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Effects of Exposure to Normobaric Hyperoxia on the Recovery of Local Muscle Fatigue in the Quadriceps Femoris of Young People

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Abstract. [Purpose] Acute development of local muscle fatigue and recovery often become large issues on sports fields. This study aimed to identify the effects of normobaric hyperoxia on the recovery of local muscle fatigue. [Subjects] Eleven healthy males participated in this study, and they all completed two protocols in a random order. [Methods] Subjects performed single-leg isometric knee extension at 70% of their maximum voluntary isometric contraction (MVIC) for as long as possible. Each participant was subsequently treated with one of two recovery conditions: 20.9% O_2 or 30.0% O_2 for 30 minutes. Afterwards, they performed an identical isometric task to measure the extent of their recovery. The following parameters were used to assess the degrees of muscle fatigue: MVIC, endurance time, surface electromyography (sEMG) power spectra, and changes in hemoglobin concentration using near-infrared spectroscopy (NIRS). [Results] The treatment of 30.0% O_2 induced a significant recovery rate in MVIC compared to the 20.9% O_2. Additionally, the data revealed a significantly higher concentration of total hemoglobin after the 30.0% O_2 treatment than after the 20.9% O_2 treatment. [Conclusion] The results of this study suggest that recovery from acute muscle fatigue can be better facilitated under 30.0% normobaric hyperoxia than a normoxic condition. Therefore, for cases requiring quicker full recovery, treatment under 30.0% O_2 environment for 30 minutes is recommended.

Key words: Maximum voluntary isometric contraction, Surface electromyography, Near-infrared spectroscopy

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INTRODUCTION

Recovery has recently been recognized to be as important as training for athletes, since the degree of recovery affects subsequent performance and the potential for injury. Although there are a number of objectives in the recovery process, such as restoration of function and performance, tissue repair, restoration of muscle soreness, and psychological recovery[1)], the premise is that there exists background fatigue. In particular, acutely developing local muscle fatigue is likely to be daily encountered; thus, it is likely to be troubling for both athletes and coaches in training and competitions.

Local muscle fatigue represents a complex phenomenon encompassing various factors, such as structural and energetic changes in local muscle tissues, and changes in activity level and efficiency of the nervous system[2)]. Muscle fatigue, therefore, is also referred to as neuromuscular fatigue, which is divided into three headings based on the levels at which fatigue is induced: central fatigue, fatigue of the neuromuscular junction, and muscle tissue fatigue[3)]. When restorations at the three levels are complete, local muscle fatigue would be fully healed, theoretically, but techniques focusing on local muscle tissue, such as hydrotherapy and soft tissue massage are more common in practice and easier to use.

One of the major causes of muscle tissue fatigue has been widely reported to be metabolic acidosis induced by exercise in the tissue[4–7)]. Metabolic acidosis due to exercise is primarily caused by an increased flow of lactate, which is anaerobically generated in working muscles, into the blood stream, which results in a decline in the pH of blood[4, 6, 8)]. Since a sufficient supply of oxygen is needed to remove the lactic acid accumulated in muscle tissue[4, 7)], supplemental oxygen should enhance recovery from metabolic acidosis, that is to say reduce local muscle fatigue.

The use of exposure to hyperoxia is an ongoing research modality in the sports field, and the effects that have been reported are controversial. When trialed in athletic populations, breathing of hyperoxic gas (99.5% O_2 for 20 minutes in total) during recovery sessions after intermittent intense exercise improved participant's perceptions of recovery significantly more than those of a control group[9)]. Howev-

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er other uses of hyperoxic gas during the recovery period between interval repetitions (40% O_2 for 29 minutes in total, and 99.5% O_2 for 10 minutes in total) were reported to have no effect on the rate of perceived exertion (RPE)[10, 11]. Furthermore, pre-exposure to hyperbaric hyperoxia (HBO, 100% O_2 at 2.8 ATA for 60 minutes) increased maximum oxygen consumption during treadmill running[12]; however, another exposure to HBO (100% O_2 at 2.0 ATA for 60 minutes) did not enhance VO_2max during subsequent cycle ergometer exercise[13].

As discussed above, it remains unclear what effect supplemental oxygen has on activities and fatigue conditions. Additionally, studies reported to date have rarely considered the use of exposure to normobaric hyperoxia in the recovery phase between anaerobic exercises, and how such a recovery protocol might affect acutely developing local muscle fatigue.

This study aimed to identify the recovery effects of exposure to normobaric hyperoxia on local muscle fatigue affecting the quadriceps, and the degree of fatigue was measured using the following parameters: maximum voluntary isometric contraction (MVIC), endurance time, surface electromyography (sEMG) power spectra, and the status of blood circulation monitored by near-infrared spectroscopy (NIRS). We hypothesized that the use of normobaric hyperoxia would result in an enhanced recovery from local muscle fatigue as a result of improvement of metabolic acidosis in the muscle tissue.

SUBJECTS AND METHODS

Local muscle fatigue is often defined as "any exercise-induced reduction in the maximum capacity to generate force or power output"[14], and it simply indicates the point at which an individual is unable to perform a certain task. To observe this state, measuring the maximum voluntary contraction (MVC) at pre- and post-work is a general procedure and 'gold standard'[14]. Additionally, the endurance time until a task can no longer be performed is the simplest indication of fatigue level. Another way to determine local muscle fatigue is the use of electromyography (EMG) that enables the observation of the physiological changes in a fatigued muscle. It is well known that the EMG power spectrum shifts from high to low frequency bands as time passes and fatigue occurs[15], and the median frequency (MF) of the spectrum is used to measure the immediate onset of muscle fatigue. While NIRS does not reflect the condition of muscle tissue fatigue directly, it is used as an indirect parameter, as it expresses the state of muscle tissue oxygenation and blood circulation, which are related to muscle metabolic acidosis and muscle tissue fatigue. The experimental design implemented in this paper was a counterbalanced repeated-measures method involving two trials.

Subjects

Eleven healthy males having a mean age of 20 years (range 18–21 years) with no self-reported cardiovascular disease, respiratory disease, or musculoskeletal disorders, volunteered to participate in this study. Their body height and mass were 170.1 ± 5.4 cm and 63.2 ± 12.2 kg, respectively. None of them were competitive athletes, but all were physically active. In order to familiarize the subjects with the protocol, all performed it once before the study session. They were instructed to avoid alcohol consumption and vigorous exercise, which might have caused delayed onset muscle soreness, for at least 24 hours in advance of the experiment. Written informed consent was obtained from all participants before the initiation of the study, and the protocol was approved by the Ethics Committee of the Koriyama Institute of Health Sciences (approval number: R1107).

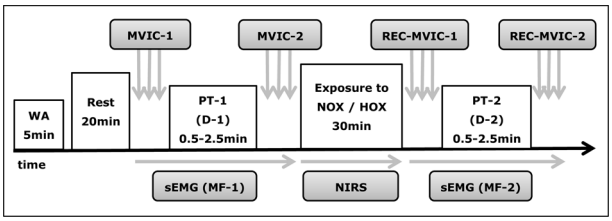

Fig. 1. Study protocol performed by subjects randomly participating in two recovery sessions on separate days: NOX and HOX between physical tasks 1 and 2. NOX = normobaric normoxia; HOX = normobaric hyperoxia; WA = warming-up; MVIC = maximum voluntary isometric contraction; REC-MVIC = MVIC after recovery session; PT = physical task; D = duration; sEMG = surface electromyography; MF = median frequency; NIRS = near-infrared spectroscopy.

Methods

Experimental Design. During the experimental sessions, all participants performed two protocols, with 30 minutes seated recovery under normobaric normoxia (NOX; 20.9% O_2), and normobaric hyperoxia (HOX; 30.0% O_2) in a room, where the concentration of oxygen was controlled, between fatiguing physical tasks (Fig. 1). Upon arrival in the experimental room, the subjects were required to complete a 5-minute warm-up for the right quadriceps followed by a 20-minute rest, which was set as a stabilization period. The subsequent physical task was designed to fatigue the quadriceps of the right leg by sustaining an isometric load of 70% of MVIC in each trial. Participants were asked to maintain this load for as long as they could, and this was set to induce exhaustion between 0.5 to 2.5 minutes. On the conclusion of the physical task, a 30-minute recovery period was provided before the start of the second identical physical task. In order to avoid carryover effects from each session, subjects performed these protocols on two separate occasions at least 5 days apart.

Maximum Voluntary Isometric Contraction. During each protocol, the MVIC was measured four times at MVIC-1 (pre-MVIC of the first physical task), MVIC-2 (post-MVIC of the first physical task), REC-MVIC-1 (pre-MVIC of the second physical task done immediately after the recovery period), and REC-MVIC-2 (post-MVIC of the second physical task) (Fig. 1). All subjects were seated on a device (T.K.K. 5710m, TAKEI, Japan) with a digital dyna-

mometer (F340, UNIPULS, Japan) which was used to measure the MVIC and test their muscle response to the endurance task. The subjects positioned their trunk and pelvis, which was secured up-right by a pelvic belt, and both hands gripped hand grips on either side. The starting position was with the right knee maintained at approximately 45° flexion against an unmovable leg bar. In this position, MVIC of the knee extensors was performed for three seconds three times, and the highest value of the three measurements was recorded as the MVIC.

Endurance Time. The muscle contraction time during the first and second physical tasks was recorded as D-1 and D-2, respectively, as one assessment of the local muscle fatigue[16].

Surface Electromyography Power Spectrum. The sEMG signals from the rectus femoris (RF) of the right quadriceps were recorded using a surface electrode bipolar configuration (special order product, Emu·ii Corporation, Matsumoto, Japan) with an inter-electrode distance of 30 mm. The electrodes were aligned parallel to the fibers on the middle portion of the muscle and the earth electrode was attached to the medial aspect of the right patella. To reduce the inter-electrode skin impedance to below 5 kΩ, the surface of the skin was wiped with alcohol swabs, and rubbed with an abrasive gel (SkinPure® Nihon Kohden, Tokyo, Japan). The sEMG data were sampled at 1024 Hz, and digitally filtered by a 15–500 Hz bandpass filter using BIMUTAS software (Nihon Kissei Comtech, Matsumoto, Japan). The sampling data were then divided into 10 equal epochs (named sample number 1 to 10) to normalize the length of data for group comparison. For frequency analysis, fast Fourier transforms (FFT) of the first 1 second of each epoch's signal were performed and the power spectrum of each epoch was derived. sEMG analysis has been used in the manner to measure the degree of local muscle fatigue during isometric exertions[15]. The median frequency (MF), which is known to be less affected by noise and more appropriate for assessing muscle fatigue, was then calculated for each sample. Each MF of sample number 2–10 was, then, normalized to the value of sample number 1 to quantify the degree of muscle fatigue over time, which is shown by a reduction in the median frequency. These signals were collected during the first and second physical tasks as MF-1 and MF-2, respectively.

Near-Infrared Spectroscopy. The status of blood circulation in the right RF muscle was monitored using NIRS (model NIRO 200, Hamamatsu Photonics, Hamamatsu, Japan). NIRS data were recorded during the 30-minute seated recovery time. The separation distance between the light source and photodetector was 40 mm, and the probe was positioned over the RF muscles just below the sEMG electrode, parallel to the major axis of the thigh. Light photons pass through the tissue and are collected by detectors with optical filters set at 775, 810, and 850 nm, in order to estimate changes of oxygen concentration (oxyhemoglobin [Oxy], deoxyhemoglobin [Deoxy], and total hemoglobin [THb]) from the baseline at the start. These changes are calculated according to the Beer-Lambert law[17, 18]. All values were continuously recorded at 0.2 Hz during the 30 minutes of recovery, and the mean value of each minute was analyzed.

Table 1. Normalized mean values (%, ± SEM) of maximum voluntary isometric contraction (MVIC) in NOX and HOX before (MVIC-1 and MVIC-2) and after (REC-MVIC-1 and REC-MVIC-2) 30 minutes recovery time

	MVIC-1	MVIC-2	REC-MVIC-1	REC-MVIC-2
NOX	100.0 (0.0)	84.6 (3.4)*	89.7 (2.9)	78.2 (4.1)*
HOX	100.0 (0.0)	86.8 (3.5)*	99.8 (3.4)*†	86.7 (4.3) *†

Significantly different (*$p < 0.05$) from the value of MVIC-1 within the same group, and significantly higher (†$p < 0.05$) in HOX than NOX.

Data are presented as mean ± SEM. Two-way (recovery environment, i.e. [NOX] or [HOX] × time) analysis of variance (ANOVA) with repeated measures was used to analyze the data for MVIC, MF, Oxy, Deoxy, and THb. When significant differences were detected by two-way ANOVA, one-way ANOVA with repeated measures and the paired t-test was additionally performed to detect significant changes from the start, and significant differences between NOX and HOX, respectively. Significant differences among mean values at $p < 0.05$ were then detected by Tukey's post-hoc test following two-way ANOVA, and Dunnett's post-hoc test following one-way ANOVA. The paired t-test was also used to analyze the data for the sustained period of the physical task. SPSS for Windows version 21.0 was used, and statistical significance was accepted for values of $p < 0.05$ in all analysis.

RESULTS

The effect of exposure to HOX on the recovery of MVIC is shown in Table 1. Values of MVIC were normalized to MVIC-1 to quantify the fatigability and recovery rates. Significant decreases ($p < 0.05$) at MVIC-2 and REC-MVIC-2 were shown in both HOX (100% to 86.8 ± 3.5% and 99.8 ± 3.4% to 86.7 ± 4.3%, respectively) and NOX (100% to 84.6 ± 3.4% and 89.7 ± 2.9% to 78.2 ± 4.1%, respectively). At REC-MVIC-1, the MVIC was measured immediately after the recovery period. There was a significant difference ($p < 0.05$) between HOX and NOX, and it can be seen that the MVIC of HOX had fully recovered (99.8 ± 3.4%), although that of NOX had a lower recovery rate (89.7 ± 2.9%) compared to the rate of MVIC-2.

The sustained time of muscle contraction at 70% of individuals' MVIC did not differ between either D-1 and D-2, or HOX and NOX. The times were 74.2 ± 7.2 sec at D-1 and 70.7 ± 9.1 sec at D-2 in HOX, and 75.6 ± 9.0 sec at D-1 and 74.0 ± 9.2 sec at D-2 in NOX.

MF during the 30-minute recovery session under the HOX and NOX conditions showed significant reductions in median frequency ($p < 0.05$) towards the end of physical task (MF-1 in HOX; 70.9 ± 3.6 Hz to 57.6 ± 3.9 Hz, MF-2 in HOX; 70.9 ± 3.3 Hz to 61.4 ± 4.1 Hz, MF-1 in NOX; 70.6 ± 3.9 Hz to 57.4 ± 4.1 Hz, MF-2 in NOX; 70.7 ± 3.5 Hz to 60.1 ± 3.9 Hz), however, there was no significant difference between either MF-1 and MF-2, or HOX and NOX.

Table 2. Time course changes in mean values (a.u., ± SEM) of oxy-hemoglobin (Oxy), deoxy-hemoglobin (Deoxy), and total hemoglobin (THb) of NOX and HOX during the 30-minute recovery sessions

	Oxy		Deoxy		THb	
time (min)	NOX	HOX	NOX	HOX	NOX	HOX
1	−0.9 (3.4)	6.0 (3.7)	3.1 (2.7)	1.7 (4.4)	2.2 (5.5)	7.7 (3.8)
2	0.5 (4.8)	12.6 (7.1)	1.7 (3.2)	2.4 (6.1)	2.2 (6.9)	15.0 (6.1)
3	4.9 (6.9)	20.5 (9.1)	2.5 (3.7)	−0.8 (6.5)	7.5 (8.6)	19.7 (7.2)
4	2.7 (7.7)	23.3 (10.1)	2.4 (4.2)	−3.5 (7.3)	5.0 (9.9)	19.8 (6.7)
5	1.3 (7.7)	30.0 (9.6)*	0.4 (6.1)	−1.6 (7.0)	1.7 (12.8)	28.3 (6.6)
6	4.8 (7.9)	33.4 (9.8)*	0.2 (6.4)	−2.4 (5.8)	5.0 (13.5)	31.0 (7.1)
7	7.1 (8.7)	39.3 (11.1)*	−1.6 (5.8)	−4.5 (6.7)	5.5 (14.1)	34.8 (8.2)*
8	2.0 (9.2)	40.2 (10.6)*	1.3 (5.4)	−2.3 (6.8)	3.3 (13.8)	37.9 (7.3)*
9	0.7 (9.6)	40.6 (10.2)*†	3.0 (5.6)	1.0 (7.1)	3.7 (14.5)	41.6 (7.0)*
10	3.5 (12.0)	38.4 (10.0)*†	2.1 (5.7)	2.2 (7.4)	5.5 (16.6)	40.7 (7.3)*
11	1.3 (13.5)	37.0 (10.2)*	4.4 (4.9)	2.9 (7.7)	5.7 (17.1)	39.9 (7.0)*
12	1.6 (13.8)	40.9 (10.4)*	0.6 (4.9)	1.8 (7.7)	2.2 (17.7)	42.7 (7.3)*
13	−0.4 (13.5)	46.3 (9.2)*†	2.2 (5.4)	2.9 (7.2)	1.8 (17.4)	49.2 (6.6)*†
14	1.6 (12.6)	47.0 (9.3)*†	−2.1 (6.9)	7.3 (7.8)	−0.5 (18.1)	54.3 (6.1)*†
15	−6.0 (12.2)	41.5 (9.3)*†	−0.3 (6.6)	9.7 (7.9)	−6.3 (17.0)	51.2 (7.7)*†
16	−0.3 (12.9)	43.2 (9.5)*†	−2.9 (7.3)	6.8 (8.0)	−3.3 (18.0)	50.0 (8.1)*†
17	0.8 (13.9)	50.3 (10.6)*†	−3.3 (7.9)	4.5 (8.6)	−2.5 (19.6)	54.8 (8.5)*†
18	2.9 (14.0)	51.4 (10.8)*†	−2.8 (8.0)	5.2 (8.9)	0.1 (20.0)	56.6 (7.5)*†
19	1.5 (14.0)	52.5 (10.3)*†	−1.9 (8.1)	4.3 (8.9)	−0.4 (19.7)	56.8 (7.7)*†
20	0.8 (14.7)	52.2 (11.2)*†	−0.1 (7.1)	3.5 (9.7)	0.7 (19.7)	55.7 (9.3)*†
21	4.0 (14.1)	54.9 (12.2)*†	−2.0 (7.9)	0.7 (10.3)	2.0 (19.9)	55.6 (9.5)*†
22	8.9 (14.8)	53.5 (11.5)*†	−1.6 (8.0)	5.2 (9.3)	7.3 (20.1)	58.6 (8.6)*
23	9.7 (15.8)	54.2 (10.9)*	−3.4 (7.5)	10.1 (9.3)	6.4 (20.5)	64.4 (9.9)*†
24	7.3 (15.4)	55.8 (12.3)*	−1.7 (7.9)	5.1 (10.2)	5.6 (20.2)	61.0 (11.0)*
25	5.3 (13.8)	54.1 (11.3)*†	−2.0 (7.00)	5.1 (10.3)	3.3 (17.8)	59.2 (10.4)*†
26	−5.3 (15.9)	53.4 (10.7)*‡	2.8 (8.3)	4.8 (10.4)	−2.5 (16.7)	58.2 (10.0)*†
27	−1.9 (16.1)	58.4 (12.2)*‡	2.0 (7.8)	3.7 (10.6)	0.1 (16.8)	62.1 (9.9)*‡
28	1.4 (16.4)	56.5 (11.1)*†	3.8 (7.7)	7.9 (10.0)	5.2 (17.6)	64.3 (8.9)*†
29	7.8 (15.7)	55.1 (11.1)*†	0.8 (8.7)	8.8 (10.2)	8.6 (18.6)	63.8 (10.8)*†
30	4.1 (14.8)	50.5 (12.4)*†	−4.1 (9.0)	7.6 (10.8)	0.0 (19.0)	58.1 (12.5)*†
ANOVA	$F_{(1, 29)} = 4.767$, $p < 0.01$		$F_{(1, 29)} = 0.958$, $p = 0.412$		$F_{(1, 29)} = 5.172$, $p < 0.01$	

Significantly different (*$p < 0.01$) from the value of the 1st minute within the same group, and significantly higher (†$p < 0.05$, ‡$p < 0.01$) in HOX than NOX. a.u. = authorized unit.

The changes in Oxy, Deoxy, and THb of the RF muscle during the 30 minutes of recovery time under the HOX and NOX conditions are presented in Table 2. Oxy and THb showed significant increases (6.0 ± 3.7 a.u. to 50.5 ± 12.4 a.u., and 7.7 ± 3.8 a.u. to 58.1 ± 12.5 a.u., respectively, $p < 0.05$) with time only under the HOX condition, and the differences between HOX and NOX became greater ($p < 0.01$) towards the end of the recovery period. For Deoxy, no significant changes were observed throughout the period under both the HOX and NOX conditions. From the above, it can be seen that only Oxy and THb under the HOX condition increased significantly; NOX data showed steady values under both conditions.

DISCUSSION

The main findings of this study are that 30 minutes exposure to normobaric hyperoxia (HOX) provided between anaerobic exercises during a recovery period significantly hastened the restoration of MVIC in locally fatigued quadriceps, and enhanced the blood circulation in the muscle tissue more than a normoxic (NOX) environment over the same time frame. On the other hand, the time to exhaustion and the sEMG power spectrum, which both have been recognized as indications of the levels of fatigue[15, 16], did not show any benefits of oxygen supplementation.

During muscle contraction under anaerobic conditions, intramuscular phosphocreatine (PCr) and glycogen are recognized as the main energy sources generating adenosine triphosphate (ATP)[19, 20]. In particular, maximum effort activities lasting for 1 to 2 minutes demand a combination of aerobic and the above anaerobic sources[21]. As a result of consumption of these energy sources, a significant amount of metabolic products, such as lactate, are accumulated,

leading to a decrease in the pH of blood[4, 8, 22]. This condition of oxygen debt is called exercise-induced metabolic acidosis, which is thought to be a contributing factor to local muscle fatigue in isometric anaerobic exercises. The physical task used in this research was set at a relatively high intensity (70% of MVIC, causing exhaustion between 0.5 to 2.5 minutes). Energy generation would have been similar to maximum effort and an oxygen debt would have been induced in subjects' muscle tissues. Once sufficient oxygen is supplied to the tissues, the accumulated metabolites would start to be removed, and function and performance would be restored[4–7]. Accordingly, the use of HOX in the present intervention would have encouraged the resolution of the oxygen debt, increasing intramuscular metabolism, which would have resulted in the acceleration of the recovery rate in MVIC (from 86.8% to 99.8%) from local muscle fatigue.

In this study, Oxy and THb measured by NIRS in RF increased during the 30 minutes of HOX recovery time, though the Deoxy value did not alter. This result implies that blood circulation, especially arterial inflow, i.e., tissue oxygenation, was augmented significantly by supplemental oxygen. This is in agreement with the results reported by Kawada et al.[6], who used exposure to hyperbaric hyperoxia (100% O_2 for 50 minutes) before high-intensity knee extensor exercises. They found an increase in oxygenation during the exposure, which lasted at least 5 minutes after the end of hyperoxia. Furthermore, Kubo et al.[17] showed that pre-exercise use of hyperbaric oxygen therapy (50% O_2 for 60 minutes) increased the values of Oxy and THb in human muscle compared with the baseline level. The present study's results suggest that increased oxygen in the tissues hastened intramuscular metabolism, metabolites accumulated, and increase of blood flow occurred in response, to remove the metabolites. The parasympathetic nervous system is activated by the use of HOX[6, 23], and relaxation of vascular smooth muscle would have caused vasodilatory effects in both arteries and veins, resulting in an increase in blood flow. The reason venous flow did not show any changes in HOX as well as NOX might be that the subjects in this study were encouraged to be completely sedentary with no muscle pumping during the recovery phase. Venous return was, therefore, not encouraged even though oxygen was supplied. HOX-enhanced tissue oxygenation and blood circulation would also have played a part in the restoration of MVIC, and this hypothesis is supported by past research showing that restricted blood flow brought about higher lactate levels and impairment of muscle metabolism resulting in significant muscle force reduction[24, 25].

There are a number of investigations to date that have verified the effects of hyperoxia, however, these previous studies have published conflicting results, which are confusing for clinical practice. The conflicting evidence can be arranged by dividing into categories regarding the timing of the exposure (pre-exposure, exposure during exercise, and exposure during recovery time) and the types of exercise (aerobic, anaerobic, and interval exercises) used in the experiments. Recently, studies focusing on "pre-exposure" in intermittent high-intensity or anaerobic exercise have been performed, and they have indicated there is no effect arising from oxygen supplementation[6, 22, 26]. Moreover, the same negative results have been reported by research in which hyperoxia exposure was done "ahead" of aerobic endurance performances[3, 27, 28]. These results suggest that increased blood levels of dissolved oxygen induced by hyperoxia would be released immediately under a normoxic environment[6, 9, 22], and that pre-exposure is incapable of producing favorable change in performance. A possible reason for the immediate oxygen release is that the subjects may be in conditions in which additional oxygen is not required, contrary to the status of oxygen debt, before any activity is performed. On the other hand, exposures to hyperoxia during aerobic activity have been shown to have beneficial effects[7, 29]. Stellingwerff et al.[7] observed that muscle glycogenolysis was decreased, and the accumulation of serum and muscle lactate was lower during aerobic exercise under hyperoxia (60% O_2) than under a normoxic condition. Tucker et al.[29] also reported that hyperoxia (40% O_2) improved endurance performance by an average of 5% during cycling time-trials and concluded that increased muscle activation, indicated by integrated electromyography activity (iEMG), resulted in a better performance. It has been proposed that the diminished lactate concentration during aerobic exercise under hyperoxia might be because of decreased lactate production, secondary to reduced glycogenolysis, glycolysis and pyruvate production, increasing lactate clearance[7]. In other words, subjects would be in conditions that require additional oxygen, and are primed to make effective use of oxygen during aerobic activities. With respect to utilization of hyperoxia during the "recovery period", there is only a small body of research to refer to about the effects of hyperoxia on local muscle fatigue caused by anaerobic tasks. Nummela et al.[10] have reported that there was no positive results with supplemental oxygen (40% O_2) during the recovery phases of intermittent exercise (3 × 3 × 300 meters of sprint) with periodic recovery times of relatively short duration (1 to 10 minutes each). Takemura et al.[30] also examined the influence of hyperoxia (28% O_2) on intermittent exercise (2 × 10 × 5 seconds of pedaling), and they showed that serum lactate accumulation was significantly decreased after oxygen supplementation. We hypothesize that this favorable result was due to the provision of a sufficient continuous recovery time of 50 minutes between exercises. In the present study, the type of physical task used was anaerobic instead of intermittent activity, but the overall order of protocol (0.5–2.5 minute isometric contractions with a 30-minute continuous recovery period in between) was similar to Takemura et al.[30] Participants who take part in this type of protocol with more than 30 minutes rest between intensive exercise lasting for few minutes, are supposed to be in a condition of oxygen debt at the end of the physical task. Thus, they would effectively use supplemental oxygen to remove metabolic waste accumulation resulting in enhancement of the recovery rate.

In summary, it appears that the use of normobaric hyperoxia is effective as a recovery strategy in local muscle fatigue induced by anaerobic exercise. It appears to speed up muscle tissue metabolism and increases tissue blood circulation, and the restoration of MVIC was significantly

hastened when compared to normoxia in the same recovery time. It was also clear that supplemental oxygen hastened the recovery from oxygen debt and improved muscle contractility in the recovery from muscle tissue fatigue, but it is not a modality that increases the ability of the muscle itself. Accordingly, it is understandable that hyperoxia neither prolonged the duration of muscle contraction after the exposure, nor changed the pattern of local muscle fatigue monitored by sEMG signals in this investigation.

Practical implications. Hyperoxia therapy has been used in the sports field for the purpose of healing musculoskeletal injuries, increasing performance level, and accelerating recovery from daily training. While the evidence is mixed, it would be obvious that when in need of oxygen, supplemental oxygen will work efficiently for athletes, such as exposure to hyperoxia during aerobic activity and the recovery phase from oxygen debt. In other words, under conditions of sufficient oxygen supply to the tissues, hyperoxia will not provide any additional effects, when administered before exercises and during anaerobic performance. Based on the present research, it is recommended to coaches and field practitioners to utilize 30 minutes of exposure to normobaric hyperoxia as a recovery strategy from local muscle fatigue acutely developed by anaerobic exercise in training and competitions. By doing this, athletes would be able to perform as well as before without experiencing the influence of local muscle fatigue, although their performance may decrease approximately 10% from the level they would have achieved if they had stayed under a normoxic environment for the same duration.

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