recovery time, in a similar manner to oxygen uptake data earlier described. The NIRS device was positioned on the left VL using the same procedures described earlier for the EMG placement (for the opposite leg). As with EMG placement, an indelible pen was used to mark the placement of the device and to ensure similar placement between trials. The NIRS device was covered with a black light-absorbing cloth to prevent contamination from ambient light. During all tests, the NIRS device was connected to a personal computer by Bluetooth for data acquisition (10 Hz). Skinfold thickness was measured at the site where the NIRS probe was attached before each trial using Harpenden skinfold calipers (British Indicators Ltd, UK). For all participants, the calculated value of skin and subcutaneous tissue thickness was less than half of the distance between the source and the detector.

Blood lactate measurement. The right ear lobe was cleaned using an alcohol swab and punctured using an automated lancet. At rest and immediately after sprints 4, 8, and 12, a blood sample was drawn using a 20- μ L capillary tube (EKF Diagnostics, Barleben, Germany). The whole blood sample was hemolyzed in a prefilled micro test tube and analyzed using a blood lactate/glucose analyzer (Biosen C-Line, EKF Diagnostics, Barleben, Germany).

Statistical Analysis

Data were analyzed using a contemporary magnitude-based inference approach (22) because small changes in performance can be meaningful in athletes. Data were log transformed to reduce nonuniformity of error except for RPE owing to its interval nature. The threshold value for the smallest meaningful change for mean and peak power output was set as 0.8% (2). For all other data, the smallest worthwhile or important effect for each dependent variable was the smallest standardized (Cohen) change in the mean: 0.2 times the between-subject SD for baseline values of all participants (8). Qualitative descriptors were assigned to the quantitative percentile scores as follows: 25%–75%, *possible*; 75%–95%, *likely*; 95%–99%, *very likely*; and >99%, *almost certain* (20). A substantial effect was set at >75%. Effect size was calculated using threshold values for Cohen *d* statistics (0.2, small; 0.5, moderate; and 0.8, large). Data are presented as mean \pm SD or percent change from placebo ($\% \Delta \pm 90\%$ confidence interval ($\pm 90\%$ CI)), percent likelihood that the difference between conditions was larger or smaller (% likelihood and effect size). An effect was deemed unclear if its confidence limits overlapped the thresholds for both the smallest beneficial and the smallest harmful effect, that is, if the effect could be substantially positive and negative.

RESULTS

The maximal peak power (mean \pm SD) obtained during the repeated sprint cycling test was 1594 ± 208 and $1630 \pm$

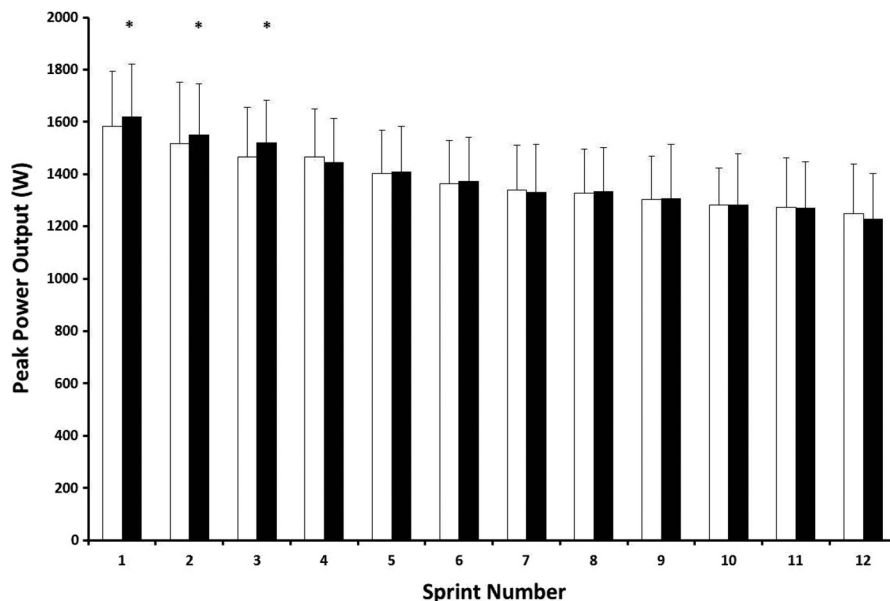


FIGURE 1—Peak power output data during 12 maximal 6-s sprints after ischemic preconditioning (solid bars) or placebo (open bars). Data are mean \pm SD. Asterisk indicates substantially different from placebo (>75% likelihood).

192 W for placebo and ischemic preconditioning, respectively. Qualitative analysis revealed that performing ischemic preconditioning before sprint activity led to a *likely* increase in maximum peak power output ($2.5\% \pm 1.9\%$, 93%, small (% Δ , % likelihood, effect size)). Raw peak and mean power output data for each sprint are presented in Figures 1 and 2, respectively. Ischemic preconditioning, relative to placebo, resulted in substantial increases in peak power output for sprints 1 ($2.4\% \pm 2.2\%$, 89% *likely*, small), 2 ($2.6\% \pm 2.7\%$, 87% *likely*, small) and 3 ($3.7\% \pm 2.4\%$, 97% *very likely*, small) only, with effects unclear for the remaining sprints. Mean power output followed a similar pattern with substantial increases in sprints 1 ($2.8\% \pm 2.5\%$,

91% *likely*, small), 2 ($2.6\% \pm 2.5\%$, 88% *likely*, small) and 3 ($3.4\% \pm 2.1\%$, 98% *very likely*, small) for the ischemic preconditioning trial, relative to placebo, and the effects on the remaining sprints were deemed unclear. During the repeated sprint cycling protocol, fatigue was evident in both trials as represented by S_{dec} values of $13.2\% \pm 5.6\%$ and $14.7\% \pm 5.9\%$ for placebo and ischemic preconditioning, respectively. Qualitative analysis revealed a *possibly* greater fatigue rate when repeated sprint cycling was performed after ischemic preconditioning ($13.5\% \pm 16\%$, 64% *possible*, small).

Blood lactate was not different at rest before the placebo and ischemic preconditioning trials (mean \pm SD, 1.1 ± 0.2

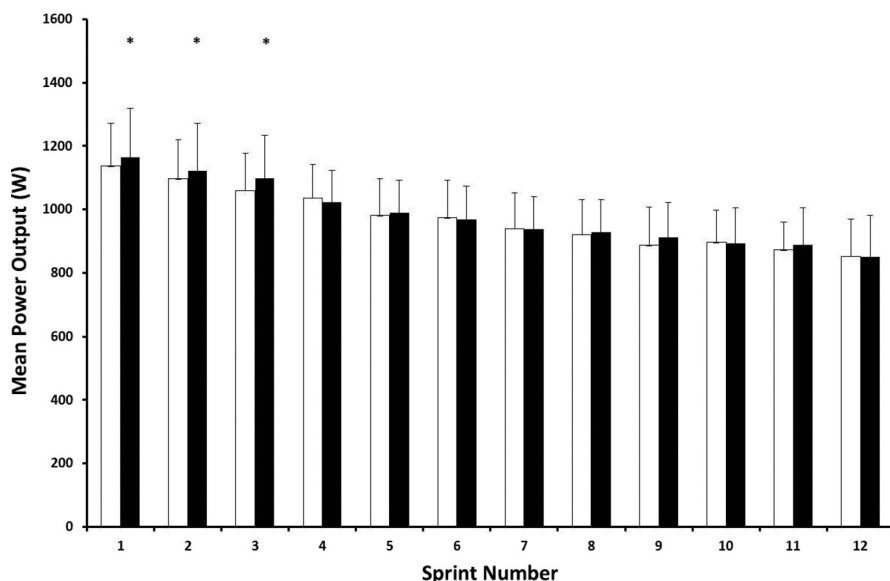


FIGURE 2—Mean power output data during 12 maximal 6-s sprints after ischemic preconditioning (solid bars) or placebo (open bars). Data are mean \pm SD. Asterisk indicates substantially different from placebo (>75% likelihood).

TABLE 1. Statistical summary of the differences between ischemic preconditioning and placebo for oxygen uptake, tissue saturation index, EMG, and blood lactate.

	Placebo	Ischemic Preconditioning	Mean Change ^a , ± 90% CI (%)	Qualitative Inference ^b (% Likelihood)	Effect Size (Qualitative Descriptor)
$\dot{V}O_2$ rest, L·min ⁻¹	0.4 ± 0.1	0.4 ± 0.1	1.9 ± 15.6	Unclear	0.08 (trivial)
$\dot{V}O_2$ exercise, L·min ⁻¹	2.6 ± 0.3	2.7 ± 0.4	4.3 ± 7.3	Unclear	0.29 (small)
TSI rest, %	71.8 ± 5.1	73.0 ± 4.0	1.7 ± 3.6	Unclear	0.24 (small)
TSI occlusion, %	72.3 ± 5.5	58.0 ± 4.2	-19.7 ± 3.6	Almost Certainly decreased (100%)	2.77 (large)
TSI exercise, %	57.7 ± 5.0	60.9 ± 6.0	5.4 ± 4.8	Likely increased (93%)	0.56 (moderate)
Rate of change in EMG MDF, Hz/sprint	-0.04 ± 0.43	-0.28 ± 0.42	48.9 ± 69.7	Possibly higher (73%)	0.37 (small)
Sprint 4 blood lactate, mmol·L ⁻¹	6.9 ± 2.1	7.5 ± 2.3	7.1 ± 11.4	Possibly higher (50%)	0.19 (trivial)
Sprint 8 blood lactate, mmol·L ⁻¹	9.6 ± 2.8	10.2 ± 2.3	4.3 ± 6.4	Possibly higher (25%)	0.12 (trivial)
Sprint 12 blood lactate, mmol·L ⁻¹	11.1 ± 3.5	11.8 ± 2.7	5.5 ± 6.4	Possibly higher (26%)	0.13 (trivial)

^aMean change refers to ischemic preconditioning minus placebo trial.

^bInference about the magnitude of the effect.

Bold inferences (% likelihood) indicate conditions with substantial change (>75% likelihood). 90% CI, 90% confidence interval.

and 1.0 ± 0.3 mmol·L⁻¹, respectively; *unclear*, trivial). Blood lactate was *possibly* higher when measured at sprints 4, 8, and 12 in the ischemic preconditioning (Table 1). Relative to placebo, the effects of ischemic preconditioning on perceived exertion at sprints 4, 8, and 12 were -0.1 ± 0.6 , 0.2 ± 0.7 , and 0.1 ± 0.8 (arbitrary units), respectively, with qualitative analysis interpretation deeming differences and effect sizes as unclear or trivial. Data for TSI are presented in Table 1. Briefly, effects for TSI at rest between trials were unclear. During the occlusion/preconditioning stimulus, there was an *almost certain* decrease in TSI during the ischemic preconditioning trial relative to placebo. During exercise, there was a *likely* higher increase in TSI in the ischemic preconditioning trial compared with placebo (Table 1). At rest and during exercise, differences in oxygen uptake between trials were unclear (Table 1). The rate of change in MDF of EMG was *possibly* higher in the ischemic preconditioning trial, relative to the placebo trial (Table 1).

DISCUSSION

The main aim of this study was to investigate the effect of ischemic preconditioning on repeated sprint cycling performance. Relative to placebo, the results showed that ischemic preconditioning was associated with a 2%–4% increase in both mean and peak power output in the early phase of the protocol. The improvement in power output is similar to other ergogenic aids used during this type of exercise (15,16) and to performance improvements observed after ischemic preconditioning using different exercise modes (12,24).

The present investigation is the first to demonstrate an improved power output after ischemic preconditioning during a repeated sprint protocol. Despite rejecting our hypothesis, we did observe substantial increases in both peak and mean power output for the first three sprints. Previous research has demonstrated an improved muscle force production after ischemia and reperfusion in animal (21,26) and human models (27). Owing to the original aim and thus the design of the study, it was not possible to determine the contribution of increased motor unit recruitment to improved performance, although it does remain a

possibility. Electromyographic amplitude has previously been demonstrated to increase in skeletal muscle of animals after ischemic preconditioning (36), suggesting increased motor unit recruitment. In the only relevant human study, muscle fiber conduction velocity, which measures the speed of action potential or excitatory impulse, is increased during isometric exercise; yet ischemic preconditioning did not play a role (37).

It is recognized that high-energy compounds are important for energy production during repeated sprint activity, with total anaerobic contributions of ATP production during a single 6-s sprint being 6%, 50%, and 44% from ATP, PCr, and anaerobic glycolysis, respectively (18). Although speculative, it is possible that the increased power production in the first three sprints in the ischemic preconditioning trial may have been a result of increased ATP production from anaerobic sources. After ischemic reperfusion injury, ATP content is maintained in rabbit and mice heart muscle as a result of ischemic preconditioning via increased concentration of PCr and PCr/ATP ratio (29) or increased anaerobic glycolysis (23). To date, little evidence is available on concentrations in skeletal muscle; however, increased PCr production has been observed using ³¹P MRS in recovery from an ischemic event (1). Therefore, it is possible that improved power output may be a result of increased anaerobic energy contribution early in the sprint protocol. Within the current study, a *possible* increased blood lactate concentration was observed after the fourth sprint in the ischemic preconditioning trial, giving further weight to this suggestion although it was not a substantial effect.

Originally, it was hypothesized that ischemic preconditioning would improve aerobic metabolism and thus improve the ability to recover between sprints. Markers of aerobic fitness such as $\dot{V}O_{2\max}$ and $\dot{V}O_2$ kinetic parameters are related to the ability to offset fatigue during a repeated sprint effort (14,30), whereas an increased aerobic energy production contributes toward power production in the latter stages of repeated intense exercise (6,18,30). Previous research using one bout of circulatory occlusion before the start of an exercise bout has demonstrated accelerated pulmonary $\dot{V}O_2$ kinetics (34). Moreover, ischemic preconditioning

has been shown to increase $\dot{V}O_{2\max}$ (13), suggesting that the method may be used to help maintain power output during repeated sprint exercise, via improved PCr resynthesis (9,18,34). However, data from the present study does not support this theory as evidenced by the similarity between trials for $\dot{V}O_2$ during the repeated sprint protocol.

Alongside an increased aerobic metabolism, ischemic preconditioning has been associated with improved muscle oxygenation during and in recovery from exercise (38). As expected, TSI was *almost certainly* decreased by 20% during the preconditioning stimulus, relative to placebo, which is similar to a previous investigation (25). However, during exercise, TSI was *likely* maintained at a higher level in the ischemic preconditioning trial. Since TSI reflects the dynamic balance between O_2 supply and O_2 consumption, the greater TSI observed during the ischemic preconditioning trial is indicative of an improved O_2 delivery at the muscle level (11). This may explain the maintenance of power output in the latter sprints in the ischemic preconditioning condition despite the higher power outputs early in the trial and place the emphasis on greater O_2 delivery. It should be noted, however, that muscle oxygenation is not a limiting factor during repeated sprint activity (39). Instead, it may be that ischemic preconditioning increases blood flow to skeletal muscle (40), thereby improving power maintenance by increasing microvascular pressure and/or by increasing metabolite washout (27). However, this mechanism is questionable given that blood flow returns to resting levels within 20 min of cuff release (7).

Previous research investigating ischemic preconditioning in exercise involving sprint activity has provided conflicting evidence. Elite swimming performance (100 m) is enhanced after ischemic preconditioning (24); however, the time taken to complete the event was ~66 s, thus not typical of a sprint experienced in team sports. Moreover, no effect of ischemic preconditioning has been demonstrated during “all-out” sprint exercise at 130% $\dot{V}O_{2\max}$ or 30-m land-based sprint running (12,19). Whereas these results differ from the ones

in the current study, they may be explained by the timing of the preconditioning strategy. Previous studies have performed a warm-up immediately after the preconditioning stimulus and moved straight into the exercise regime (19). In the current study, the warm-up was started 30 min after the ischemic preconditioning stimulus to make the research more applicable to an athletic setting. Current research investigating performance immediately after the ischemic preconditioning stimulus may be confined to a laboratory setting owing to the impracticality of performing a similar action in an athletic event. It may be that the extra recovery time after the ischemic preconditioning stimulus is more beneficial for sprint-related activity as demonstrated by the increased power output in the first three sprints. Owing to the evidence in a controlled laboratory environment and protocol in the current study, future research should focus on the mechanisms for improved performance and application of ischemic preconditioning in events that mimic actual performance events.

In conclusion, ischemic preconditioning of skeletal muscle *likely* increases both mean and peak power output in the first three sprints by 2%–4% during the early stages of repeated sprint cycling. This was in contrast to our hypothesis that ischemic preconditioning would improve S_{dec} through aerobic metabolism. Moreover, S_{dec} was not substantially different between the trials, possibly owing to maintenance of TSI in the ischemic preconditioning condition. Further research is required to establish the mechanisms for increased power output during repeated sprint cycling after ischemic preconditioning. Overall the results of this study suggest that ischemic preconditioning is a potential aid for improving sprint-based performance.

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