Intermittent lower-limb occlusion enhances recovery after strenuous exercise

Article *in* Applied Physiology Nutrition and Metabolism · September 2012

DOI: 10.1139/h2012-101 · Source: PubMed

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Intermittent lower-limb occlusion enhances recovery after strenuous exercise

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Abstract: Repeated cycles of vascular occlusion followed by reperfusion initiate a protective mechanism that acts to mitigate future cell injury. Such ischemic episodes are known to improve vasodilation, oxygen utilization, muscle function, and have been demonstrated to enhance exercise performance. Thus, the use of occlusion cuffs represents a novel intervention that may improve subsequent exercise performance. Fourteen participants performed an exercise protocol that involved lower-body strength and power tests followed by repeated sprints. Occlusion cuffs were then applied unilaterally (2 × 3-min per leg) with a pressure of either 220 (intervention) or 15 mm Hg (control). Participants immediately repeated the exercise protocol, and then again 24 h later. The intervention elicited delayed beneficial effects (24 h post-intervention) in the countermovement jump test with concentric (effect size (ES) = 0.36) and eccentric (ES = 0.26) velocity recovering more rapidly compared with the control. There were also small beneficial effects on 10- and 40-m sprint times. In the squat jump test there were delayed beneficial effects of occlusion on eccentric power (ES = 1.38), acceleration (ES = 1.24), and an immediate positive effect on jump height (ES = 0.61). Thus, specific beneficial effects on recovery of power production and sprint performance were observed both immediately and 24 h after intermittent unilateral occlusion was applied to each leg.

Key words: ischemia, reperfusion, muscle function.

Résumé : Des cycles répétés d'occlusion vasculaire suivie d'une reperfusion mettent en œuvre un mécanisme protecteur atténuant les lésions cellulaires ultérieures. D'après des études, ces épisodes ischémiques améliorent la vasodilatation, l'utilisation de l'oxygène, la fonction musculaire et la performance physique. Dès lors, les manchons compressifs constituent une nouvelle approche susceptible d'améliorer une performance subséquente. Quatorze sujets participent à une séance d'exercices constituée d'épreuves de force et de puissance du bas du corps suivies de sprints répétés. On installe unilatéralement les manchons (2 × 3 min par jambe) pour exercer une pression de 220 mm Hg (intervention) ou de 15 mm Hg (contrôle). Puis les sujets refont la séance d'exercices immédiatement et 24 h plus tard. L'intervention suscite des effets bénéfiques retardés (24 h post) au cours de l'épreuve de saut avec contremouvement : on observe une récupération plus rapide des vélocités miométrique (ampleur de l'effet (« ES ») = 0,36) et pliométrique (ES = 0,26) comparativement à la condition de contrôle. On observe aussi un mince effet bénéfique sur le temps de performance aux 10 m et 40 m. Au saut en position accroupie, on observe un effet bénéfique retardé de l'occlusion sur la puissance pliométrique (ES = 1,38), l'accélération (ES = 1,24) et un effet positif immédiat sur la hauteur de saut (ES = 0,61). Ainsi, on observe des effets bénéfiques spécifiques sur la récupération de la production de puissance et sur la performance au sprint, et ce, immédiatement et 24 h après l'occlusion unilatérale intermittente dans chaque jambe.

Mots-clés : ischémie, reperfusion, fonction musculaire.

[Traduit par la Rédaction]

Introduction

It has been demonstrated that brief repeated periods of occlusion followed by reperfusion can mitigate the injurious effects of prolonged ischemia in cardiac muscles, as well as attenuate other cellular damage (Eisen et al. 2004; Iliodromitis et al. 2007). The cardioprotective effect of ischemic preconditioning was first described by researchers who demonstrated that multiple brief ischemic episodes resulted in a reduction in infarct size in canine hearts subsequently exposed to prolonged ischemia (Murry et al. 1986). Subsequently, the beneficial effects of cycles of ischemia and reperfusion have been observed in a number of mammalian species, including humans, with ischemic preconditioning

Received 17 October 2011. Accepted 5 June 2012. Published at www.nrcresearchpress.com/apnm on .

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eliciting “robust and reproducible” protective effects (Hausenloy and Yellon 2008). The mechanism of such protection, however, has not been fully elucidated.

It is known that remote application of an ischemic stimulus to the upper arm using a blood pressure cuff can produce cardioprotective effects in humans (Bøtker et al. 2010). Current knowledge suggests that cardioprotective effects of cycles of ischemia and reperfusion arise through activation of the reperfusion injury salvage kinase and survivor activating factor enhancement pathways (Gonon et al. 2011). Endogenous ligands, such as adenosine, bradykinin, prostaglandins, and opioids, have been implicated as initiating and mediating the protective effects of an ischemic intervention (Hausenloy and Yellon 2008; Li et al. 2009). Importantly, elevated adenosine levels can increase oxygen delivery and blood flow because of the dilation of resistance vessels. Interestingly, increased blood flow has been purported as a mechanism behind many recovery strategies via a repletion of ATP (Connolly et al. 2003) and metabolic waste product removal (Gill et al. 2006; Hamlin 2007; Higgins et al. 2011; Vaile et al. 2011).

Cycles of ischemia and reperfusion have also been associated with the preservation of ATP levels in canine myocardium, indicating a decreased energy demand (Jennings et al. 2001). Furthermore, it has been reported that an ischemic stimulus can improve muscle function (Lawson and Downey 1993) as well as exercise performance and maximal oxygen uptake in humans (de Groot et al. 2010; Jean-St-Michel et al. 2011). However, the potential for cycles of ischemia and reperfusion to stimulate the recovery process following exhaustive exercise has received little research attention. As a result of the reported effects of ischemia on blood flow, oxygen extraction, and muscle function, the present study examined the effectiveness of an alternating unilateral occlusive protocol as a recovery intervention to enhance subsequent exercise performance in healthy participants.

Materials and methods

Participants

Fourteen healthy individuals (10 males and 4 females; age, 32 ± 7 years; body mass, 76.4 ± 12.9 kg) volunteered to participate in this study. All participants were recreationally trained nonsmokers. Based on their medical history, all participants were free of contraindications that would preclude participation in strenuous exercise and gave their written informed consent. The Ethics Committee of the Waikato Institute of Technology approved the study protocol.

Protocol and measurements

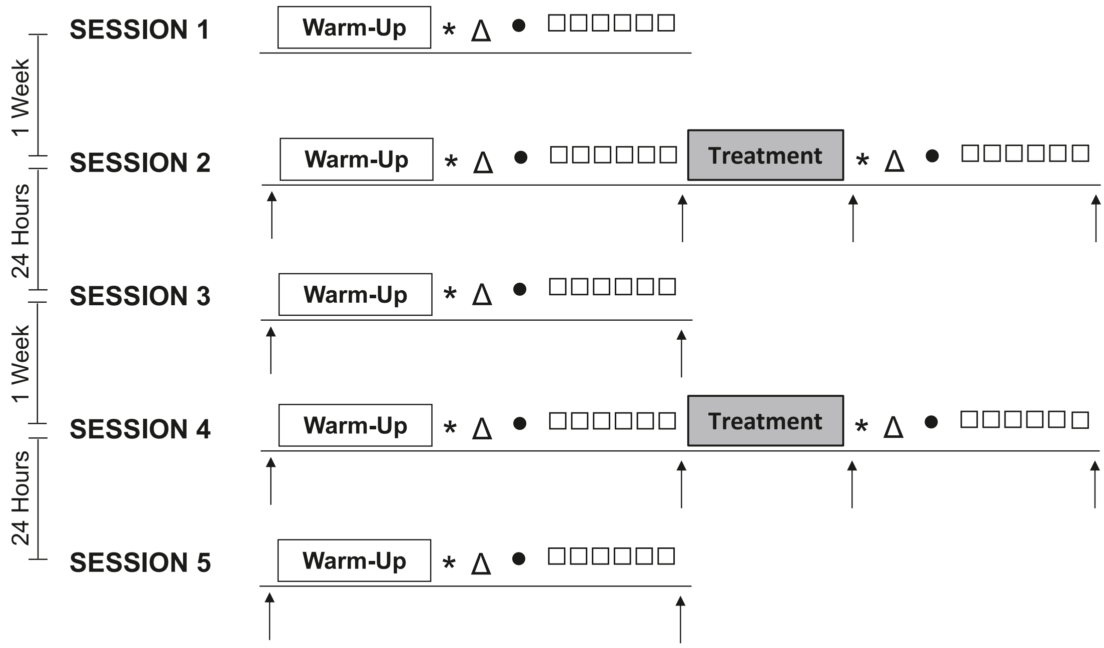
All participants refrained from alcohol and intense physical exercise for at least 24 h prior to testing. The exercise protocols were performed at the same venue and at the same time of day to minimize any confounding effects of daily biorhythms. On the days of testing, as much as possible, all participants ensured they had achieved a previous good night’s sleep and were hydrated and fed. Across the trials themselves, water was available and participants encouraged to stay well hydrated. To eliminate possible confounding training or familiarization effects, the application of 220 mm Hg occlusive recovery intervention and the 15 mm Hg control condition were assigned in a counterbalanced, cross-over design. Participants were not informed about the rationale of the study to reduce any placebo effect.

Each participant reported to the testing venue on 5 occasions (Fig. 1). The first visit (session 1) allowed the participants to become familiar with the standardized warm-up and exercise protocols. The standardized warm-up that was completed before every session consisted of a 400-m jog followed by dynamic stretches that targeted the muscles of the lower limbs (incorporating lunges, leg swings, and skipping) and lasted approximately 10 min. Session 1 was completed at least 1 week prior to the start of the exercise trial.

In session 2, following the warm-up, the participants performed a set of 3 squat jumps with a 90° knee angle. In a nonfatigued state the participants then performed a set of 3 countermovement jumps (CMJ). For the CMJ, participants lowered themselves into a self-selected half-squat position, and utilized the stretch-shortening cycle to jump explosively in an effort to achieve maximal height. Participants were instructed to sink to absorb their mass on landing. All jumps were performed with a 6-kg bar resting on the posterior deltoids at the base of the neck. The characteristics of power production (maximum and mean values for eccentric and concentric peak power, velocity, and acceleration as well as work, jump height, time to peak power, and time to peak velocity) during each squat jump and CMJ repetition were quantified with a Gymaware optical encoder (50-Hz sample period with no data smoothing or filtering; Kinetic Performance Technology, Canberra, Australia). The GymAware system recorded displacement-time data using a signal driven sampling scheme where position points were time-stamped when a change in position was detected, with time between samples limited to a minimum of 20 ms. The first and second derivate of position with respect to time was taken to calculate instantaneous velocity and acceleration, respectively. Acceleration values were multiplied by the system mass to calculate force, and the given force curve multiplied by the velocity curve to determine power. Mean values for force and power were calculated over the concentric and eccentric portions of each movement and peak values for velocity, force, power, and time taken to achieve these values were derived from each of the respective curves. Jump height was determined as the highest point on the displacement-time curve. The validity and reliability of the power optical encoder to provide data regarding peak and mean values of force and power as well as jump height have been reported previously (Crewther et al. 2011; Drinkwater et al. 2007; Taylor et al. 2010). Specifically, Taylor and colleagues (2010) reported coefficients of variation of day-to-day reliability of between 0.8% and 6.2%, with smallest worthwhile changes of between 1.9% and 4.3% for power, velocity, force, and height measurements with the GymAware system. System mass (mass of the bar plus body mass) was used for the calculation of maximal and mean concentric and eccentric power generated in the squat jump and countermovement jump exercises (Dugan et al. 2004). Immediately following the jump tests, the participants were instructed to perform a set of 6 leg press repetitions on a flywheel dynamometer (Concept 2, Vermont, USA) with the intention to produce maximal velocity.

The participants then performed 3 submaximal 40-m efforts at 50%, 70%, and 90% intensity before performing a ser-

Fig. 1. Experimental Design. *, Squat jump test; open triangle, counter movement jump test, filled circle, dynamometer leg press test; open square, 40-m sprint. Treatment: Occlusion cuff fitted unilaterally and inflated to 220 or 15 mm Hg for two 3-min periods per leg in a cross-over experimental design. Arrows represent the recording of perceived exertion (Borg 1987).



ies of 6 maximal 40-m sprints departing every 30 s. Dual-beam timing lights (Swift Performance Equipment, Australia) were used to monitor 10- and 40-m sprint times over the 6 repeated sprints. A rating of perceived exertion on a scale of 6 (no exertion) to 20 (maximal exertion) was assessed after each exercise protocol (Borg et al. 1987).

Cumulative sprint times for the 6 sprints are presented as, due to the variability inherent in fatigue indices, it has been suggested that total sprint time better reflects the ability of a participant to repeatedly produce maximal sprint efforts (Oliver 2009). Repeated 40-m sprints have been shown to be a reliable means of evaluating repeated sprint ability (Fitzsimmons et al. 1993; Glaister et al. 2009). Six repeated sprints were used as it has been suggested that less than 8 sprints should be used to avoid a pacing strategy (Oliver 2009) and greater than 3 sprints are required to observe a performance decrement (Balsom et al. 1992). Verbal encouragement and performance feedback was provided at each stage of the exercise protocol.

Immediately following the completion of the exercise protocol, participants adopted a comfortable supine position in a gymnasium and were fitted with a unilateral occlusion cuff (BJ Dare Medical Equipment, China) around the proximal portion of the leg as a recovery intervention. The cuff contained a pneumatic bag along its inner surface that was connected to a pressure gauge that was manually inflated to either 15 or 220 mm Hg for 3 min. The 220-mm Hg occlusion protocol was selected as a stimulus likely to induce lower limb ischemia, while the 15-mm Hg protocol was a control condition. The occlusion pressure was constantly monitored and the cuff was alternated to the contralateral leg for a further 3 min. This cycle was repeated twice for a total of 12 min, and both legs received an "ischemic dose" of 6 min per leg. The participants then repeated the squat jump, CMJ, dynamometer leg press, and 40-m sprint exercise protocol within 5 min of the cuff being removed. Twenty-four hours later, each participant returned and repeated the exercise protocol (session 3). One week later the participants returned again and repeated the 2 days of testing (sessions 4 and 5) using the alternate occlusion pressure treatment during the recovery period in a cross-over manner (Fig. 1).

Statistical analyses

The dependent variables were log-transformed before analysis and no observations were excluded as outliers. Back transformation provided estimates of mean effects as percentages and errors as coefficients of variations. Standardised changes in the mean of each measure were used to assess magnitudes of effects by dividing the changes by the appropriate between-participant standard deviations in the control condition. Magnitudes of the standardized effects were interpreted using thresholds of 0.2, 0.6, and 1.2 for small, moderate, and large, respectively (Hopkins et al. 2009). Standardised effects of between –0.19 and 0.19 were termed trivial. To make inferences about the true (large sample) value of an effect, the uncertainty in the effect was expressed as 90% confidence limits. The effect was deemed unclear if its confidence interval overlapped the thresholds for small positive and negative effects (Batterham and Hopkins 2006). Thresholds for assigning the qualitative terms to chances of substantial effects were <1%, almost certainly not; <5%, very unlikely; <25%, unlikely; 25%–75%, possibly; >75%, likely; >95%, very likely; and >99%, almost certain. The significance level was set at $p \leq 0.05$.

Results

The occlusion intervention of 220 mm Hg had a clear beneficial effect on the mean squat jump height immediately fol-

Table 1. Effect of occlusion as a recovery intervention on measures of lower-limb function.

	Immediately post–pre-intervention		24 h post–immediately postintervention		24 h post–pre-intervention	
Variable	%	Qualitative outcome	%	Qualitative outcome	%	Qualitative outcome
Peak values						
CMJ (W)	–0.1±8.1	Unclear	–0.9±7.4	Unclear	–1.0±11.2	Unclear
Squat jump (W)	*-5.2±8.5*	*Possibly –ve*	***9.8±12.6***	***Likely +ve***	4.1±13.9	Unclear
LP maximum strength (kg)	0.7±3.7	Trivial	–1.8±4.1	Likely trivial	–1.1±3.6	Likely trivial
LP average strength (kg)	–0.6±3.1	Trivial	–1.0±3.6	Likely trivial	–1.6±3.5	Likely trivial
LP total power (W)	–5.8±8.9	Possibly –ve	*10.4±11.7*	*Possibly +ve*	4.0±7.9	Possibly +ve
LP work (J)	1.3±3.8	Likely trivial	0.7±2.8	Trivial	2.0±3.8	Likely trivial
LP velocity ($m\cdot s^{-1}$)	0.0±2.2	Unclear	1.2±1.8	Likely trivial	1.1±2.0	Likely trivial
Mean values — CMJ						
Con PP (W)	–1.9±15.1	Unclear	*4.9±8.8*	*Possibly +ve*	2.9±17.7	Unclear
Ecc PP (W)	*–7.0±12.8*	*Possibly –ve*	11.6±20.8	Unclear	3.7±16.2	Unclear
Con PV ($m\cdot s^{-1}$)	–1.0±5.2	Unclear	***4.2±4.2***	***Likely +ve***	3.2±7.6	Unclear
Ecc PV ($m\cdot s^{-1}$)	***–32.4±54.5***	***Likely –ve***	***16.4±11.4****	*Possibly +ve*	–21.3±47.0	Unclear
Con PA ($m\cdot s^{-1}\cdot s^{-1}$)	5.6±23.9	Unclear	1.6±27.0	Unclear	7.2±12.3	Unclear
Ecc PA ($m\cdot s^{-1}\cdot s^{-1}$)	***–11.6±11.6****	***Likely –ve***	6.4±10.4	Unclear	–5.9±17.3	Unclear
Con work (J)	–3.9±7.8	Possibly –ve	2.3±4.9	Likely trivial	–1.7±10.6	Unclear
Jump height (cm)	0.7±12.2	Unclear	2.7±12.2	Unclear	3.5±9.0	Unclear
Time to PP (s)	*–9.5±15.1*	*Possibly –ve*	8.8±42.0	Unclear	–1.5±35.7	Unclear
Time to PV (s)	–4.7±12.9	Unclear	2.2±15.3	Unclear	–2.6±21.2	Unclear
Mean values — squat jump						
Con PP (W)	–2.9±11.6	Unclear	8.0±16.7	Unclear	2.2±18.1	Unclear
Ecc PP (W)	***–14.2±17.6***	***Likely –ve***	***38.5±14.7*****	***Almost certainly +ve***	14.5±24.9	Unclear
Con PV ($m\cdot s^{-1}$)	1.7±3.9	Unclear	–2.9±5.5	Unclear	–1.3±4.5	Unclear
Ecc PV ($m\cdot s^{-1}$)	–8.1±31.9	Unclear	–2.7±43.7	Unclear	–10.6±48	Unclear
Con PA ($m\cdot s^{-1}\cdot s^{-1}$)	5.7±16.9	Unclear	–0.4±28.5	Unclear	5.3±30.8	Unclear
Ecc PA ($m\cdot s^{-1}\cdot s^{-1}$)	–4.6±13.1	Unclear	***28.1±19.4****	***Very likely +ve***	***22.1±20.5***	***Likely +ve***
Con work (J)	3.6±4.8	Possibly +ve	***–5.4±2.9*****	***Likely –ve***	–2.0±3.1	Likely trivial
Jump height (cm)	***9.0±9.1***	***Likely +ve***	–2.5±9.7	Unclear	***6.9±8.5***	***Likely +ve***
Time to PP (s)	5.6±22.3	Unclear	–2.6±15.5	Unclear	2.8±18.2	Unclear
Time to PV (s)	0.8±7.9	Unclear	–2.5±8.4	Unclear	–1.6±9.6	Unclear

Note: Difference in the mean change (%) (intervention – control) ± 90% confidence limit. Italics represent effects that are non-trivial; Bold type indicates effects that are both likely and non-trivial. PP, peak power; PV, peak velocity; PA, peak acceleration; LP, leg press; CMJ, countermovement jump; Con, concentric; Ecc, eccentric. *, $p < 0.05$; **, $p < 0.01$.

lowing the occlusion intervention (effect size = 0.63, moderate effect) compared with the 15 mm Hg control condition. There were also likely detrimental effects on mean eccentric peak velocity and peak acceleration in the CMJ and mean eccentric peak power in the squat jump immediately post-intervention (Table 1). Other immediate effects were generally unclear or trivial.

Twenty-four hours after the occlusion intervention there was a likely beneficial effect on the rate of recovery of maximal power production (W) in the squat jump compared with the control condition (ES = 0.50, small effect). This delayed beneficial effect appeared to be more pronounced in males (ES = 1.02, moderate effect). In the squat jump test there were also large, clear beneficial effects of the occlusion intervention on the recovery of mean eccentric peak power and eccentric peak acceleration 24-h postexercise (Fig. 2). In the CMJ test there were clear beneficial effects of the occlusion intervention compared with the control condition on the mean concentric (ES = 0.36, small effect) and eccentric peak velocity (ES = 0.26, small effect) 24-h postexercise.

Total power produced in the dynamometer leg press (W) test 24 h postexercise was also clearly enhanced as a result of the occlusion intervention (ES = 0.30, small effect), and again the magnitude of this effect was greater in the male participants (ES = 0.68, moderate effect). Participants that performed the occlusion intervention also recovered at a greater rate compared with those in the control group when the cumulative 10- and 40-m sprint times 24 h post-intervention were assessed (Fig. 3). A likely detrimental effect of the occlusion intervention was observed in the change in the rate of recovery on the mean concentric work performed in the squat jump at the 24-h time point. Other delayed effects were generally unclear or trivial (Table 1).

Discussion

Cuff occlusion has previously been suggested as improving acute exercise performance; however, to our knowledge these benefits have not been proposed as a recovery strategy. We report that the treatment of both legs with intermittent unilateral cycles of occlusion at 220 mm Hg and reperfusion were effective at eliciting substantial beneficial effects on specific aspects of subsequent maximal exercise performed both im-

Fig. 2. Comparison of the eccentric peak power and peak acceleration before, after, and 24 h following the occlusion and control interventions when performing a static jump test. ES, Effect size difference between the post- and 24-h-poststatic jump tests. *, p = 0.004; †, p = 0.033.

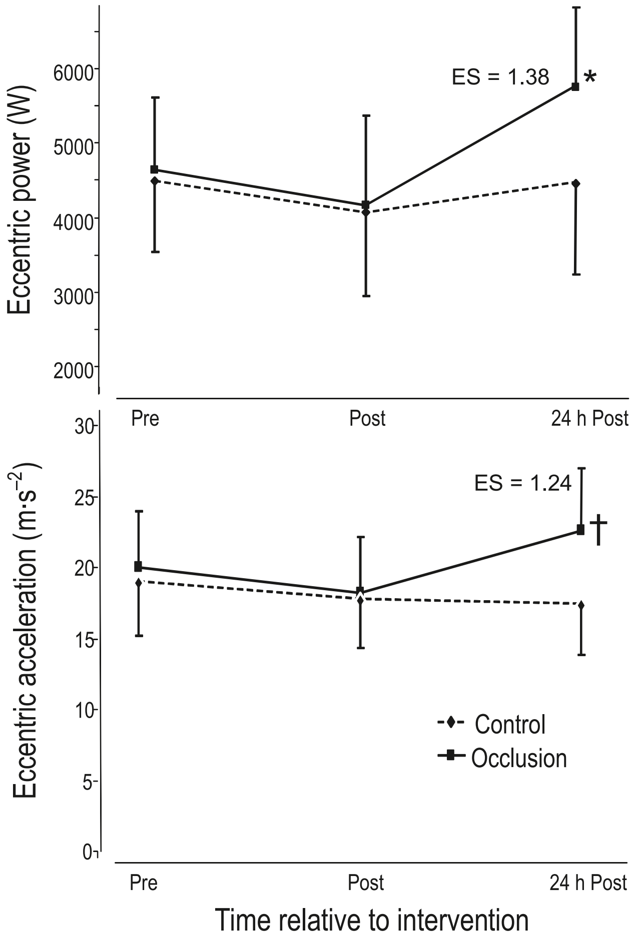
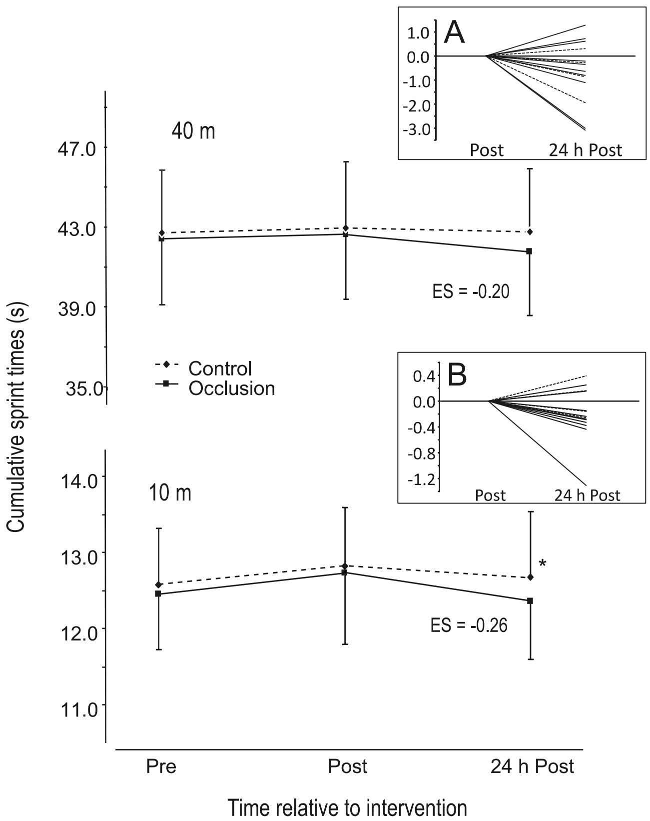


Fig. 3. Comparison of the cumulative 10- and 40-m sprint times before, after, and 24 h following the occlusion and control interventions. ES, Effect size difference between the change in mean post- and 24-h-post-sprint times. *, p = 0.06 difference in the change in the mean post- to 24-h-post-sprint time. Inset A: Individual change in 40-m sprint times difference between occlusion intervention and control. Inset B: Individual change in 10-m sprint times difference between occlusion intervention and control. Dashed lines in inserts represent female participants.



mediately following exposure and 24 h later. Previously, acute beneficial effects of ischemic preconditioning on exercise performance have been observed and speculatively attributed to vasodilation and improved oxygen delivery associated with increases in adenosine, activation of ATP-sensitive potassium channels (de Groot et al. 2010) and inhibition of afferent fatigue signalling increasing neural drive (Crisafulli et al. 2011). The preconditioning effects of adenosine are mediated via interaction A_3 adenosine receptors (Liu et al. 1994). Interestingly, stimulation of adenosine A_3 receptors has been reported to decrease creatine kinase responses to a muscle-damaging eccentric exercise protocol (Wang et al. 2010).

Our study showed small but meaningful improvements in cumulative 10- and 40-m sprint times 24 h after the occlusion intervention. Ischemic preconditioning has been associated with a reduced rate of anaerobic glycolysis, elevated glucose levels, and ATP preservation (Jennings, et al. 2001; Pang et al. 1995). Enhanced muscle oxygenation (Saito et al. 2004), vasodilation, and oxygen delivery have also been attributed to cycles of ischemia and reperfusion, which could have contributed to the observed results (de Groot et al. 2010). Functional sympatholysis associated with activation of ATP-sensitive potassium channels (Joyner and Thomas 2003) and changes in teleoanticipation (Noakes 2011) may have also contributed to the observed recovery of maximal sprint performance.

The occlusion intervention was also associated with specific delayed beneficial effects on both concentric and eccentric force produced during CMJ and squat jumps. Previous research has indicated that ischemia can enhance measures of skeletal muscle contractile function, such as maximal isometric force production, Ca^{2+} handling, and EMG amplitude in animal models (Kohin et al. 2001; Lawson and Downey 1993; Phillips et al. 1997). It should, however, be noted that a number of measures of lower limb function were impaired immediately after the occlusion intervention. These impairments were particularly apparent in the eccentric measures. The mechanism behind this impairment is not obvious; however, hypoxia has also been reported to impair muscle spindle reactivity and such alterations of sensorimotor control may have contributed to the observed effects on eccentric measures (Delliaux and Jammes 2006; Hoshikawa et al. 2010). It is also possible that the occlusive pressure was not well tolerated and a greater degree of familiarization or incremental application of this pressure may have alleviated the detrimental effects to some degree. It should also be noted that because of the large number of variables measured in non-highly trained participants, the possibility that type I errors are apparent cannot be discounted despite rigorous statistical attempts to present reliable and worthwhile effects.

In the current study, the majority of the beneficial effects of the occlusion intervention were observed 24 h after the occlusion intervention. This observation suggests that the increased blood flow because of the reperfusion phenomena and improved muscular oxygen utilization led to a more rapid return of muscle function. Alternatively, the observed results may have been partially mediated by a bimodal time course of ischemic preconditioning that has been described previously (Kuzuya et al. 1993). However, the physiological mechanism responsible for the effects on exercise performance were not examined in the current study as it was the intention to evaluate functional benefits of the occlusive intervention.

The occlusion pressure of 220 mm Hg was selected for the ischemic stimulus as this pressure has been suggested to restrict venous blood flow, cause pooling of blood in capacitance vessels distal to the belt, restrict arterial blood flow, and can elicit meaningful physiological responses in strength (Abe et al. 2005), sprint (Jean-St-Michel, et al. 2011), and endurance exercises (de Groot, et al. 2010). The cycle of 3 min occlusion and 3 min of reperfusion that comprised the occlusion intervention was repeated twice by both legs to give a total of a 6-min ischemic dose per leg. Unilateral occlusion has previously been demonstrated to elicit systemic cardioprotective effects (Bøtker, et al. 2010), although our occlusion intervention was designed to specifically target the musculature of the lower limbs. The 3-min cycles of occlusion and reperfusion were also selected to fulfil the duration threshold criterion that has been reported previously (Van Winkle et al. 1991). This threshold has been suggested to reflect the period of ischemia required to accumulate sufficient localized adenosine concentrations where the adenosine receptors are sufficiently populated to elicit a preconditioning effect. Importantly, a total ischemic stimulus of 4–6 min has been shown to be most effective at eliciting a protective effect in human myocardium, regardless of the number of cycles of ischemia and reperfusion (Ghosh et al. 2000). Although the occlusion intervention used in the current study was effective in improving specific aspects of subsequent exercise performance, it is worth noting that other protocols may be equally, or more, effective in eliciting effects of intermittent ischemia. Also, because of the study design we were also unable to identify the specific time course and duration of the effects observed.

Interestingly, although the small female sample size ($n = 4$) made it difficult to draw any firm conclusions regarding gender differences associated with the intervention, our data suggested that the observed beneficial effects of the occlusion protocol on exercise appeared to be more pronounced in the male subjects. While our participants would not be considered trained athletes, they were all currently participating in competitive or semi-competitive sports (e.g., football, touch rugby, and volleyball) and the females were of a similar training level to the males. In rats, it has been demonstrated that preconditioning required testosterone to increase heat shock protein 70 synthesis, which mediated delayed onset cardioprotection in the male via an androgen receptor-mediated mechanism (Liu et al. 2006). More recently, testosterone has been shown to confer cardioprotection by upregulating the α_1-adrenoceptors (Tsang et al. 2008). Thus, the greater effect of the occlusion intervention on specific aspects of performance in males in our study could speculatively be due to the presumably higher levels of testosterone.

Conclusion

The unilateral occlusive recovery intervention applied in the current study elicited both positive and negative effects on specific aspects of neuromuscular function. Importantly though, beneficial effects on functional measures of athletic performance, including repeated sprint ability and jump height, were observed 24 h after the intervention. Although the mechanism is yet to be defined, improved blood flow and enhanced efficiency of muscular oxygen utilization associated with cycles of lower limb occlusion and reperfusion may have contributed to a more rapid return of muscle function.

References

Abe, T., Kawamoto, K., Yasuda, T., Kearns, C.F., Midorikawa, T., and Sato, Y. 2005. Eight days KAATSU-resistance training improved sprint but not jump performance in collegiate male track and field athletes. Int. J. KAATSU Training Res. **1**(1): 19–23. doi:10.3806/ijktr.1.19.

Balsom, P.D., Seger, J.Y., Sjödin, B., and Ekblom, B. 1992. Physiological responses to maximal intensity intermittent exercise. Eur. J. Appl. Physiol. Occup. Physiol. **65**(2): 144–149. doi:10.1007/BF00705072. PMID:1396638.

Batterham, A.M., and Hopkins, W.G. 2006. Making meaningful inferences about magnitudes. Int. J. Sports Physiol. Perform. **1**(1): 50–57. PMID:19114737.

Borg, G., Hassmén, P., and Lagerström, M. 1987. Perceived exertion related to heart rate and blood lactate during arm and leg exercise. Eur. J. Appl. Physiol. Occup. Physiol. **56**(6): 679–685. doi:10.1007/BF00424810. PMID:3678222.

Bøtker, H.E., Kharbanda, R., Schmidt, M.R., Bøttcher, M., Kaltoft, A.K., Terkelsen, C.J., et al. 2010. Remote ischaemic conditioning before hospital admission, as a complement to angioplasty, and effect on myocardial salvage in patients with acute myocardial infarction: a randomised trial. Lancet, **375**(9716): 727–734. doi:10.1016/S0140-6736(09)62001-8. PMID:20189026.

Connolly, D.A.J., Brennan, K.M., and Lauzon, C.D. 2003. Effects of active versus passive recovery on power output during repeated bouts of short term, high intensity exercise. J. Sports Sci. Med. **2**: 47–51.

Crewther, B.T., Kilduff, L.P., Cunningham, D.J., Cook, C., Owen, N., and Yang, G.-Z. 2011. Validating two systems for estimating force and power. Int. J. Sports Med. **32**(4): 254–258. doi:10.1055/s-0030-1270487. PMID:21380970.

Crisafulli, A., Tangianu, F., Tocco, F., Concu, A., Mameli, O., Mulliri, G., and Caria, M.A. 2011. Ischemic preconditioning of the muscle improves maximal exercise performance but not maximal oxygen uptake in humans. J. Appl. Physiol. **111**(2): 530–536. doi:10.1152/japplphysiol.00266.2011. PMID:21617078.

de Groot, P.C.E., Thijssen, D.H.J., Sanchez, M., Ellenkamp, R., and Hopman, M.T.E. 2010. Ischemic preconditioning improves maximal performance in humans. Eur. J. Appl. Physiol. **108**(1): 141–146. doi:10.1007/s00421-009-1195-2. PMID:19760432.

Delliaux, S., and Jammes, Y. 2006. Effects of hypoxia on muscle response to tendon vibration in humans. Muscle Nerve, **34**(6): 754–761. doi:10.1002/mus.20633. PMID:16941658.

Drinkwater, E.J., Galna, B., McKenna, M.J., Hunt, P.H., and Pyne, D. B. 2007. Validation of an optical encoder during free weight resistance movements and analysis of bench press sticking point power during fatigue. J. Strength Cond. Res. **21**(2): 510–517. PMID:17530976.

Dugan, E.L., Doyle, T.L., Humphries, B., Hasson, C.J., and Newton, R.U. 2004. Determining the optimal load for jump squats: a review of methods and calculations. J. Strength Cond. Res. **18**(3): 668–674. PMID:15320681.

Eisen, A., Fisman, E.Z., Rubenfire, M., Freimark, D., McKechnie, R., Tenenbaum, A., et al. 2004. Ischemic preconditioning: nearly two decades of research. A comprehensive review. Atherosclerosis, **172**(2): 201–210. doi:10.1016/S0021-9150(03)00238-7. PMID: 15019529.

Fitzsimmons, M., Dawson, B., Ward, D., and Wilkinson, A. 1993. Cycling and running tests of repeated sprint ability. Aust. J. Sci. Med. Sport, **25**: 82–87.

Ghosh, S., Standen, N.B., and Galiñanes, M. 2000. Preconditioning the human myocardium by simulated ischemia: studies on the early and delayed protection. Cardiovasc. Res. **45**(2): 339–350. doi:10.1016/S0008-6363(99)00353-3. PMID:10728354.

Gill, N.D., Beaven, C.M., and Cook, C.J. 2006. Effectiveness of post-match recovery strategies in rugby players. Br. J. Sports Med. **40** (3): 260–263. doi:10.1136/bjsm.2005.022483. PMID:16505085.

Glaister, M., Hauck, H., Abraham, C.S., Merry, K.L., Beaver, D., Woods, B., and McInnes, G. 2009. Familiarization, reliability, and comparability of a 40-m maximal shuttle run test. J. Sports Sci. Med. **8**: 77–82.

Gonon, A.T., Jung, C., Yang, J., Sjöquist, P.-O., and Pernow, J. 2011. The combination of l-arginine and ischaemic post-conditioning at the onset of reperfusion limits myocardial injury in the pig. Acta Physiol. (Oxf.), **201**(2): 219–226. doi:10.1111/j.1748-1716.2010.02168.x. PMID:21241456.

Hamlin, M.J. 2007. The effect of contrast temperature water therapy on repeated sprint performance. J. Sci. Med. Sport, **10**(6): 398–402. doi:10.1016/j.jsams.2007.01.002. PMID:17339133.

Hausenloy, D.J., and Yellon, D.M. 2008. Preconditioning and postconditioning: new strategies for cardioprotection. Diabetes Obes. Metab. **10**(6): 451–459. doi:10.1111/j.1463-1326.2007.00762.x. PMID:17825079.

Higgins, T.R., Heazlewood, I.T., and Climstein, M. 2011. A random control trial of contrast baths and ice baths for recovery during competition in U/20 rugby union. J. Strength Cond. Res. **25**(4): 1046–1051. PMID:20661161.

Hopkins, W.G., Marshall, S.W., Batterham, A.M., and Hanin, J. 2009. Progressive statistics for studies in sports medicine and exercise science. Med. Sci. Sports Exerc. **41**(1): 3–13. doi:10.1249/MSS.0b013e31818cb278. PMID:19092709.

Hoshikawa, M., Hashimoto, S., Kawahara, T., and Ide, R. 2010. Postural instability at a simulated altitude of 5,000 m before and after an expedition to Mt. Cho-Oyu (8,201 m). Eur. J. Appl. Physiol. **110**(3): 539–547. doi:10.1007/s00421-010-1530-7. PMID:20549513.

Iliodromitis, E.K., Lazou, A., and Kremastinos, D.T. 2007. Ischemic preconditioning: Protection against myocardial necrosis and apoptosis. Vasc. Health Risk Manag. **3**(5): 629–637. PMID: 18078014.

Jean-St-Michel, E., Manlhiot, C., Li, J., Tropak, M., Michelsen, M. M., Schmidt, M.R., et al. 2011. Remote preconditioning improves maximal performance in highly-trained athletes. Med. Sci. Sports Exerc. **43**(7): 1280–1286. doi:10.1249/MSS.0b013e318206845d. PMID:21131871.

Jennings, R.B., Sebbag, L., Schwartz, L.M., Crago, M.S., and Reimer, K.A. 2001. Metabolism of preconditioned myocardium: Effect of loss and reinstatement of cardioprotection. J. Mol. Cell. Cardiol. **33**(9): 1571–1588. doi:10.1006/jmcc.2001.1425. PMID: 11549338.

Joyner, M.J., and Thomas, G.D. 2003. Having it both ways? Vasoconstriction in contracting muscles. J. Physiol. **550**(2): 333. doi:10.1113/jphysiol.2003.044628. PMID:12777450.

Kohin, S., Stary, C.M., Howlett, R.A., and Hogan, M.C. 2001. Preconditioning improves function and recovery of single muscle fibers during severe hypoxia and reoxygenation. Am. J. Physiol. Cell Physiol. **281**(1): C142–C146. PMID:11401836.

Kuzuya, T., Hoshida, S., Yamashita, N., Fuji, H., Oe, H., Hori, M., et al. 1993. Delayed effects of sublethal ischemia on the acquisition of tolerance to ischemia. Circ. Res. **72**(6): 1293–1299. doi:10.1161/01.RES.72.6.1293. PMID:8495557.

Lawson, C.S., and Downey, J.M. 1993. Preconditioning: state of the art myocardial protection. Cardiovasc. Res. **27**(4): 542–550. doi:10.1093/cvr/27.4.542. PMID:8324783.

Li, R., Wong, G.T., Wong, T.M., Zhang, Y., Xia, Z., and Irwin, M.G. 2009. Intrathecal morphine preconditioning induces cardioprotection via activation of delta, kappa, and mu opioid receptors in rats. Anesth. Analg. **108**(1): 23–29. doi:10.1213/ane.0b013e3181884ba6. PMID:19095826.

Liu, G.S., Richards, S.C., Olsson, R.A., Mullane, K., Walsh, R.S., and Downey, J.M. 1994. Evidence that the adenosine A3 receptor may mediate the protection afforded by preconditioning in the isolated rabbit heart. Cardiovasc. Res. **28**(7): 1057–1061. doi:10.1093/cvr/28.7.1057. PMID:7954592.

Liu, J., Tsang, S., and Wong, T.M. 2006. Testosterone is required for delayed cardioprotection and enhanced heat shock protein 70 expression induced by preconditioning. Endocrinology, **147**(10): 4569–4577. doi:10.1210/en.2006-0297. PMID:16794012.

Murry, C.E., Jennings, R.B., and Reimer, K.A. 1986. Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium. Circulation, **74**(5): 1124–1136. doi:10.1161/01.CIR.74.5.1124. PMID:3769170.

Noakes, T.D. 2011. Time to move beyond a brainless exercise physiology: the evidence for complex regulation of human exercise performance. Appl. Physiol. Nutr. Metab. **36**(1): 23–35. doi:10.1139/H10-082. PMID:21326375.

Oliver, J.L. 2009. Is a fatigue index a worthwhile measure of repeated sprint ability? J. Sci. Med. Sport, **12**(1): 20–23. doi:10.1016/j.jsams.2007.10.010. PMID:18078786.

Pang, C.Y., Yang, R.Z., Zhong, A., Xu, N., Boyd, B., and Forrest, C. R. 1995. Acute ischaemic preconditioning protects against skeletal muscle infarction in the pig. Cardiovasc. Res. **29**(6): 782–788. PMID:7656281.

Phillips, D.J., Petrie, S.G., Zhou, B.-H., Guanche, C.A., and Baratta, R.V. 1997. Myoelectric and mechanical changes elicited by ischemic preconditioning in the feline hindlimb. J. Electromyogr. Kinesiol. **7**(3): 187–192. doi:10.1016/S1050-6411(97)84627-5. PMID:20719704.

Saito, T., Komiyama, T., Aramoto, H., Miyata, T., and Shigematsu, H. 2004. Ischemic preconditioning improves oxygenation of exercising muscle in vivo. J. Surg. Res. **120**(1): 111–118. doi:10.1016/j.jss.2003.12.021. PMID:15172197.

Taylor, K., Cronin, J.B., Gill, N., Chapman, D.W., and Sheppard, J. 2010. Sources of variability in isoinertial jump assessments. Int. J. Sports Physiol. Perform. **5**(4): 546–558. PMID:21266738.

Tsang, S., Wu, S., Liu, J., and Wong, T.M. 2008. Testosterone protects rat hearts against ischaemic insults by enhancing the effects of α_1-adrenoceptor stimulation. Br. J. Pharmacol. **153**(4): 693–709. doi:10.1038/sj.bjp.0707624. PMID:18157169.

Vaile, J., O'Hagan, C., Stefanovic, B., Walker, M., Gill, N.D., and Askew, C.D. 2011. Effect of cold water immersion on repeated cycling performance and limb blood flow. Br. J. Sports Med. **45** (10): 825–829. doi:10.1136/bjsm.2009.067272. PMID:20233843.

Van Winkle, D.M., Thornton, J.D., Downey, D.M., and Downey, J. M. 1991. The natural history of preconditioning: cardioprotection depends on duration of transient ischemia and time to subsequent ischemia. Coron. Artery Dis. **2**: 613–619.

Wang, R., Urso, M., Zambraski, E.J., Rader, E.P., Campbell, K.P., and Liang, B.T. 2010. Adenosine A_3 receptor stimulation induces protection of skeletal muscle from eccentric exercise-mediated injury. Am. J. Physiol. Regul. Integr. Comp. Physiol. **299**(1): R259–R267. doi:10.1152/ajpregu.00060.2010. PMID:20427727.