Doctoral Dissertation (Shinshu University)

Influences of Hyperoxia and Psychological Task on Fluctuations in Local Muscle Fatigue

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Contents

Preface and Acknowledgments

The process of writing this thesis is a great catalyst for growth of myself. I read a quote quite a while ago and now remember:

‘To learn, read. To know, write. To master, teach.’

Although I am still on the way of learning, knowing and mastering physiotherapy, this thesis work surely upgraded me as a reader, writer, and teacher.

There are so many people without whose support this thesis would never have been completed and to whom I owe my heartfelt thanks. To Professor Koji Abe, who is an amazing researcher, for your understanding and flexibility in the PhD process. You have always impressed me and given me the immense learning journey. I am also thankful to Professor Iwao Yamamoto, Professor Masayoshi Kamijo, Associate Professor Yoshiro Ogoma, Associate Professor Tetsuya Fujimoto, Associate Professor Hiroaki Yoshida, who have refined my thinking, and I am grateful for all the experience and growth they have fostered. To Professor Takayuki Fujiwara, who continues to be passionate and to inspire me as a researcher, teacher, and physiotherapist. I am forever grateful to you for giving me this PhD opportunity and continual encouragement. Thanks to Professor Ryuya Yanagihashi, who have spent much time of him to supervise and lead me throughout the PhD process. I owe you for your great supports, understanding, flexibility and expertise. To all stuff in the Koriyama Institute of Health Sciences, especially to Professor Noboru Goto, Professor Ken Yanagisawa, Dr. Masaaki Hanaoka, Dr. Kenji Handa, Mr. Toshihiro Endo, Dr. Kazunori Morozumi, Dr. Kunio Yoshizaki, Professor Hiroshi Karasuno, Mr. Hisayoshi Ogihara, Mr. Masao Hashimoto, for their warm supports and efficiencies that allow me freedom to do research work smoothly. Thanks to Mr. Yoetsu Ogata, an amazing clinical expert to influence my path, and who gave me this great opportunity. I forever appreciate everything you have done for me. To Mr. Katsuyuki Morishita, my PhD fellow, for your expertise and continuous encouragement. I have learned much from you. To Mr. Robert Holt, who used his fine English editing skills to improve my grammar and punctuation. My papers dramatically read much better thanks to your contributions. I also wish to thank to all

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I wish that my PhD work would help and support someone in the future as I have been supported by people around me. In order to do so, I strongly feel the need of my further growth as a researcher, clinician and teacher. Yes, this is the start!

Abbreviations

ANOVA	analysis of variance
ATA	atmosphere absolute
ATP	adenosine triphosphate
Ca^{2+}	calcium ion
CFF	critical flicker frequency
Deoxy-Hb	deoxy-hemoglobin
EMG	electromyography
FFT	fast Fourier transform
fMRI	functional magnetic resonance imaging
H^{+}	proton
HBO	hyperbaric hyperoxia
HOX	normobaric hyperoxia
iEMG	integrated electromyography
LDH	lactate dehydrogenase
MEXT	Ministry of Education, Culture, Sports, Science and Technology
MF	median frequency
MVC	maximum voluntary contraction
MVIC	maximum voluntary isometric contraction
NCAA	National Collegiate Athletic Association
NIRS	near-infrared spectroscopy
NOX	normobaric normoxia
O_2	oxygen
Oxy-Hb	oxy-hemoglobin
PCr	phosphocreatine
pH	potential Hydrogen
Pi	phosphate
PT	psychological task

R	rest
RF	rectus femoris
RPE	rate of perceived exertion
SD	standard deviation
SEM	standard error of the mean
sEMG	surface electromyography
THb	total (tissue) hemoglobin
VAS	visual analog scale
VL	vastus lateralis
VO_2max	maximum oxygen uptake

Chapter 1.

Introduction

1.1 Background

The number of athletic participation has been increased for these decades with the increased interest in sports as not only participating competitions but also enjoying recreational activities and being healthier. According to the National Collegiate Athletic Association (NCAA) which organizes the athletic programs of colleges and universities in the United States and Canada, the total player number has increased 20% in males and 80% in females from 1988 to 2004 [1)]. The similar tendency has been observed in Japan, and furthermore the annual survey of 3,000 people done by the Ministry of Education, Culture, Sports, Science and Technology (MEXT) revealed that the frequency of playing sports for each person has been also increased from 1982 to 2009 (Figure 1.1). It reported that people who play sports more than once per week increased approximately 1.6 times, on the other hand, people who play only once to three times in a year decreased to about 0.7 times [2)].

Accompanied with the increased athletic players and frequency, sports-related musculoskeletal injuries also have been increased in spite of continued evolution of the practice of sports medicine. Causes of sports injuries are divided into two major cases; contact trauma and noncontact trauma. The survey done on sports injuries during the

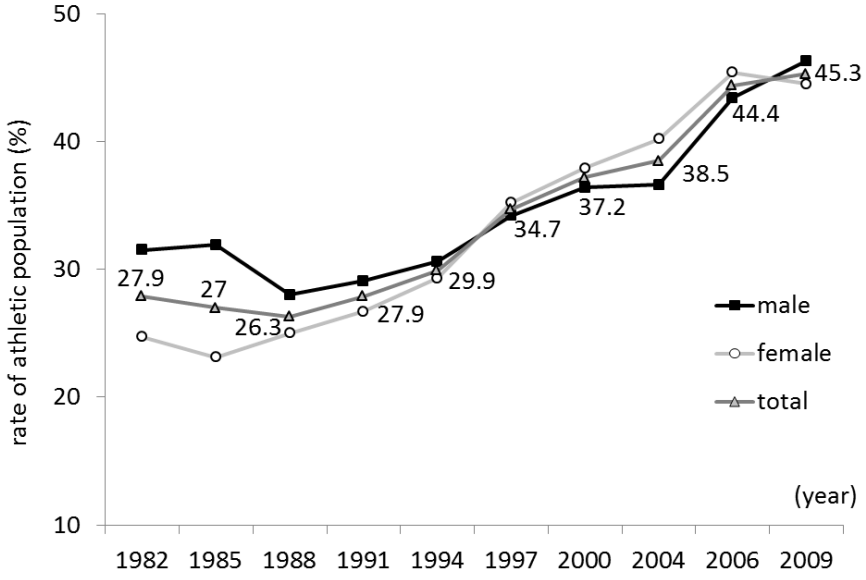

Figure 1.1 Rate of athletic participants who play more than once per week in Japan (1982-2009).

Table 1.1 Overuse injuries; predisposing factors (Cited from Burkner et al. [4]).

Extrinsic factors	Intrinsic factors
Training Errors	***Malalignment***
Excessive volume	Pes planus
Excessive intensity	Pes cavus
Rapid increase	Rearfoot varus
Sudden change in type	Tibia vara
Excessive fatigue	Genu valgum
Inadequate recovery	Genu varum
Faulty technique	Patella alta
Surfaces	Femoral neck anteversion
Hard	Tibia torsion
Soft	***Leg length discrepancy***
Cambered	***Muscle imbalance***
Shoes	***Muscle weakness***
Inappropriate	***Lack of flexibility***
Worn out	Generalized muscle tightness
Equipment	Focal areas of muscle thickning
Inappropriate	Restricted joint range of motion
Environmental	***Sex, size, body composition***
Conditions	***Other***
Hot	Genetic factors, endocrine factors, metabolic conditions
Cold	
Humid	
Psychological factors	
Inadequate nutrition	

Summer Olympic Games 2008 have shown that 32.9% of the injuries were caused by contact with another athlete, while noncontact trauma and overuse were numbered in 41.9% as more frequent causes [3]. The causes of overuse injuries are usually divided into extrinsic factors such as training errors and environment, or intrinsic factors such as leg length discrepancy and muscle imbalance (Table 1.1) [4]. Training errors are especially common pitfalls for most coaches and athletes since they assumed that increased training, i.e., overload, is the ultimate prescription for improvement. Overload is definitely one of training principles, however it is sometimes manipulated by allowing to decrease recovery time between efforts of a given volume and intensity. Inadequate recovery, such as excessively shortened recovery time is obvious to result in overtraining, thus overuse injuries. In term, if the recovery was improved, greater training volumes would be accepted and higher level of performance would be maintained without incurring the negative aspect of overloading.

Given the above, it is clear that recovery is as important to athletes as training in order to avoid overuse injuries and optimize chronic improvements in physiological capacities since

most exercise induced adaptations take place during recovery. In particular, the recovery from local muscle fatigue is a critical issue for athletic participants, since the acutely developed muscle fatigue would be the most commonly encountered thus the most troubling form of fatigue in training and competitions.

1.2 Local Muscle Fatigue

Local muscle fatigue is a daily encountered physiological phenomenon, and it often restricts people from staying active and requires temporal rest. This might be a biological defense mechanism, since it works at preventing muscles being injured by maintaining required force production [5, 6]. It is, however, obvious that the occurrence of muscle fatigue adversely influences on people's performance, and there have been a great number of studies done in this field to clarify the mechanisms.

Local muscle fatigue represents a complex phenomenon that encompasses various factors; structural and energetic changes in local muscle tissues, and changes in activity level and the efficiency of the nervous system [7]. Muscle fatigue, therefore, is also referred to as neuromuscular fatigue and is divided into three headings based upon the location of

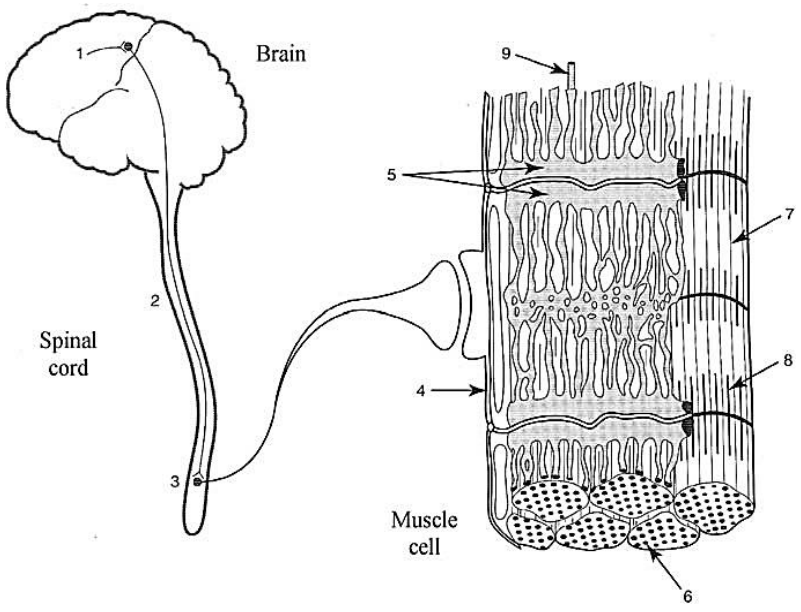

Figure 1.2 Body sites which contribute to local muscle fatigue (Cited from Boyas et al. [8]).

where the fatigue is induced; central fatigue (sites 1 to 3 in Figure 1.2), fatigue of the neuromuscular junction (site 4 in Figure 1.2), and muscle tissue fatigue (sites 5 to 9 in Figure 1.2) [8, 9]. Furthermore, muscle fatigue is often conveniently divided into just two categories;

i. Central fatigue (sites 1 to 3 in Figure 1.2), which encompasses all the supraspinal and spinal physiological phenomena resulting in a decrease in motoneuron excitation.
ii. Peripheral fatigue (sites 4 to 9 in Figure 1.2), which indicates a decrease in the contractile strength via changes in the structure and the metabolism of muscle tissue and includes the fatigue at the neuromuscular junction [8, 10].

When both forms of fatigue are fully treated and the fatigue eliminated then local muscle fatigue is theoretically fully healed. However, the reality is that the most common techniques for recovery, such as hydrotherapy or soft tissue massage, focus on peripheral fatigue as they are easier and more convenient to use as practical techniques.

1.3 Methods for Recovery from Local Muscle Fatigue

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 [11]. The methods for recovery would be different based on the objectives and which level of fatigue, i.e., central or peripheral fatigue, trainers and therapists target to treat after vigorous trainings and competitions. A number of methods are commonly used to hasten the recovery process such as cryotherapy, soft tissue massage, active recovery*[1], analgesics, nutrition, dehydration and psychological relaxation whereas those effects are often controversial. An establishment of adequate recovery methods is crucial for coaches and athletes, therefore it is natural that the attention of sports sciences practitioners continues to evolve and seek new methods of recovery. With this in mind, a relatively under researched recovery modality is the use of supplemental oxygen.

1.3.1 Normobaric Hyperoxia

Widely reported as a major cause of peripheral fatigue is exercise induced metabolic acidosis in the muscle tissue [8, 12, 13, 14]. Metabolic acidosis resulting from exercise is particularly caused by the increase in intracellular concentrations of proton (H^+) and inorganic phosphate (Pi), which are established by the activity of anaerobic metabolic pathways and result in a decline in blood pH [8, 12, 13, 15]. During the process of cellular acidosis, increased lactate production coincides to prevent pyruvate accumulation [15], thus blood lactate concentration is a good indirect marker of muscle metabolic acidosis levels. A sufficient supply of oxygen enhances the activation of the mitochondria which consumes H^+ and Pi as substrates for mitochondrial respiration during recovery [8, 12, 14]. Given this, oxygen supplementation is easily imagined to accelerate the improvement of such acidotic condition. Therefore, the use of supplemental oxygen would enhance metabolic acidosis recovery and thus peripheral muscle fatigue would reduce quicker than without the use of oxygen supplementation.

The use of normobaric hyperoxia environment is the one of options to increase oxygen concentration in the artery. Compared to other methods of oxygen supplementation such as utilization of the hyperbaric hyperoxia environment and inhalation of oxygen gas, the use

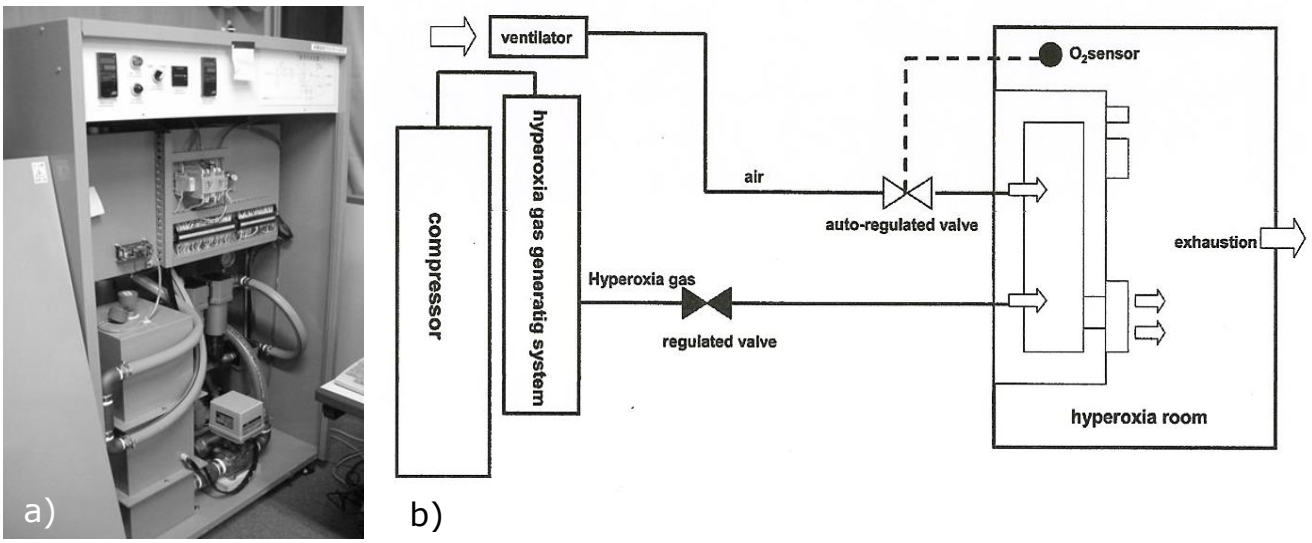

Figure 1.3 a) Oxygen concentration control device. b) Oxygen concentration control system (Cited from Ohkura et al. [16]).

of normobaric hyperoxia environment is safer and more convenient, thus more practical for clinical situation. The oxygen control system used in this study was membrane separation using polyimide, and the oxygen concentration in the control room was maintained by an auto-regulated valve and an oxygen sensor (Figure 1.3) [16]. The concentration of oxygen in the room was possible to manipulate between 14.5% and 35.0% in the air.

Increases in arterial oxygen content via atmospheric hyperoxia would affect not only peripheral fatigue but also central fatigue [17, 18]. This is because that the increased oxygen concentration in the artery influences on oxygen transport throughout the organism [19, 20], therefore, changes in performance under the hyperoxic environment may occur due to the central fatigue mechanisms which dictate the increases and decreases of volitional motor output into the muscle tissue. Tucker et al. [18] found that the integrated electromyographic (iEMG) activity, an indirect measure of local muscle activation, was greater during 20 km

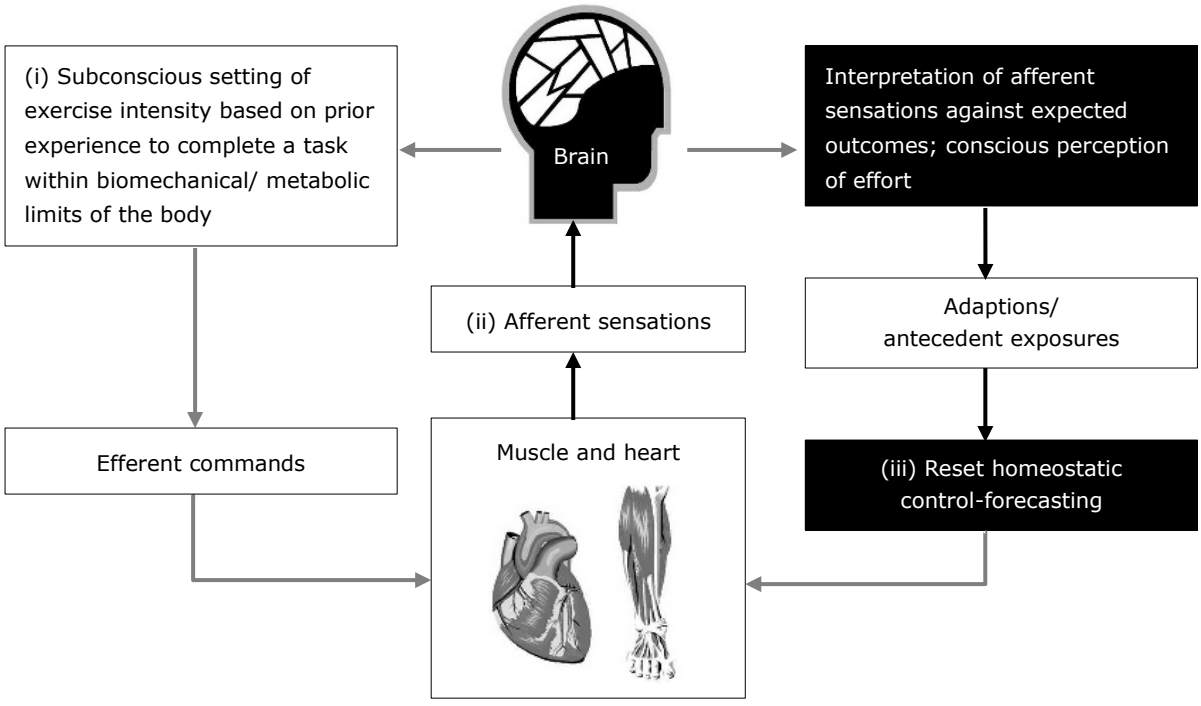

Figure 1.4 The model of teleoanticipation*[2] (Modified from Lambert et al. [22]). (i): There is a subconscious setting of exercise intensity based on the previous experience. (ii): Afferent feedback from peripheral organ system is then interpreted by the central nervous system against expected outcomes. (iii): Based on the interpretation, the exercise intensity and teleoanticipatory set points are reset.

cycling time-trials in hyperoxia (40% O_2) than in normoxia, concluding that improved performance in hyperoxia is partly due to a centrally mediated increase of the local muscle activation. Further, Peltonen et al. [21] and Amann et al. [17] found that central motor output represented by iEMG was reduced in hypoxia (15.8% O_2 and 15% O_2, respectively), and hypothesized that performance capacity was controlled by central neural drive as a result of the brain responding to changes in peripheral muscle tissue conditions such as metabolite accumulation. This mechanism is also supported by other researchers who suggest the model of teleoanticipation*[2] as a control system for optimization of performance during physical activity (Figure 1.4) [22].

1.4 Local Muscle Fatigue and Psychological Task

As mentioned in the section 1.2, local muscle fatigue is divided into central and peripheral fatigue based on the sites where the fatigue is induced. Central fatigue is a mechanism in the central nervous system which blocks off the motor unit before the muscle is impaired [5] by declining impulses of motor neuron pool, and this is thought to be also governed by a psychological statement. In other words, neuromuscular fatigue is influenced by not only the physical workload, but also psychological factors such as motivation, mental concentration and alertness. From the above, it is easily imagined that those factors are affected by psychological tasks like a series of successive numerical calculations, and such a task would influence the level of local muscle fatigue as a result of central fatigue.

One of the objectives of recovery from fatigue is psychological recovery. If there is insufficient recovery of the nervous system, athletes remain sympathetically aroused [11]. Sympathetic over-arousal delays absorption of nutrients from the gastrointestinal tract as well as elevating the metabolic rate. Needless to say, prolonged these conditions lead to more serious physical and psychological symptoms. Given the above, it is clear that understanding the relationship between local muscle fatigue and psychological factors is important in attempting to make athletes fully recovered.

1.5 Assessment of Local Muscle Fatigue and Recovery Rate

1.5.1 Maximum Voluntary Isometric Contraction and Endurance Time to Exhaustion

The term, 'local muscle fatigue', was first introduced by Chaffin [23], and thereafter many researchers have defined it, such as "failure to maintain the required or expected force" [24] and "any exercise-induced reduction in the maximum capacity to generate force or power output" [25]. Simply these situations indicate the onset that an individual is unable to perform certain work, such as maintaining a defined level of static muscle contraction (isometric contraction). To observe this state, measuring the maximum voluntary contraction (MVC) before and after work is a general procedure and 'gold standard' [25]. Additionally, the endurance time until the task fails is also the simplest indication to measure fatigue level.

1.5.2 Surface Electromyography

Another way to determine local muscle fatigue is the use of electromyography (EMG) that enables the observation of the physiological changes in a fatiguing muscle. It is well known that increased EMG amplitude, i.e., increased integrated EMG (iEMG), is recognized, and EMG power spectrum shifts from high to low frequency band with time and as fatigue occurs [26]. The median frequency (MF) of EMG power spectrum is generally used to measure the immediate onset of muscle fatigue (Figure 1.5). There are a number of studies using various fatigue indexes, for example, increased brain activity with functional magnetic resonance imaging (fMRI) [27], and biochemical changes detected by muscle biopsies [28]. Nevertheless, those techniques are unlikely to monitor the state of fatigue in real-time and real daily living situations. Hence, EMG data i.e., both iEMG and MF are good direct markers to measure muscle fatigue.

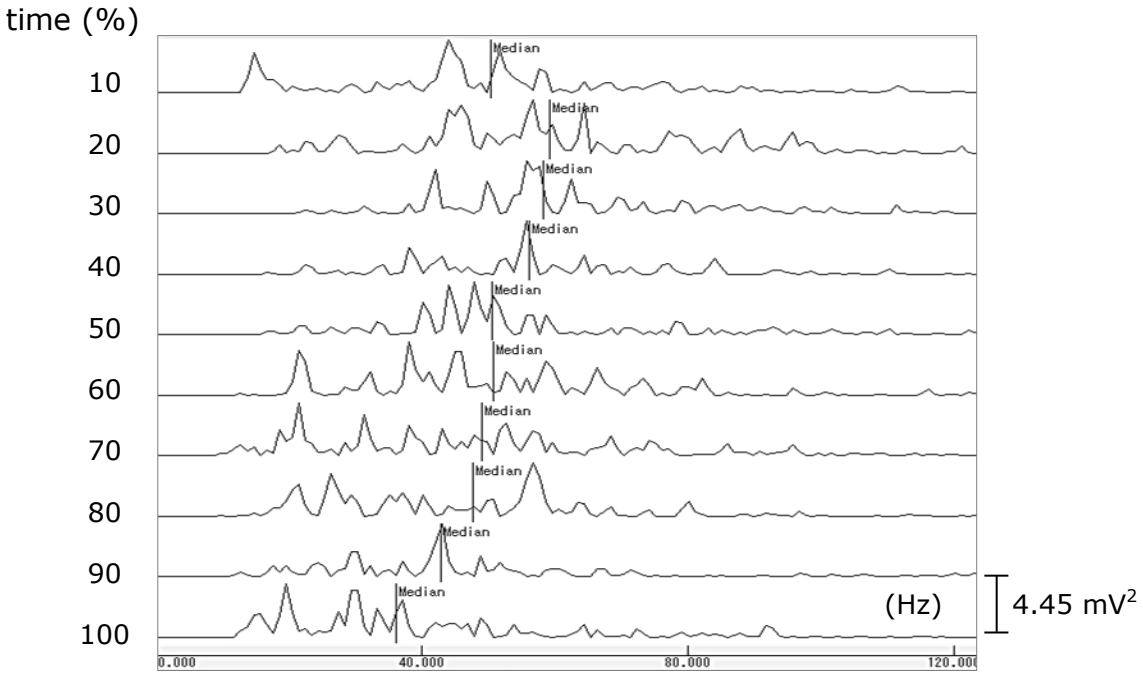

Figure 1.5 An Example of EMG power spectrum shifts from high to low frequency band during isometric contraction. The sampling data was divided into 10 equal epochs to normalize the length of data, i.e., 10 to 100%.

1.5.3 Near-infrared Spectroscopy

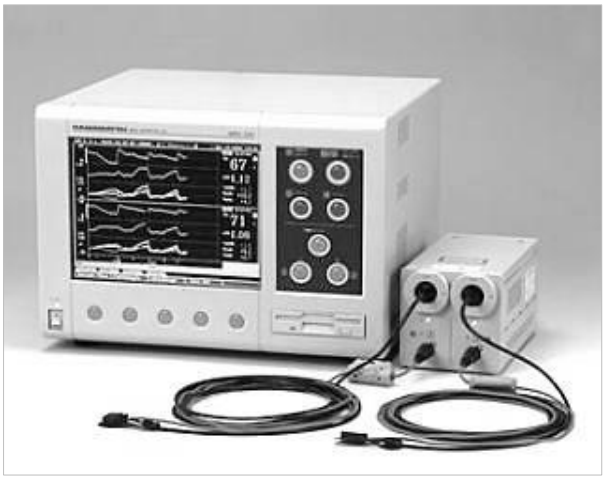

Figure 1.6 Near-infrared spectroscopy; NIRO-200 (Hamamatsu Photonics).

Recent developments in optical instrumentation using near-infrared spectroscopy (NIRS) have made it possible to monitor changes in tissue oxygenation noninvasively. The device used in this study was NIRO-200 (Hamamatsu Photonics, Hamamatsu, Japan; Figure 1.6). This technique has been used to evaluate the balance between oxygen delivery and its use, thus blood circulation, in the skeletal muscle of human by showing the changes in oxy-hemoglobin (Oxy-Hb) and deoxy-hemoglobin (Deoxy-Hb) in the tissue. Total tissue hemoglobin (THb) is calculated by adding Oxy-Hb and Deoxy-Hb. Changes in Oxy-Hb

and Deoxy-Hb are measured according to the Beer-Lambert law [29, 30]. While NIRS does not reflect the condition of muscle tissue fatigue directly, it was used in this study as an indirect parameter by expressing the state of muscle tissue oxygenation and blood circulation which are related with muscle metabolic acidosis and muscle tissue fatigue.

1.5.4 Blood Lactate Concentration

The development of metabolic acidosis in the muscle tissue during intense exercise has traditionally been explained due to the accumulation of lactic acid resulting in a decrease in cellular pH. However, the main cause of acidosis has been recently revealed to be the increased proton (H^+) release into the cytosol rather than the increased lactate concentration in the tissue and blood [10, 12, 13]. When exercise intensity is steady and moderate, mitochondrial metabolism which consumes protons and electrons mainly functions to produce adenosine triphosphate (ATP) (Figure 1.7a). On the other hand, when the rate of ATP demand and consumption exceeds the mitochondrial capacity during non-steady intense exercise, lactate production aids to buffer the rate of proton release (Figure

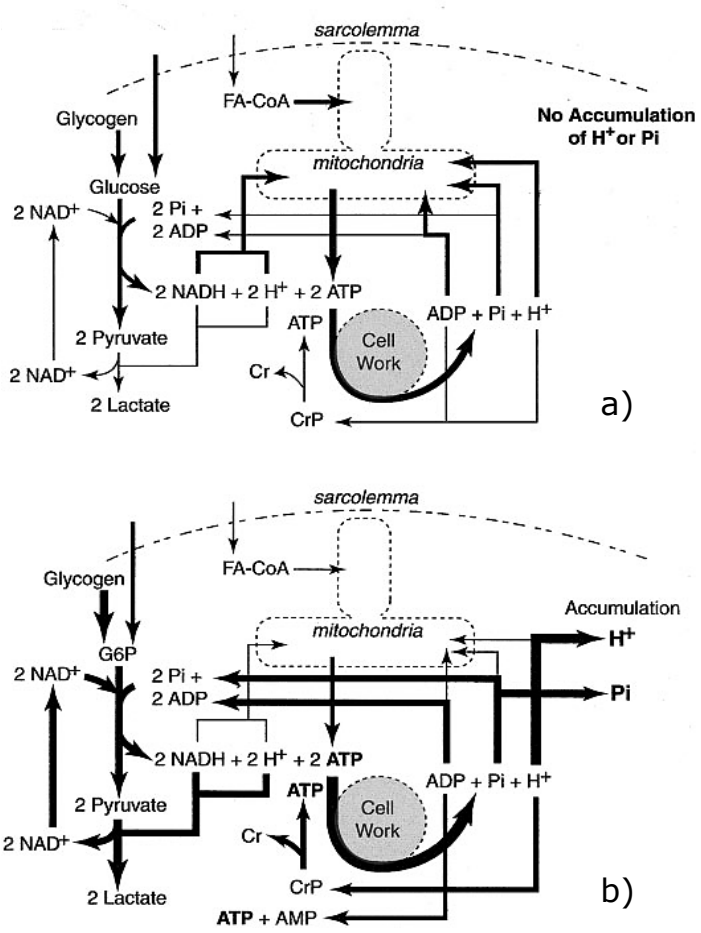

Figure 1.7 Diagrams of metabolism under two different exercise intensities (Cited from Robergs et al. [15]). The magnitude of the arrows represents proportions of substrate flux. a) moderate intensity steady-state exercise. b) short-term intense exercise causing volitional fatigue in two to three minutes.

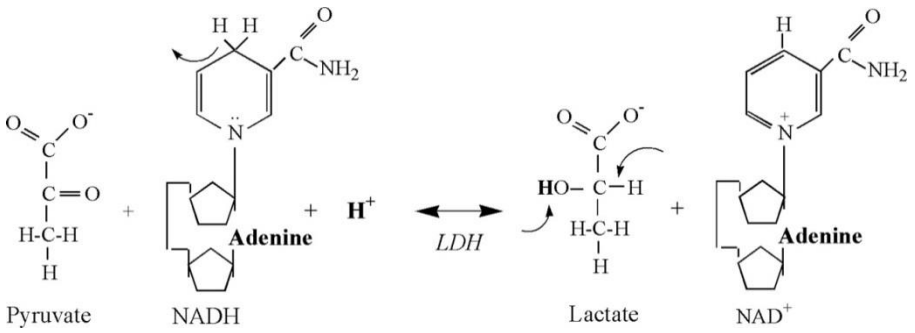

Figure 1.8 Lactate dehydrogenase (LDH) reaction (Cited from Robergs et al. [15]). A proton is consumed to reduce pyruvate and produce lactate.

1.7b and 1.8). Eventually, the metabolic proton buffering is exceeded by the rate of ATP hydrolysis in the cell and it results in proton accumulation leading to a decrease in the cellular pH. From the above, although lactate is not the direct cause of metabolic acidosis, blood lactate concentration would be still a good indirect marker for conditions of muscle metabolic acidosis since the increased lactate production coincides to prevent pyruvate accumulation during the process of cellular acidosis.

The instrument for evaluation of blood lactate used in this study was a portable blood lactate analyzer; Lactate Pro (LT-1710$^{(R)}$ Arkray KDK, Kyoto, Japan; Figure 1.9). This analyzer was demonstrated to have a good reliability and accuracy when compared to another laboratory-based analyzer [31-33].

Figure 1.9 Portable blood lactate analyzer; Lactate Pro (LT-1710$^{(R)}$ Arkray KDK)

1.5.5 Perceived Exertion

To quantify perceptions of physical workload and the recovery rate in the targeted muscle, ratings of perceived exertion were collected using visual analog scale (VAS). Subject was asked to mark on a line of 10 cm length to express the locally perceived muscle fatigue (Figure 1.10). The use of VAS is simple and easy to understand for subject,

thus practically convenient in evaluating perceived exertion. In addition, since every time the marking was required a new paper was provided, subject was unlikely to be biased by previous rates differently from confirming the rates orally. The way of rating VAS was explained to subject as Table 1.2 in order to have a common understanding.

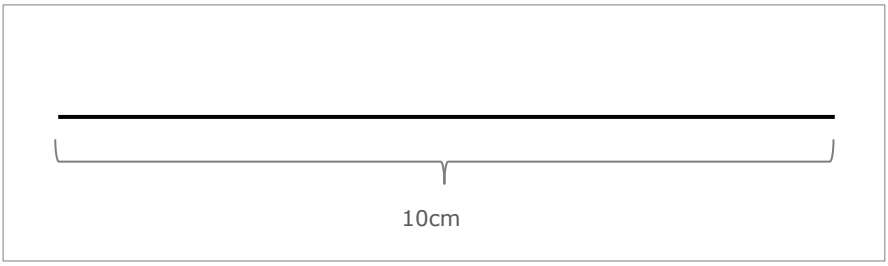

Figure 1.10 Visual analog scale (VAS).

Table 1.2 Common understanding in rating VAS

Rating of VAS	
0	no fatigue
2~3	a little bit of fatigue
5~6	fatigue which needs to be treated e.g.) massage, streching, cryotherpy, etc
10	maximum fatigue which is ever perceived

1.6 Experimental Approach

In order to clarify the effects of normobaric hyperoxia on local muscle fatigue, Chapter 2 and 3 are going to present studies focusing on the recovery effects. Chapter 2 considers the peripheral factor in muscle fatigue, i.e., conditions of muscle tissue metabolism, by attempting to reveal the relationships between MVIC, endurance time, MF and tissue blood circulation. In Chapter 3, while it will continue to observe the recovery effects of normobaric hyperoxia, it goes into more detailed mechanism by dividing it into central and peripheral factors using various evaluation indexes (Table 1.3).

The influences of psychological element on local muscle fatigue are presented in Chapter 4. It discusses and hypothesizes the involvement of attentional resources of the prefrontal

cortex, i.e., central factors, consumed during both psychological and physical workloads.

In closing, Chapter 5 concludes the overall presentation in this thesis, and it also brings several applications to athletes and coaches working in clinical situation.

Table 1.3 Summery of purposes and evaluation indexes in each Chapter.

Chapter	Purpose	Intervention	Evaluation of Fatigue and/or Recovery
2	To identify the 30 minute recovery effects of exposure to normobaric hyperoxia on local muscle fatigue as induced by single isometric exercise.	30 minute rest in the normobaric hyperoxia environment	① MVIC; Maximum Voluntary Isometric Contraction ② Endurance time to exhaustion ③ Median Frequency (MF) of sEMG power spectrum ④ Tissue oxygenation/ blood circulation (using NIRS; Near-Infrared Spectroscopy)
3	To identify the repeated 15 minute recovery effects of exposure to normobaric hyperoxia on local muscle fatigue as induced by intermittent isometric exercise.	2 x 15 minute rests in the normobaric hyperoxia environment	① MVIC; Maximum Voluntary Isometric Contraction ② Endurance time to exhaustion ③ Integrated EMG (iEMG) ④ Blood lactate concentration ⑤ Perceived exertion (using VAS; Visual Analog Scale)
4	To identify if local muscle fatigue is influenced by a prior psychological task.	20 minute psychological task (numerical calculation)	① MVIC; Maximum Voluntary Isometric Contraction ② Endurance time to exhaustion ③ Median Frequency (MF) of sEMG power spectrum

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Terminology

*[1] active recovery; is one of warm-down strategies and is done by actively exercising after training or competition. This has been recognized to remove lactate from the circulation more quickly than passive recovery. This kind of strategies appears particularly important if the next bout of activity is within 2 to 4 hours.

*[2] teleoanticipation; is an integrated system in the body that afferent input of metabolic activity in the muscle tissue influences and modifies the muscle output by interpreting the afferent sensations against expected outcomes in the brain. This is specific for exercise and associates programming of power output.

Chapter 2.

Recovery Effects of Exposure to Normobaric Hyperoxia

~ Peripheral Factor in Local Muscle Fatigue~

2.1 Background

The use of exposure to hyperoxia is an ongoing research modality in the sports field, and the effects that have been reported were considerably conflicted and complicated. When trialed in well trained populations, breathing of hyperoxic gas (99.5% O_2 for 20 minutes in total) during recovery sessions after an intermittent intense exercise was shown that the participant's perceptions of recovery had a significantly better result than the control group [1)], though other uses of hyperoxic gas during recovery period between interval repetitions (40% O_2 for 29 minutes in total, and 99.5% O_2 for 10 minutes in total) were presented to have no effect on the rate of perceived exertion (RPE) [2, 3)]. Furthermore, pre-exposure to hyperbaric hyperoxia (HBO, 100% O_2 at 2.8 ATA for 60 minutes) increased maximum oxygen consumption during treadmill running [4)], however, another exposure to HBO (100% O_2 at 2.0 ATA for 60 minutes) did not enhance VO_2max during the following cycle ergometer exercise [5)].

From the above, it remains unclear what effect supplemental oxygen would have on activities and fatiguing conditions. Additionally, studies reported to date 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 aims to identify the recovery effects of exposure to normobaric hyperoxia on local muscle fatigue affecting the quadriceps femoris*[1], and the degree of fatigue was measured by the following parameters; maximum voluntary isometric contraction (MVIC), endurance time, surface electromyography (sEMG) power spectrum signals, and the status of blood circulation monitored by near-infrared spectroscopy (NIRS). It was 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.

2.2 Methods

2.2.1 Subjects

11 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. Body height and mass (mean ± *SD*) were 170.1 ± 5.4 cm and 63.2 ± 12.2 kg, respectively. None of them are competitive athletes, but all are physically active. In order to familiarize subjects with the protocol, all experienced it once before the study session. They were instructed to avoid alcohol consumption and vigorous exercise which might cause 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 from the Koriyama Tohto Academy Educational Foundation Ethical Committee (approval number: R1107).

2.2.2 Procedures

Experimental Design. During the experimental sessions, all participants performed two protocols including 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 is controlled, between fatiguing physical tasks (Figure 2.1). Upon arrival to the experimental room, subjects were required to complete a 5 minute warm-up for the right quadriceps femoris followed by a 20 minute rest set as a stabilization period. The subsequent physical task was designed to fatigue quadriceps femoris of the right leg by sustaining isometric load set at 70% of MVIC for each trial (Figure 2.2). Participants were asked to maintain this load for as long as they could, and this was set for them to reach exhaustion at between 0.5 to 2.5 minutes. At the conclusion of the physical task, a 30 minute recovery period was provided before the start of a 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.

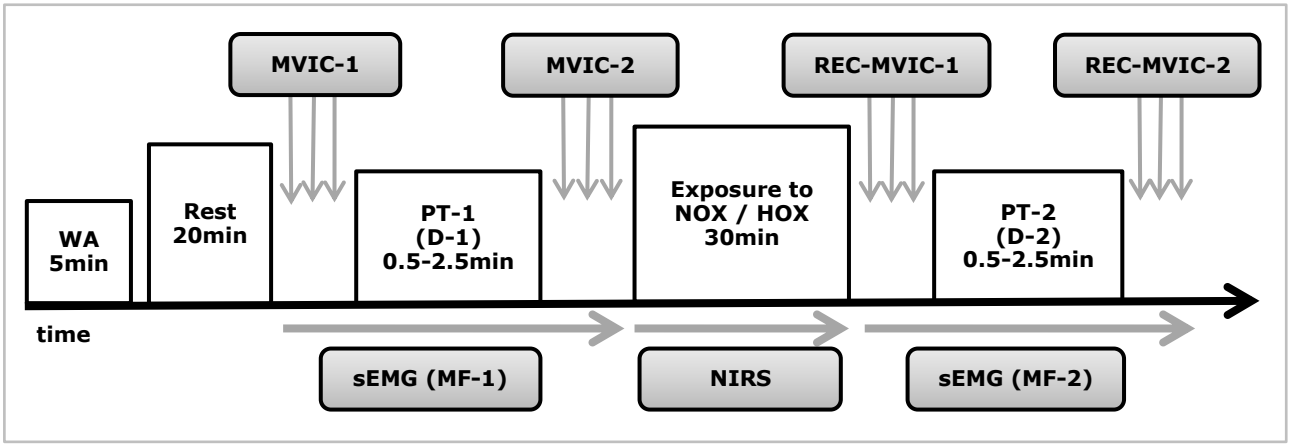

Figure 2.1 Study protocol performed by subjects randomly participating two recovery sessions on each separate day; NOX and HOX between physical task-1 and -2. Abbreviations: NOX = normobaric normoxia; HOX = normobaric hyperoxia; WA = warming-up; MVIC = maximum voluntary isometric contraction; REC-MVIC = MVIC after recovery session; PT = physical task; D = duration; sEMG = surface electromyography; MF = median frequency; NIRS = near-infrared spectroscopy.

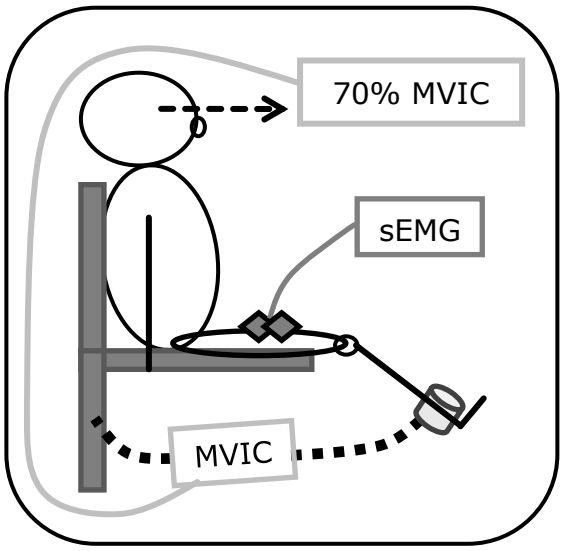

Figure 2.2 Experimental set-up for MVIC and physical task. Subjects used visual feedback while maintaining 70% MVIC by monitoring the digital dynamometer. Abbreviations: MVIC = maximum voluntary isometric contraction; sEMG = surface electromyography.

Maximum Voluntary Isometric Contraction. During each protocol, the MVIC was measured four times:

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 (Figure 2.1).

All subjects were seated on a device (T.K.K. 5710m, Takei, Japan) set with a digital dynamometer (F340, Unipuls, Japan) which was used to measure the MVIC and test their muscle response to the endurance task (Figure 2.2). The subjects positioned their trunk and pelvis, which were 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, their 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 of assessment of the local muscle fatigue [6].

Surface Electromyography Power Spectrum Signals. The sEMG signals from the rectus femoris (RF) of the right quadriceps femoris were recorded using a surface electrode bipolar configuration (special order product, Emu･ii Corporation, Matsumoto, Japan) with an inter-electrode distance of 30 mm (Figure 2.2). 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(R) 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 signals 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 [7]. 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 muscle just below the sEMG electrode, parallel to the major axis of the thigh. Light photons pass through the tissue and are collected by the detectors with optical filters set at 775, 810, and 850 nm, in order to estimate changes of oxygen concentration (oxy-hemoglobin [Oxy-Hb], deoxy-hemoglobin [Deoxy-Hb], and total hemoglobin [THb]) from the baseline at the start. These changes are calculated according to the Beer-Lambert law [8, 9]. All values were continuously recorded at 0.2 Hz during the 30 minutes of recovery, and the mean values of each minute were analyzed.

2.2.3 Statistical Analysis

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-Hb, Deoxy-Hb, 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 any 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 endurance time. SPSS for Windows version 21.0 was used, and the statistical significance was accepted for values of $p < 0.05$ in all analysis.

2.3 Results

2.3.1 Maximum Voluntary Isometric Contraction

The effect of exposure to HOX on the recovery of MVIC is depicted in Figure 2.3. 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 within groups of both HOX (100% at MVIC-1 to 86.8 ± 3.5% at MVIC-2, and 99.8 ± 3.4% at REC-MVIC-1 to 86.7 ± 4.3% at REC-MVIC-2) and NOX (100% at MVIC-1 to 84.6 ± 3.4% at MVIC-2, and 89.7 ± 2.9% at REC-MVIC-1 to 78.2 ± 4.1% at REC-MVIC-2). At REC-MVIC-1, the MVIC was measured immediately after the recovery period. There was a significant difference ($p < 0.05$) at REC-MVIC-1 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.

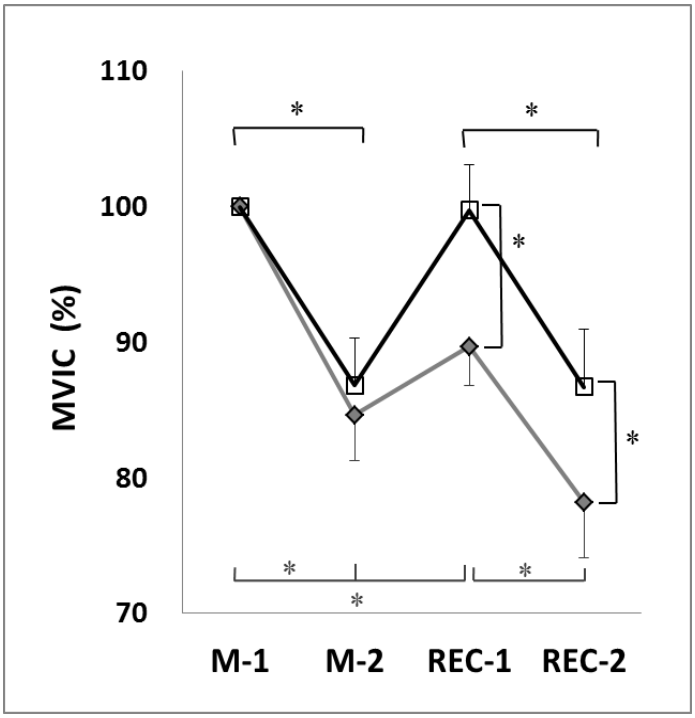

Figure 2.3 Normalized mean values (± *SEM*) of maximum voluntary isometric contraction (MVIC) in NOX (closed diamond) and HOX (opened square) before (M-1 and M-2) and after (REC-1 and REC-2) 30 minutes recovery time. Abbreviations: M = MVIC; REC = REC-MVIC; MVIC after the recovery time. *Significant differences (*$p < 0.05$).

2.3.2 Endurance Time

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.

2.3.3 Surface Electromyography Power Spectrum Signals

MFs during the first and second physical tasks were presented in Figure 2.4. All of MF had 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.

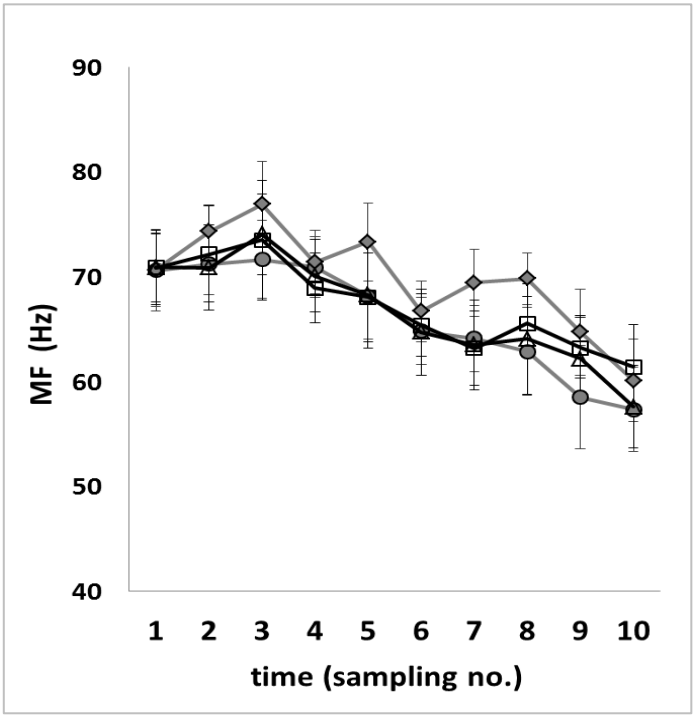

Figure 2.4 Time course changes in mean values (± *SEM*) of median frequency (MF) in NOX (MF-1: closed circle, MF-2: closed diamond) and HOX (MF-1: opened triangle, MF-2: opened square) before (MF-1) and after (MF-2) 30 minute recovery time. The length of individual's data was normalized by dividing it into 10 equal epochs shown as sampling numbers (time) for the group comparison.

2.3.4 Near-Infrared Spectroscopy

The changes in Oxy-Hb, Deoxy-Hb, and THb of the RF muscle during the 30 minutes of recovery time under the HOX and NOX conditions are expressed in Figure 2.5. Oxy-Hb and THb showed significant increases (6.04 ± 3.7 a.u. to 50.5 ± 12.4 a.u., and 7.74 ± 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-Hb, 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-Hb and THb under the HOX increased significantly, though all NOX data showed steady values.

2.4 Discussion

There are three main findings revealed in this study:

i. 30 minute exposure to normobaric hyperoxia (HOX) provided between anaerobic exercises as a recovery period hastened the restoration of MVIC in locally fatigued quadriceps femoris significantly.
ii. The 30 minutes of recovery under the HOX condition also enhanced the blood circulation in the muscle tissue more than staying in a normoxic (NOX) environment for the same time frame.
iii. 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 [6, 7], did not show any benefits of oxygen supplementation.

During muscle contraction under anaerobic conditions, intramuscular phosphocreatine (PCr) and glycogen are well recognized as the main energy sources to generate adenosine triphosphate (ATP) [10, 11]. In particular, maximum effort activities lasting for 1 to 2 minutes demand a combination of aerobic and the above anaerobic sources (Figure 2.6) [12, 13]. As a result of consumption of these energy sources, a significant amount of metabolic products, such as proton (H^+) and lactate, are accumulated, and it eventually induces a decrease in the

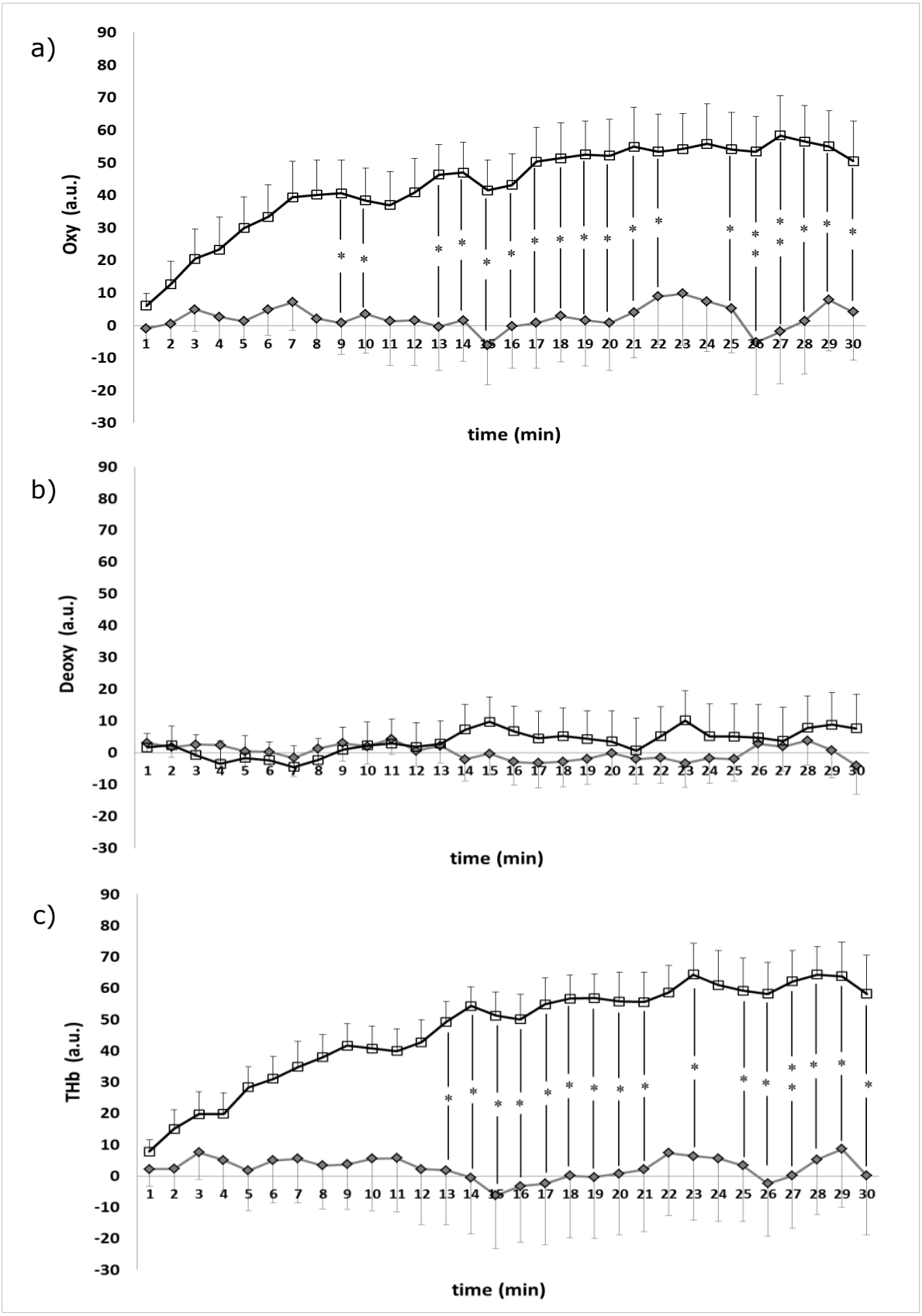

Figure 2.5 Time course changes in mean values (± *SEM*) of oxy-hemoglobin (Oxy; a), deoxy-hemoglobin (Deoxy; b), and total hemoglobin (THb; c) of NOX (closed diamond) and HOX (opened square) during 30 minutes recovery sessions. *Significant differences (*$p < 0.05$, **$p < 0.01$).

pH of blood [14-16]. 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. Since 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 [14, 17-19]. 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%, in Figure 2.3) from local muscle fatigue.

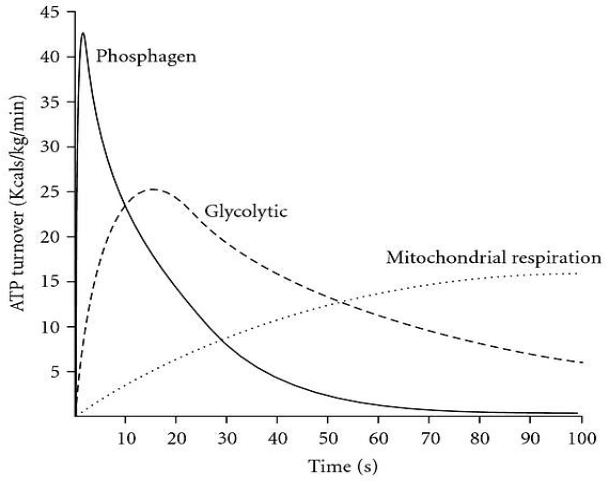

Figure 2.6 Time course of changes in ATP generating sources during exercise (Cited from Baker et al. [13]).

In this study, Oxy-Hb and THb measured by NIRS in RF increased during the 30 minutes of HOX recovery time, though the Deoxy-Hb 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. [18], 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. [8] showed that the pre-exercise use of hyperbaric oxygen therapy (50% O_2 for 60 minutes) increased the values of Oxy-Hb 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. Additionally, the parasympathetic nervous system is activated by the use of HOX [18, 20], thus the relaxation of vascular smooth muscle would have caused vasodilatory effects in both arteries and veins, resulting in the increase of 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 the hypothesis is supported by the past research showing that restricted blood flow brought about higher metabolites levels and impairment of muscle metabolism resulting in significant muscle force reduction [21, 22].

There are a number of investigations to date that have verified the effects of hyperoxia, however, the previous reports have demonstrated conflicting results, which are confusing for clinical practice. The conflicting evidence can be arranged by dividing into categories regarding the timing of 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 (Table 2.1). Recently, studies focusing on "pre-exposure" in intermittent high-intensity or anaerobic exercise have been performed, and they have indicated no effects arising from the oxygen supplementation [16, 18, 23]. Moreover, the same negative results have been reported by research in which hyperoxia exposure was done "ahead" of aerobic endurance performances [5, 24, 25]. These results suggest that increased blood levels of dissolved oxygen induced by hyperoxia would be released immediately under a normoxic environment, and the pre-exposure is incapable of producing favorable change in performance. A possible reason for the immediate oxygen release is that the

Table 2.1 Summary of hyperoxia effects (timing of exposure × types of exercise). Dark gray boxes and light gray boxes indicate negative effects and mixed effects reported by authors, respectively. White boxes indicate positive effects.

	aerobic	anaerobic	Interval
pre-exposure	Webster et al. (1998) [5] McGaverock et al. (1999) [25]	Rozenek et al. (2007) [16]	Kawada et al. (2008) [18] Sperlich et al. (2010) [23]
during exercise	Stellingwerff et al. (2005) [19] Tucker et al. (2007) [26]	Okushima et al. (2009) [28]	Hiraiwa et al. (2009) [29] Takemura et al. (2011) [27]
recovery	not found	Fordy et al. (2012) [30] this study	Nummela et al. (2002) [2] Takemura et al. (2011) [27]

participants may be in conditions in which additional oxygen is not required, contrary to the status of oxygen debt, before activity is performed. On the other hand, exposures to hyperoxia "during" aerobic activity have been shown to have beneficial effects [19, 26]. Stellingwerff et al. [19] 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. [26] 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 the better performance. It has been proposed that the diminished lactate concentration during aerobic exercise under hyperoxia might be because of reduced glycolysis and pyruvate production, therefore increased lactate clearance [19]. 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. [2] have reported that there was no positive result with supplemental oxygen (40% O_2) during the recovery phase 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. [27] also examined the influences 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. It is hypothesized 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. [27]. Participants who take part in this type of protocol, i.e. having more than 30 minutes rest between intensive exercises lasting for few minutes, are supposed to be in a condition of oxygen debt at the end of the physical task, so that supplemental oxygen would be used effectively to remove metabolic waste accumulation and resulted in the enhancement of the recovery rate.

2.5 Conclusions

To sum up, it appeared that the use of normobaric hyperoxia was effective as a recovery strategy in local muscle fatigue induced by anaerobic exercise. It would speed up the muscle tissue metabolism and increase tissue blood circulation, and the restoration of MVIC was significantly hastened when compared to normoxia in the same recovery time frame. It was also clear that the 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 (Figure 2.4) in this investigation.

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Terminology

*[1] quadriceps femoris (Figure 2.7); is a muscle containing rectus femoris and vasti, and they locate in front of the femur. The rectus femoris is a flexor of the hip and extensor of the knee joint. The vasti is are further divided into three heads; medialis, lateralis and intermedius. They are powerful extensors of the knee joint.

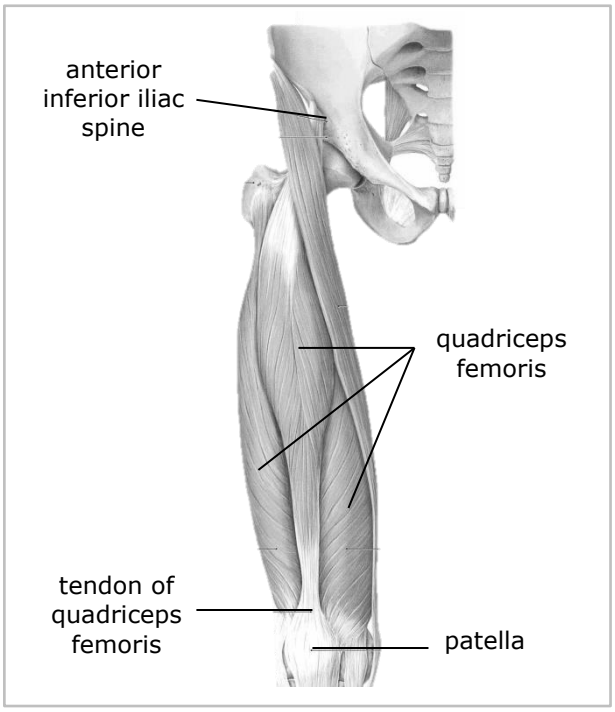

Figure 2.7 Quadriceps feromis (Vastus intermedius is not shown because it is underneath the rectus femoris).

Chapter 3.

Recovery Effects of Exposure to Normobaric Hyperoxia

~ Central and/or Peripheral Factors in Local Muscle Fatigue ~

3.1 Background

As showed in the Chapter 2, it is clear that 30 minutes of recovery strategy under the normobaric hyperoxia environment is effective to improve blood circulation and oxygen debt, thus to recover force output in the quadriceps femoris. Table 2.1 shows that oxygen supplementation will work when the organs are in need of oxygen such as during aerobic exercises and after anaerobic exercises. The evidence presenting the recovery effects for intensive intermittent exercises, however, is mixed and conflicted. Nummela et al. [1)] applied hyperoxia during recovery sessions between intermittent exercises (40% O_2 for 29 minutes in total), and reported that there was no effect in removing the lactate accumulation. On the other hand, Takemura et al. [2)] who had a similar hyperoxia strategy (28% O_2 for 50 minutes) to Nummela et al. [1)] showed a positive result on lactate concentration. The difference caused between them might be due to the length of recovery sessions. Nummela et al. [1)] had three times of recovery time and each was no more than 10 minutes. Although Takemura et al. [2)] had only one recovery session, it was for 50 minutes and much longer.

Given this, it still remains unclear what effect supplemental oxygen would have on recovery from local muscle fatigue acutely developed by intermittent exercises. Based on the above reports and the results in the Chapter 2, the present study uses repeated 15 minute recovery strategies (30 minutes in total) under the hyperoxia environment. Additionally, the oxygen supplementation is thought to influence not only metabolism and tissue oxygenation but also central factors in muscle fatigue such as activation level of motoneuron which controls strength of contraction in peripheral muscles.

This study aims to identify the recovery effects of exposure to normobaric hyperoxia on local muscle fatigue in quadriceps femoris as induced by intermittent isometric exercises. We also aimed to clarify the mechanisms of recovery resulting from hyper-oxygenation of the muscle tissue, by focusing on the factors of central and peripheral fatigue. It was hypothesized that the use of normobaric hyperoxia would enhance local muscle fatigue recovery as a result of improvement in both central and peripheral factors.

3.2 Methods

3.2.1 Subjects

12 healthy males having a mean age of 20 years (range: 18-21 years) with no self-reported cardiovascular and respiratory disease, or musculoskeletal disorders, volunteered to participate in this study. Body height, mass and body mass index (mean ± *SD*) were 170.1 ± 6.2 cm, 61.7 ± 10.6 kg, and 21.3 ± 2.6 kg/m^2, respectively. None of them are competitive athletes, but all are physically active. In order to familiarize subjects with the protocol, all experienced it once before the study session. They were instructed to avoid alcohol consumption and vigorous exercise which might cause 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 Koriyama Tohto Academy Educational Foundation Ethical Committee (approval number: R1107).

3.2.2 Procedures

Experimental Design. During the experimental sessions, all participants performed 3 sets of 3 repetitions, with each repetition to last for no less than 30 seconds (3 × 3 × best effort > 30 seconds) of isometric quadriceps femoris contraction with two recovery sessions between (Figure 3.1). The recovery strategy was a 15 minute seated rest in an oxygen controlled room [3)] in one of two different conditions: normobaric normoxia (NOX; 20.9% O_2), and normobaric hyperoxia (HOX; 30.0% O_2). Upon arrival to the experimental room, subjects were required to complete a 5 minute warm-up for the right quadriceps femoris followed by a 15 minute rest set as a stabilization period. The subsequent physical task was designed to fatigue the quadriceps femoris of the right leg by sustaining isometric load set at 70% of an individual's MVIC as previously measured. Participants were asked to maintain this load for as long as they could, and it was envisioned that exhaustion would be reached between 0.5 to 2.0 minutes. They repeated the physical task three times with two 30 second short breaks separating them. All subjects were able to maintain the required

70% of MVIC in the first task of each set. However, excluding the first task, four of the twelve subjects were unable to maintain the required 70% of MVIC for more than 20 seconds. Consequently, they were asked to maintain the contraction as a “best effort” for at least 30 seconds. The lowest recorded MVIC was 55% at the end of the “best effort”. At the conclusion of the first set of three physical tasks, a 15 minute recovery period was provided before the start of second identical set, and again prior to the third identical set. In order to avoid carryover effects from each session, subjects performed these protocols on two separate occasions at least 5 days apart. The order of testing was randomized so that six participants performed NOX first and other six participants performed HOX first. They were blinded to the oxygen concentration levels and were not informed of the hypothesis of the experiment. Subjects were instructed to maintain their daily activity levels for the duration of their involvement in the study.

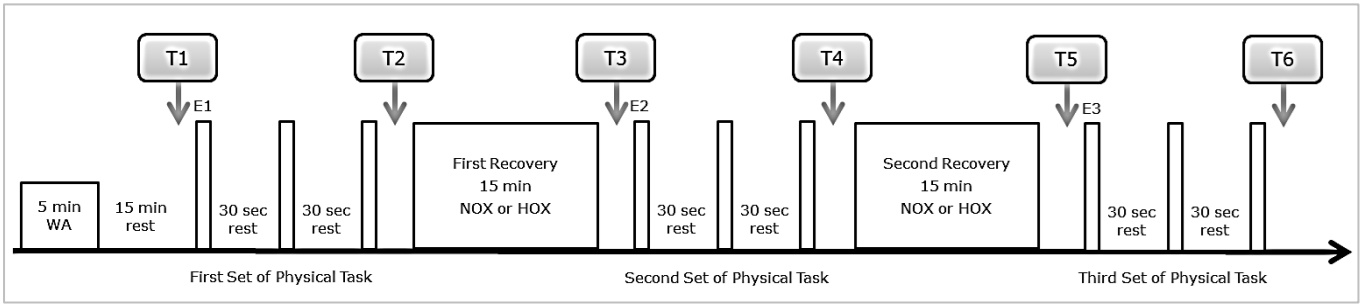

Figure 3.1 Experimental design. Subjects performed 3 sets of 3 intermittent, isometric, submaximal quadriceps contractions with two recovery sessions under different conditions: NOX and HOX. MVIC, EMG activity, blood lactate and perceived exertion were measured at T1 to T6, and endurance time to exhaustion was also measured at E1, E2 and E3. Abbreviations: NOX = normobaric normoxia (20.9% O_2); HOX = normobaric hyperoxia (30.0% O_2); MVIC = maximum voluntary isometric contraction; EMG = electromyography; T = time; E = endurance time; WA = warming-up.

Maximum Voluntary Isometric Contraction. During each protocol, the MVIC of the quadriceps femoris was measured six times:

T1 = Before the first set of physical tasks.

T2 = After the first set of physical tasks.

T3 = Before the second set of physical tasks, measured immediately after the first recovery period.

T4 = After the second set of physical tasks.

T5 = Before the third set of physical tasks, measured immediately after the second recovery period, and

T6 = Aafter the third set of physical tasks (Figure 3.1).

All subjects were seated on a device (T.K.K. 5710m, Takei, Japan) accompanied with a digital dynamometer (F340, Unipuls, Japan) set to measure their MVIC and test their muscle response to the fatiguing physical task. The subjects positioned their trunk and pelvis against the back-rest and were secured up-right by a pelvic belt. Both hands gripped hand grips by their side. The starting position was set as the right knee maintained at a 45° angle from full knee extension against an immobile leg bar. In this position, the MVIC of knee extensors, which lasted for four seconds, was performed three times, and the highest of the three values was recorded as the subject's MVIC. Subsequent MVIC values were then normalized based on the MVIC at T1 in order to see the rates of fatigue and recovery over T2 to T6.

Surface Electromyography Activity. The surface electromyography (sEMG) signals from the rectus femoris (RF) of the right quadriceps femoris were recorded using a surface electrode bipolar configuration (special order product, Emu･ii Corporation, Matsumoto, Japan) during the measurement of MVIC. The electrode was aligned parallel to the fibers on the middle portion of the muscle with an inter-electrode distance of 30 mm, and an earth electrode was attached on the medial aspect of the right patella. To have inter-electrode skin impedance below 5 kΩ, the surface of the skin was wiped with alcohol swabs, and rubbed with an abrasive gel (SkinPure$^{(R)}$ Nihon Kohden, Tokyo, Japan). The sEMG data were sampled at 1024 Hz, and digitally filtered by 15-500 Hz bandpass filter using BIMUTAS software (Nihon Kissei Comtech, Matsumoto, Japan). For subsequent analysis of the raw sEMG signals, iEMG over the middle three seconds of four seconds obtained from the highest MVIC was calculated, and the values at T2-6 were normalized by the value at T1.

Endurance Time to Exhaustion. All subjects reached to exhaustion at between 0.5 to 2.0 minutes during the first physical tasks which were set at 70% of individual's MVIC. The muscle contracting continuances during the first physical tasks of each set were recorded as

E1, E2, and E3 (Figure 3.1), and used as one of the measurements criteria in assessing the local muscle fatigue [4].

Blood Lactate Concentration. Blood samples were collected for lactate analysis from the tip of the finger and the lactate concentration was determined by a portable lactate analyzer (Lactate Pro LT-1710$^{(R)}$ Arkray KDK, Kyoto, Japan) [5-7]. Before blood collection, the tip of finger was cleaned with an alcohol swab and a small incision was made by a lancet. The initial drop of the blood was removed, and the second drop of the blood was collected for analysis. In order to see the changes in blood lactate, measurements were repeated over T1-6.

Perceived exertion. A visual analog scale (VAS) was used as a representation of perceived exertion at T1-6. Subjects were asked to mark on a 10cm long line to express the locally perceived muscle fatigue in the right quadriceps femoris. This scale was explained to the participants during the familiarization session and prior to each protocol. The marked point was then measured manually using a ruler and the value was recorded.

3.2.3 Statistical Analysis

Data are presented as mean ± *SEM*. Two-way (recovery environment, i.e., [NOX] or [HOX] × time, i.e., [T1-6] for MVIC, iEMG, lactate concentration and VAS, or [E1-3] for endurance time) analysis of variance (ANOVA) with repeated measures was used for all data gained in the present study. When significant differences were detected by two-way ANOVA, one-way ANOVA with repeated measures and paired *t*-test were additionally performed to detect significant changes from the start, and any 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. SPSS for Windows version 21.0 was used, and the statistical significance was accepted for values of $p < 0.05$ in all analysis.

3.3 Results

3.3.1 Maximum Voluntary Isometric Contraction

The normalized MVIC at T1-6 was depicted in Figure 3.2. The recovery pattern differed significantly between NOX and HOX ($F_{(1, 5)} = 3.194$, $p < 0.05$). In NOX, values at T2-6 were significantly lower than the baseline MVIC at T1 ($p < 0.05$). In HOX, although the MVIC at T2, T4 and T6 showed significant decreases compared to the basal value at T1 ($p < 0.01$), the rates at T3 and T5 recovered to 92.3 ± 3.1% and 96.6 ± 5.2%, respectively. This resulted in a significantly greater recovery rate in HOX (96.6 ± 5.2%) than in NOX (82.5 ± 3.5%), especially at T5 ($p < 0.05$) which was at the end of total 30 minute seated recovery period. Further, the mean value in HOX at T6 continued to be higher than in NOX (80.2 ± 4.4% and 72.5 ± 2.4%, respectively) although there was no significant difference statistically ($p = 0.066$).

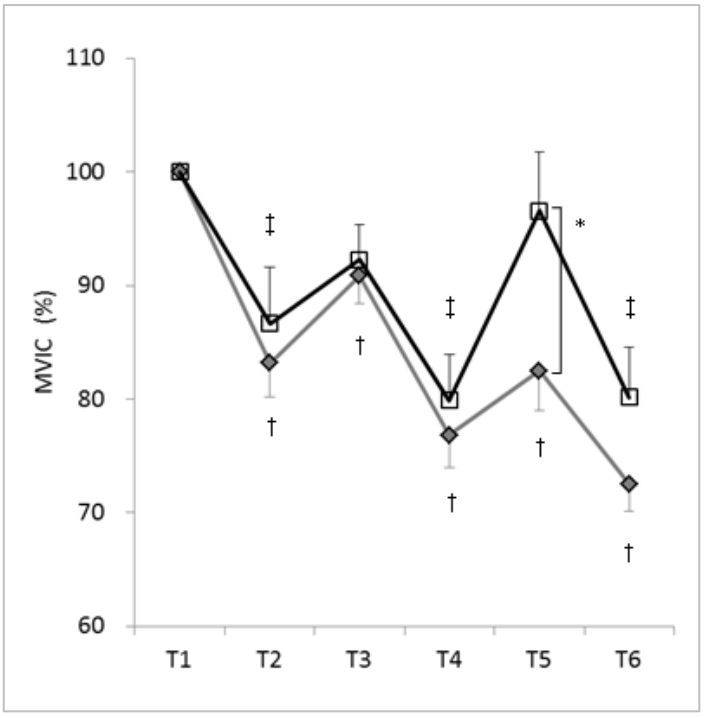

Figure 3.2 Normalized mean values (± *SEM*) of maximum voluntary isometric contraction (MVIC) in NOX (closed diamond) and HOX (opened square) between T1 and T6. †‡Significantly different from the start within the same condition (†$p < 0.05$, ‡$p < 0.01$). *Significantly different between conditions (*$p < 0.05$).

3.3.2 Integrated EMG Activity

The changes in iEMG rate over time monitored during T1-6 were presented in Figure 3.3. A significant condition (NOX vs. HOX) × time (T1 to T6) interaction was found for iEMG ($F_{(1, 5)} = 2.985$, $p < 0.05$). That is, between T1 and T4, iEMG was gradually decreased in both NOX and HOX, while greatly increased in HOX and continuously decreased in NOX afterwards. The iEMG was significantly higher in HOX compared to NOX at T6 (86.6 ± 8.0% and 80.0 ± 6.4%, respectively, $p < 0.05$), and the mean value at T5 was also higher in HOX than in NOX (93.0 ± 8.0% and 81.4 ± 4.5%, respectively, $p = 0.164$). From the above, it could be seen that the iEMG in HOX was greater after the second recovery compared to that in NOX.

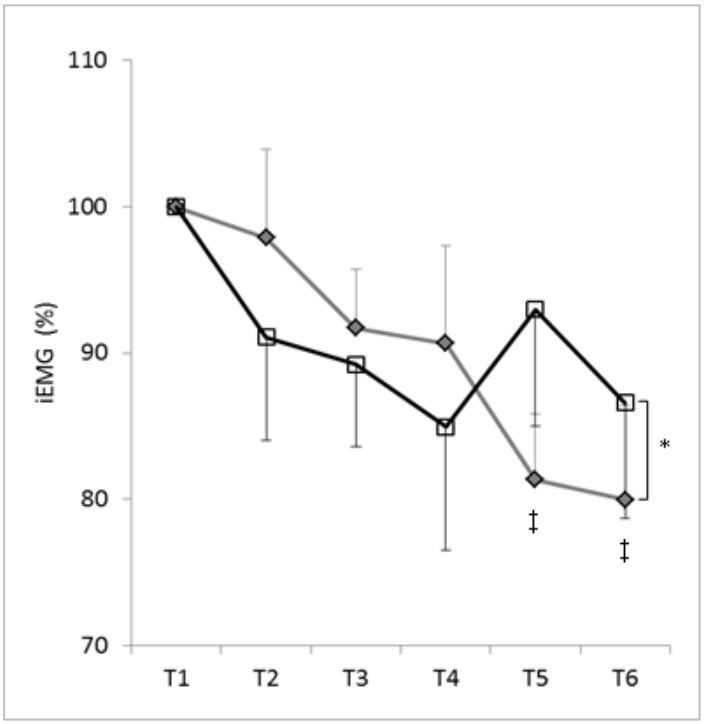

Figure 3.3 Normalized average values (± *SEM*) of integrated EMG (iEMG) in NOX (closed diamond) and HOX (opened square) between T1 and T6. ‡Significantly different from the start in NOX (‡$p < 0.01$). *Significantly different between conditions (*$p < 0.05$).

3.3.3 Endurance Time to Exhaustion

The sustained periods to the limit of muscle contraction at 70% MVIC did not differ either between E1, E2 and E3 or NOX and HOX. However the time during E2 and E3

tended to be shorter in both NOX and HOX. In NOX, the duration of 70% MVIC changed marginally from 54.6 ± 7.6 sec at E1 to 46.1 ± 5.2 sec at E2 and 47.1 ± 5.8 sec at E3. As in NOX, similar small changes in the endurance time were observed in HOX; 55.0 ± 7.2 sec at E1, 47.3 ± 4.6 sec at E2 and 43.2 ± 3.5 sec at E3.

3.3.4 Blood Lactate Concentration

Figure 3.4 expresses the changes in blood lactate concentrations over T1-6. In both NOX and HOX conditions, blood lactate levels significantly increased at T2 (6.0 ± 1.1 mmol/L and 6.1 ± 0.9mmol/L, respectively), T4 (5.1 ± 0.8 mmol/L and 7.0 ± 1.4 mmol/L) and T6 (5.4 ± 0.9 mmol/L and 6.6 ± 1.2 mmol/L) when compared to the baseline at T1 (2.4 ± 0.3 mmol/L and 2.7 ± 0.4 mmol/L, $p < 0.05$). Throughout the protocols, the patterns of change in lactate concentration did not show any significant difference between two conditions, although the mean lactate levels in HOX are higher than in NOX between T4 and T6.

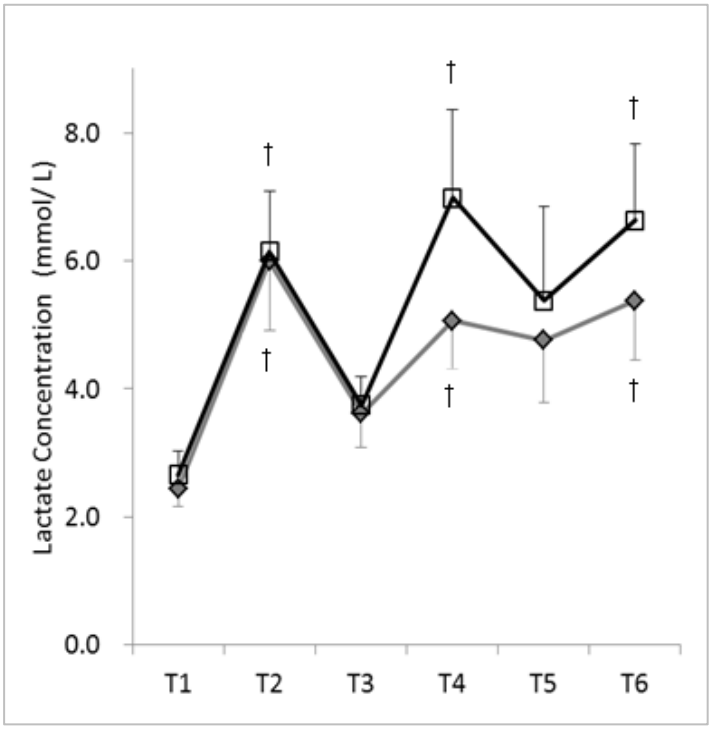

Figure 3.4 Average values (± *SEM*) of blood lactate concentration in NOX (closed diamond) and HOX (opened square) between T1 and T6. †Significantly different from the start within the same condition (†$p < 0.05$).

3.3.5 Perceived exertion

Perceived exertions expressed by VAS across T1-6 are shown in Figure 3.5. VAS increased significantly over the course of the protocols in both oxygen conditions ($p < 0.05$). There are no significant differences in perceived exertion between NOX and HOX. The VAS rate at T3 showed a significant increase only in NOX compared to the beginning (0.2 ± 0.1 cm at T1 to 2.4 ± 0.6 cm at T3, $p < 0.05$) but it did not occur in HOX (0.7 ± 0.3 cm at T1 to 2.2 ± 0.5 cm at T3). The final VAS values (T6) in NOX and HOX were 6.8 ± 0.7 cm and 6.7 ± 0.6 cm, respectively.

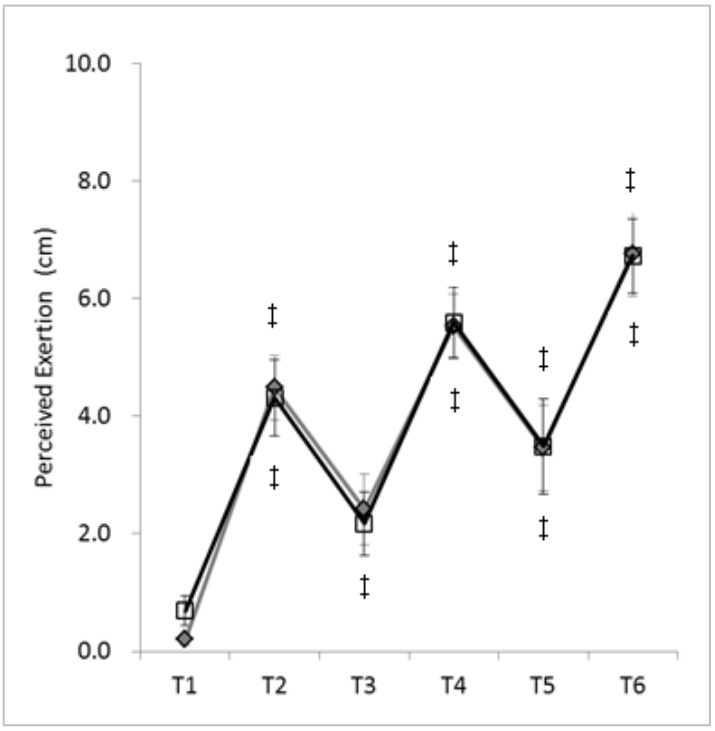

Figure 3.5 Average values (± *SEM*) of perceived exertions expressed by visual analog scale (VAS) in NOX (closed diamond) and HOX (opened square) between T1 and T6. ‡Significantly different from the start within the same condition (‡$p < 0.01$).

3.4 Discussion

There are two main findings revealed in this study:

i. Two 15 minutes seated recovery sessions in normobaric hyperoxia (HOX) significantly hastened the MVIC restoration in locally fatigued quadriceps (Figure 3.2).

ii. The MVIC result mainly associated with the influence of the central motor output, since the iEMG increased after the completion of two recovery sessions in HOX but it continued to decrease in NOX (Figure 3.3).

The above results were not associated with changes in other parameters, i.e., endurance time, blood lactate concentration or perceived exertion between two oxygen conditions.

We interpret this to mean that hyperoxia influences the regulation of central motor output to the activated muscle and therefore of muscle force output. Conversely, a peripheral parameter in muscle fatigue, represented by the blood lactate accumulation, did not show a significant difference between NOX and HOX (Figure 3.4), suggesting that the levels of peripheral fatigue were similar across the conditions. Although it has been understood that hyperoxia improves the peripheral factors in fatigue, such as metabolite accumulation, our findings cannot explain the peripheral factor as only a cause of MVIC restoration. The central factor, an increased capacity for the motor unit activation, would rather be responsible for the present result. While the causes of supraspinal and spinal fatigue are poorly known [8], central factors affecting muscle fatigue have been recognized as a reduction or accumulation of certain brain neurotransmitters and substances such as serotonin [9] and glycogen [10], the limitation of cortical activity caused by muscle afferents relating to the muscle's biochemical conditions and force generation capacity [11, 12], depletion in sensitivity of neuromuscular spindles [11], and a decrease in motoneuron activity [13]. Gandevia [11] summarized evidence of supraspinal factors in fatigue in a literature review, and concluded that feedback from muscle afferents on the muscle's biochemical conditions and force generation capacity is likely to diminish the activation of cortical sites. Especially, in the presence of oxygen debt and lactate accumulation, the group III and IV muscle

afferents, which are metaboreceptors and are sensitive to metabolites generated during muscle fatigue, continue to discharge and result in an inhibition of alpha motoneuron activity [14, 15]. Considering the above, the brain would increase central motor output in HOX than NOX in this study by responding to the status of peripheral fatigue, i.e., lesser metabolic acidosis level in HOX. The magnitude of peripheral muscle fatigue would be a considerable dependent variable in terms of decreases and increases of centrally-mediated neural stimulation into muscles thus of force output (Figure 1.4).

The findings in the present study seem to be in agreement with those of Amann et al. [16] and Tucker et al. [17], whose subjects performed cycling time-trials under a hyperoxic environment (40% O_2 and 100% O_2, respectively). They found shortenings of time to completing the time-trials, and parallel increases in power output and central motor output confirmed by the results of iEMG. These studies used aerobic endurance cycling exercise and the average force outputs were approximately 25 to 35% of MVC and 30 to 40% of MVC, respectively. This type of exercise consumes energy from the aerobic metabolic pathways [6]. In contrast, the adenosine triphosphate (ATP) supplied during the trials of intermittent intense anaerobic exercise (70% MVIC) in our study would be generated by nonmitochondrial sources, thus it would increase proton (H^+) release more than the former studies (Figure 1.7) [8, 18, 19]. This causes greater metabolic acidosis in muscle tissues, i.e., oxygen debt, which is more sensitive to the oxygen supplementation.

As a result of consuming the nonmitochondria sources during anaerobic exercises, a significant amount of metabolic products such as proton (H^+) and inorganic phosphate (Pi) are accumulated, and they induce a decrease in the pH of blood [18-20]. This has been well documented as a major cause of peripheral fatigue, leading to a decrease in the contractile strength of muscles. Similarly, fatigue at the neuromuscular junction such as insufficient propagation of the action potentials and neurotransmitter depletion [21], reductions in the quantity of calcium ion (Ca^{2+}) released by the sarcoplasmic reticulum [22], and decreases in blood flow [23, 24] are also reported as peripheral factors in muscle fatigue. Additionally, the concentration of lactate has been understood as a good marker for tissue metabolic conditions although it might not be directly attributed to metabolic acidosis in muscle

tissues [19, 20]. The lactate accumulation levels, however, did not show a significant difference between NOX and HOX in our study, and the mean values even showed to be higher in HOX than in NOX between T4 and T6 (Figure 3.4). There are two reasons that can explain this. Firstly, since lactate is not a dead-end waste product of glycolysis but is rather an important intermediate metabolite under the anaerobic metabolism pathway [19, 20, 25], the values of blood lactate concentrations might not express the exact instantaneous changes in metabolism processes. Secondly, as the result of higher central neural drive and therefore of greater force output in HOX via feedback mechanism from the muscle afferents, it is not unexpected that more lactate was produced in HOX than in NOX. Hence, it seems reasonable to suggest that the threshold of maximum peripheral fatigue was increased in HOX and a greater force output, i.e., recovery in MVIC, could be achieved without harmful metabolic acidosis occurring.

There are a number of investigations to date that have verified the effects of hyperoxia on blood lactate accumulation as a recovery strategy, however, the previous reports have demonstrated conflicting results [1, 2, 26-28]. One of the reasons for this confliction might be due to the difference in the type of exercise used in the experiments. When the exercise was performed at the fixed work load, lactate concentrations were reported to be lower in the hyperoxic environment [2, 27], whereas, when the exercise was done maximally or self-paced, there were no differences in lactate levels between normoxia and hyperoxia [26]. Although those authors did not discuss the central factors in fatigue, there might be some elements in central neural activation leading to those results as with the present study.

Perceived exertion represented by VAS in our study showed identical changes throughout the trials in both conditions (Figure 3.5). Ratings of perceived exertion have been shown to be strong indicators of physical fatigue and not to be influenced by central factors in fatigue [29, 30]. Therefore, it would be reasonable to recognize the scale as a reflection of peripheral fatigue rather than central fatigue. Based on this, which is similar to the result of blood lactate concentrations, our findings on VAS would express that levels of peripheral fatigue were similar in NOX and HOX, although the force output was significantly recovered mainly by increasing the central motor output in HOX.

3.5 Conclusions

To sum up, it appeared that the use of two sets of 15 minute seated recovery under normobaric hyperoxia was effective in the MVIC restoration from local muscle fatigue induced by intermittent intense exercises. The characteristic parallel increases of force output and iEMG activity suggest that the effective recovery under the hyperoxic environment was due to a combination of peripheral effects and an enhanced capacity to activate neural drive. From this, it was revealed that the recovery rate is regulated by a complex mechanisms relating to both peripheral and central elements which are sensitive to the oxygen concentration in the air.

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Chapter 4.

Influences of Psychological Task on Local Muscle Fatigue

4.1 Background

Local muscle fatigue, i.e., neuromuscular fatigue, is clear to be affected by both central and peripheral factors as shown in the Chapter 2 and 3. Since the central factor in fatigue is governed by psychological statement, such as motivation and concentration, mental tasks are easily imagined to influence fatigability in the local muscle.

Numerous research has been done to identify the interactive effects of physical and mental workload [1-6], and many of them have shown that the additional psychological task resulted in increases in muscle tension during low-level exertions. However, there is little evidence regarding the physiological fatigability under high-intensity activities with and without psychological demand. Mehta and Agnew [4] observed that concurrent psychological tasks decreased muscle activities in upper extremity muscles during the performance of physical tasks at 45%, 65% and 85% MVC, while such neuromuscular responses did not occur under lower physical loads (5% and 25% MVC). They implied that this is due to the decreased joint steadiness caused by reduced output of postural muscles at higher exertion levels in the presence of mental demand. Moreover, Melin and Lundberg [6] suggested that influences on physical conditions led by an additional psychological task, such as increased muscle tension, may not only be caused during the task processing but also held even after work.

This study aims to identify if local muscle fatigability in quadriceps femoris is influenced by a prior psychological task. One of the important recovery objects is psychological recovery. It has been recognized that if athletes continue to fatigue psychologically, sympathetic over-arousal is caused and it eventually leads to serious physical and psychological symptoms, such as musculoskeletal injuries and depression [7]. Therefore, it is necessary to understand the relationship between muscle fatigue and psychological factors, which mainly affect central fatigue, in order to comprehend full recovery mechanism from muscle fatigue. Because increased muscle tension induced by mental demands can lead to continuous motor unit firing [2], it is hypothesized that prior loaded psychological task would adversely affect the following muscle activity and the fatigability.

4.2 Methods

4.2.1 Subjects

15 healthy males [mean ± *SD*, age 18 ± 1 year, height 170.1 ± 5.4 cm, weight 63.2 ± 12.2 kg, BMI 21.7 ± 3.2 kg/m^2] with no self-reported cardiovascular disease, respiratory disease, or musculoskeletal disorders, volunteered to participate in this study. None of them are competitive athletes, but all are physically active students at a college. Written informed consent was obtained from all participants and the protocol was approved by the Koriyama Tohto Academy Educational Foundation Ethical Committee (approval number: R1107).

4.2.2 Procedures

To assess the degree of local muscle fatigue in quadriceps femoris, three parameters were used; maximum voluntary isometric contraction (MVIC), endurance time to exhaustion, and surface electromyography (sEMG) power spectrum signals.

Maxmam Voluntary Isometric Contraction. All subjects were seated on a device (T.K.K. 5710m, Takei, Japan) set with a digital dynamometer (F340, Unipuls, Japan) to measure the MVIC and test their muscle response to the fatiguing task. The subjects positioned their trunk and pelvis and were secured up-right by a pelvic belt, and both hands gripped hand grips by their side. The starting position was set as the right knee maintained an approximately 45° flexion against the unmovable leg bar. In this position, their MVIC of knee extensors, which lasted for three seconds, was performed three times at each pre- and post-endurance task, and the highest value was recorded as the pre- and post-MVIC. The values of post-MVIC were then normalized with respect to pre-MVIC to observe the rate of local muscle fatigue in different conditions.

Endurance Time to Exhaustion. The physical task was designed to fatigue the right quadriceps femoris by sustaining isometric load set at 70% MVIC for each trial. Participants were asked to maintain this load for as long as possible. The duration of muscle fatigue while isometrically contracting quadriceps femoris was recorded as one of the measurements assessing the local muscle fatigue.

Surface Electromypgraphy Power Spectrum Signals. The sEMG signals from the vastus lateralis (VL) and rectus femoris (RF) of the right quadriceps femoris were recorded using a surface electrode bipolar configuration (special order product, Emu･ii Corporation, Matsumoto, Japan) with an inter-electrode distance of 30 mm. They were aligned parallel to the fibers on the middle portion of the muscles and an earth electrode was attached on the medial aspect of the right patella. To have inter-electrode skin impedance below 5kΩ, the surface of the skin was wiped with alcohol swabs, and rubbed with an abrasive gel (SkinPure(R) Nihon Kohden, Tokyo, Japan). The sEMG data were sampled at 1024 Hz, and digitally filtered by 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. In order to analyze the frequency, fast Fourier transform (FFT) for the first 1 second of each epoch's signal was used and the power spectrum for each sample number was derived. Similar sEMG analysis has been used to measure the degree of local muscle fatigue during isometric exertions [8, 9]. Median frequency (MF), which is known to be less affected by noise and more appropriate to assess muscle fatigue, was then calculated for each sample. The sample number 1 was excluded due to the dispersion that was probably caused by the beginning of physical task. Sample number 3-10, therefore, were normalized based on the sample number 2 to see the degree of muscle fatigue over time, which is shown by the shortening of the power spectrum.

Experimental Design. All subjects performed two protocols; 20 minute seated rest (R) and 20 minute psychological task (PT) followed by a physical task (Fig. 4.1). In order to avoid influences of delayed onset muscle soreness in the second session, subjects performed these protocols on two separate occasions at least 5 days apart. The psychological task was a series of numerical calculations taken from a Kraepelin census for a period of 20 minutes; a test using a series of additions to see how calculations were conducted in terms of speed and accuracy. To make it a more time-pressured situation, participants were asked to do the addition as quickly and accurately as possible and to move to the following line every minute, regardless of completing questions. All of the

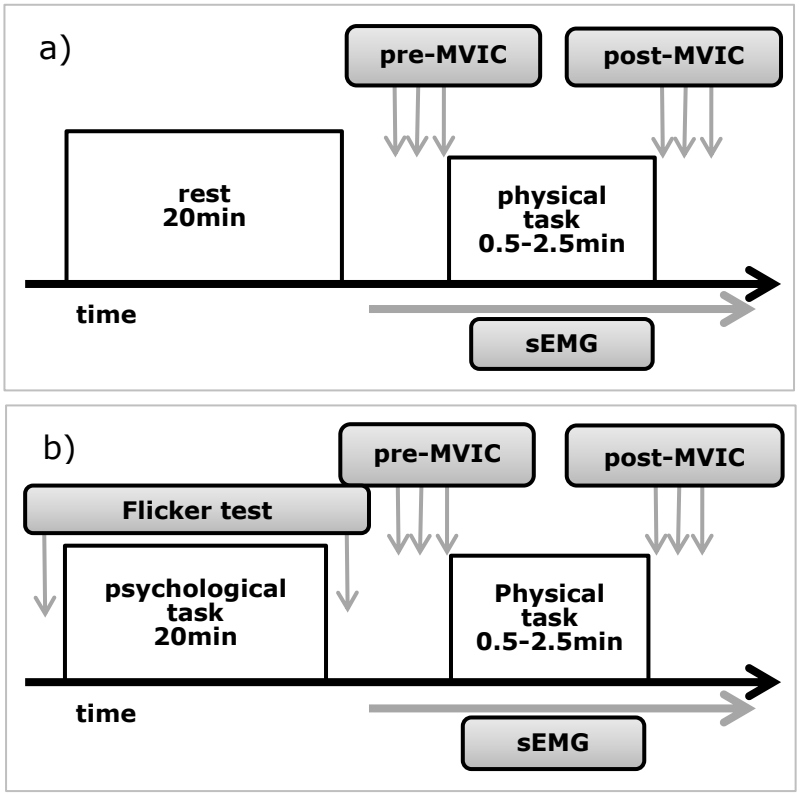

Figure 4.1 Experimental protocols. Subjects performed rest (R; a) or psychological task (PT; b) prior to submaximal quadriceps contractions. Abbreviations: MVIC = maximum voluntary isometric contraction; sEMG = surface electromyography.

subjects were students at the same grade in the same college department, thus effects caused by differences in educational background were assumed to be minimal. The accuracy rate of each line was then calculated and normalized based on the first line's number of correct answers to see the trend of reaction during the psychological task. A Flicker test was also used to confirm the levels of excitation in the cerebral cortex before and after the calculation task. A Flicker test, which evaluates critical flicker frequency (CFF; rate of successive light flashes at the point that the sensation of the flicker disappears and it becomes steady light), had been originally used on the physiology of vision due to the measuring method, but it has been applied to evaluate the level of cortex excitation for decades [10-12]. Gellhorn and Hailman [12] elicited that CFF changes nearly parallel to the levels of excitability in the neocortex assessed by electroencephalogram, and stated CFF could be a good index of the measurement of cortical excitation.

4.2.3 Statistical Analysis

All data are presented as mean ± *SD* except the values of sEMG signals which are shown by median ± *SD*. Two-way (difference in condition; R vs. PT × time; pre- vs. post-, or shown as sample number 2 to 10) analysis of variance (ANOVA) with repeated measures was used for the statistical evaluation of MVIC and the MF in VL and RF, and they were followed by Tukey post-hoc tests in cases of significant condition × time interaction. One-way (time; 1-20 minutes) ANOVA with repeated measures was also used for the accuracy rate in numerical calculation followed by a Dunnett post-hoc test in order to reveal any significant changes from the start. The effects of condition on the endurance time and values of a Flicker test before and after PT were tested using paired *t*-tests. SPSS for Windows version 21.0 was used, and the statistical significant level was set to $p < 0.05$ for all analysis.

4.3 Results

4.3.1 Degree of Psychological Task

Figure 4.2 shows the trend of the accuracy rate in the psychological calculation task for 20 minutes. As time progresses, the rate on a minute-by minute basis rose, and it became more obvious towards the latter half, although noted decreases of the mean score were seen in the 17th and 18th minutes. Relative to the first minute's rate, there were significant increases in the 16th (121.54 ± 6.97%, $p = 0.000$), 19th (121.33 ± 7.91%, $p = 0.000$), and 20th (123.79 ± 8.73%, $p = 0.000$) minutes. In addition, another parameter of the psychological task, a Flicker value, was also significantly increased post-psychological task (39.32 ± 1.87 cps, $p = 0.008$) in comparison with pre-task (36.55 ± 1.58 cps) (Figure 4.3).

4.3.2 Value of Maximum Voluntary Isometric Contraction

Figure 4.4 depicts a change in the mean rate of MVIC at pre- and post-physical fatiguing task for both rest (R) and psychological task (PT). The significant effect of R and PT was observed for the MVIC rate ($F_{(1,14)} = 5.108$, $p = 0.04$), and it was confirmed with a

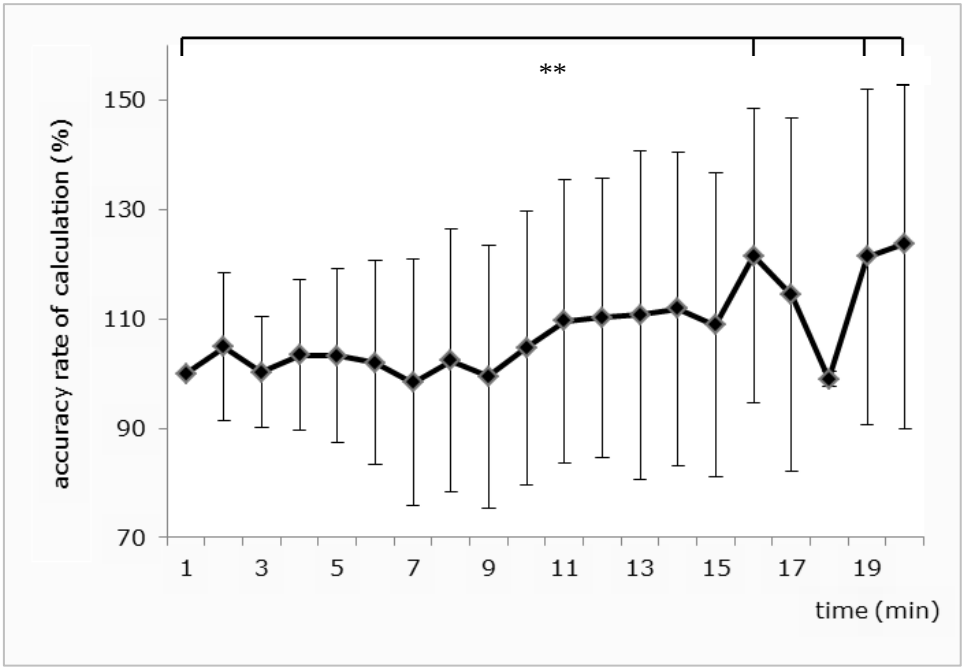

Figure 4.2 Accuracy rate in psychological calculation task. For 20 minute calculation, the accuracy rate tended to increase towards the last, and compared with the first minute, 16th, 19th and 20th minutes presented significant increases (121.54 ± 6.97%, 121.33 ± 7.91%, and 123.79 ± 8.73%, respectively, $^{**}p < 0.01$).

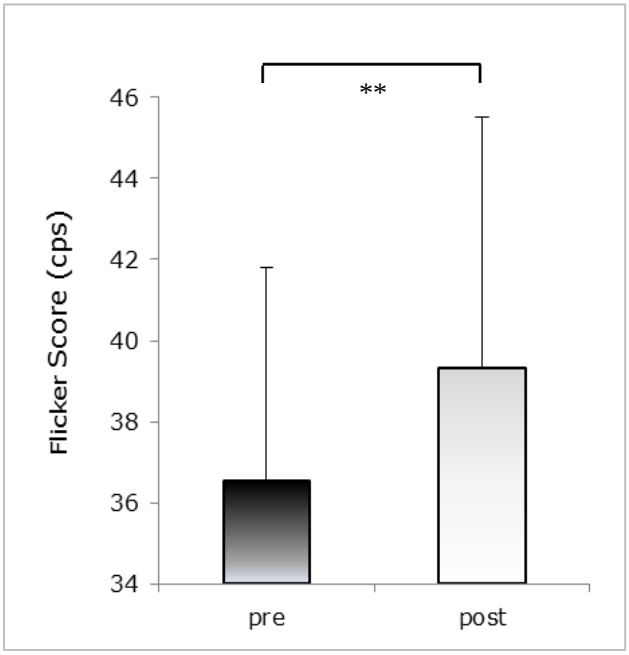

Figure 4.3 Values of Flicker test. Before performing the psychological task, the mean of Flicker value is 36.55 ± 1.58 cps, and it significantly climbed to 39.32 ± 1.87 cps ($^{**}p < 0.01$) at the post-calculation task.

significant difference at post-MVIC between R and PT (98.14 ± 15.25% and 88.77 ± 12.14%, $p = 0.045$, respectively). In terms of the difference in time (i.e., pre- vs. post-), PT showed a significant decrease ($p = 0.012$) at post-MVIC compared to pre-MVIC, though R

did not elicit such neuromuscular response (p = 0.949).

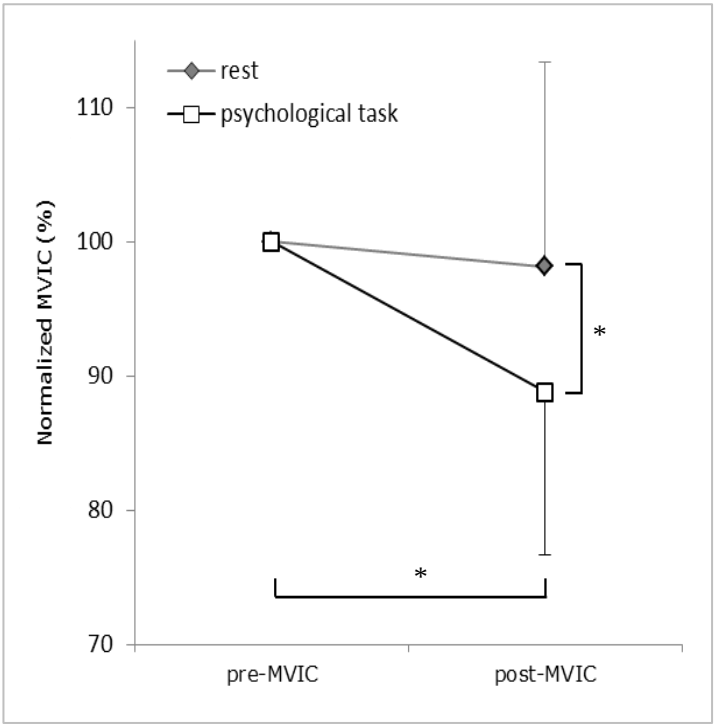

Figure 4.4 Mean values of MVIC at R and PT. There was a significant decline in post-MVIC in PT compared to pre-MVIC although it did not show any difference in R between pre- and post-exercise (*p < 0.05).

4.3.3 Endurance Time to Exhaustion

The sustained period of muscle contraction at 70% of individuals MVIC showed no significant difference between R and PT. Compared with R (81.0 ± 34.5 sec), PT tended to shorten the duration (56.0 ± 30.9 sec), however, the p-value remained at 0.076. Nine of the participants (n = 15) demonstrated a shorter muscle contracting period following PT than R, though 2 of them did not change, and 4 of them even prolonged it.

4.3.4 Surface Electromyography Power Spectrum Signals

Figure 4.5 presents changes in MF of VL and RF of quadriceps femoris respectively, for R and PT across sample numbers 2 to 10 normalized by the MF of no. 2. All power spectrums in quadriceps femoris demonstrate significant shifts from high frequency band to low frequency band towards the end (R of VL; 92.4 ± 14.3%; p = 0.048, PT of VL; 86.4 ±

16.1%; p = 0.002, R of RF; 87.3 ±9.5 %; p = 0.000, and PT of RF; 82.1 ± 9.1%; p = 0.000 at all no.9), which is known as an indication of local muscle fatigue [8, 9, 13]. The lines of R in VL and RF each drew a diphasic figure when the power spectrum shortened, although those diphasic figures disappeared after the psychological task in both LV and RF, and these lines went down smoothly over time. This tendency is more definite in RF than those in VL. There was no significant difference in MF between R and PT through sample number 2 to 10 for both VL and RF (VL: $F_{(8,112)}$ = 1.123, p = 0.353; RF: $F_{(8,112)}$ = 84.253, p = 0.193).

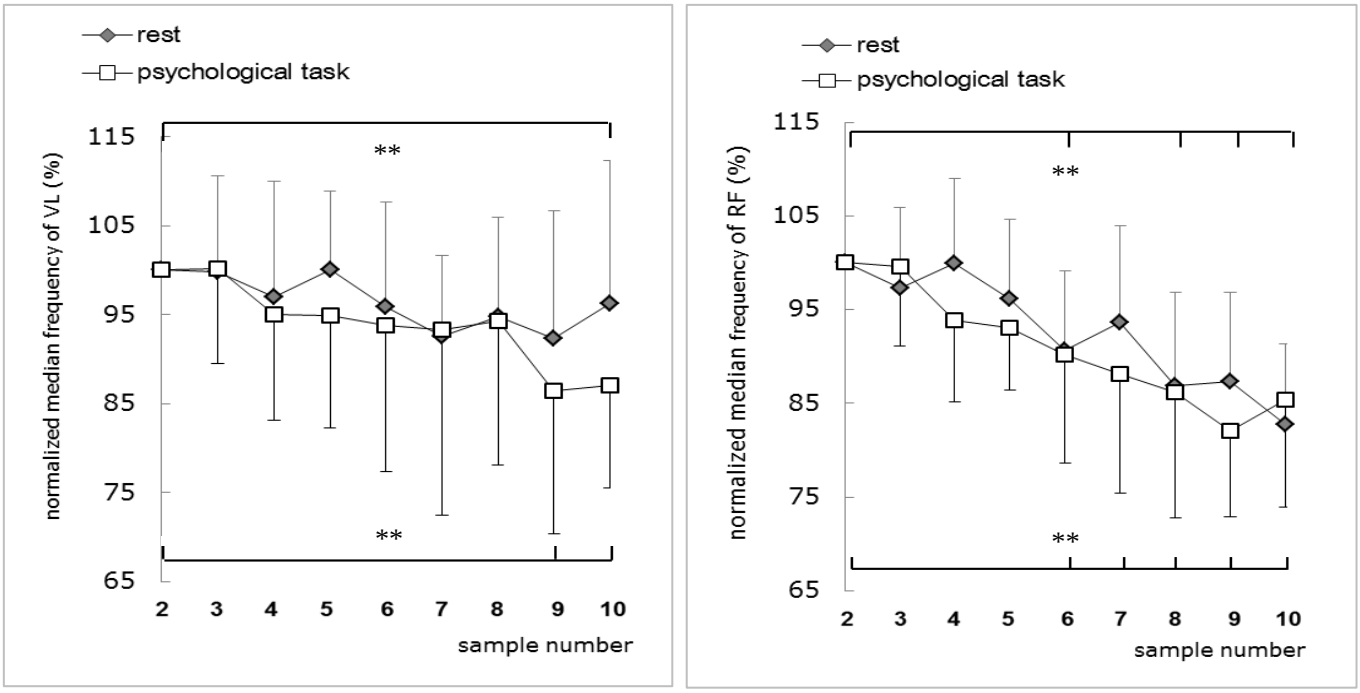

Figure 4.5 Changes in sEMG median frequency (MF) of VL (a) and RF (b) (**p < 0.01). MF in R showed a diphasic figure when the frequency is shortening, however, it disappeared in PT. Though the same tendency is seen in the result of VL, it was more marked in RF.

4.4 Discussion

The main findings in this study have shown that a 20 minute psychological workload increased the excitation level in the cerebral cortex (i), and the psychological workload followed by a physical task at 70% MVIC adversely influenced on the post-force production and changed the patterns in MF of sEMG signals over time (ii).

More Profound Local Muscle Fatigue Caused by Psychological Task. Considering the extent of the psychological task, the task might make participants' psychological states, such as concentration and attention levels, to be more facilitated based on the result of an increase in the calculation efficiency towards the end (Figure 4.2). It has been recognized that accuracy rate in Kraepelin test generally draws a U-curve or V-curve due to tiredness in the middle and increased motivation at the end of calculation [14]. In the present study, the rate slowly increased in the first half and it showed a V-curve only between the 16th and 20th minutes. This might be because participants were not familiar with the test at the start, and then they became used to it as they progressed with the calculation, which resulted in the steady increase in the first half and a notable V-curve in the final quarter. Moreover, indications on the calculating task may not be sufficient to maximally force subjects at the first part, therefore they might not fatigue till the last part of the test. In addition to the result of Kraepelin test, since an increment of Flicker values (Figure 4.3) indicates that the levels of excitation in the cerebral cortex became higher, it could be seen that the psychological task used in this study raised the excitation level in the cerebrum.

In terms of local muscle fatigue in the quadriceps femoris, the sEMG power spectrum presented significant shifts from high to low frequency bands in both VL and RF (Figure 4.5) during static exertions. From this finding, the target muscles were understood to reach states of acute local muscle fatigue in all conditions. This acute muscle fatigue could recover quickly as shown in the MVIC result of R (Figure 4.4), however the application of a 20 minute calculating task prior to the exercise increased cerebral excitation levels and was thought to cause a more profound local muscle fatigue in the knee extensors. This could be interpreted that the excitation in the cerebral cortex after PT was brought by increased activation of dorsolateral prefrontal cortex, where the facilitated activity during psychological demands has been recognized [15]. Since muscular contraction requires the activation of the same area of the cortex [1], the following physical task might be performed under less available attentional resources than that in condition of R, resulting in a decreased rate of muscular force immediately after the exertion.

Changes in Values of MVIC. Fatigue is often defined as a reduction in the capacity to

generate maximal force of the muscle [16], and strength decline has been accepted as a gold standard of muscle fatigue indicator [17]. The present study showed a greater reduction in the rate of post-MVIC following PT than R, which implies that PT negatively influenced the level of local muscle fatigue in the quadriceps femoris. Similar results have been reported in studies done by Mehta and Agnew [4], and Macdonell and Keir [3], who applied psychological demands during high force production. Although the current study did not require physical and psychological tasks simultaneously, the effect of PT was thought to be sustained after the task, and it resulted in a prolonged increase muscle tension [2, 6]. This might lead to maintained firing in low threshold motor units [2], which mainly innervate slower type 1 muscle fibers*[1] and are related with postural control. Mehta and Agnew [4] discuss that mental demand accompanied with physical task decreased shoulder joint steadiness, and it might be caused by a decreased output in the postural muscles. Since reduced joint steadiness demands more superficial muscle, i.e., type 2b muscle fibers*[2], activity to maintain the required force production [3], it would be understandable that the value of post-MVIC, governed by mainly type 2b fibers, decreased after PT, and showed quicker progression of fatigue in this study.

Trend of Shorter Duration of Muscle Fatigue. Though the endurance time following PT tended to be shortened compared to that of R, it did not show a significant difference between them statistically. A similar result was reported in the research of Mehta and Agnew [5] who revealed that the effects of mental demands on endurance time was more evident at 35% MVC than the higher rate of 55% MVC. There are several possibilities for these findings under high force levels to be considered.

First of all, local muscle fatigue is divided into three components based on the levels where the causes of fatigue occur; central fatigue (1), fatigue at the neuromuscular junction (2), and muscle tissue fatigue (3) (Figure 1.2) [18]. Central fatigue (1) is associated with the central nervous system that blocks off the motor unit to avoid damage in muscles [19], and it has been widely recognized to be influenced by psychological statement, such as motivation and mental fatigue [20]. Fatigue at the neuromuscular junction (2) is a mechanism that the available resource of the neurotransmitter, i.e., acetylcholine, is drained by

repetitive or continuous muscle contraction, and muscle tissue fatigue (3) relates to metabolization in muscle tissue including the accumulation of proton and structural changes [21, 22]. Due to the high force (70% MVIC) used in the present study, peripherally induced fatigue, like muscle tissue fatigue, might influence more powerfully than central fatigue, therefore, it might mask the centrally caused effects, i.e., psychological task effects, resulting in no significant changes in endurance time between R and PT.

Secondly, a demonstration of longer time to exhaustion has been used to imply improvements in fatigability in many areas [16], in other words, time to exhaustion has been recognized as an indication of the levels of fatigue. However, some authors state that sports tests which use time to exhaustion are unreliable and have a high coefficient of variation [23], and there is also a large variability between time to exhaustion and decreases in MVIC [24]. This is because endurance time indicates the exact point which local muscle fatigue has completely occurred and is no longer maintaining the required force level [25], while reduced muscle strength is thought to be associated with the degree of progression in muscle fatigue [17), 16].

Diphasic Figure and the Disappearance in sEMG Power Spectrum Signals. When muscle was fatigued, the sEMG power spectrum has been recognized to shift from high frequency band to low frequency band, as it demonstrates that faster type 2b muscle fibers reflecting the higher frequency components of the sEMG spectrum to become less active over time [13]. Although this tendency was seen in both R and PT of both VL and RF, there was no significant difference in MF between R and PT throughout the exertions. This finding indicates that similar successive changes in quadriceps femoris occurred, and similar fatigued conditions were reached at the end of the physical task, in the absence and presence of a prior psychological task. Those spectrums in R of VL and RF, however, showed diphasic figures in contrast to those in PT, which smoothly decreased to the end point. Physical demand isometrically done at higher force level tends to demonstrate more fluctuated changes in sEMG spectrums than that done at lower force level [8], and the current study also presented the same trend in R. Force production at high intensity may require participants to be psychologically motivated, thus the psychological emphasis might

be represented as the diphasic figures [8]. Since the motor pool appears to be wholly activated when muscle contraction is done at levels between 50-85% MVC [27], the force used in this study required higher demands on the excitability of the motor cortex in order to maintain the neural drive. Moreover, given that a psychological task decreases the available attentional resources in the brain and the effects are sustained afterwards [28], the prior psychological task in the current study has the potential to cause such central effects, and thus it resulted in the reduced psychological emphasis in the sEMG spectrum in the following physical task.

4.5 Conclusions

Psychological task; a series of single figure calculations for 20 minutes, used in this study increased the excitation levels in the cerebral cortex, and it induced a more profound local muscle fatigue in the quadriceps femoris. From the point of neuromuscular aspects, the results of MVIC and sEMG power spectrum signals emerged that the psychological task would have an impact on available attentional resources of the prefrontal cortex which is also activated by muscular contractions. This would be because greater local muscle fatigue was observed in PT compared to R.

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Terminology

*[1] type 1 muscle fibers; are tonic and slow-twitch muscle fibers, and they generate a low level of muscle tension but can sustain the contraction for a long time. These fibers are geared toward aerobic metabolism and are very slow to fatigue.

*[2] type 2b muscle fibers; are phasic and fast-twitch muscle fibers, and they generate a great amount of tension in a short period of time. These fibers are geared toward anaerobic metabolic activity and tend to fatigue quickly.

Chapter 5.

Conclusions and Practical Applications

This study examined if recovery from local muscle fatigue is enhanced by supplementation of oxygen under the normobaric hyperoxic environment and attempted to reveal the relevance with central and peripheral factors in neuromuscular fatigue. Furthermore, in understanding the comprehensive recovery mechanism, it examined the influences of psychological task, which is thought to cause central fatigue, on fatigability in a lower extremity muscle.

Hyperoxia therapy has been considered and applied in sports field for the purpose of healing musculoskeletal injuries, increasing performance level, and accelerating recovery from daily training. While the evidence is mixed, it became obvious from the results in the Chapter 2 and 3 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, when in conditions that sufficient oxygen is already supplied in the tissues, hyperoxia will not provide any additional effects, such as the use of oxygen supplementation "before" exercises.

Based on the results in the Chapter 2, 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 leaving the influence of local muscle fatigue, although performance may decrease approximately 10% if they had stayed under normoxic environment for the same duration. Chapter 3, which used intensive intermittent exercise as the physical workload, also revealed that two 15 minute recovery sessions in normobaric hyperoxia enhanced the restoration of MVIC and the recovery rate would be approximately 14% greater in hyperoxia than in normoxia.

While the favorable influences of hyper-oxygenation to the muscle tissue have been recognized, there are several considerations that should be noted in terms of applying the findings in this study to practical situations. Firstly, although each recovery session was set up for 15 minutes in the Chapter 3, the required time for recovery would need to be longer such as 30 minutes in the Chapter 2. This is because that the recovery rates of MVIC in

HOX followed the same course as those in NOX even after the completion of the first recovery session, i.e., at T3 and T4 in the Figure 3.2. Given that the value of MVIC was significantly restored at T5 which was just after the conclusion of the second recovery session, it would be more effective for subjects to rest for more than 30 minutes under HOX. Second, the muscle used throughout the study was the quadriceps femoris whose muscle volume corresponds to approximately 2.0 to 2.5 kg per lower extremity [1)], thus there should be caution when migrating the results to whole body training and competitions. However, it is still possible that a large muscle group exercise is affected in a similar way to a single muscle and improved by an increase in oxygen concentration in the air.

Chapter 4 made clear that 20 minute calculating psychological task increased the cerebral excitation levels and it caused a greater local muscle fatigue in the lower extremity. Given that these findings were brought under high-intensity isometric contraction, it is possible to consider the application of this result into sports field which people are often required to play under stressful conditions physically and psychologically. This kind of activity demands high levels of concentration, resulting in increased excitation in the cortex. Along with maximum physical exertions, it would accelerate the progression of local muscle fatigue and delay the recovery from the fatigue due to the prolonged effects of psychological demands. From this point of view, athletes and coaches may need to be careful about mentally more stressful conditions, like crucial and important matches and games, and to prepare adequate strategies for quicker recovery from the muscle fatigue in order to avoid the onset of sports related musculoskeletal injuries.

As stated in the introduction of the Chapter 1, local muscle fatigue is a complicated phenomenon containing influences of numerous factors such as central and peripheral elements. This might be the major reason why athletes have continued to develop sports-related injuries in this medically developed modern society. It is, therefore, our responsibility to keep up searching the recovery mechanism from local muscle fatigue, and providing new evidence regarding how athletes could be recovered psychologically as well as physically. I would hope that this study would help coaches and trainers to understand a part of recovery mechanism and eventually help athletes to reach their dreams. I would be

definitely glad to support these dreamers by continuing to develop my research work.

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