วารสารวิทยาศาสตร์และเทคโนโลยีการกีฬา ปีที่ 21 ฉบับที่ 2, ธันวาคม 2564

Journal of Sports Science and Technology Volume 21, No. 2, 2021

Original article

THE EFFECT OF STATIC CORE MUSCLE EXERCISES ON RECOVERY AFTER HIGH-INTENSITY EXERCISE IN YOUNG HEALTHY MEN

Chakrit AMORASETH, Rungchai CHAUNCHAIYAKUL, Amornpan AJJIMAPORN, Kornkit CHAIJENKIL, and Suchada SAOVIENG*

College of Sports Science and Technology, Mahidol University, Nakhonpathom, THAILAND

ABSTRACT

This study aimed to evaluate the effectiveness of static core exercises on physiological markers of recovery after Wingate test. Young healthy males (N = 11) participated in this randomized cross-over designed study. Following baseline measurements, each subject performed the 30-sec Wingate anaerobic test which was followed by a 15-min of either static core exercises (4 set of plank and side plank with 10:20 sec work/rest ratio, repeated two rounds) or passive (resting) recovery. Blood lactate was measured at baseline, immediately after, and at 15 minutes of recovery. Cardiorespiratory variables were measured at baseline, immediately after and at 5, 10, and 15 minutes of recovery. Data was analyzed with repeated measured ANOVA (2 conditions x5 times). Results showed no significant differences between recovery interventions for blood lactate levels. However, significant differences for respiratory gas exchange variables were observed. VE/VCO_2 ratio was significantly higher in the passive group than that in the static core exercises at 5 and 10 min of recovery (passive = 36.77 ± 3.79, 37.10 ± 4.00, static = 32.71 ± 2.96, 32.32 ± 2.19, respectively, $p < 0.05$). While no difference in cardiac outputs among conditions were found, higher systemic vascular resistance in the passive group versus static group was detected at 5 min of recovery (573.17 ± 104.87, 417.68 ± 41.72 $dyn.s/cm^5$, $p < 0.05$). It is likely that passive group exhibited lower blood flow and higher metabolic acidosis during recovery period. These results indicate that static core exercise exerts its positive effects via respiratory and metabolic variables and somehow involves with cardiac function. Thus, the active recovery methods, via static core exercises, could potentially be applied for a brief intense exercise.

(Journal of Sports Science and Technology 2021; 21 (2):26-43)

(Received: 3 August 2021 , Revised: 7 December 2021, Accepted: 8 December 2021 2021)

Keywords: BLOOD ACIDOSIS / ACTIVE RECOVERY / V_E/VCO_2 RATIO / SYSTEMIC VASCULAR RESISTANCE

* Corresponding author: Suchada Saovieng, PhD.

College of Sports Science and Technology, Mahidol University Salaya, Phutthamonthon, Nakhonpathom, 73170, THAILAND

Email: mook.suchadasaovieng@gmail.com

วารสารวิทยาศาสตร์และเทคโนโลยีการกีฬา ปีที่ 21 ฉบับที่ 2, ธันวาคม 2564

Journal of Sports Science and Technology Volume 21, No. 2, 2021

INTRODUCTION

Intensive exercise is known to cause an accumulation of metabolic by-products resulting from high rate of anaerobic energy pathways, such as blood lactate and blood acidosis[1]. These metabolic by-products have long been proved to associate with fatigue and performance reduction, and widely used as biomarkers for recovery[1-4]. Typical recovery strategies being used include active and passive recovery methods. While passive recovery can be done at rest with rhythmic pneumatic compression, massage, and electrical stimulation[5-7], active recovery following an intense exercise has been well established to enhance performance restoration[8, 9]. Indeed, the active recovery was reported to be effective in reducing blood lactate level more than the passive one[5, 10-13]. The mechanisms behind this effect is unclear but may involve an increase in blood flow, and a recruitment of oxidative muscle fibers, thus enhancing lactate removal[14-16]. Although the active recovery mainly involved dynamic movement was extensive researches, the efficacy of a static movement remains unknown[6].

Core exercise has been originally developed to improve performance and prevent injury[17-20]. It involves the recruitment of two groups of local and global muscles of the trunk[18, 20]. Local group consists of deep muscles that act as stabilizer for lumbar and pelvic regions, preventing any injury that could occur during movement from impact of high force on spine [18, 20]. This exercise involves static core muscle training[17-19]. Global muscle group is located the outer layer of core muscles, and related to dynamic movement [18, 20]. This muscle group is considered to support the body and help transferring force between upper and lower parts[18]. Both muscle groups mostly consist of oxidative muscle fibers[21, 22], in which highly related to lactate removal via oxidation [16].

There are very few researches addressing on the use of core muscle exercises as an active recovery strategy, even though it is widely used for improving core stability and injury prevention. In one study, Navalta and Hrncir (2007) found that combined static and dynamic core exercises was more effective in reducing blood lactate level more compared with the passive rest [23]. However, blood acidosis is involved with recovery from high intensity exercise, since it may impair gluconeogenesis and oxidative phosphorylation in muscle[3, 4]. The effect of static core exercises on blood acidosis still unclear. Also, active recovery mostly involved with dynamic muscle contraction, the efficacy of static muscle contraction is unclarified yet. We hypothesize that static core exercises would enhance muscle recovery after high intensity exercise as determined by blood lactate, anaerobic performance, and respiratory parameters.

OBJECTIVE

To investigate the effect of static core muscle exercises on blood lactate, respiratory gas exchange, cardiac function, and anaerobic performance after the Wingate test.

HYPOTHESIS

Static core muscle exercises would improve blood lactate and respiratory gas exchange, cardiac function, and anaerobic performance in a greater extent than the passive rest.

METHODS

Subjects

Eleven healthy young male volunteered to participate in this randomized, controlled crossover study. The mean age, height, BMI, and weight were 20.36 ± 0.67 years, 174.91 ± 5.89 cm, 22.6 ± 1.3, and 69.09 ± 6.19 kg, respectively. Inclusion criteria included those with a history of active lifestyle (exercise longer than 30 minutes, at least 3 days/week), no cardiorespiratory and musculoskeletal problems, and passed trunk endurance plank test [24]. This study was approved by the Mahidol University Central Institutional Review Board, COA No. MU-CIRB 2020/107.2008.

Procedures

After giving an informed consent, experimental procedures, benefits, possible risks and self-preparatory instruction were explained to each subject. Each subject visited the laboratory for two occasions, at least 7 days apart, for randomized interventions (passive rest or static core exercises). On both visits, subject was allowed to rest for 10 to 15 minutes after the arrival. During resting, heart rate and blood pressure were measured to determine readiness of subjects before begin experiment. Then, a blood sample was collected from the fingertip for resting lactate level (Lactate scout analyzer, UK). Gas exchanges (VE/VCO_2, RER, VE, VO_2, VCO_2) were monitored throughtout the experiment using breath-by-breath mode via a face mask (CORTEX MetaLyzer®, Germany). Cardiac function variables (HR, CO, SVR) were non-invasively measured throughout the experiment (PhysioFlow®, Manotec, France). After baseline measurement, subjects performed the all-out 30 sec Wingate test on a cycle ergometer

(Cyclus 2, Germany). Following a 5-min warme up (at 50 rpm with unloaded), the resistance (kp) was increased to the predetermined workload (0.075 x subject's body weights (kg)). Each subject was instructed to perform and sustain their maximum speed and was verbally encouraged throughout the test. , Blood sample was obtained at 3-min post exercise with zero load at 50 rpm. Thereafter, subjects were asked to randomly picked up balls in a blinded box to specify an intervention of passive rest or static core exercises, each lasting for 15 minutes. Then, the blood lactate was colleted again and the 2nd Wingate test was repeated to observe the change in subsequent performance. Through out experiment session, subjects were not allowed to drink fluid. After all variables were measured, subject were given a 200 ml orange juice.

Recovery interventions

In this studypassive rest was performed in sitting position for 15 minutes with no movement, whereas static core exercises were carried out using modified protocol as previously described [23, 25].

In brief, static core exercises consisted of two exercises with plank and side plank. Plank was done in the prone position, with weight bearing on elbows and toes by keeping a back straight. Subject had to hold this position by 10 seconds and relax for 20 seconds for a set, repeated for 4 sets. Side plank was done on a side-lying position with weight bearing on feet and one forearm positioned directly below the shoulder, then lifting the hip until the body was in a straight line. Subject had to maintain this position for 10 seconds and relax for 20 seconds for a set, and repeated for 2 sets on both side. Subjects were instructed to repeat these static core exercises for two rounds with 1 minutes rest between round. Static core exercises were performed within 15 minutes. The static exercise protocol was modified from previous works [23, 25].

วารสารวิทยาศาสตร์และเทคโนโลยีการกีฬา ปีที่ 21 ฉบับที่ 2, ธันวาคม 2564

Journal of Sports Science and Technology Volume 21, No. 2, 2021

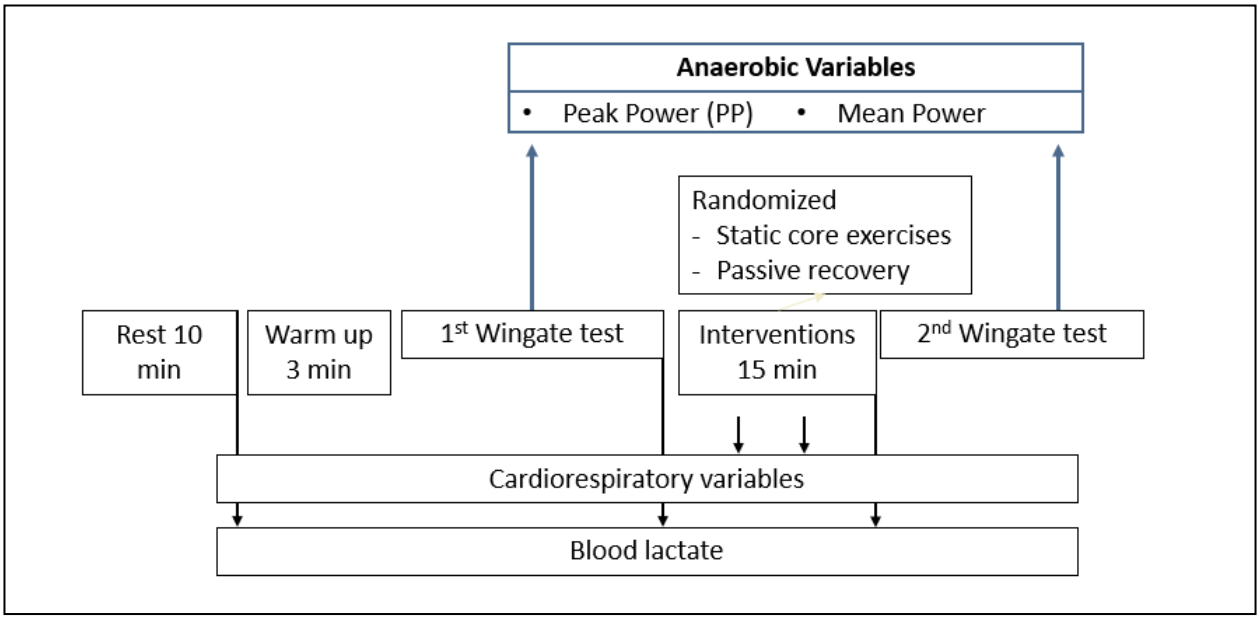

Figure 1. Experimental design

Statistical Analyses

Statistical analyses were conducted by JASP Version 0.14.1 (JASP team, 2020). Normality of data was checked with Shapiro-Wilk test. Blood chemistry data was analyzed using a 2 (conditions) x 3 (times) repeated measure ANOVA test. Anaerobic performance data was analyzed using a 2 (conditions) x 2 (times) repeated measure. Respiratory gas exchange and cardiac output data were analyzed using a 2 (conditions) x 5 (times) repeated measure ANOVA. Differences were identified using the Tukey test. All statistical significant was accepted at $p < 0.05$.

RESULTS

The mean VO_2 max of participants was 48.37 ± 5.93 ml/kg/min. The mean VO_2 at lactate threshold of participants was 22.61 ± 1.54 ml/kg/min.

For blood lactate concentration, there was no significant difference among conditions, but has significant difference for times effect ($F_{2,20} = 296.55$, $p<0.001$) (Table 1).

There were also no significant differences for main interaction effects for peak power and mean power output between conditions [Peak power, ($F_{1,10} = 0.062$, $p=0.808$); Mean power, ($F_{1,10} = 1.211$, $p=0.297$)] (Table 1).

วารสารวิทยาศาสตร์และเทคโนโลยีการกีฬา ปีที่ 21 ฉบับที่ 2, ธันวาคม 2564
Journal of Sports Science and Technology Volume 21, No. 2, 2021

Table 1. Blood Lactate and anaerobic performance parameters at rest (Pre), Immediately after the Wingate test (Post), and 15-min post intervention (15-min Post).

Blood lactate (mmol/L)			
Conditions	**Pre**	**Post**	**15-min Post**
Passive	1.72 ± 0.42	13.06 ± 1.51	10.33 ± 3.49
Static	1.78 ± 0.58	13.62 ± 2.13	9.14 ± 1.94

Anaerobic performance (Watts)				
Conditions	**Peak power**		**Mean power**	
	1st Wingate	**2nd Wingate**	**1st Wingate**	**2nd Wingate**
Passive	732.47 ± 95.40	725.45 ± 95.36	522.38 ± 51.50	551.26 ± 65.67
Static	724.57 ± 88.37	737.78 ± 78.19	542.30 ± 61.44	559.66 ± 60.03

Respiratory gas exchange was presented as the ventilatory equivalent of carbon dioxide (VE/VCO_2 ratio), respiratory exchange ratio (RER), minute ventilation (VE), oxygen consumption (VO_2), carbon dioxide production (VCO_2), and percentage of VO_2 relative to VO_2 at Lactate threshold. The measurement timepoints were baseline (pre), immediately post-exercise, and at 5-, 10-, and 15-minute post interventions (Int 5, Int 10, Int 15).

For the VE/VCO_2 ratio, the passive group showed significantly higher results compared with the static core exercises at 5 and 10 minutes of intervention [$F(2, 20) = 18.144$, $p < .001$]. Whereas, no significantly different was observed between groups at 15 minutes (Figure 2).

วารสารวิทยาศาสตร์และเทคโนโลยีการกีฬา ปีที่ 21 ฉบับที่ 2, ธันวาคม 2564

Journal of Sports Science and Technology Volume 21, No. 2, 2021

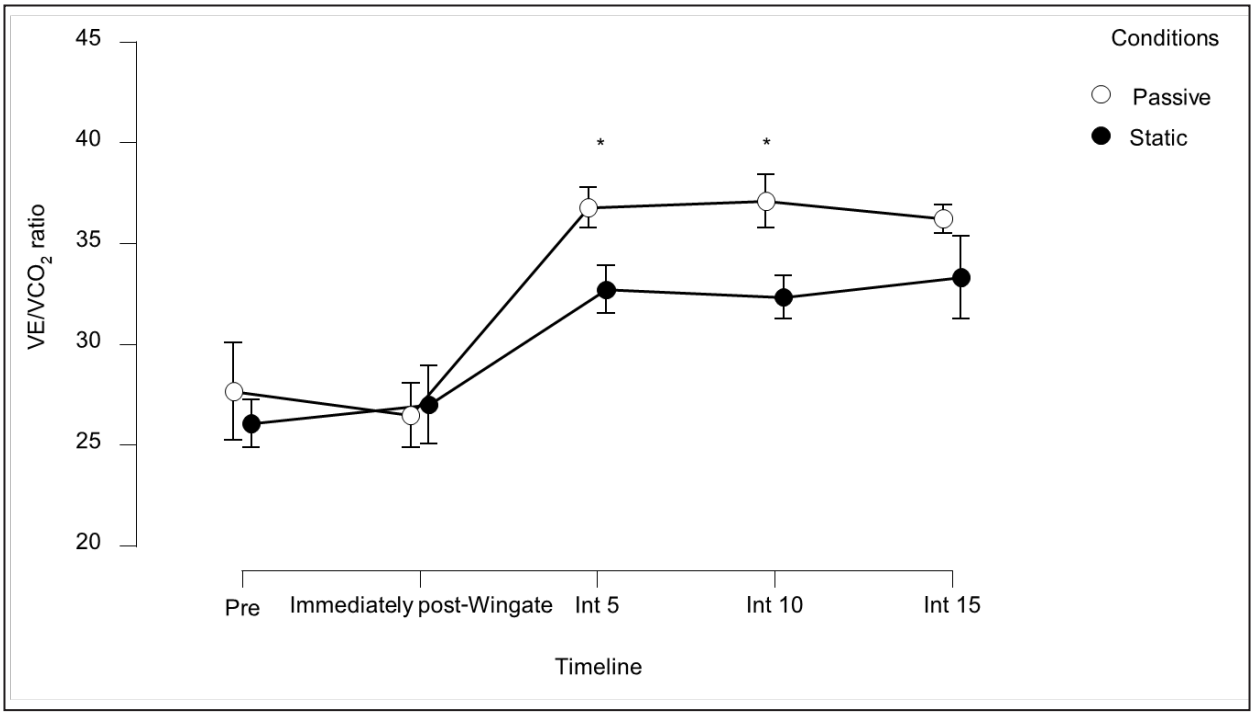

Figure 2. Ventilatory equivalent of carbon dioxide (VE/VCO2) at baseline (Pre), immediately post-exercise, and at 5, 10, and 15 post interventions (Int 5, Int 10, Int 15). Data were presented as means ± (SD). * Passive vs static, $p<0.05$.

Respiratory exchange ratio of passive group was significantly higher than the static group at 5 minutes post-intervention [$F(2, 20) = 7.36$, $p < .004$] (Figure 3).

วารสารวิทยาศาสตร์และเทคโนโลยีการกีฬา ปีที่ 21 ฉบับที่ 2, ธันวาคม 2564

Journal of Sports Science and Technology Volume 21, No. 2, 2021

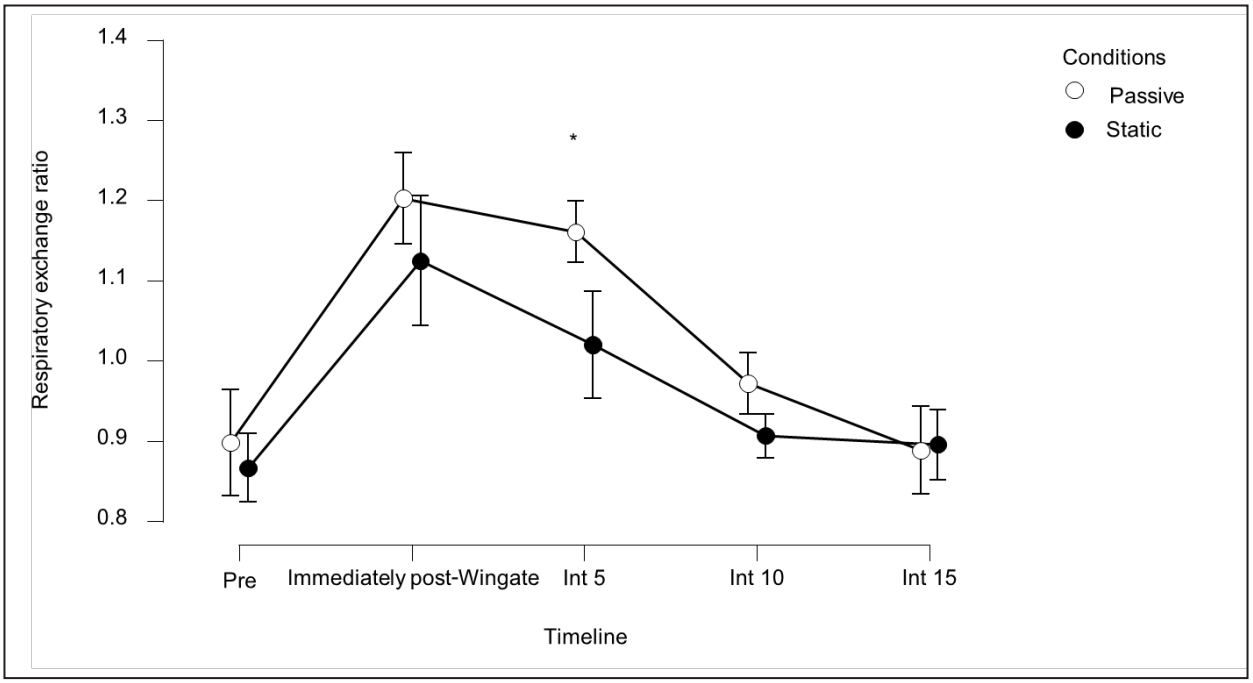

Figure 3. Respiratory exchange ratio (RER) at baseline (Pre), Immediately post-exercise, and at 5-, 10-, and 15-minutes post- interventions (Int 5, Int 10, Int 15). Data was presented as means (SD). * passive vs static, $p<0.05$.

Table 2. Respiratory variables at baseline (Pre), Immediately post-exercise, and at 5, 10, and 15 minutes pos-t interventions (Int 5, Int 10, Int 15). Data were presented as means (SD). * passive vs static, $p<0.05$. The data was analyzed with RM ANOVA (3 conditions x 5 timepoints). Tukey test was used for post hoc analysis with a p-value < 0.05.

Minute Ventilation (L/min)					
	Baseline	Immediate-post Wingate	Int 5	Int 10	Int 15
Static core exercise	11.59 ± 3.71	86.48 ± 16.79	31.44 ± 4.38	26.02 ± 4.84	16.39 ± 4.49
Passive rest	12.17 ± 4.10	90.61 ± 20.79	25.41 ± 1.91	20.05 ± 4.18	16.55 ± 3.88
There was no significant intervention effect for minute ventilation at $p<0.05$ [$F(1,10) = 3.047$, $p=0.111$]					

Oxygen consumption (ml/min)					
	Baseline	Immediate-post Wingate	Int 5 *	Int 10 *	Int 15 *
Static core exercise	397 ± 118.27	2551.22 ± 340.53	840.54 ± 100.49	781.65 ± 117.13	437.90 ± 90.11
Passive rest	379.11 ± 78.88	2591.75 ± 299.36	544.64 ± 36.55	455.20 ± 49.85	424.88 ± 65.83
There was significant intervention effect for oxygen consumption at $p<0.05$ [$F(1,10) = 38.472$, $p < 0.001$]					
Carbon dioxide production (ml/min)					
	Baseline	Immediate-post Wingate	Int 5	Int 10	Int 15
Static core exercise	344.33 ± 113.36	2897.23 ± 291	837.88 ± 80.80	696.95 ± 102.24	393.99 ± 86.89
Passive rest	341.87 ± 77.09	3112.22 ± 435.14	622.11 ± 57.42	447.79 ± 70.51	374.26 ± 84.44
There was no significant intervention effect for carbon dioxide production at $p<0.05$ [$F(1,10) = 3.097$, $p = 0.109$]					
* passive vs static, $p < 0.05$					

There was no significant difference in minute ventilation between both conditions.

we found statistically significant difference in VO_2 but not VCO_2. Passive group had lower VO_2 values than the static group through out recovery session (Table 2).

The intensity of interventions measured as percentage of VO_2 relative to VO_2 at lactate threshold in both conditions was found to be lower than 80%, which is the ideal intensity to remove lactate accumulation in blood stream according to a study of Menzie et al., in 2010 [(14)], represented via the dot line (Figure 4). At 5 and 10 minutes, this outcome in the passive group showed significantly lower than static group [$F(1, 10) = 70.62$, $p < .001$].

วารสารวิทยาศาสตร์และเทคโนโลยีการกีฬา ปีที่ 21 ฉบับที่ 2, ธันวาคม 2564

Journal of Sports Science and Technology Volume 21, No. 2, 2021

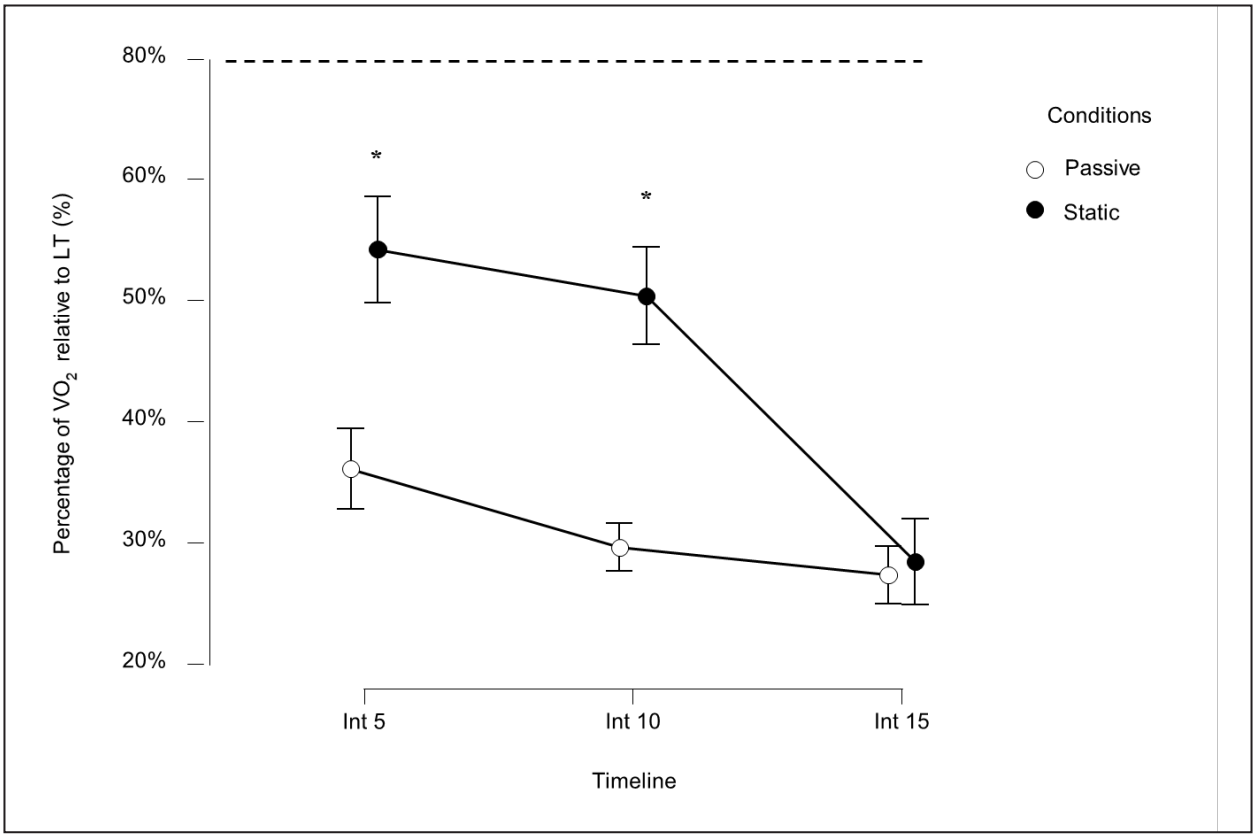

Figure 4. Percentage of oxygen consumption (VO_2) relative to VO_2 at lactate threshold at 5, 10, and 15 minutes of interventions (Int 5, Int 10, Int 15). Data were presented as means (SD).

* passive vs static, $p<0.05$. Dot line represents intensity which higher than 80% of VO2 relative to VO2 at lactate threshold. The data were analyzed with RM ANOVA (3 conditions x 3 timepoints). Post hoc analysis used Tukey test with p-value < 0.05.

At 10 minutes of interventions, passive group had lower HR compare to static core exercises group. For cardiac output (CO), static core exercises group had higher than passive group at 5 minutes of intervention (Table 3). Systemic vascular resistance (SVR) showed significant difference at 5 and 10 minutes post-interventions. Passive group had higher SVR than the static group at 5 minutes and 10 minutes (Figure 5).

วารสารวิทยาศาสตร์และเทคโนโลยีการกีฬา ปีที่ 21 ฉบับที่ 2, ธันวาคม 2564

Journal of Sports Science and Technology Volume 21, No. 2, 2021

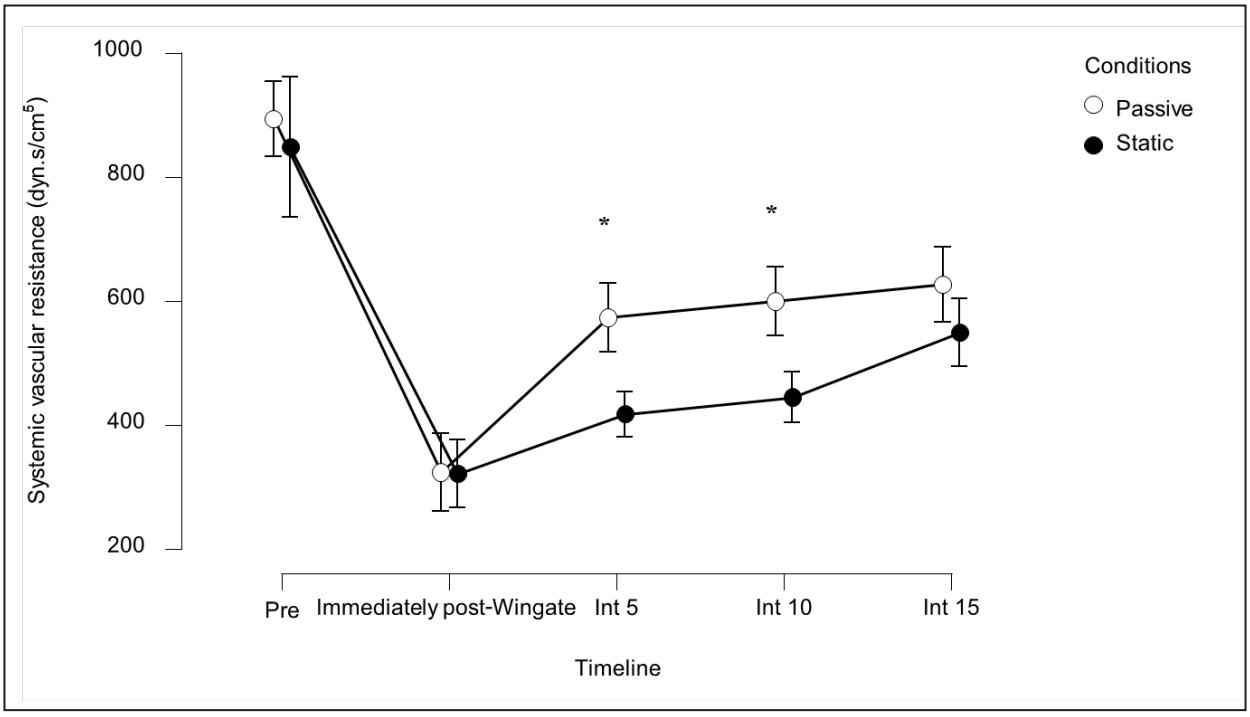

Figure 5. Systemic vascular resistance (SVR) at baseline (Pre), Immediately post-exercise, and at 5-, 10-, and 15-minutes post- interventions (Int 5, Int 10, Int 15). Data were presented as means (SD).

* passive vs static, $p<0.05$. The data were analyzed with RM ANOVA (2 conditions x 5 timepoints). Post hoc analysis used Tukey test with p-value < 0.05.

Table 3. Cardiac function variables at baseline (Pre), Immediately post-Wingate, and at minute 5, 10, and 15 of interventions (Int 5, Int 10, Int 15). Data was presented as means (SD). * passive vs static, $p<0.05$. The data was analyzed with RM ANOVA (3 conditions x 5 timepoints). Post hoc analysis used Tukey test with p-value < 0.05.

Heart rate (bpm)					
	Baseline	Immediate-post Wingate	Int 5	Int 10 *	Int 15
Static core exercise	79.97 ± 14.65	170.17 ± 10.97	120.53 ± 15.28	118.31 ± 13.68	109.11 ± 17.48
Passive rest	82.06 ± 10.96	170.72 ± 6.49	109.18 ± 11.52	105.64 ± 15.29	103.49 ± 14.13
There was significant intervention effect for heart rate at $p<0.05$ [$F(1,7) = 5.802$, $p= 0.047$]					

Cardiac output (L/min)					
	Baseline	Immediate-post Wingate	Int 5 *	Int 10	Int 15
Static core exercise	8.27 ± 2.08	20.95 ± 3.10	15.85 ± 2.18	14.43 ± 1.86	12.28 ± 2.19
Passive rest	7.46 ± 0.70	21.24 ± 5.43	11.76 ± 2.00	11.16 ± 1.62	10.81 ± 2.37
There was significant intervention effect for cardiac output at $p<0.05$ [$F(1,8) = 6.429$, $p= 0.035$]					
* passive vs static, $p < 0.05$					

DISCUSSION

This study aimed to assess the benefit of using static core muscle exercises as an active recovery tool, on blood acidosis accessed vis respiratory gas exchange. We found that after performing static core exercises for 15 minutes, blood lactate did not show significantly different among conditions. No significant difference was found for anaerobic performance. Interestingly, disproportion of carbon dioxide production relative to minute ventilation in passive condition was observed (VE/VCO_2 ratio). At 5 and 10 minutes of intervention, we found higher respiratory exchange ratio (RER) in passive condition compared to static core exercise condition. Also, we found that passive condition has higher systemic vascular resistance (SVR) compared to static core exercise condition.

We found similar blood lactate in both conditions measured at 15 minutes after the Wingate test. However, comparing to Navalta & Hrncir, they found lactate reduction measured at 5 minutes after Wingate test[23]. The contrasting outcome might be interfered with the different measurement timepoint. Another possibility is that, the intensity of static core exercises used in this study might not be enough to speed up lactate clearance. According to Menzies et al., they studied different intensity for oxidizing lactate after high intensity exercise. They reported that the most efficient intensity for lowering blood lactate is around 80% to 100% of VO_2 relative to VO_2 at lactate threshold[14]. In this study, we found that the intensity of static core exercises, determined by oxygen consumption values, was higher than the passive group at 5 and 10 minutes of intervention. However, none of all conditions were high enough to reach 80% of VO_2 at lactate threshold. The causation of this lower intensity is possibly caused by reduced work rest ratio of the static core exercises. The static core exercises protocol in this experiment was modified form the Parkhouse et al., 2011. Their workload was longer than 20 sec, compared to this study that use

10 sec. In addition, the reduction of the workload in this study may be due to the different fitness of subjects, since Parkhouse's work was conducted on university level sport athletes[25].

Anaerobic performance was not different among conditions. Also, we found no significant difference for anaerobic performance including peak power and mean power compared between the 1st Wingate (before performing intervention) and the 2nd Wingate (after performing intervention). This indicated that two consecutive rounds of the 30 seconds Wingate test were not enough to induce anaerobic performance reduction. This effect expressed similarly to the works conducted by Signorile et al., their work consisted of 8 rounds of 6-second power test on a cycle ergometer. They found that total work retrieved from the 1st round and the 2nd round were not difference. However, when comparing to the other rounds, the total work was reduced with trial number dependent fashion [26]. Also, Ahmaidi et al., studied effect of passive rest and active recovery between 6-second of force velocity test. They found that mean power of the 1st round and the 2nd round of the test was not difference between groups of passive rest and active rest [27]. Another study by Greenwood et al., who studied four repeated Wingate tests, showed that peak power of the 1st and the 2nd round of Wingate test were not difference. However, the power reduction from Wingate test occurred at the 3rd round and the 4th round[10]. Based on these findings, we concluded that only two consecutive rounds of Wingate test were not enough to induce anaerobic fatigue. Therefore, the effectiveness of core exercises on recovery from power reduction required a further investigation.

High intensity exercise is known to cause an increase production of hydrogen proton (H^+), leading to metabolic acidosis after exercises[1]. In this study, we found that core exercises had lower metabolic acidosis as estimated indirectly via respiratory gas exchange. During acidosis, bicarbonate (HCO_3) in blood traps hydrogen ion (H^+) and transforms it into carbon dioxide (CO_2), and excrete it out of body via expiration[28-30]. Therefore, blood acidosis can be attenuated with the increase in ventilation (VE) and CO_2 expiration (VCO_2). One respiratory gas exchange commonly used as a compensatory mechanism due to accumulation of H^+ is VE/VCO_2 Ratio [31]. When VE/VCO_2 ratio values increase, it represents that VE is increased disproportionately to carbon dioxide production. This refers to additional H^+ in circulation[29-32]. In this study, we successfully induced lactic acid production. The relationship between lactic acid production and respiration could be explained by excessive lactic acid production can lower blood pH. This effect was found to be reduced after performing active recovery compare to passive rest [33, 34]. We did not find the difference in lactic acid reduction in active recovery and passive control. However, higher CO_2 excretion was observed at minute 5 and 10 in the static core exercise group. In this regard, respiratory gas

exchange could be another monitoring tool for efficiency of active recover relative to blood acidosis. Since, the accumulation of blood acidosis could potentially inhibit oxidative phosphorylation, and gluconeogenesis during and after exercise resulting in reduction of substrate replenishing and delay recovery period [3, 4].

Respiratory gas exchange ratio (RER) is normally ranged between 0.7 to 1. These values indicate which substrate is being used for metabolism. If carbohydrate is being oxidized the ratio of CO_2 production divided by O_2 consumption will equally at 1.00. For fatty acid oxidation, the value will be 0.70. However, after high intensity exercise, RER can be increased higher than 1.00. This is because the additional carbon dioxide production comes from metabolic acidosis (H^+) that was converted by bicarbonate to CO_2 [35]. In our study, RER value for the passive group was higher than 1.00 and higher than in the static core exercises group during 5 minutes of recovery. This result supports and confers with the result obtained from VE/VCO_2 ratio, indicating that the passive group had higher blood acidosis compared to the core exercise group.

For cardiovascular function, SV and HR of passive group were lower than that of static core exercises group at 5- and 10- minute of recovery, respectively. Further, both were reduced throughout the recovery period, reflecting a gain in recovery.

Furthermore, passive group tended to show higher systemic vascular resistance (SVR) at 5- and 10-minute during recovery. Similar to this finding, Sinoway et al.[36] reported the relationship of muscle blood flow with muscle acidosis with calf vasoconstriction. They found that static contraction of forearm (30% of maximum voluntary contraction) for 2 minutes, following by occlusion for 3 minutes, caused an increase in SVR and a decrease of blood pH. This reduction of blood pH was maintained throughout the occlusion period with increasing SVR. Furthermore, they reported correlation of Δ% of vascular resistance with blood pH. The correlation was negative, which meant that the more vascular resistance (less blood flow), the lower blood pH [36]. Our finding confirmed with Sinoway et al result. Passive group who had higher VE/VCO_2 ratio also had higher SVR at 5 and 10 minutes of intervention compared with the active group.

Even though, this study used different design from Sinoway et al. but the mechanism of muscle blood flow interfered with blood acidosis may occur. Due to difficulty of measuring internal muscle blood flow especially, core muscles, the clarification of this relationship remains unclear, further investigation may require. Also, core muscles training might affect cardiorespiratory function. As reported by Datta et al.,[37] who found that core strength training every day for four weeks cloud increased VO_2 max of participants [37].

Although the SVR was used as a surrogated measure of total resistance of blood vessel, it does not seem to reflect the increases blood flow return to core muscles, thus a further study is still need to clarify this static core muscle exercise inducing effect. Another limitation of this study was blood chemical analysis, since we were not capable to measure several recovery markers in blood that can clarify the benefits of static core muscle exercises in terms of active recovery. Therefore, the evaluation of these blood markers may help us to better understand the mechanism underpinning its beneficial effects.

CONCLUSION

We concluded that static core muscle exercises may have the potential to be used as an active recovery strategy after training based on respiratory gas exchange analysis aligned with systemic vascular resistance result even the exercises were not involved with dynamic movement as typical active recovery. Even though, it seemed that physiological response of performing core exercise was last at 5 minutes according to the results. We suggest that performing static core exercises as an active recovery tool should be no longer than 5 minutes. Otherwise, the benefit would be decreased and shifted to the other purposes such as enhanced postural control rather than recovery. Since core exercises training widely incorporated in performance enhancement and injury prevention program, performing core exercises following muscular work may add recovery benefits into the training session.

REFERENCES

1. Robergs RA, Ghiasvand F, Parker D. Biochemistry of exercise-induced metabolic acidosis. Am J Physiol-Reg I. 2004;287(3):R502-R16.

2. Hoff J, Støren Ø, Finstad A, Wang E, Helgerud J. Increased blood lactate level deteriorates running economy in world class endurance athletes. J Strength Cond Res. 2016;30(5):1373-8.

3. Iles RA, Cohen RD, Rist AH, Baron PG. The mechanism of inhibition by acidosis of gluconeogenesis from lactate in rat liver. Biochem J 1977;164(1):185-91.

4. Jubrias SA, Crowther GJ, Shankland EG, Gronka RK, Conley KE. Acidosis inhibits oxidative phosphorylation in contracting human skeletal muscle in vivo. J Physiol. 2003;553(Pt 2):589-99.

5. Martin JS, Friedenreich ZD, Borges AR, Roberts MD. Acute Effects of Peristaltic Pneumatic Compression on Repeated Anaerobic Exercise Performance and Blood Lactate Clearance. J Strength Cond Res. 2015;29(10):2900-6.

6. Mor A, Ipekoglu G, Arslanoglu C, Acar K, Arslanoglu E. The Effects of electrostimulation and core exercises on recovery after high-intensity exercise. Int J Appl Exerc Physiol. 2017;6(4):46-53.

7. Gupta S, Goswami A, Sadhukhan AK, Mathur DN. Comparative study of lactate removal in short term massage of extremities, active recovery and a passive recovery period after supramaximal exercise sessions. Int J Sports Med. 1996;17(02):106-10.

8. Barnett A. Using recovery modalities between training sessions in elite athletes. Sports Med. 2006;36(9):781-96.

9. Bishop PA, Jones E, Woods AK. Recovery from training: a brief review: brief review. J Strength Cond Res. 2008;22(3):1015-24.

10. Greenwood JD, Moses GE, Bernardino FM, Gaesser GA, Weltman A. Intensity of exercise recovery, blood lactate disappearance, and subsequent swimming performance. J Sports Sci. 2008;26(1):29-34.

11. Miladi I, Temfemo A, Mandengué SH, Ahmaidi S. Effect of recovery mode on exercise time to exhaustion, cardiorespiratory responses, and blood lactate after prior, intermittent supramaximal exercise. J Strength Cond Res. 2011;25(1):205-10.

12. Neric FB, Beam WC, Brown LE, Wiersma LD. Comparison of swim recovery and muscle stimulation on lactate removal after sprint swimming. J Strength Cond Res. 2009;23(9):2560-7.

13. Ortiz RO, Jr., Sinclair Elder AJ, Elder CL, Dawes JJ. A Systematic Review on the Effectiveness of Active Recovery Interventions on Athletic Performance of Professional-, Collegiate-, and Competitive-Level Adult Athletes. J Strength Cond Res. 2019;33(8):2275-87.

14. Menzies P, Menzies C, McIntyre L, Paterson P, Wilson J, Kemi OJ. Blood lactate clearance during active recovery after an intense running bout depends on the intensity of the active recovery. J Sports Sci. 2010;28(9):975-82.

15. Gladden LB, Crawford RE, Webster MJ. Effect of blood flow on net lactate uptake during steady-level contractions in canine skeletal muscle. J Appl Physiol. 1992;72(5):1826-30.

16. Donovan CM, Pagliassotti MJ. Quantitative assessment of pathways for lactate disposal in skeletal muscle fiber types. Med Sci Sports Exerc. 2000;32(4):772-7.

17. Akuthota V, Ferreiro A, Moore T, Fredericson M. Core stability exercise principles. Curr Sports Med Rep. 2008;7(1):39-44.

18. Hibbs AE, Thompson KG, French D, Wrigley A, Spears I. Optimizing performance by improving core stability and core strength. Sports Med. 2008;38(12):995-1008.

19. Huxel Bliven KC, Anderson BE. Core stability training for injury prevention. Sports Health. 2013;5(6):514-22.

20. Willardson JM. Core stability training: applications to sports conditioning programs. J Strength Cond Res. 2007;21(3):979-85.

21. Bajek S, Bobinac D, Bajek G, Vranic TS, Lah B, Dragojevic DM. Muscle fiber type distribution in multifidus muscle in cases of lumbar disc herniation. Acta Med. 2000;54(6):235-42.

22. Rantanen J, Rissanen A, Kalimo H. Lumbar muscle fiber size and type distribution in normal subjects. Eur Spine J. 1994;3(6):331-5.

23. Navalta JW, Stephen P Hrncir JR. Core stabilization exercises enhance lactate clearance following high-intensity exercise. J Strength Cond Res. 2007;21(4):1305-9.

24. Tong TK, Wu S, Nie J. Sport-specific endurance plank test for evaluation of global core muscle function. Phys Ther Sport. 2014;15(1):58-63.

25. Parkhouse KL, Ball N. Influence of dynamic versus static core exercises on performance in field based fitness tests. J Bodyw Mov Ther. 2011;15(4):517-24.

26. Signorile JF, Tremblay LM, Ingalls C. The effects of active and passive recovery on short-term, high intensity power output. Can J Appl Physiol. 1993;18(1):31-42.

27. Ahmaidi S, Granier P, Taoutaou Z, Mercier J, Dubouchaud H, Prefaut C. Effects of active recovery on plasma lactate and anaerobic power following repeated intensive exercise. Med Sci Sports Exerc. 1996;28(4):450-6.

28. Salway JG. Medical biochemistry at a glance: John Wiley & Sons; 2012.

29. Wasserman K, Beaver WL, Sun X-G, Stringer WW. Arterial H+ regulation during exercise in humans. Respir Physiol Neurobiol. 2011;178(2):191-5.

30. Wasserman K, Cox TA, Sietsema KE. Ventilatory regulation of arterial H+ (pH) during exercise. Respir Physiol Neurobiol. 2014;190:142-8.

31. Neder JA, Berton DC, Muller PT, Elbehairy AF, Rocha A, Palange P, et al. Ventilatory Inefficiency and Exertional Dyspnea in Early Chronic Obstructive Pulmonary Disease. Ann Am Thorac Soc. 2017;14(Supplement_1):S22-S9.

32. Lindinger MI, Heigenhauser GJF. Effects of Gas Exchange on Acid-Base Balance. Compr Physiol. 2012;2(3):2203-54.

33. Coso JD, Hamouti N, Aguado-Jimenez R, Mora-Rodriguez R. Restoration of blood pH between repeated bouts of high-intensity exercise: effects of various active-recovery protocols. Eur J Appl Physiol. 2010;108(3):523-32.

34. Siegler JC, Keatley S, Midgley AW, Nevill AM, McNaughton LR. Pre-Exercise Alkalosis and Acid-Base Recovery. Int J Sports Med. 2008;29(07):545-51.

35. John P Porcari CXB, Fabio Comana. Exercise physiology: Philadelphia, PA : F.A. Davis Company; 2015. 905 p.

36. Sinoway L, Prophet S, Gorman I, Mosher T, Shenberger J, Dolecki M, et al. Muscle acidosis during static exercise is associated with calf vasoconstriction. J Appl Physiol. 1989;66(1):429-36.

37. Aashima Datta SS, Shivpriya. Effects of Core Strengthening on Cardiovascular Fitness, Flexibility and Strength on Patients with Low Back Pain. J Nov Physiother. 2014;4(2).