

Effect of ischemic preconditioning on lactate accumulation and anaerobic performance in physically active male students

Bilgehan Baydil

School of Physical Education and Sport, Kastamonu University, Kastamonu, Turkey.

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ABSTRACT

Ischemic preconditioning (IPC) has recently been experimented with the hypothesis that it can acutely improve exercise performance. IPC's ergogenic effects have been a novel approach to improve anaerobic power and sprint performances. Since the beneficial effects of the underlying mechanisms on anaerobic power have not been clearly understood, the results obtained from different approaches are not sufficient to reach a clear opinion. The purpose of this study was to determine the effect of IPC on anaerobic power and capacity in the physically active students to provide new findings to the literature. A randomized crossover order, twenty-four physically active male students voluntarily participated in this study. Students completed familiarization before pretest intervention and measurements. They performed a cycle ergometer Wingate 30s protocol resistance with 7.5% bodyweights in IPC and control conditions. The IPC protocol was performed at 220 mmHg, 5 min, 3 times as ischemia interspersed with 5 min of reperfusion. Participants were applied pre and post-test to elicit the effects of IPC on anaerobic performance. Paired sample T-Test was used to determine differences between pre and post-tests. All statistical procedures processed by using the significance level at $p < 0.05$. The response to IPC treatment did not change anaerobic power and capacity performance outputs (PP $t = 0.064$, $p = 0.95$; MP $t = -.151$, $p = 0.881$; PD % $t = -.328$ $p = 0.746$). The application of IPC has not improved significantly any parameters of anaerobic performance. However, non-significant IPC effect on post-exercise lactate level to the extent of 15.32% ($t = 1.43$, $p = 0.17$) appeared.

Keywords: Anaerobic performance, physically active, ischemic preconditioning, blood lactate.

*Corresponding author. E-mail: bilgehan@kastamonu.edu.tr.

INTRODUCTION

Remote ischemic preconditioning is a technique of ischemia and reperfusion cycles created by pneumatic cuff or tourniquet into the limbs. The positive effects of IPC against the ischemia-reperfusion injury on all muscle tissues especially the myocardial muscle have been clearly demonstrated in clinical studies (Murry et al., 1986). This protective mechanism is generally explained by the neuronal, humoral, and systemic response of remote IPC effects (Veighey and MacAllister, 2012). Ischemia and reperfusion cycles created (3-4 sets 5 min) the endogenous substance which triggers neuronal and humoral pathways adenosine, bradykinin opioids start to

generate distance muscle (Ylitalo and Peuhkurinen, 2001; Andreas et al., 2011). This mechanism on muscle is evidenced by increases in the secretion of catecholamines in ischemic conditions, increasing levels of adenosine in the cell and enhancement of the function of ATP-sensitive potassium channels (K⁺ ATP channels) (Andreas et al., 2011; Incognito et al., 2017). De Groot et al. (2010) firstly adopted the method as a sports performance enhancer based on these indicators. They reported significant benefits on the power output and maximal oxygen consumption during an incremental maximum cycling test. Several following studies

highlighted the positive effect of IPC on exercise performance (Incognito et al., 2017; Caru et al., 2019; Yılmaz, 2019). Crisafulli's study results indicated that the application of IPC led to better power output and total work time during incremental maximum cycling trail. The positive effects were corroborated by literature findings on time trails with cycling (Hittinger et al., 2014; Cocking et al., 2017; Gürses et al., 2017; Cocking et al., 2018; Gürses et al., 2018). In addition, aerobic and anaerobic cycling test performances were improved by IPC in performance trails (Lalonde and Curnier, 2015; Lindsay et al., 2017). The most featured result was Wingate cycling test results (Kraus et al., 2015). Kraus et al indicated that IPC affected significantly Wingate peak and mean power output. In addition to relevant findings, two other interventions found that IPC decreases blood lactate accumulation. However, the positive effect generally occurred in the cycling trend. Whenever IPC is applied in-field performance, especially with special techniques, it has no effects (Richard and Billaut, 2018). In running trails, IPC has no effect on sprint, maximal acceleration, endurance running and short distance running (Caru et al., 2019). These data can be supported by rugby, rowing basketball and badminton (Tocco et al., 2015; Garcia et al., 2017; Gürses et al., 2018; Richard and Billaut, 2018). The effects of IPC on sports performance have been very confusing so far. Moreover, the number of studies related to cycling, maximal performance and peak power is limited. In this context, it is important to demonstrate the effect of IPC on performance by eliminating technique specific to a unique sport with a Wingate anaerobic Test on people with less experience in the sport. Thus, the aim of this study was to determine the effect of IPC on anaerobic power and capacity in the physically active individual.

MATERIALS AND METHODS

Subjects

Twenty-four physically active male students who were studying at School of Physical Education, Kastamonu University (age: 23.24 ± 2.86 yr; height 175.82 ± 7.15 cm; weight 70.48 ± 6.29 kg; Blood pressure: Systole 119.1 ± 7.3 mmHg, diastolic 78.3 ± 5.2 mmHg) voluntarily participated in this study. The participants regularly attended exercise 3 to 5 times a week for at least three years. None of the participants had any health problems, chronic disease or smoking habits and used any medication. The information about the study was given in detail and explained about the vigorous exercise to all participants. They were asked to avoid strenuous physical activities, alcohol, and caffeine consumption 24 hours prior to performance interventions. The subject was excluded from the study when they voluntarily wanted to leave the study.

Experimental design

A randomized crossover study design was performed to determine the acute effect of IPC on anaerobic Wingate Performance Test (Figure 1). To determine anaerobic power and capacity the 30-second Wingate test protocol was used. Participants performed 3 times a 30-second Wingate test protocol with 72 hours of recovery intervals. 10.00 to 12.00 and 16.00 to 18.00 hours of the day were selected for the measurements. Anthropometric measurements and familiarization were carried out on the first day when they visited the laboratory. After familiarization, participants were randomly assigned to either IPC (n = 12) or Control (n = 12) groups. Then all participants performed two other 30-second Wingate test protocols during different conditions. In IPC conditions participants were applied preconditioning cycles before warm-up routine. IPC was carried out 50 minutes before the measurements similar to the implementation of Crisafulli et al. (2011). For the control group, the only warm-up routine was used. Trails differed from each other only in the warm-up stage. The trails took place at the same time as intervention days. Pre- and post-exercise blood lactate and systolic and diastolic levels (with determining criteria $< 140/100$ mmHg) were measured during these two visits.

Ischemic preconditioning protocol

IPC protocol was performed 220 mmHg bilateral arterial occlusion on both legs with a manual pneumatic tourniquet (Reister, 5255, Germany). 5 min bilateral arterial occlusion applied 3 times interspersed with 5 min of reperfusion. IPC was implemented on the nearest point of the leg by using sleeve pressure 50 mmHg higher than blood pressure for each experimental. After blowing up leg-sleeve it was checked whether the circulation was completely interrupted from the foot artery.

Blood lactate measurement

The blood lactate levels were determined by using lancing with drilling at the distal phalanx of the index finger (AccutrendPlus GCTL with reagent strips BM-Lactate, Roche, Germany). Before collecting samples, participants' toes were cleaned with cotton soaked in alcohol 90° and dried well and then ~ 25 μ l of blood was collected. The blood sample was placed directly on the test strips for the lactate measurement by using the same drill.

30-second WINGATE test protocol

The lower body 30-second Wingate (WANT) test was

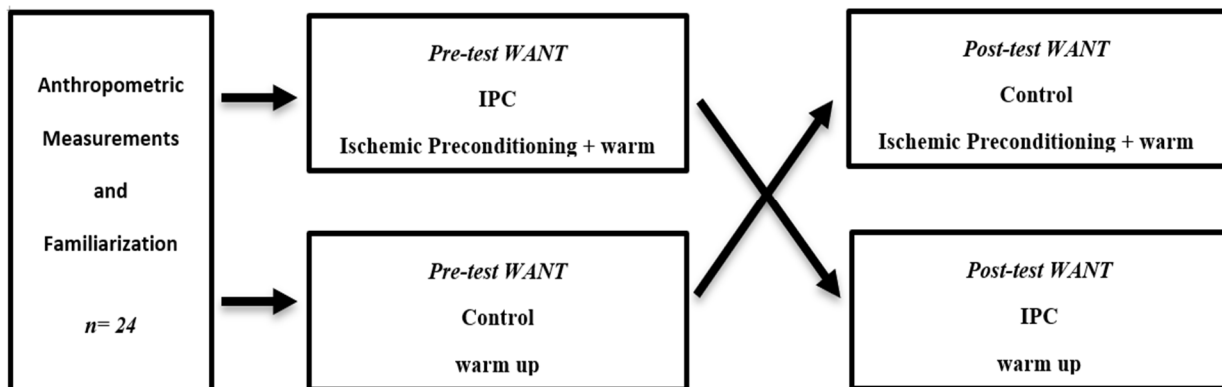


Figure 1. The crossover study design to determine the acute effect of IPC on 30-second Wingate Protocol.

performed in the performance laboratory (Bar-Or, 1987). The test was conducted on a cycle ergometer (Monarch 894E Ergomedic, Sweden). A 5-minute standardized warm-up was performed at the inertial resistance, including 4 bouts of 4 s performed at the beginning of each minute on the cycle ergometer. After a 10-min rest, the participants performed the test using a resistance of 0.75 kg/body mass load for 30 seconds at maximal speed. Verbal encouragement was provided throughout the test.

Statistical analysis

The statistical analysis was carried out with the SPSS 23 software program. Data normality was verified using the Shapiro Wilk test. A descriptive statistical method was used to calculate the mean and standard deviation for all variables. The paired sample T-test was used to determine the differences in IPC and control test conditions results. All statistical procedures were processed by using the significance level at $p < 0.05$. Effect sizes were calculated following the recommendations by Rhea (2004). Thresholds values to effect size were < 0.25 trivial, 0.25 to 0.50 small, 0.50 to 1.00 moderate and > 1.00 large.

RESULTS

Table 1 shows performance and blood lactate responses during both conditions. No significant difference was found for resting blood concentrations and trivial effects size was observed. All participants were at the same resting level. Concerning IPC and normal conditions no significant difference was found for post-exercise blood lactate levels, peak power, mean power and power drop. Trivial and large effect sizes observed. IPC affected post-exercise blood lactate levels non-significantly -15.32%, peak power -0.08%, mean power 0.24, power drop 1.20%. (Figures 2 and 3)

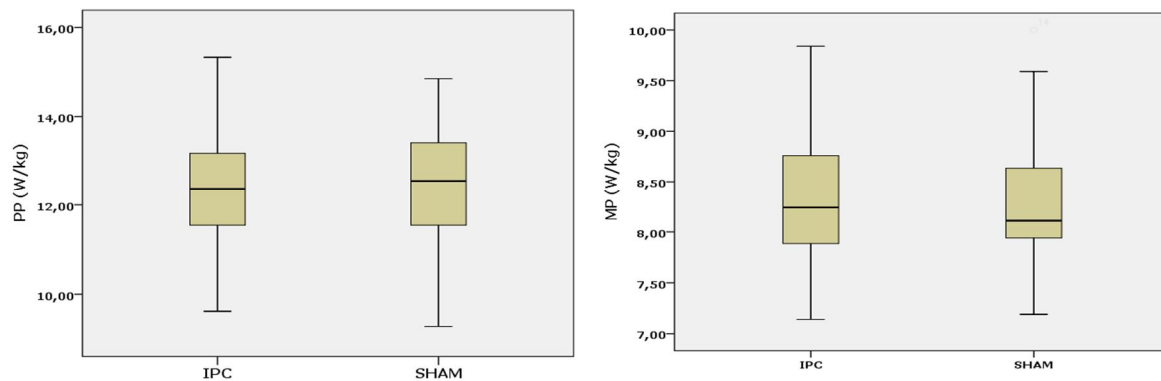
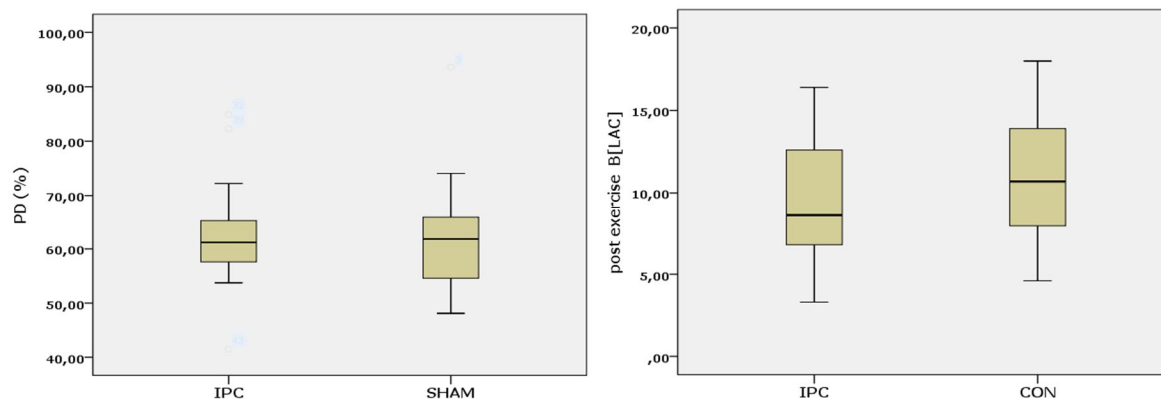
DISCUSSION

The main finding of the present study was that acute IPC treatment has no significant effect on anaerobic performance and blood lactate responses. Observations suggested that IPC has shown neither advantageous or nonadvantages impact on maximal cycling exercise performance and blood lactate accumulation in active males. However, the benefits of skeletal muscle metabolic efficiency which can occur during exercise performance has been suggested. Clinical studies have proved that the IPC inhibits ATP depletion (Ylitalo and Peuhkurinen, 2001), glycogen depletion (Andreas et al., 2011), and lactate accumulation during the ischemic condition (Murry et al., 1986; Yilmaz, 2019). In addition, IPC enhances muscle blood flow during ischemia by enhancing both arterial vasodilation and endothelial and microvascular functions (Veighey and MacAllister, 2012; Andreas et al., 2011). IPC may be acknowledged as an ergogenic aid to improve exercise and competitive performances and physiological responses to exercise stress. We hypothesized that IPC would increase anaerobic cycling performance and lower blood lactate accumulation.

The findings are not in line with studies showing that acute IPC treatment has both positive and negative impact on cycling performance in the literature (Caru et al., 2018). De Groot et al. investigated IPC treatment applied with 3 times \times 5 min IPC at 220 mmHg for maximal cycling performance ($\sim 1.6\%$) of healthy active male individuals. It has been clearly pointed out that the treatment of IPC beneficially alters the maximal cycling performance level in males. Those findings were later supported by Crisafulli et al. They found that IPC (with 3 times \times 5 min at 50 mmHg plus systolic blood pressure) was able to induce a significant enhancement of maximum power output in particular. Patterson et al. (2015) investigated bilateral upper body cycling performance for 12 \times 6 s cycle sprints in team sport athletes. They demonstrated significant improvement in the first 3 sprint peak power and mean power values.

Table 1. Lactate accumulations and WanT outputs of physically active male.

Variables	IPC		Control		t	p	d
	Mean	Std.	Mean	Std.			
Rest B[LAC] (mmol/L)	1.68	0.51	1.61	0.53	0.11	0.87	0.13
Post exercise B[LAC] (mmol/L)	9.53	3.74	10.99	4.20	1.43	0.17	-0.37
PP (W/kg)	12.39	1.29	12.40	1.52	0.06	0.95	-0.01
MP (W/kg)	8.32	0.70	8.30	0.68	-0.15	0.88	0.03
PD (%)	61.67	9.79	62.41	9.14	-0.33	0.75	5.83

**Figure 2.** Both IPC and control conditions Peak Power (PP) and Mean Power (MP) response of moderately trained university students. Values are expressed as mean \pm std.; n=24.**Figure 3.** Both IPC and control conditions Peak Power (PP) and Mean Power (MP) response of moderately trained university students. Values are expressed as mean \pm std.; n=24.

These results are supported by recent findings that determined the positive effect of IPC upon cycling exercise performance (Tocco et al., 2015; Cruz et al., 2016; Lindsay et al., 2017). Accordingly, as for the peak power improvement, a possible explanation of this positive effect may be explained with the fact that muscle residual strength of muscle contraction and muscle contractility capability which is affected by IPC (Ylitalo and Peuhkurinen, 2001). The protective mechanism of IPC is to open potassium-sensitive adenosine

triphosphate (ATP) potassium gates (KATP-Channels) of mitochondrial phosphorylation by triggering Gi proteins, thereby allowing calcium to accumulate in the mitochondria (Ylitalo and Peuhkurinen, 2001; Andreas et al., 2011). This accumulation plays a role in protein phosphorylation (Incognito et al., 2017). As a result, ATP synthesis increases compared to resting values. Accelerated ATP synthesis results in increased high-energy phosphate levels in intracellular areas (Andreas et al., 2011; Kocman et al., 2015; Lee et al., 2015; Incognito

et al., 2017). In this context, increased pre-exercise PCR storage level may increase anaerobic power and capacity output by improving the intensity and duration of the explosive exercise. In addition, it is known that IPC accelerates neurotransmitters into the cell by triggering the actions of the Gi protein (Lee et al., 2015). Neurotransmitters regulate the intracellular voltage level required for contraction in the skeletal muscle so that the muscle may respond more strongly and quickly to stimuli from neurons. This may be the other mechanism that can improve peak power. However, IPC's possible mechanism did not work in our investigation model.

Paxio et al. investigated the acute IPC treatment effects in male cyclist and they found a negative effect with a notable decrease in maximum power and mean anaerobic power on a Wingate test contrary to the abovementioned information. Gibson et al. observed a similar finding of this investigation. Negative effects have been reported in female participants' sprint performance with trivial (< 0.2) effect sizes. However, no significant effects were observed at any sprint speed in males. Similarly, it has been demonstrated that IPC treatment (3 x 5 min at 220 mmHg) did not affect maximal 10-m and 20-m sprinting performance (Thompson et al., 2018). Hittinger et al. observed no change in peak power during a graded cycling test. Lalonde and Curnier applied the 6 x 6 sec short repeated sprint and Wingate protocol after IPC 3 x 5min at 50 mmHg above systolic blood pressure; they reported no IPC effect in peak power and mean power. Our findings are similar in contrast to what we expected.

There was no statistically significant difference between the post blood lactate levels ($p > 0.05$). The similar effect of IPC has been reported previously (De Groot et al., 2010; Hittinger et al., 2014; Barbosa et al., 2015; Gürses et al., 2017). Paixão et al. (2014) showed that blood lactate levels were not affected by IPC during the repeated Wingate Test. It was seen that all the measurements after exercise were lower in the IPC group. This is because more oxygenation in the cell increases the return of pyruvic acid to alanine as a result of increasing endothelial vascular function (Ylitalo and Peuhkurinen, 2001; Veighey and MacAllister, 2012; Lee et al., 2015). This increases the use of hydrogen ions in the transport chain (Andreas et al., 2011). Thus, when the cell opens the potassium channels, calcium is stored in the mitochondria, and conditions such as the presence of anti-oxidant enzymes remain active while maintaining the intracellular pH level may accelerate lactate removal. Furthermore, no change in fatigue index calculated during repetitions can be explained by this mechanism and it can be said that this is due to the physiological effect of IPC.

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