Effect of ischemic preconditioning on repeated sprint ability in team sport athletes

Title: Effect of Ischemic Preconditioning on repeated sprint ability in team sport athletes.

Running Title: Ischemic preconditioning and repeated sprinting.

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Abstract

This study investigated whether ischemic preconditioning in a trained population affected repeated sprint performance. A secondary aim was to assess responses according to gender. Sixteen (nine females and seven males) well trained team sport athletes took part in a randomised crossover study design. Participants underwent an ischemic preconditioning and placebo treatment involving three periods of 5 min occlusion applied unilaterally (3 x 5 min occlusion to each leg) at either 220 mmHg or 50 mmHg respectively. Each period of occlusion was followed by 5 min reperfusion. Following treatment 5 x 6 s maximal effort sprints were undertaken on a cycle ergometer against 7.5 % body mass, each interspersed by 24 s recovery. Measured parameters included peak power, total power, percentage decrement, post exercise blood lactate and ratings of perceived exertion. No within subject main effect for ischemic preconditioning was observed, neither was there an interaction effect with gender. Effect sizes were trivial (ES<0.2) with the exception of a moderate (ES<1.2) change in post exercise blood lactate in the female cohort (1.6 ± 0.4 $mmol^{-1}$ lower following IPC). Results suggest no benefit to team sport players in utilising ischemic preconditioning as a means of enhancing repeated sprint performance. A lower blood lactate response in female participants following ischemic preconditioning may suggest improved blood flow through vasodilation.

Introduction

Since early research investigating the effect of ischemic preconditioning on the myocardium the beneficial effect of the treatment has been observed in different tissues within the body, including skeletal muscle (de Groot, Thijssen, Sanchez, Ellenkamp, Hopman, 2010; Crisafulli et al., 2011; Beaven, Cook, Kilduff, Drawer, Gill, 2012; Jean-St-Michael et al., 2011). Research investigating the effect of ischemic preconditioning on exercise has included measures of aerobic capacity (de Groot et al., 2010; Crisafulli et al., 2011), speed (Gibson, White, Neish, Murray, 2013) and recovery following strength and power tasks (Beaven et al., 2012). Data from these studies is equivocal with the suggestion that a pattern of responders and non-responders may exist and that the intervention may be less effective in female populations (Beaven et al., 2012; Gibson et al., 2013).

The ability of ischemic preconditioning to exert a beneficial effect on exercise and recovery thereafter appears to be dependent on metabolites, including bradykinin, opioids and adenosine, reaching a critical level (Downey, Davis, Cohen, 2007). Inhibition of one of these metabolites removed the beneficial effect of a single bout of ischemic preconditioning suggesting the existence of a threshold below which occlusion may prove ineffective (Goto et al. 1995). Adopting multiple episodes of ischemic preconditioning has been advocated within the literature to ensure such a threshold is met (de Groot et al., 2010, Crisafulli et al., 2011, Jean-St-Michael et al., 2011).

Much of the research to date has focused on the usefulness of ischemic preconditioning on exercise tasks of an endurance nature with positive effects reported for total work, power, exercise time (Crisafulli et al., 2011) and $\dot{V}O_2$max (de Groot et al., 2010). The effect of ischemic preconditioning on activities of a shorter and more anaerobic nature is less clear: a significant improvement has been reported in elite level swimmers over a distance of 100m (Jean-St-Michael et al., 2011) whilst no improvement was noted when cyclists used the intervention prior to performing supra-maximal efforts (Crisafulli et al., 2011). It should be noted however that in both these studies exercise time was in excess of 60s, considerably longer than typical anaerobic efforts observed in team sport environments (Spender, Bishop, Dawson & Goodman, 2005). When utilised prior to land based sprint activities no effect was reported (Gibson et al., 2013) however the intervention was found to be beneficial on measures of acute and chronic recovery when used between tasks requiring maximal force generation (Beaven et al., 2012). This may in part be due to enhanced muscle recruitment via a desensitizing of the afferent groups III and IV, increased neural drive and force output (Noakes, 2011) Interestingly, it has been suggested that ischemic preconditioning is less beneficial in female populations (Beaven et al., 2012; Gibson et al., 2013)

Whilst a number of individual events are characterised by short but intense single efforts, team invasion sports require the performance of multiple bouts of maximal and at times supra-maximal activity (Spencer et al., 2005; Gabbett, 2012; Dwyer & Gabbett, 2012; Impellizeri et al., 2006; Gabbett, 2009). The importance of these repeated sprint efforts to successful performance has been illustrated within rugby league (Gabbett, 2012). Given that ischemic preconditioning has been shown to exert a beneficial effect on performance in high intensity tasks when administered 45 minutes before competition (Jean-St-Michael et al., 2011) its use within the warm up prior to team sports would seem plausible. Ischemic preconditioning has been evidenced to enhance vasodilation, oxygen delivery and ATP sparing (Beaven et al., 2012; Liu et al., 1991; Jennings, Sebbag, Schwartz, Crago & Reimer, 2001), adaptations similar to those that could be expected following endurance training. With this in mind a positive effect on repeated sprint activities may be expected given the

large aerobic component that exists in exercise of this nature (Dupont, McCall, Prieur, Millet & Berthoin, 2013; Bucheit & Laursen, 2013). The current investigation is designed to assess whether ischemic preconditioning exerts a beneficial effect when used prior to repeated sprint activity performed on a cycle ergometer. Given the large aerobic component that is associated with exercise of this nature it is hypothesised that enhanced oxygen delivery, facilitated through adenosine mediated vasodilation and/or ATP sparing, will provide a beneficial effect on performance. A secondary aim is to compare responses between gender groups to further investigate the assertion that ischemic preconditioning may be less suitable for female populations (Beaven et al., 2012; Gibson et al., 2013)

Methods

Participants and design

Sixteen participants (7 males and 9 females) volunteered to take part in the study, all with a recognised competition history within the invasion sports of Soccer, Field Hockey and Rugby Union. Mean age, stature and body mass are presented in table I. All participants signed an informed consent document and the study received institutional ethical approval conforming to the code of ethics of the World Medical Association (Declaration of Helsinki).

A counterbalanced randomised crossover design was used to assess the impact of a brief period of remote ischemic preconditioning on repeated sprint performance under two separate conditions, experimental and placebo. All participants undertook a prior control with no treatment. In both placebo and ischemic preconditioning trials participants were fitted with a blood pressure cuff positioned around the upper thigh and inflated to 50 mmHg or 220 mmHg respectively. Following treatment participants followed a standardised warm up followed by 5 x 6 s sprints against an external load of 7.5 % body mass. Measured variables were peak power, relative peak power (Watts per kilogram body mass), total power, percentage decrement, post assessment blood lactate and ratings of perceived exertion (RPE).

INSERT TABLE 1 NEAR HERE

Baseline assessment and control

All participants were required to visit the laboratory on three separate occasions each no more than seven days apart for the collection of control, placebo and experimental data. On their first visit participants' age, stature and body mass were recorded along with a measure of resting blood pressure (Omron RX-3, Kyoto – Japan). Any participants presenting with a blood pressure higher than 140/100 mmHg (systolic/diastolic) were precluded from taking part in the study. These guidelines were in line with ethical approval of the study. Prior to data collection the cycle ergometer (MonarkErgomedic 814c, Stockholm, Sweden) was calibrated according to the manufacturers guidelines and configured to suit the participants preferred cycling position. Participants completed a standardised warm up consisting of five minutes stationary cycling at 60 rpm and against 1kg of external resistance. This was followed by two 3s sprints separated by 60 s to habituate themselves with the requirements of the assessment. The repeated sprint assessment required the performance of 5 x 6 s sprints against 7.5 % of body mass, each separated by a recovery period of 24 s, a protocol used previously in team sport athletes (Blee, Goodman, Dawson & Stapff, 1999; Bishop, Spencer, Duffield & Lawrence, 2001). This protocol was chosen to limit the impact of pacing and provide a sufficient number of sprints for the accurate and reliable assessment of peak power and percentage decrement (Hachana, Attia, Nassib & Shephard, 2012). Blood lactate samples were collected three minutes post the fifth and final sprint using a Lactate Pro

analyser (ArkrayInc, Kyoto, Japan. CV 5.66%). RPE data was collected following each of the five sprints. Data from the repeated sprint protocol was collected and analysed using specific software (Cranlea Wingate Software version 3). Following the collection of control data all participants underwent both placebo and experimental conditions in a randomised counterbalanced fashion.

Placebo trial

On arrival at the laboratory participants had their blood pressure measured as described above to screen for any contraindications to the experimental procedure. Participants were then instructed to adopt a semi-recumbent position on a medical plinth with both legs outstretched. A blood pressure cuff (Boso-roid I aneroid sphygmomanometer, Bosch and Son, Germany) was positioned around the upper thigh, distal to the inguinal fold. For placebo treatment the cuff was inflated by hand to 50mmHg. Each leg was exposed to 5 min of pressure followed by 5min of reperfusion for three consecutive cycles eliciting a total treatment time of 30 min. During reperfusion the contralateral leg was fitted with the cuff and inflated to 50 mmHg in accordance with the protocol for ischemic preconditioning administration described elsewhere (Gibson et al., 2013). During the treatment participants were asked at regular intervals (every minute) to confirm they were able to continue with the protocol. Any participant indicating light headedness, nausea or discomfort had the pressure cuff removed and were omitted from the study (n = 0). Following the final 5 min of reperfusion the participant was supported whilst they stepped down from the plinth and given a moment to ensure they were steady on their feet before commencing the standardised warm up detailed above. The time delay between removing the pressure cuff and commencing the warm up was five minutes.

Ischemic preconditioning treatment

The ischemic preconditioning treatment followed an identical format to that of the protocol as described above however the blood pressure cuff was inflated to 220 mmHg which has been shown to elicit ischemia by occluding arterial blood flow to the lower legs (Koojiman et al., 2008).

Statistical analysis

Data was checked for homogeneity of variance using Lavene's test and did not violate the assumption of sphericity using Mauchly's test. All results were non-significant ($P<0.05$) and as such deemed appropriate for parametric analysis. Data were analysed using SPSS for windows (PASW statistics 17.0) and a 3 x 2 mixed factorial ANOVA with significance calculated at $P<0.05$. Due to the practical nature of the investigation effect sizes (Cohen's d) were also used. Effect sizes of <0.2, <0.6, <1.2, <2.0 and >2 were considered trivial, small, moderate, large and very large respectively (Hopkins, Marshall, Batterham & Hanin, 2009).

Results

Table I details the physical characteristics of participants included in the study as a pooled cohort and separated by gender groups. Table II details means ± SD's for performance variables calculated during the repeated sprint protocol along with corresponding effect sizes comparing control with ischemic preconditioning trials. No significant main effect or interaction with gender was observed for occlusion on peak power or relative peak power ($P>0.05$) as shown in figure 1. Calculated effect sizes were classified as trivial (ES<0.2) for peak power, total power and relative peak power. Within the female cohort a small effect

size was detected for percentage decrement whilst for the blood lactate response effect sizes of small and moderate magnitude were calculated for the male and female cohort respectively as shown in figure 2.

INSERT TABLE II NEAR HERE

INSERT FIGURE I NEAR HERE

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Discussion

Data collected in the current investigation suggest ischemic preconditioning to exert neither a beneficial nor deleterious effect on absolute and relative peak power, total power or percentage decrement during a repeated sprint protocol. Unlike previously reported data (Beaven et al., 2012; Gibson et al., 2013) there appears to be no difference in response to the intervention when compared by gender group with the exception of post exercise blood lactate. Results would suggest that for events requiring short (<6 s) maximal efforts ischemic preconditioning is not a suitable pre exercise intervention for performance enhancement. The finding that post exercise blood lactate levels may be reduced, especially in a female cohort, combined with non-significant changes in total power and percentage decrement warrants further investigation into the effect of ischemic preconditioning on activities requiring repeated forceful yet sub-maximal efforts, such as those occurring in team sports (Spencer et al., 2005; Gabbett, 2012; Dwyer & Gabbett, 2012; Impellizeri et al., 2006; Gabbett, 2012). The ability to produce similar amounts of work whilst attenuating the production of lactate and/or augmenting its clearance may suggest the intervention to facilitate a greater contribution from aerobic pathways and the sparing of ATP generated via glycolysis (Bailey et al., 2012).

Equivocal results are apparent in response to power output following ischemic preconditioning administration. When used as a recovery modality following activities requiring high power output ischemic preconditioning was shown to attenuate reductions in performance, both acutely and chronically (Beaven et al, 2012). In a study conducted with international level swimmers the intervention was shown to improve performance by 0.7 s, a change paralleled with an increased stroke count. This change may be interpreted as being the result of less force exerted per stroke (Jean-St-Michael, 2011). The event duration (~60 s) may have provided sufficient time for any decrement in initial peak power following ischemic preconditioning administration to be compensated for by a higher sustained average power in the latter stages of the race. It should be noted that in the present study ischemic preconditioning exerted no significant effect on average, total or peak power. In cyclists exercising supra-maximally for approximately 120 s no beneficial effect of ischemic preconditioning was realised in terms of exercise time or power output (Crisafulli et al., 2011). In studies examining the effect of ischemic preconditioning on swimmers and cyclists however exercise duration was substantially longer than that which has been reported for team sports (Spencer et al., 2005), a sporting population from which the current cohort was drawn. Considering the present study's findings and those reported when ischemic preconditioning was used prior to short land based sprinting (<5 s) (Gibson et al., 2013) there appears to be evidence that would support the existence of a threshold in exercise duration below which the intervention has no effect on performance.

A mechanism postulated for the beneficial effect of ischemic preconditioning on exercise is increased blood flow and oxygen delivery to the working musculature via adenosine

mediated vasodilation (Beaven et al., 2012; Liu, 1991). There is also evidence of ATP preservation, albeit in canine models (Jennings et al., 2001). In the current investigation no changes in performance following ischemic preconditioning with respect to percentage decrement were reported however moderate effect sizes were measured for post exercise blood lactate in the female cohort with lower values reported following IPC. Lower blood lactate levels following exercise preceded by ischemic preconditioning have been reported elsewhere (Bailey et al., 2012). Following 5 x 3 minute stages of incremental treadmill running ranging from 10 to 14 $km.h^{-1}$ blood lactate was observed to be 1.07 ± 0.11 $mmol^{-1}$ lower when ischemic preconditioning preceded exercise. In the present study repeated sprints preceded by ischemic preconditioning were shown to illicit a blood lactate response 1.6 ± 0.4 $mmol^{-1}$ lower than control within the female cohort and a corresponding moderate effect size. This reduction was paralleled by non-significant changes in peak power, total power and percentage decrement. It is suggested that future research includes a greater number of sampling points to more fully explain lactate kinetics following exercise preceded by ischemic preconditioning.

Whilst improvements in aerobic capacity have been associated with reduced blood lactate following sub-maximal exercise of a given workload (Lorenzo, Minson, Babb & Halliwell, 2011) this mechanism is unlikely to explain changes in hematology during the present study. An alternative hypothesisis that ischemic preconditioning mimics some of the chronic changes associated with training and its associated improvements in aerobic capacity may be postulated. These include but are possibly not limited to, vasodilation and the associated increases in blood flow that facilitate energy provision via aerobic pathways, sparing of ATP derived from anaerobic glycolytic pathways and potential augmentation of blood lactate clearance. Previous results have shown that the provision of energy via aerobic pathways increases during repeated sprint exercise to compensate for the reduction in glycolysis (Bailey et al., 2012). A strong relationship has also been shown between repeated sprint performance and aerobic capacity (Dupont et al., 2010; Bishop et al., 2004) characterised by an enhanced ability for energy provision via increased capilliarisation, blood flow and mitochondrial density. Indeed it was augmentation of blood flow which was suggested as a potential mechanism for ischemic preconditioning providing a beneficial stimulus when used as a recovery modality following tasks requiring maximal force generation (Beaven et al., 2012). Reductions in blood lactate following the use of ischemic preconditioning may be a result of enhanced energy provision via aerobic pathways, ATP sparing and/or enhanced oxidation and clearance rates. Such mechanisms would lend support to the contention that during repeated sprint activities where energy derived from anaerobic processes is compromised as a result of intensity, duration or insufficient between effort recovery, ischemic preconditioning may be beneficial to performance.

It was hypothesised that ischemic preconditioning would result in a greater decrement in performance within the female cohort, something that was not evident in the results. Previous studies have shown the intervention to negatively affect performance in female athletes when used prior to land based sprinting activities and as a recovery tool following strength and power activities (Beaven et al., 2012; Gibson et al., 2013). Individual variations in thigh circumference, muscle mass and limb composition and the corresponding level of occlusion caused at an absolute pressure of 220 mmHg (Dempsey & Wagner, 1999) have been cited as potential causes of this discrepancy, along with the perception of discomfort associated with the intervention. RPE data collected in the present study would not support the contention that a greater perception of effort and/or discomfort was associated with ischemic preconditioning in male or female participants. It is acknowledged however that

given the relatively low participant numbers in the present study drawing firm conclusions regarding differences that may exist between gender is difficult.

Time motion analysis in team sports has suggested mean sprint durations to be between 2-3 s for elite level Soccer, Field Hockey and Australian Rules Football rising to 4.1 ± 1.1 s when mean maximal sprint duration is considered (Spencer et al., 2005). As such the protocol in the present study characterised by 5 x 6 s sprints has been suggested to be representative of field based invasion game activity. For sports that incorporate short (<1 s) accelerative efforts requiring high force output results from the present study would suggest ischemic preconditioning to be an inappropriate pre exercise intervention. If however the sport is more reliant on running sprints (Impellizeri et al., 2006) characterised by high running speeds over longer durations and potentially less forceful accelerations (Gabbett, 2012), the use of ischemic preconditioning may be warranted given non-significant changes in percentage decrement and lower post exercise blood lactate levels in the female cohort. Future research should focus on investigating the effectiveness of IPC as a precursor to land based repeated sprint activities and/or sport specific simulation protocols (Twist & Sykes, 2011)

Conclusion

Ischemic preconditioning exhibited no beneficial effect on markers of performance associated with repeated sprinting characterised by 5 x 6 s efforts, including total power, peak power and relative peak power. Additionally there appears to be no difference in response between gender groups following the intervention as has been reported for single sprint activity and recovery. Interestingly however a moderate reduction in post exercise blood lactate following ischemic preconditioning in the female cohort was observed. This finding may suggest that for repeated sprint protocols of a longer duration, or those involving actions that more closely mimic the demands of team sports, such as collisions or changes of direction ischemic preconditioning may be beneficial to markers of performance.

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Table 1. Means ± SD for physical characteristics of participants as a pooled cohort and separated by gender.

Physical characteristic	Participants (n = 16)	Female (n = 9)	Males (n = 7)
Age (years)	24.1 ± 2.6	24.0 ± 3.71	24.2 ± 1.6
Stature (cm)	174.0 ± 6.1	171.1 ± 4.4	177.6 ± 6.2
Mass (kg)	73.7 ± 11.8	67.6 ± 7.1	81.4 ± 12.5

Table 2. Means ± SD for peak power (PP), peak power adjusted for body mass (RPP), total power (TP), percentage decrement (%Dec) and delta rating of perceived exertion (RPE) along with corresponding effect sizes for control, placebo and ischemic preconditioning repeated sprint trials. Effect sizes correspond to the change between control and ischemic preconditioning trials. Data reported for all participants and by gender group.

.	Pooled data (n = 16)				Females (n = 9)				Males (n = 7)			
	Control	Placebo	IPC	ES	Control	Placebo	IPC	ES	Control	Placebo	IPC	ES
PP (W)	1583.2 ± 368.6	1611.7 ± 461.7	1577.4 ± 374.1	0.02	1360.4 ± 247.0	1319.9 ± 102.3	1353 ± 184.1	0.03	1869.7 ± 297.5	1987.0 ± 476.4	1865.6 ± 363.9	0.01
RPP (W.kg)	21.4 ± 3.6	21.7 ± 4.3	21.3 ± 2.9	0.04	20.1 ± 2.7	19.8 ± 2.7	20.1 ± 2.0	0.01	23.1 ± 4.1	24.2 ± 4.7	22.9 ± 3.1	0.07
TP (W)	6748.6 ± 1413.6	6842 ± 1712.0	6668.6 ± 1364.0	0.06	5883.5 ± 895.0	5707.3 ± 489.2	5788.3 ± 630.1	0.12	7860.7 ± 1167.1	8301.0 ± 1611.5	7800.2 ± 1211.0	0.05
%Dec	14.1 ± 5.3	14.4 ± 5.2	14.8 ± 4.3	0.15	12.8 ± 6.2	13.4 ± 6	14.1 ± 4.4	0.23	15.8 ± 3.8	15.7 ± 3.9	15.8 ± 4.4	0.01
Bla ($mmol^{-1}$)	9.3 ± 2.1	9.0 ± 2.6	8.2 ± 2.3	0.51	9.0 ± 2.4	8.8 ± 2.4	7.4 ± 2.0	0.72	9.7 ± 1.9	9.4 ± 2.9	9.2 ± 2.3	0.25
Delta RPE	4.6 ± 2.3	5.0 ± 2.9	5.0 ± 2.3	0.19	3.8 ± 1.6	4.2 ± 1.4	4.2 ± 1.8	0.26	5.6 ± 2.8	6.0 ± 2.9	6.0 ± 3.7	0.18

Effect sizes of <0.2, <0.6, <1.2, <2.0 and >2 were considered trivial, small, moderate, large and very large respectively.

Figure 1. Mean ± SD for relative peak power (RPP) and percentage decrement (%Dec) across 5 x 6 s sprints on a cycle ergometer against 7.5% of body mass.

Figure 2. Mean ± SD for the blood lactate (Bla) response following baseline ischemic preconditioning and placebo trials for all participants and separated by gender. * denotes a moderate effect size for differences in Bla response within female participants following ischemic preconditioning compared to baseline trials